Born to crime : the genetic causes of criminal behavior / Lawrence Taylor.

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BORN TO CRIME

The genetic causes of criminal behavior

Lawrence Taylor

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by Howard Abadinsky
Contributions in Criminology and Penology, No. 2

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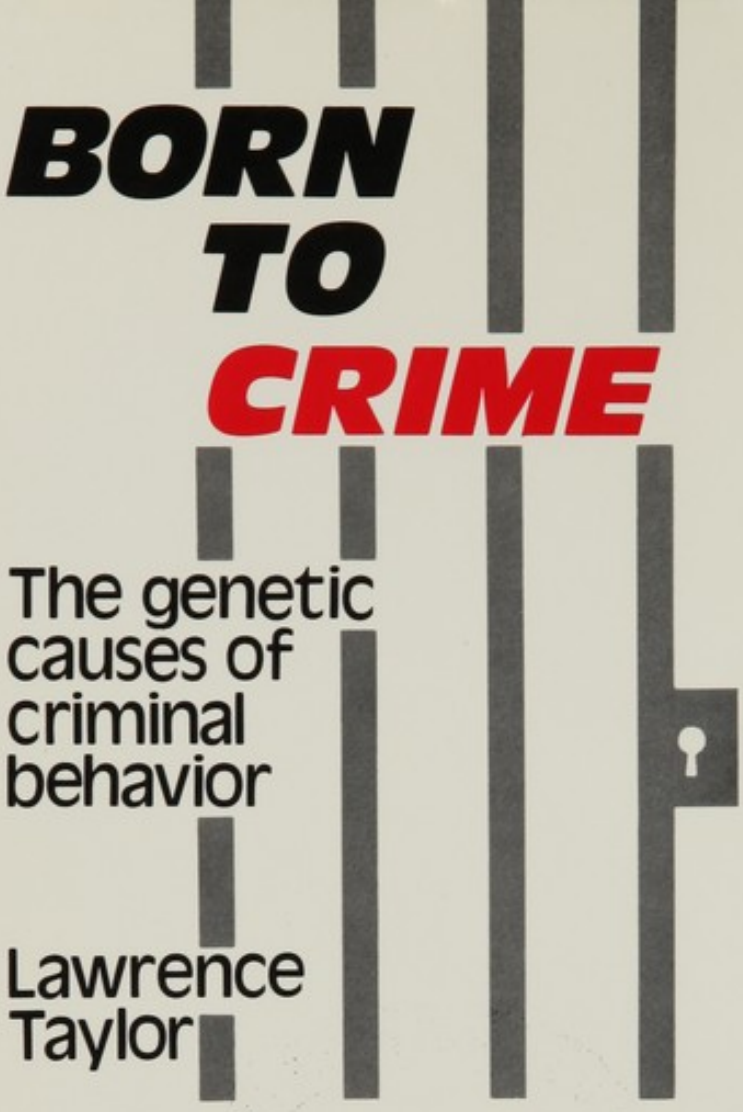

Lawrence Taylor

The vast majority of violent crimes are committed by recidivists, "hard core criminals." Many of these individuals act antisocially because of inherited physiological conditions that genetically predispose them to criminal behavior. In **Born to Crime** Lawrence Taylor summarizes recent studies in this area, then evaluates our legal system's traditional concepts of guilt and punishment.

Taylor reviews the foundation of today's criminal justice system and its failures, including theories of punishment and rehabilitation. He then summarizes recent theory and research in areas such as Wilson's "sociobiology," identical twin studies, the XYY chromosomal deviation, premenstrual syndrome, the male hormone correlation with violent crime, biological origins of schizophrenia and alcoholism, temporal lobe epilepsy, and biochemical triggering of hostile behavior. The author then reassesses the legal system's traditional concepts of guilt and punishment in light of this newly emerging body of

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Criminology and Penology

The Criminal Elite: Professional and Organized Crime
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[I]f, in the course of further developments in genetic science and technology, the law—either judicial decision making or the legislative process—is invoked, it will very likely reflect contemporary mores. What concerns some people, myself included, is the good possibility that because the rate of discovery of new scientific knowledge exceeds the rate of adaptability of this knowledge on the part of our social and legal institutions, we are today running the risk that, as with nuclear fission, we shall have the knowledge of the means of radically changing the traditional nature of man or his institutions without a concomitant development of our social sciences and of our competence to control the use of this new knowledge for humane purposes.

—Professor Horace Krever, K.C., University of Western Ontario Faculty of Law, in an address at the 18th Annual Meeting of the Genetics Society of Canada.

Contents

Preface

It is the limited purpose of this book to present emerging scientific evidence that genetics plays a key role in the origins of criminal behavior. The *ethical* considerations raised by such evidence are considerable. The far-reaching implications present vast—and terrifying—possibilities. Certainly, knowledge of a genetic cause of criminal conduct can be misused in ways limited only by the imagination.

I have refrained from imposing my personal views concerning the moral issues necessarily raised by the application of this new knowledge to the criminal justice system, primarily because of my concern that such moral discussions would interfere with the reader's consideration of the validity of the scientific facts themselves. It will be argued that in presenting this evidence and taking no position on the moral issues involved, I am violating my own ethical obligations as lawyer, teacher and citizen. Perhaps. But, rightly or wrongly, I see my personal views on the disturbing implications of this emerging evidence as of minor interest and consequence; such views would only be seen as coloring the presentation of evidence. If this book were interpreted in terms of my own irrelevant personal judgments, rather than in terms of the existence or nonexistence of relevant new evidence, its purpose would be defeated. The validity of new concepts is too often associated with the moral or ethical positions of those presenting them.

I do not mean to suggest that the moral questions posed by

these new discoveries are of secondary importance. A primary purpose in writing this book is, in attempting to establish the existence of a genetic factor in criminal behavior, to present the resultant ethical issues for serious consideration. These questions will have to be answered—possibly sooner than we anticipate. But before the ethical problems inherent in recognition of the genetic component of criminal behavior can be addressed, the existence of that fact must be established.

The "message" of this book, then, is only that new evidence on the origins of criminal behavior exists; it is not that society should, or should not, use this knowledge in any given way.

Knowledge of genetics, as with knowledge of almost anything, can be used to further the good: it represents the potential of eliminating much crime, easing pain, instilling common trust and security. It also contains the seeds of totalitarian control of the individual to a degree never dreamed of by Huxley or Orwell. However, it is for another time and place to debate the larger questions posed by the influence of genetic factors on criminal behavior. For now, I will simply attempt to present evidence that this influence exists.

BORN TO CRIME

1

Breakdown

American citizens are afraid—afraid of being mugged, afraid of being burglarized, afraid of being murdered. They are afraid to talk with strangers, afraid to walk the streets of their own neighborhoods, afraid to sit in their own homes. They are afraid that the police can no longer protect them, afraid that the courts and prisons no longer have any effect—and these fears are growing.

In a 1981 Harris survey taken across the United States, the question was asked, "In the past year, do you feel the crime rate in your area has been increasing, decreasing, or has it remained the same as it was before?"[1] Fully 68 percent of those polled replied that the crime rate in their area had increased over the previous year. Asked the same question in 1969, twelve years earlier, only 46 percent had replied in the affirmative.

In a 1981 Gallup poll, the question was asked of the public, "Is there any area around here—that is, within a mile—where you would be afraid to walk alone at night?"[2] Forty-five percent replied that they were afraid to walk alone at night in their own neighborhoods; in the larger cities the figure rose to 53 percent. In 1967 the overall figure was only 31 percent. Nor was the fear limited to venturing outside of the home: in yet another survey, conducted in 1980, 51 percent of the public were found to use extra locks on their doors, and 52 percent owned a gun that was kept in the home.[3]

How serious does the public think the crime problem is? According to a survey conducted by the U.S. Department of

Housing and Urban Development in 1978, 72 percent of Americans believe that crime is a severe problem in their community; this figure was greater than for any other problem, including drugs, unemployment and pollution.[4] In another opinion poll, conducted by Gallup, crime was viewed as the single most serious problem facing America—more important even than unemployment or inflation—and a problem that is continually growing worse.[5] In 1949 a similar Gallup poll reflected a prevailing view that crime was only the sixth most important national problem—surpassed by poor housing, traffic congestion, unsanitary conditions, high taxes and corrupt politics.

In a 1981 Harris survey, members of the American public were asked, "Do you feel that our system of law enforcement works to really discourage people from committing crimes?" Four-fifths of the public replied that the system had no effect in preventing crime.[6] This figure represents a significant increase over the 56 percent figure obtained in a similar poll conducted in 1967. When asked in yet another survey, "How much confidence do you have in the police to protect you from crime?," fully half of those questioned replied that they had little or no such confidence.[7]

Are these increasingly widespread fears justified by the facts? They are. Without question, the crime rate in the United States has experienced a fairly steady climb during recent years. Statistics compiled by the U.S. Department of Justice clearly reflect such a trend: During the decade of 1972 to 1981, for example, the department's "Crime Index" for the nation indicates a nearly 50 percent increase in the incidence of crime.[8] The increase was even greater in crimes of violence such as rape and aggravated assault, where the figures approached a 75 percent increase for the ten-year period.

Another way of looking at the incidence of crime is to view the Department of Justice's statistics for crime in each household. In 1980 more than 24 million American households were victimized by some form of crime—a figure that includes one-third of all households in the nation. As the department concluded in reporting this troubling statistic, "it is clear that Americans are at risk to an extent previously unknown."[9]

What is the cause of this epidemic increase in criminal conduct? Is it, as some have suggested, the result of an increasingly

"sick" society? Or is there a breakdown of the criminal justice system itself? Either society is producing a higher percentage of individuals who are criminals or the justice system is failing to deal adequately with a relatively constant percentage.

The prevailing view, of course, is that the increasing crime rate is simply a reflection of a troubled society. Unemployment, racial inequality, poor educational opportunities and the lack of social programs all contribute toward greater numbers of individuals who turn to crime. The problem, however, is that the rise in crime has taken place against a national backdrop of greater equality and opportunity than ever before. Certainly, the crime rate was much lower in years past when unemployment was higher, racial equality nonexistent, educational opportunities limited and social programs nowhere near as extensive as at present.

Is the problem, then, to be found within the criminal justice system itself? One explanation is that the system is simply overloaded. There is too much crime for the courts and correctional facilities to accommodate. The system is being inundated. The answer, it is often suggested, is the same as that so often given for governmental failures. More tax dollars are needed for more law enforcement, more courts, more prisons. But is this overwhelming flood of crime a cause of the breakdown—or an *effect*?

What if the very basic premises of our criminal justice system are wrong? What if the entire system of dealing with criminal offenders rests upon an incorrect assumption? A clue to the possible existence of such an answer lies in the statistics on a very critical aspect of the criminal justice system: recidivism. *Recidivism* refers to repeated or habitual criminal behavior. The assumption is always made that when the crime rate increases, the number of individuals committing the crimes is also increasing. But, there is an alternative explanation: The percentage of the population committing offenses may be constant, but these individuals are committing increasingly greater numbers of criminal acts. In other words, the rate of recidivism among a relatively stable criminal segment of the population is increasing. If the rate of recidivism is increasing, then clearly, there is something wrong with the criminal justice system in failing to deal

with identifiable hard-core criminals. In fact, it appears that the vast majority of our crime today *is* committed by repeat offenders.

A number of recent studies have provided some disturbing statistics. In a 1981 study of the incidence of crime in the United States, researchers concluded that "the probability of re-arrest for [a serious] crime is estimated as 85 to 90 percent. . . . The offenders who commit the more serious crimes display [an even] higher recidivism probability."[10] The chances then "are extremely high that an individual arrested for a felony will be arrested again and again. Those individuals committing the most serious felonies are almost *certain* to continue in their criminal careers.

These statistics have been confirmed by other studies. In the well-known Belkin study, for example, a nationwide recidivism rate for all offenses was found to be 87.5 percent.[11]

A statistical survey of crime in New York City is of some interest. Researchers there initially observed that "Twenty years ago the average city dweller had a rather small chance of being a victim of a violent crime; in contrast, at present crime rates, he has a very small chance to escape becoming such a victim." They concluded after reviewing all of the available data:

> The first and most important (conclusion) was that most crimes are committed by recidivists. The evidence is rather convincing that most crimes that are solved by either arrest or conviction are committed by recidivists. One can arrive at this conclusion at several different ways, and it is backed by massive research. . . .
>
> The data indicate that more than 80% of solved crimes are committed by recidivists.[12]

The researchers concluded furthermore that "the minimum average number of crimes committed by each criminal is at least 25" and may be as high as 50. Hence, the vast majority of serious crimes is committed by a relatively small number of individuals who repeatedly engage in criminal conduct.

Yet another study was conducted by researchers at the University of Pennsylvania.[13] The criminal records of ten thousand juveniles were carefully researched, and their criminal involve-

ment was analyzed up to the age of eighteen. The results again indicated an impressive recidivistic influence, this time at an early age: Although only 6 percent of the juveniles had committed five or more offenses, these 6 percent accounted for more than *66 percent of all violent crimes committed by the ten thousand.*

Thus it is difficult to avoid concluding, as did the noted scholar James Q. Wilson, that "most serious crime is committed by repeaters. What we do with first offenders is probably far less important than what we do with habitual offenders."[14]

What *do* we do with these habitual offenders? If our criminal justice system is failing in some significant respect and the crime rate is thereby increasing, the answer must lie in how this hard core of recidivists are dealt with by that system. It is perhaps here that we may discover if a fundamental premise of our system is faulty.

NOTES

1. *Sourcebook of Criminal Justice Statistics* (Washington, D.C.: U.S. Department of Justice, 1982) at p. 178.
2. *Id.* at p. 181.
3. Research and Forecasts, Inc., *The Figgle Report on Fear of Crime: America Afraid* (Willoughby, Ohio: A-T-O Inc., 1980) at p. 84.
4. U.S. Department of Housing and Urban Development, *The 1978 HUD Survey on the Quality of Community Life* (Washington, D.C.: Government Printing Office, 1978) at p. 217.
5. *Gallup Opinion Index, Report Number 124*, October 1979.
6. *The Harris Survey* (New York: The Chicago Tribune-New York News Syndicate, February 23, 1981).
7. "The plague of violent crime," *Newsweek*, March 23, 1981, at p. 49.
8. Federal Bureau of Investigation, *Uniform Crime Reports, 1981* (Washington, D.C.: Department of Justice 1981).
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11. J. Belkin et al., "Recidivism as a feedback process: An analytical model and empirical validation," 1 *Journal of Criminal Justice* 7 (1972).

12. S. Shinnar and R. Shinnar, "The effects of the criminal justice system on the control of crime," 9 *Law and Society Review* 581 (1975) at 592, 597–598.

13. National Institute of Law Enforcement and Criminal Justice, Law Enforcement Assistance Administration, *Curbing the Repeat Offender* (Washington, D.C.: U.S. Department of Justice, 1977) at p. 8.

14. J. Q. Wilson, *Thinking About Crime* (New York: Basic Books, 1975) at p. 199.

2

Foundations

The key to the prevention of crime appears to be in the recidivist, or hard-core criminal. In subsequent chapters I examine who this individual is and, specifically, where the origins of his or her criminal behavior lie. In the meantime, however, we must take a close look at exactly how our system of criminal justice deals with this person.

We deal with hard-core criminals the same way we deal with any individual who breaks the law. Their guilt is based upon the same premise, and their punishment is determined by application of the same theories. In short, a criminal offender may be found "guilty" and then fined, sent to jail or prison, placed under either informal or supervised probation or ordered into rehabilitative programs. Whatever the basis for a finding as to guilt, the premise is the same whether recidivist or not; whatever the sentence, the theories determining it are the same.

But those theories are wrong. Consider first how our criminal justice system assesses guilt. Our society has decided (at least for legal purposes) that man is a creature born of *free will*. At the same time, our system recognizes that attitudes may be influenced by *environmental* factors. A poor family environment or social or economic deprivation can forge the individual's mental and emotional outlook and thereby his or her behavioral patterns. Thus adults who engage in criminal conduct are seen as having a sense of values distorted by adverse environmental conditions such that they exercise their free will in ways that are

antisocial. People have complete control over their actions but choose to exercise them in criminal ways because of their environmentally influenced attitudes.

Guilt, then, is premised upon the concept that everyone who commits an act does so out of an exercise of free will: A person is fully capable of not committing the act, should he or she so desire. We recognize this in our establishment of the defense of insanity. We grant complete exoneration to (although we may require medical commitment of) a person who commits a crime while insane, that is, while suffering from a mental disease or defect that precludes the person from understanding the significance of what he or she is doing or being able to control himself or herself. The person is legally absolved of guilt because he or she does *not* act with "free will." If we did not recognize an insanity defense, we would have to deny the concept of free will as the basis for culpability, for if free will is the basis for guilt, an absence of free will (such as is recognized in insanity) must surely mean absence of guilt. As the eminent legal scholar Herbert L. Packer once observed, "We must put up with the bother of the insanity defense because to exclude it is to deprive the criminal law of its chief paradigm of free will."[1]

Just as guilt is founded upon the concept of free will, so punishment is premised upon certain assumptions about the human condition. Why do we punish? How do we determine what is the best method of punishment? What goals or objectives are being attempted in the imposition of punishment?

There are basically four theories behind the punishing of an offender. The first of them is *retribution*, the biblical concept of "an eye for an eye." The essential purpose of punishment, so this theory goes, is to satisfy what must be recognized as essentially a community thirst for vengeance. There is some social utility in this approach, for in institutionalizing vengeance we satisfy those harmed by the offender's criminal acts and thereby make it less likely that they will seek revenge themselves: The father of the child that has been run down by a drunken driver is less likely to "go gunning for him" if he realizes the courts will extract the vengeance he seeks.

Generally, however, most experts in the area will concede that

there is relatively little value in basing punishment upon a retributive theory. Aside from the temporary satisfying of a blood lust, there is minimal social utility involved. Retribution does not change the offender nor does it by itself inhibit the likelihood of future harm from that or any other offender.

The second theory of punishment involves *incapacitation*. Under this theory, the primary justification for incarceration (or execution) is preventive: to remove offenders from society so that they will not harm anyone. Clearly, there is utility in this approach, for as long as criminals are behind bars they will be unable to engage in burglary, robbery, rape and other crimes. The problem with this approach, however, is that the individual remains what he or she is—a potential offender. This theory does not attempt to correct the individual; in fact, there is considerable agreement that our system of prisons serves only to make the individual's attitude *more* antisocial. Thus the preventive, or incapacitative, theory of punishment is effective only as long as the offender is *confined*; once released, statistics indicate that an offender represents an even greater threat to society. Yet we cannot simply incarcerate every offender permanently for preventive purposes. The economic costs alone would be prohibitive, and the moral dilemmas would also prove to be a problem: Should we permanently isolate a petty thief just as we would a mass murderer?

The third approach to punishing an offender is one of *deterrence*. The primary purpose of punishment, according to this theory, is to deter future criminal conduct. A criminal act and its harm cannot be undone, but future acts may be prevented through the deterrent effect of punishing the existing act. The theory of deterrence can be broken down further into two subtheories: specific deterrence and general deterrence. With *specific deterrence*, we are concerned with frightening the individual whom we are punishing. Much as with spanking a child, we attempt to inhibit future criminal acts through showing offenders how they will suffer. *General deterrence* deals with the setting of an example for the general population: "If any of you out there commit this act, this is what will happen to you, too."

The deterrent theory is currently one of the most popular ap-

proaches to punishment of criminal behavior. Certainly, along with rehabilitation, it is held up by experts as the primary justification for punishing. Yet there is considerable evidence that this theory is ineffective as it applies to the hard-core recidivist. The statistics on repeat offenders clearly indicate that deterrence—at least specific deterrence—is ineffective: If punishment deters the individual from committing further criminal acts, why is our rate of recidivism so terribly high? Why is one who has already been punished so much more likely to commit an offense again?

The deterrent theory is based upon the economic model propounded in a previous century by Jeremy Bentham:

> It assumes a perfectly hedonistic, perfectly rational actor whose object it is to maximize pleasure and minimize pain. To such an actor contemplating the possibility of a criminal act the decision is based on a calculus: How much do I stand to gain by doing it? How much do I stand to lose if I am caught doing it? What are the chances of my getting away with it? What is the balance of gain and loss as discounted by the chance of apprehension?[2]

More recently, Bentham's aged concepts have been modernized and applied to our system of criminal justice in the form of the widely accepted "Becker-Ehrlich model":

> Criminals maximize their expected gains [according to some utility functions] from illicit activity. A person commits an offense if the expected utility he will receive exceeds the utility he would receive by engaging in other activities. Thus the criminal's decision is based on benefits and costs of both a monetary and psychic nature. . . . An increase in the probability of apprehension and punishment, with no change in other variables, reduces the incentive to participate in illegitimate activities.[3]

Yet as many critics have pointed out, this assumption is unrealistic. Criminals do not, in fact, pause to reflect upon the possible punishments for their conduct. There is usually little, if any, calm and rational weighing of alternatives and consequences. Perhaps the most often cited example is that of public hangings in Great Britain. Despite the obvious general deterrent purposes

of the execution, the number of pickpockets plying their trade among the onlooking crowd always increased dramatically during such hangings.[4]

It would be nice if rational approaches to punishment such as the Becker-Ehrlich model were valid. But they are not. In fact, the Becker-Ehrlich model itself has been repeatedly challenged on the grounds that it "has glaring shortcomings and, when examined critically, does not lead to the claimed testable hypothesis regarding the [deterrent] effect of punishment of crime."[5] In fact, after reviewing extensive research in the area, two scientists have concluded that "little or no progress has been made during the past 10 years in our understanding of the potential deterrent effects of punishment on crime."[6] Yet deterrence continues to be a primary purpose behind the imposition of punishment.

Assume that the deterrence theory is correct, that potential criminals operate in accordance with Bentham's views and rationally weigh the possible punitive consequences of each contemplated criminal act. Does this theory not take for granted that potential criminals are *capable* of altering their conduct in accordance with this rational thought process? But what happens to the deterrent theory if we find that a potential criminal's behavior has *biological* origins, that is, that this conduct is strongly influenced, if not determined, by genetic factors? We know we cannot deter an insane person because his or her conduct is the result of a mental disease or defect. Can we effectively deter an individual whose conduct is the result of a *genetic* disease or defect?

The fourth and final theory of punishment is the one currently in the greatest vogue: *rehabilitation*. This approach to punishment suggests that both society and the offender receive the greatest benefit if the offender can be reformed. Thus any "punishment" should be designed to reshape the offender's attitudes. Past negative environmental influences must be countered with future positive environmental influences. The prison should serve not to isolate or punish individuals but as an opportunity to educate them, teach them a trade and "socialize" them. If supervised probation or rehabilitative programs can better serve the purposes of rehabilitation than can prison, they should be considered the most effective form of "punishment."

As Packer observed, however, there is at least one major problem with the rehabilitation theory:

> Very simply, we do not know how to rehabilitate offenders, at least within the limit of the resources that are now or might reasonably be expected to be devoted to the task. The more we learn about the roots of crime, the clearer it is that they are non-specific, that the social and psychic springs lie deep within the human condition. . . .
>
> Rehabilitation . . . suffers simply from a lack of appropriate means. The measures that we can take are so dubiously connected with the goal that it is hard to justify their employment. We can use our prisons to educate the illiterate, to teach men a useful trade, and to accomplish similar benevolent purposes. The plain disheartening fact is that we have very little reason to suppose that there is a general connection between these measures and the prevention of future criminal behavior.[7]

Packer is far from being alone in this view. A New York State Governor's Special Committee on Criminal Offenders spent considerable time and money in studying the effects of rehabilitation on hard-core criminals, concluding that "with few and isolated exceptions, the rehabilitative efforts that have been reported so far have had no appreciable effect on recidivism".[8] George F. Cole of the University of Connecticut observed that there has "been an accumulating literature pointing to the ineffectiveness of rehabilitative programs. . . . The dominating influence of rehabilitation is seen to have made little impact on the control of crime while at the same time greatly interfering with due process."[9] John P. Conrad declared that "the rehabilitative ideal has always been a distant goal, unattainable by any system of practice that has yet been devised; its effects on the routines of criminal justice have been slight."[10] More than one commentator has pointed to the fact that California, the state that has most completely adopted the rehabilitative model of punishment, has one of the highest recidivism rates in the nation.

It seems that, like the deterrence theory, rehabilitation as a basis for punishment is not working—at least as it is presently conceptualized. Why has it failed, despite the overwhelming

support of social scientists for decades? Perhaps the answer lies in its premise: Human behavior is molded by environmental influences and may therefore be modified by environmental influences. Assume that this premise is false, that human behavior is molded by *inherited physiological* influences. If this is true, current methods of rehabilitation would be ineffective. You cannot "rehabilitate" genetic conditions with environmental therapy. An environmentally created problem may be cured with environmental methods; a physiologically created problem must be treated with physiological methods.

Our system of criminal justice is not working. The crime rate continues to increase, reflecting primarily the activity of a relatively small group of hard-core repeat offenders who are not being reached by a penal system that has increasingly become oriented toward behavioral rehabilitation and deterrence. Clearly, our theories and methods of punishment are not affecting this type of individual. Why not? Is it possible that our basic premises of free will and environmental influence are wrong? Who are these so-called recidivists, what makes them commit crimes, and why are they so impervious to our current theories of punishment?

Is it possible that at least some forms of criminal behavior may have biological origins?

NOTES

1. H. L. Packer, *The Limits of the Criminal Sanction* (Stanford, Calif.: Stanford University Press, 1968) at p. 132.
2. *Id.* at pp. 40–41.
3. S. S. Brier and S. E. Fienberg, "Recent econometric modeling of crime and punishment: Support for the deterrence hypothesis?" in *Indicators of Crime and Criminal Justice: Quantitative Study* (Washington, D.C.: U.S. Department of Justice, 1980) at p. 84.
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5. Brier and Fienberg, *supra* note 3 at 95.
6. *Id.*
7. Packer, *supra* note 1 at pp. 55–56.

8. R. Martinson, "What works? Questions and answers about prison reform," 35 *The Public Interest* 22 (1974).

9. *New York Times*, February 19, 1978, section 23 at p. 20.

10. J. P. Conrad, "A lost ideal, a new hope: The ways toward effective correctional treatment," 72 (4) *The Journal of Criminal Law and Criminology* 1699 (1981).

3

Nature or Nurture?

The idea that human behavior may have biological origins is not new—nor, for that matter, is the theory that the likelihood of criminal conduct in an individual may be predicted to an extent through the identification of biological traits. Such views have been expressed for years by members of the scientific community in the continuing controversy over the relative effects of heredity and environment on behavior. It has not been until relatively recently, however, that the environmental determinists have achieved near-total victory, and that, as a result, our system of criminal justice has been predicated upon the concept that socially deviant behavior is completely the result of the molding effect of environmental influences. The social scientists have finally succeeded in having our social institutions reflect the view that it is "nurture," not "nature," that shapes conduct. Consequently, our institutions have attempted to modify deviant conduct through environmental influences exclusively, such as with prison and rehabilitation programs.

Unfortunately, many of the early proponents of the "nature" school of behavior were handicapped by a lack of existing knowledge of genetics. Developments in the field of genetics have taken place relatively recently; in fact, it has been estimated that as of 1982 we were *doubling* our total knowledge of genetics every two years. Those early scientists who advocated heredity as the primary determinant of criminal behavior were, therefore, extremely limited in their ability to provide answers. They could

only come to conclusions based upon what they observed and offer theories resting upon the state of scientific knowledge at the time. By modern standards, that knowledge was primitive, and the theories reflected the fact. As a result, many of these theories, although reflecting underlying truths, appear ridiculous today. Thus, for example, some scientists in the nineteenth century theorized that criminal behavior was directly related to specific defects in the structure of the skull, that is, phrenology.[1] Because today such theories appear ludicrous, there has been a tendency in the scientific community to associate modern views of genetic causes of criminal behavior with these earlier discredited theories.

Perhaps the best-known early genetic approach to criminality was proposed in the latter part of the nineteenth century by the Italian physician Cesare Lombroso. Lombroso developed a theory that a "criminal type" could be identified by physical characteristics such as excessive hair, long earlobes, large jaw and slanting forehead.[2] These characteristics indicated a biological reversion in the individual to primitive stages of evolution and were attended by primitive levels of response to the environment. Thus, Lombroso believed such an inferior "throwback" would have a biological predisposition to act in aggressive and even violent ways, that is, to commit criminal offenses.

A half-century later, in the late 1930s, the American anthropologist E. A. Hooton conducted extensive studies involving 13,173 men incarcerated in state prisons.[3] After taking numerous physical measurements and comparing them to those he had taken of 3,203 men without criminal records, he also concluded that the primary cause of crime was "biological inferiority." Hooton went further, however, and claimed that particular types of crime are caused by particular types of biological inferiority. He went further yet when he wrote that different racial and antisocial groups commit characteristic patterns of crimes. He concluded that "It follows that the elimination of crime can be effected only by the extirpation of the physically, mentally and morally unfit or by their complete segregation in a socially asceptive environment."[4]

In 1949, William Sheldon set forth his theories of so-called constitutional psychiatry. According to Sheldon, human behav-

ior was strictly a function of body structure. Given careful measurement of an individual's physiological characteristics, fairly accurate predictions could be made about how he would behave.[5] Thus, for example, humans could be physiologically classified as *ectomorphs* (characterized by linearity and fragility of body structure), *endomorphs* (softness of structure) or *mesomorphs* (muscular). After conducting studies comparing the body types of known criminal offenders with those of nonoffenders, Sheldon claimed that the proportion of mesomorphs among the criminals was nearly twice that among the noncriminals.[6]

Today, such approaches to absolute genetic determinism are widely rejected and even ridiculed by both the scientific and legal communities. (It should be noted, however, that a Neo-Lombrosian so-called constitutional school of criminology is today widely accepted in Central and South America, as well as in parts of Europe.)[7] In fact, the reaction in recent years to such attitudes toward genetic determination and the undemocratic concept of biological inferiority—with the inevitably attendant shadow of racism always present—has caused a violent antibiological bias among both scientists and those in the criminal justice system. Partly because of the obvious fallacies expressed by Lombroso, Hooton and others, medical and social scientists have swung to the other extreme of the spectrum, almost universally adopting a strict environmental theory of crime: Criminal conduct is purely the result of the totality of social factors operating on an individual from early childhood on into the adult stages.

The extremes of this view were originally expressed by the early psychologist John Broadus Watson: Behavior, in its simplest terms, can be reduced to a stimulus that brings about a particular response. An individual's behavior is nothing more than a function of learned or conditioned responses gained during the ongoing process of trial and error. The human being is simply a blank nervous system that learns to respond in predictable ways to given environmental stimuli; human behavior is nothing more than a complex of learned responses shaped by the environment. Watson rejected even the concept of human consciousness or a "mind," dismissing their existence as being "as useless as monk's lore and old wives' tales."[8]

Current views of environmental determinism are not so ex-

treme. Theories such as Watson's denial of the human mind have apparently not had the same tainting effect on the environmental position that those like Hooton's on racial behavior have had on the heredity position. Yet the modern view, commonly associated with the views of Harvard psychologist B. F. Skinner, still sees the human being as a mechanism whose behavior is little more than learned responses to stimuli. According to Skinner, humans behave in a certain way because their responses to stimuli have been conditioned and reinforced by the environment. Thus undesirable responses, for example, criminal behavior, can be eliminated either by eliminating the reinforcing mechanism (by taking the individual away from the environment that has conditioned him or her to act criminally) or by substituting negative or positive reinforcers (punishing the individual for acting criminally or rewarding him or her for acting in a noncriminal manner).[9]

Today, then, the prevalent view among our scientists, criminologists, politicians and members of the legal community is that criminal conduct has little or no basis in biology but is almost exclusively the result of environmental factors: The criminal is simply the product of society. Ashley-Montague once expressed the prevailing view: "There is not the slightest evidence to believe that anyone ever inherits a tendency to commit criminal acts; crime is a social condition, not a biological condition."[10]

Is crime a social condition? Or is the well-known (and controversial) English psychologist H. J. Eysenck right in arguing recently that Lombroso was, perhaps, not far wrong, that there may be individuals who are truly "born criminals" due to "some kind of gene, chromosome or other structure which could be the physiological or neurological basis for differences between the criminal and non-criminal kind of person"?[11]

NOTES

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4

A Second Look at Evolution

There are only two ways of viewing the human being as a developing organism. The first is that this creature is apart from other animal life: he is so unique that his nature and development follow laws of nature separate and distinct from those by which all other animals are governed. The second is that *homo sapiens*, although certainly the most complex and advanced of organisms, nevertheless are governed by the same immutable principles that apply to all forms of life.

If humans are, after all, subject to the same biological principles as any other animal—and the weight of scientific knowledge clearly supports this view—the key to the origins of human behavior may be found in the origins of *animal* behavior. If animal behavior can be proven to be influenced (or possibly even determined) by genetic factors, it seems to follow that the same should be true of humans: The origins of human behavior is in the genes.

The fact is that animal behavior *is* largely inherited. As a psychology professor at the State University of New York observed, "We can state with confidence that in animals there is overwhelming evidence for the heritability of many forms of social behavior, such as courtship, mating, caretaking, and aggression."[1] Thus, for example, scientists have been able to take a strain of mice and through selective inbreeding develop two separate strains—one peaceable and one violently aggressive.[2] In another interesting experiment, a species of parrot that carries

nesting material with the help of small hooklike appendages under the tail feathers was bred with another species that had no such hooks and instead carried the material in its beak. The resulting strain of parrot had no hooklike appendages; yet these birds consistently attempted to tuck nesting material under their tail feathers, clearly indicating a genetically transmitted behavior pattern.[3]

Although we cannot conduct such experiments with humans to determine the effect of genetics on behavior, we can attempt to compare humans with the animal to which they are most similar (and to which they are the closest in evolutionary terms): the chimpanzee. It has repeatedly been noted by biologists that humans and chimpanzees are remarkably similar to each other in their physiological details—as would be expected if we accept Darwin's theory of evolution. What is not so well known, however, is how very similar the two species are in their *genetic* makeup. Two biochemists have carefully analyzed the molecular structure of the human and chimpanzee genes and concluded that the total genetic differences between the two species is roughly the same as the difference between two nearly identical species of fruit flies.[4]

If the chimpanzee is so similar to humans in physiological and genetic structure, and if behavior is determined or strongly influenced by genetic considerations, it could be expected that this animal's behavior patterns would be comparatively similar to a human's. This is the case, too: Chimpanzees live in rudimentary cultures, they are able to use tools in constructive ways, and they are capable of a considerable degree of intelligence. Recent research has shown that chimpanzees can be taught to learn vocabularies of two hundred words and to use them in sentences—complete with basically correct syntax—in carrying on continuing "conversations" with humans.[5]

In comparing humans and chimpanzees, then, it is not unreasonable to conclude along with Professor Edward Wilson of Harvard University:

> Chimpanzees are close enough to ourselves in the details of their social life and mental properties to rank as nearly human in certain domains where it was once considered inappropriate to make compari-

sons at all. These facts are in accord with the hypothesis that human social behavior rests on a genetic foundation—that human behavior is, to be more precise, organized by some genes that are shared with closely related species and others that are unique to the human species. The same facts are unfavorable for the competing hypothesis which has dominated the social sciences for generations, that mankind has escaped its own genes to the extent of being entirely culture-bound.[6]

The fact of the similarity of behavior patterns between humans and their closest biological cousins is certainly evidence of a genetic origin for that behavior. If genes influence conduct, a common genetic ancestry would be reflected in some remaining common patterns of behavior. There is, however, more compelling evidence for the inherited nature of human conduct: the innate behavior of human infants.

If behavior in *homo sapiens* has genetic roots, as it has in other animals, one could expect to observe this behavior in the absence of any environmental influences. Although we cannot isolate humans from such influences for the purpose of study, we can simply look to the newborn human baby. Should there be no universal behavioral patterns in such infants, the environmental determinists would seem to be correct. Should there, on the other hand, be commonly observed behavior in all newborn humans—despite the obvious lack of cultural or familial influence—the argument for genetic influence would appear to grow stronger.

Such behavior is, of course, routinely noted among human infants. Perhaps the most basic behavior is the rhythmic searching of the newborn for the nipple, ceasing only when the lips finally close around it and the infant begins the sucking behavior.[7] How did the infant know to look for a nipple? How did it know what to do with it when it was found?

Another uniformly encountered behavioral pattern of the newborn is the grasping reflex of the hand. If the palm of an infant's hand is touched with an object, the tiny hand will close firmly around the object—and always in an ordered sequence with the middle finger closing first. This reaction, it has been discovered, is especially quick when the touching object is hairy, presumably because this behavior is left over from earlier evolutionary stages when the child held on to the mother's fur.[8]

Other, more complex, patterns of behavior that would seem to require learning appear in newborns. Thus even prematurely born infants can be observed to "know" how to climb with well-coordinated arm and hand movements. The ability to crawl is also inherent, and if supported in an upright position a newborn baby will begin to place one leg before the other in a pattern of walking behavior. When infants are placed in water, they will exhibit a coordinated hand-and-leg swimming motion, an intrinsic behavior that disappears at three to four months (after which swimming must be learned).[9] Yet none of these examples of behavioral patterns was the result of any environmental influence. How could the infant "learn" to act in these uniform ways?

There are other examples of behavior in children that appear not to be the result of any learning experience. In one study of children who were born blind, it was found that the supposedly learned behavior patterns of smiling, laughing and crying—as well as the facial expressions for anger, pouting, fear and sadness—were present despite the obvious inability of the children to "learn" these reactions by mimicking others.[10] These universal behavior patterns have even been observed in children who are deaf, blind and without limbs—without any means for seeing, hearing or touching to "learn."[11] If the environment did not supply the learning experience, how did the children know the appropriate expressions and behavior? It is interesting to note that expressions such as the grimace of fear, the smile, the bared teeth of threat and laughter are found in chimpanzees, as might be expected if we share a distant heredity.

Yet another approach to resolving the nature versus nurture controversy is to study the behavior patterns of various human cultures. If the sociologists are correct and basic human behavior is shaped by the environment, we can expect to find elementary behavioral differences among differing cultures. If, however, there appear to be basic similarities of behavior among all humans regardless of contrasting cultural influences, the common denominator would appear to be our mutual genetic heritage.

Perhaps the most commonly compared types of elementary human behavior involve expressions and gestures. More complex forms of behavior can represent the mixed effects of ge-

netics and environment, but the simpler forms of behavior should be a valid source for resolving whether patterns of behavior can, in fact, be inherited. If the easily identified simpler forms of behavior can be inherited, there is certainly no reason why more complex forms may not also be determined—or at least strongly influenced—by genetic factors.

Studies have been conducted in this area. In one, psychologists took photographs of the facial expressions of Stone Age Papuan tribesmen as they told stories involving happiness, surprise, fear, loathing and anger. They then took photographs of Americans acting out these same emotions. The facial expressions were essentially the same. When tribesmen were shown the various photographs of an American, they were able to interpret the emotion with a better than 80 percent accuracy, despite the fact that each had been raised in very different environments isolated from the other.[12] In a similar study, two scientists took motion pictures of differing cultures around the world. The resulting film revealed an apparent universality of expressions and gestures among cultures having no connection with one another. For example, the scientists noted that an apparently universal behavior used in friendly greeting over a distance is the "eyebrow flash," that is, the rapid raising and lowering of eyebrows in combination with smiling, headtossing and nodding.[13]

These comparative studies necessarily involve very basic behavorial patterns—necessarily, because of the increasing difficulty in isolating the comparative effects of genetics and culture as the behavior becomes more complex. Yet the very existence of certain kinds of complex patterns of behavior in all cultures tends to be evidence of a genetic factor. If, for example, bodily adornment were shown to be present in every society known to humans, this would tend to support the existence of a common thread—and in the absence of a similarity in culture, it is certainly arguable that the thread is a mutual heredity. In fact, one American anthropologist compiled a list of sixty-eight social characteristics that have been found in every culture throughout history.[14] This list includes traits such as bodily adornment, courtship, dancing, etiquette, funeral rites, gestures, hair styles, incest taboos, marriage, personal names, puberty customs and

status differentiation. Yet as Wilson observed, "few of these unifying properties can be interpreted as the inevitable outcome of either advanced social life or high intelligence."[15] Then why their universality? The most reasonable explanation appears to be that a common genetic "memory" is involved.

A closer examination of one of these cultural traits will highlight the difference between the environmental and the genetic theory for its existence. As reflected in the list, incest taboos are universally found in every culture—most commonly, the taboo against sexual relations between brother and sister. Why this universality? The social scientist explains the taboo as a means of preventing the confusion in family roles that would result. Thus a brother-sister role relationship in a family would be clouded—and the family's structure threatened—by a sexual relationship such as in the husband-wife roles. The genetic explanation, however, recognizes the hereditary effects of inbreeding. The practice of inbreeding has been shown to cause hereditary diseases, create a high risk of stillbirth and result in physical or mental defects. As a result, the offspring of incestuous relationships leave far fewer descendants than the offspring of normal relationships, and so succeeding generations increasingly reflect the genetic predisposition of individuals who avoided incestual relationships. As Wilson put it, "natural selection has probably ground away along these lines for thousands of generations, and for that reason human beings intuitively avoid incest through the simple, automatic rule of [the incest taboo]. To put the idea in its starkest form . . . human beings are guided by an instinct based on genes."[16]

Thus there appear to be clear indications that behavior patterns are inherited in human beings just as they are in all other animals. But if this is so, what is the mechanism by which behavior is genetically passed from generation to generation? What are the distant origins of human behavior? Most importantly to our system of criminal justice, are patterns of antisocial conduct included within these inherited behavioral traits? If so, to what extent—if any—can they be modified by the rehabilitative or deterrent techniques of our present penal system?

The answers to at least some of these questions have been suggested by Wilson in his recent book *Sociobiology: The New*

Synthesis.[17] Widely recognized by supporters and critics alike as a revolutionary work of science, the book has been compared in importance to Darwin's *Origin of Species.*[18] In the book, Wilson presented his distillation of evidence from various scientific disciplines in a central hypothesis. This hypothesis is that Darwin's concepts of natural selection apply not only to physical but to behavioral traits.

> The heart of the genetic hypothesis is the proposition, derived in a straight line from neo-Darwinian evolutionary theory, that the traits of human nature were adaptive during the time that the human species evolved and that genes consequently spread through the population that predisposed their carriers to develop those traits. Adaptiveness means simply that if an individual displayed the traits he stood a greater chance of having his genes represented in the next generation than if he did not display the traits. . . . If the possession of certain genes predisposes individuals toward a particular trait, say a certain kind of social response, and the trait in turn conveys superior fitness, the genes will gain an increased representation in the next generation. If natural selection is continued over many generations, the favored genes will spread throughout the population, and the trait will become characteristic of the species. In this way human nature is . . . shaped by natural selection.[19]

This new theory of sociobiology states that behavior patterns have developed during the evolution of humans through exactly the same process of natural selection as have physical characteristics. Just as an opposable thumb and a larger brain developed in the ongoing process of "survival of the fittest," so too were patterns of behavior that made humans better able to survive genetically favored.

Certainly, this theory of the origins of human behavior explains the existence of behavioral characteristics found in infants, as well as those universally encountered in isolated cultures. It is consistent, too, with the view of the human being as an animal rather than as an exception to the laws of nature.

The contrast in the approaches of sociobiology and the traditional social sciences becomes clearer when humans and their conduct are viewed in terms of society. The traditional view today, put forth by our social scientists, is that society itself is, in

a sense, the constantly evolving organism. As an organism, its values and needs change to meet the needs of the time—in effect, a process of natural selection for societies. Humans are only a reflection of this changing society. They are born with inherited physical qualities (eye color, height, and so on) but with a "blank tablet" for behavior. This blank tablet will be filled in by the society, that is, one's family, peers and social institutions teach one behavioral patterns and provide role models. Thus in the view of our social scientists, humans are but a rubber mold that is being constantly reshaped by the surrounding world.

Sociobiology, however, views the process in reverse. Humans are the causative part of the equation, and society is the blank tablet. Humans arrive in this world with basic behavioral patterns already determined by evolutionary processes to some unknown extent; society is simply the aggregate reflection of the totality of a given group of people, changing within the limitations set by human evolutionary patterns of behavior. "Despite the imposing holistic traditions of Durkheim in sociology and Radcliffe-Brown in anthropology," concluded Wilson, "cultures are not superorganisms that evolve by their own dynamics. Rather, cultural change is the statistical product of the separate behavioral responses of large numbers of human beings who cope as best they can with social existence."[20]

How did humankind's present behavioral patterns evolve? Wilson argued that the vast majority of these patterns were developed through natural selection during the 5 million years before recorded civilization—when people existed in isolated tribes of hunters and gatherers. Since genetic evolution takes time, only a small fraction of the wide range of human traits developed in the ten thousand years since the appearance of agriculture and cities. The vast majority of human behavioral patterns, then, were developed millions of years ago in response to a very different world from the one in which we live today.

> The question of interest, then, is the extent to which the hereditary qualities of hunter-gatherer existence have influenced the course of subsequent cultural evolution. I believe that the influence has been substantial. In evidence is the fact that the emergence of civilization has everywhere followed a definable sequence.[21]

Wilson saw the relatively recent emergence of civilization as an explosive growth from the modest environmental adaptations of a hunter-gatherer society to the present complexities of today's industrial world. The uniquely rapid development of the human brain, with the resultant dominance over the surrounding environment, facilitated this accelerated change in social structures. "Yet the directions this change can take and its final products are constrained by the genetically influenced behavioral predispositions that constituted the earlier, simpler adaptations of preliterate human beings."[22] Many of our primitive behavioral patterns genetically remain despite the context of a more complex society. Much of our behavior has changed through natural selection and has in turn shaped society, but that change has outpaced the genetic ability to shed other primitive behavior patterns.

This view is not one of complete genetic determination, however. We are not genetic automatons, with a gene that dictates each type of conduct. Rather, we are a complex array of two hundred fifty thousand genes and 10 billion neurons, which interract with an almost incomprehensible range of theoretical possibilities. "But to what extent," asked Wilson, "does the wiring of the neurons, so undeniably encoded in the genes, preordain the directions that social development will follow?"[23] The answer is that the genetic "circuitry" describes the *possibilities* of conduct that often, in conjunction with environmental influences, will determine behavior. "Rather than specify a single trait, human genes prescribe the *capacity* to develop a certain array of traits. In some categories of behavior, the array and the outcome can be altered only by strenuous training—if ever. In others, the array is vast and the outcome easily influenced."[24] Nevertheless, much of an individual's patterns of behavior will be significantly influenced by his genetic structure—often through the indirect impact on his biochemistry.

[S]ubtle behavioral controls are known that incorporate alterations in levels of hormones and transmitter substance acting directly on nerve cells. The recently discovered enkephalins and endorphins are protein-like substances of relatively simple structure that can profoundly affect mood and temperament. A single mutation altering the chemical na-

ture of one or more of them might change the personality of the person bearing it, or at least the predisposition of the person to develop one personality as opposed to another in a given cultural surrounding. Thus, it is possible, and in my judgement even probable, that the positions of genes having indirect effects on the most complex forms of behavior will soon be mapped out on the human chromosomes. These genes are unlikely to prescribe particular patterns of behavior; there will be no mutations for a particular sex practice or mode of dress. The behavioral genes more probably influence the ranges of the form and intensity of emotional responses, the thresholds of arousals, the readiness to learn certain stimuli as opposed to others, and the pattern of sensitivity to additional environmental factors that point cultural evolution in one direction as opposed to another.[25]

The relevance of this important new theory to the criminal justice system is apparent. We have premised both the concept of guilt and the subsequent punishment (in terms of behavioral deterrence and rehabilitation) on the theory that humans are creatures of completely free will who have total control over their conduct and whose behavior can be modified by environmental influences. It now appears that this approach may be futile, that antisocial behavior may be the result of genetic influences. In a sense, a given criminal offender may have been genetically "programmed" to steal, rape or kill—or, perhaps more accurately, programmed in a manner that favored stealing, raping or killing as a behavioral response to certain environmental conditions.

Certainly, aggressive behavior seems to be programmed into *homo sapiens*. Freud saw aggression as the product of an inner drive that is constantly seeking release.[26] Konrad Lorenz noted that humans share an instinct for aggressive behavior with certain other animals, an instinct that is commonly vented in activities such as competitive sports.[27] Wilson agreed that aggressive behavior exists in humans, but he distinguished between different types of aggression—each a genetically influenced response to certain stimuli. In other words, there is no general instinct of aggression but a complex array of various behavioral reactions to different situations—reactions that are largely influenced by genetic structure. Genes provide the potential for aggressive or even violent behavior. The environment triggers that be-

havior and may even give form to it—such as in sports or war.

Aggressive and violent behavior patterns were of value to early humans: The more aggressive and violent they were, the more likely they were to survive. Thus natural selection favored these behavioral traits and they were genetically transmitted through the ongoing evolution of humans. In today's society, some degree of aggressive behavior is still deemed beneficial. Violence is condoned and even rewarded in activities such as ice hockey, boxing, police work, and warfare. But these same genetically programmed patterns of violent behavior are not acceptable in most aspects of our complex societies. As a result, we punish it. We attempt to change these patterns through behavior modification (rehabilitation) and through deterring others from acting similarly by setting punitive examples. Yet this approach can meet with only superficial success. Certainly, the inherited predisposition to react violently in a given situation can be modified—in some individuals and to some extent. But equally certainly, this environmental-determinism approach will fail in other situations. Thus it becomes important to reassess our methods for dealing with antisocial conduct and learn to change them in accordance with the genetic realities involved.

The chapters that follow present scientific evidence that, in fact, biological factors are strongly influential—possibly determinative—in the existence of antisocial behavior. These findings tend to support Wilson's new theory of sociobiology. It is, of course, possible that Wilson is wrong and that all of the scientific findings that are presented are wrong. It is also possible that they are right. In light of the apparent failures of our criminal justice system today, can we afford to ignore the possibilities?

NOTES

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11. Eibl-Eibesfeldt, *supra* note 7 at p. 454.

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16. *Id.* at p. 38.

17. E. O. Wilson, *Sociobiology: The New Synthesis* (Cambridge, Mass., and London: The Belknap Press of Harvard University, 1975).

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19. *Id.* at pp. 32–33.

20. *Id.* at p. 78.

21. *Id.* at p. 88.

22. *Id.* at p. 89.

23. *Id.* at p. 55.

24. *Id.* at pp. 56–57.

25. *Id.* at p. 47.

26. S. Freud, "Why war?" in *Collected Papers*, vol. 5, ed. J. Strachey (New York: Basic Books, 1959) at pp. 273–287.

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5

Nature's Laboratory

The old argument of whether heredity or environment determines personality and behavior (and if both, in what proportions and for what traits) has raged for years, with theories periodically thrown out to support various opposing positions. Theories are fine, but how can we determine by scientific method the true effect—if any—of genetics on behavior? We are, after all, not dealing with mice and rabbits: Human beings do not take well to controlled breeding and laboratory dissection.

The ideal test of heredity versus environment would be to create genetically identical beings and rear them separately under very different environmental conditions, in other words, create *clones*—individuals who are biological replicas of one another—and separate them at birth, subjecting them thereafter to contrasting social, parental, geographic, economic, educational and nutritional influences. After twenty to thirty years, these human carbon copies could then be carefully compared for intellect, behavioral patterns and personality traits. The results should be conclusive: If these physically identical beings turned out to be considerably different in their personalities and patterns of behavior, the victory would clearly go to the environmentalists. If they turned out to be very similar, despite having been subjected to dissimilar environmental influences, it would clearly seem to indicate that genetics is the primary force in shaping human personality and conduct.

Obviously, we cannot create human beings for the purpose of

experimentation. Fortunately, however, we do not need to create them. Nature has already provided us with subjects for the experiment: identical twins. There are two kinds of twins—fraternal and identical. *Fraternal*, or *dizygotic*, *twins* are created by the independent fertilization of two ova by two spermatazoa. As a result, the two fertilized eggs grow independently, even though in the same womb, and their genetic makeup will be no more similar than siblings born years apart; fraternal twins will show only the ordinary physical resemblance of brothers and sisters. *Identical*, or *monozygotic*, *twins* come from the fertilization of a single egg cell by a single sperm cell. This egg cell (and its genes) later divides into two separate entities at a very early stage of embryonic development. The genes in the two resulting embryos are therefore identical—in effect, mirror images of each other. Unlike fraternal twins, identical twins develop into individuals with strikingly similar physical characteristics, often indistinguishable in appearance even to close personal friends.

Thus if we were able to locate identical twins who had been separated for various reasons at birth, and who had been reared independently of each other under differing conditions, the experimental conditions would have been met. Since monozygotic twins are genetically identical, any significant behavioral differences between them would have to be attributed to social rather than biological causes. But if there were substantial behavioral similarities between the twins, despite their different upbringing, it would be difficult to avoid the logical conclusion: Behavior is inherited or is at least strongly influenced by heredity. Such findings among separated identical twins would almost dictate the disturbing conclusion that our personalities and behavioral patterns are "programmed" to a great extent even before birth. The entire concept of "free will" would have to be reappraised.

As we all know, identical twins are rare. Identical twins who have been separated at birth and reared separately are even more rare. Locating such twins—most of whom would never have seen each other and might not even be aware of the other's existence—and bringing them together for intensive comparative study make the entire proposition all the more difficult. But it has been done, and the results have been fascinating.

The first such reputable study was conducted in the 1930s by

three American specialists in different fields working together—a psychologist, a statistician and an expert in the biology of twins.[1] The three men were able eventually to locate sixty-nine pairs of identical twins, nineteen of whom had been reared separately. Many of these twins were enticed to visit the scientists for close study by the offer of a free trip to the Chicago World's Fair.

The three men used the fifty pairs of identical twins who had been reared together, along with fifty-one pairs of fraternal twins also reared together, as a "control group," that is, as a source of comparison. Most of these twins, as well as the identical twins reared apart, were adults, with a small number of children over eleven years of age included.

Each of the twins had their physical measurements taken, and each was then given a battery of tests that reflected the existing state of psychological tools in the 1930s: the Stanford Educational Age Achievement Test, the Otis Intelligence Test, the Stanford-Binet Intelligence Test, the Woodworth-Matthews Neurotic Inventory and various personality tests. The personal history of each twin was also carefully recorded.

The results of the tests, applying the comparatively primitive psychological tools at hand, indicated that as to intelligence the identical twins reared apart were slightly less similar than identical twins reared together but more similar than fraternal twins reared together. The debate concerning the effects of heredity versus environment on intelligence appeared to be a standoff. However, the same was not true of the relative effects on personality and neurotic behavior. The personality tests indicated that neither social nor educational differences had any appreciable effect on the development of the personalities of the twins. More interestingly, the Woodworth-Matthews Neurotic Inventory questionnaire demonstrated that the identical twins brought up apart were actually a little *more* similar in their emotional makeup than were either the identical or fraternal twins reared together.

When the tests were completed and the data analyzed, the leader of the research team concluded that he was "much more impressed with the very great intra-pair similarities after the twins had been exposed to all sorts of environmental differences" than he was with the relatively insignificant differences between them.

No further major developments in the study of personality and

behavior of identical twins took place for the next quarter of a century. Then in the 1950s a British scientist set out to conduct another such study.[2] Aided by an appeal on a BBC television program, James Shields, a sociologist and a lecturer at London's Institute of Psychiatry, was able to obtain some five thousand completed questionnaires from twins all over the British Isles. Armed with the resulting information, Shields was able to identify forty-four identical twins who had been separated at birth or at an early age and reared separately; most of them were in their thirties or forties, and so more than enough time had passed for environmental factors to affect their behavior patterns. As a control group, he selected an equal number of identical twins who had been brought up together. As was done in the earlier American study, an additional group of thirty-two fraternal twins reared together was added here for purposes of further comparison.

Each of the twins was interviewed and his or her personal history carefully recorded, habits and behavioral traits noted, to confirm whether they were monozygotic or dizygotic. The histories indicated that thirty of the forty-four separately reared identical twins had been separated during the first year of life, with twenty-one of them separated at the time of birth; ten had been separated some time during the first five years, with the final four pairs of twins separated by the age of nine. The differences in environment varied, of course; interestingly, one pair was reared, respectively, in South America by an English doctor married to a French woman and in Scandinavia by a Scandinavian carpenter married to an Englishwoman.

Batteries of tests were administered by Shields to the various twins, including the Dominoes Intelligence Test, the Mill Hill Vocabulary Scale and the Self-Rating Personality Questionnaire. The resulting data were finally gathered and analyzed.

Addressing first the question of intelligence, Shields's studies indicated that the hereditary influence was clearly dominant. This was clearly established, he concluded, by the significant resemblance between those monozygotic twins raised apart and those raised together. He contrasted his results with those of the earlier American study, concluding that there was little to indicate the importance of the early family environment as a cause of differences in intelligence.

The mannerisms, habits and other aspects of personality were then compared. Shields found a remarkable similarity between the identical twins—regardless of whether reared together or separately—in mannerisms, voice, temperament, sociability, interests, abilities, occupations, sexual behavior, drinking and smoking habits and thought processes and feelings. Thus, for example, he noted in an interview between two identical twins reared separately that both liked to tap the table when making a point, would flick their fingers while attempting to express themselves, and nodded their heads energetically when agreeing. Another set of twins both had a habit of picking the skin over the left eye, and yet another pair each sat with one hand placed between crossed legs. In studying the speech patterns of one set of separated identical twins, he observed that from their tone of voice it would have been difficult to distinguish them immediately from their voices alone; their tone of voice, rate of speaking and even what they said on many topics were nearly identical.

The smoking habits of the twins were particularly interesting. Confirming earlier German and Swedish studies, Shields discovered not only that there was a striking similarity in identical twins' smoking habits but that the correlation was even greater among such twins who had been reared in different environments![3] Shields concluded that there was a very strong presumption that genetic factors were influential in determining differing tastes for tobacco.

Shields then turned to the degree and quality of extroversion or introversion present in sets of identical twins. Heredity was again found to be the primary influence. Shields concluded that the data offered no general support for the view that differences in childhood family environment were important for this trait.

Addressing the question of the existence of neurotic behavior, the studies again indicated that monozygotic twins showed significant resemblance in both separated and raised-together groups. Thus identical twins were much more likely to have similar neurotic tendencies than were the fraternal twins, and the similarity existed regardless of the fact that many of the identical twins were reared separately and under different influences. In one pair of separated identical twins, both brothers suffered the

onset of schizophrenia at the same age—twenty-two; Shields noted that their striking resemblance in personality and symptomatology was strong evidence of the importance of genetic factors. He went on to comment that previous studies indicating that an identical twin had a high chance of being schizophrenic if his twin brother was had been borne out by his studies. Similarity seemed to be no lower in monozygotic twins brought up apart than in those brought up together; personality characteristics such as quick temper, anxiety, emotional instability, rigidity and cyclothymic (alternate lively and depressed moods) tendencies were often concordant in monozygotic twins, whether brought up together or apart.

Thus Shields's exhaustive studies indicated that many types of neurotic behavior were the result of inherited traits or were strongly influenced by hereditary factors. It is possible that the personality traits or behavior patterns are themselves imprinted in the combinations of a fertilized egg's thousands of genes. Another possibility, suggested by Shields, is that genes are similar to a blueprint that tends to channel the individual's reaction according to the environment in which it finds itself; the individual is "programmed" to react in a given fashion to given environmental stimuli. If this is the case, it would be easy to understand how we have for so long assumed that it was the environment that was shaping behavior, rather than just triggering it. Shields observed that each individual will probably have a particular set of genetically determined traits. Similarly, each individual's unique genetic makeup will make him particularly sensitive or insensitive to various environmental stimuli.

Finishing his work, Shields concluded that differences in personality development were clearly demonstrated to be dependent upon variations in an individual's genetic makeup.

The next significant study of the relative impact of genetics and heredity on identical twins was conducted by Danish psychiatrist Niels Juel-Nielsen in the late 1950s.[4] The study is particularly interesting for its in-depth focus on the similarities and differences in mental abnormalities between twins.

Juel-Nielsen went to the Institute of Human Genetics at the University of Copenhagen, where a register of every twin birth occurring in Denmark had been kept between 1870 and 1910;

the list included thirty-eight thousand pairs. He narrowed the potential subjects to twins that were monozygotic and reared apart. Of the thirty-eight thousand, seventeen pairs of twins remained; of them, seven were excluded because of a death of one of the pair, and two were ruled out because they had not been separated until relatively late in their childhoods. Only eight pairs of identical twins from the register remained for Juel-Nielsen's study. From other sources, he was able to locate an additional four sets of identical twins who also fulfilled all of the requirements. The resulting twelve pairs of twins ranged in age from twenty-two to seventy-seven; most had been separated at birth or within the first few months of life.

Juel-Nielsen then conducted an intensive study of each of the subject twins, making up in depth what the previous studies had accomplished in numbers alone. He interviewed each of the twins one at a time, obtaining personal histories, noting personal traits and probing with questions into the psyche of the subject. Most subjects were interviewed from fifteen to twenty-five times, although some were questioned and observed on as many as thirty-eight occasions. In addition to these psychiatric interviews, each twin was given a medical examination and two intelligence and two personality tests.

The results were—again—impressive. As to intelligence, despite the influence of different environments, Juel-Nielsen noted a close similarity between test scores of the twin partners. He then concluded that intelligence test scores are predominantly determined by genetic factors, although environment and education were less influentially contributing factors.

Discussing the personality traits of the separated twins next, the Danish scientist commented upon the similarity in their movements, gestures, carriage, gait and facial expressions, especially smiles and laughter and the tone and pitch of voice. More profound emotional and characterological personality traits were also noted to be similar. Juel-Nielsen concluded that such similarities were a clear indication of the twins' genetic similarity, and constituted strong proof of the predominant impact of genetic factors in determining personality structure.

The most fascinating findings, however, came to light when the data on psychiatric disorders was compiled and analyzed. Juel-

Nielsen indicated that six of the twenty-four subjects suffered from serious mental problems. The first of them was diagnosed as a severe neurotic, with dominant symptoms of psychosomatic, sexual-neurotic, anxious-hysterical and periodic depression traits. Her twin had the identical diagnosis. Both, it developed, were frigid, both had sought and obtained sterilization and both had been hospitalized for their psychiatric problems. Yet these twins had been separated in their first year of life and reared completely apart.

The third emotionally disturbed subject was diagnosed as suffering from neurosis characterized by hypochondriacal and psychosomatic traits and symptoms of anxiety and depression. This subject's twin also had an identical diagnosis; both had been hospitalized repeatedly. This set of twins had been separated three weeks after their birth and reared apart.

The fifth twin was classified as having a characterologically deviating or psychopathic personality. His twin had an identical diagnosis. These two, also, had been separated in their first year.

It is difficult to avoid concluding, as did Juel-Nielsen, that heredity is influential in the formation of personality disorders. He then commented that many psychiatrists and psychologists tend to belittle the influence of genetic factors on the development of neuroses. This is especially remarkable, he noted, since Freud himself had recognized as late as 1949 that the human mental condition had its origin in the relationship between inherited disposition and environmental experiences.

Some years after the Juel-Nielsen study, two American scientists at the University of Texas initiated a different approach to the problem. Rather than studying in great detail a small number of twins reared apart, John Loehlin and Robert Nichols chose to investigate with less depth the largest number of twins ever attempted.[5] Rather than compare the personalities and behavioral characteristics of identical twins who had been brought up in different environments, they would take both identical and fraternal twins who had been reared together or apart and compare them. If the identical twins were more similar to each other than were the fraternal, a good argument would be made that the only major difference—genetic identity—plays a significant role in shaping the human psyche.

Loehlin and Nichols chose a simple way to obtain their subjects. The answer sheet for the National Merit Scholarship test given to 600,000 high school seniors had a question concerning whether the individual was a twin or not. With access to this information, the men were able to identify 1,507 pairs of twins who were of the same sex. They then mailed each of these twins a four-page questionnaire designed to determine whether they were identical or fraternal, together with a solicitation to participate further in the study. Almost 80 percent of the twins returned the completed questionnaire and volunteered to take part.

The scientists then mailed a battery of personality and interest tests to the 1,188 sets of twins who had volunteered. In addition, questionnaires were also mailed to their parents, teachers and friends asking them to rate each twin as to various personality traits.

When all of the materials had been returned to the scientists, complete data was available for 850 sets of twins—514 of them identical, 336 fraternal. The two men and their assistants then set about correlating the data and looking for indications of whether there were any differences between identical and fraternal twins in their personality development.

The conclusions, although limited only to personality assessment, were consistent with the previous studies: The identical twins demonstrated greater similarity in personality, ability and interests than did fraternal twins. As to the identical twins reared apart compared with those reared together, the results were familiar: Similarity or dissimilarity in family environment had little impact on the personalities of the twins. Rearing the identical twins separately did not make their personalities less similar, and rearing them together did not make them more similar.

About the same time that the University of Texas data were being collected, German psychiatrists at the Psychosomatic Clinic of the University of Heidelberg initiated a study of the hereditary and environmental influences in the development of neurotic behavior.[6] From 26,799 patients who had been psychiatrically treated and for whom medical records were available, 240 twins were identified. From these 240, 50 pair were selected as satisfying all of the requirements: Both twins were still alive, they were accessible and cooperative and at least one of the twins had

a history of neurosis. Of the 50, 21 were identical twins and 29 were fraternal; 32 were adult, 18 were children. Once again, an extensive battery of tests was administered to the twins, and each was carefully interviewed using psychoanalytic methods. Again, the results were startling.

The psychiatrists concluded that there were almost exactly twice as many neurotic symptoms shared by the identical twins as were shared by the fraternal twins. In other words, if one identical twin suffered from a neurotic symptom such as overly aggressive behavior, his counterpart was much more likely to exhibit the same neurotic aggressiveness than would a fraternal twin's counterpart. Yet each of the 50 sets of twins had been reared together. Since environment was the same for each brother or sister in a set, the conclusion is inescapable: The only difference between the identical and fraternal twins was the genetic sameness of the identical twins. The German doctors concluded that the manifestation of neurotic symptoms was statistically proven to be the result of hereditary factors.

The doctors then considered different behavioral characteristics and assessed the comparative genetic influence on each as indicated by the study. Symptoms with a strong hereditary component included maladaptive oral behavior, depressive disturbances, maladaptive aggressive behavior, sleep disturbances and *enuresis* (bed-wetting). Those symptoms affected by genetics but more receptive to environmental influence were listed as obsessive-compulsive behavior, fears and phobias and gastrointestinal disorders. The research team concluded that neurotic cardiovascular problems, skin lesions, headaches, psychomotor disorders and functional sexual-genital disorders had little if any hereditary origin.

The finding of genetic influence on "maladaptive aggressive behavior" is particularly interesting when dealing with the causes of criminal conduct.

Perhaps the most impressive of the studies of twins, however, has only relatively recently been completed. That effort, begun in March 1979, involved a team of psychologists, psychiatrists, geneticists and medical doctors from the University of Minnesota and dealt with the intensive examination of twenty pairs of identical twins who had been separated at or near birth.[7] Inter-

estingly, however, the purpose of the study appears to have been to prove that environment—not heredity—is the prime determinant of behavior. As the leader of the research team, Thomas Bouchard, later admitted to a reporter, "I frankly expected far more differences [between twins] than we have found so far. I'm a psychologist, not a geneticist. I want to find out how the *environment* works to shape psychological traits."[8]

Each of the twins was subjected to six days of intensive testing and interviews. The team of scientists and physicians examined them physically and verbally, probing into their personal experiences, medical histories, habits, tastes, abilities and intelligence. They were given x-rays, heart stress tests, reaction tests, electrocardiograms and pulmonary examinations; they were wired to electroencephalographs to determine brain wave responses to stimuli, injected with various substances to detect allergies and examined in dozens of other ways. Each twin was asked to answer more than fifteen thousand questions during the one-week period of study, covering family and childhood environment, personal interests, fears and phobias, moral feelings and values, reading and TV watching habits and so on. Each was asked to complete an intelligence test (Wechsler Adult Intelligence Scale), as well as tests for verbal processing (Posner NI-PI task), speed of memory scan (Sternberg memory search) and spatial processing (Shephard-Metzler cube comparison).

The results were, once again, very interesting. The compiled data clearly indicated that intelligence was predominantly an inherited characteristic, relatively unaffected by environmental factors. In fact, the scores of identical twins who had been raised separately were so similar that more than one member of the research team remarked that they were actually closer than would be expected if one individual were given the tests twice.

The similarities in even the most minute details of the twins' lives are striking—similarities that go beyond coincidence and raise the question of just how far genetic determination of behavior extends. Consider the case of one set of male twins studied by the group. Both men, reared entirely apart, liked math and disliked spelling in school, worked part time as deputy sheriffs, drove Chevrolets and habitually vacationed in Florida. Both had married women named Linda, and both had divorced them;

both had remarried women named Betty. Each had one son—named James Allan and James Alan, respectively. Both had dogs that they had named "Toy." Both had similar drinking and smoking patterns, and both chewed their fingernails to the nubs. Each of the twins had hemorrhoids, identical pulse and blood pressure and similar sleep patterns. Each had suddenly gained ten pounds at the same time in their lives. Both men suffered from a combination of migraines and tension headaches, which had begun for both at the age of eighteen.

Or consider a pair of British housewives who were reared in opposite socioeconomic environments. Each had given birth to one son and one daughter. The names given the sons were Richard Andrew and Andrew Richard; the daughters' names were Catherine Louise and Karen Louise. When the women were reunited in Minnesota for the tests, each wore seven rings on her fingers, two bracelets on one wrist and a bracelet and a watch on the other.

Perhaps the most interesting pair of twins involves two men from as nearly opposite environments as it is possible to imagine. One of the men was reared by his grandmother in Germany as a Catholic and a Nazi youth; the other was brought up by his father in the Caribbean as a Jew and spent a number of years on a kibbutz in Israel. The natural expectation would be that the men would be completely dissimilar in every way. Yet when the two were reunited, each had a mustache and wore wire-rimmed glasses and two-pocket shirts with epaulets. Both men, it turned out, were absent-minded, liked spicy foods, flushed the toilet before using it, kept rubber bands stored on their wrists, were fond of sweet liqueurs, read magazines from back to front, dipped buttered toast in their coffee and thought it humorous to sneeze in a group of strangers. Not surprisingly, the men had very similar profiles in the personality tests.

Yet another pair of British sisters again demonstrated the amazing resemblances in behavioral patterns among the identical twins. Although reared in different environments, both women proved to be chronic gigglers (there were no "gigglers" in their adoptive families), and each had identical methods of coping with stress: They avoided it. Despite the fact that stress avoidance is almost universally considered to be learned behavior, both sis-

ters had developed similar psychological mechanisms for "blanking out" any conflict or controversy.

The team members repeatedly expressed amazement at the similarity of personality traits and behavior patterns. Psychologist David Lykken commented that "more of these unique characteristics than we previously thought may be determined by a particular combination of genes." He went on to observe that "people get so upset when you suggest that the wiring diagram can influence the mind." But to conclude otherwise, he added, "requires a naive dualism . . . an assumption that mental events occur independent of the physical substrate."[9]

The psychiatrist on the team, Leonard Heston, noted that the twins reared together had very similar psychiatric histories. Past studies have indicated, for example, that if one identical twin suffers from schizophrenia, the other—if they have been reared in the same environment—stands a 45 percent chance of also having the condition. In the past, this had been attributed to the similarity of environment that twins normally encounter. But Heston observed with some surprise that "what we see [with twins reared apart] is pretty much the same as in twins brought up together."[10] The separately reared twins generally share very similar phobias and fears and often suffer from the same emotional/mental problems. In one case, for example, a studied set of twins both exhibited very similar neurotic and hypochondriacal traits; yet one had been reared in a strict disciplinarian family, the other by a warm, tolerant, loving mother.

Nor are the similarities limited to major mental or emotional problems. Heston noted, "Things that I would never have thought of—mild depressions, phobias—as being in particular genetically mediated" are evidently strongly influenced by heredity.[11] Even the smallest peculiarities in human behavior appear to some—perhaps appreciable—extent the product of an individual's genetic "program."

A final striking example of the similarity between identical twins has been provided by Michel Jouvet of France. Jouvet studied the sleep patterns of such twins, recording their rapid eye movement (REM) while each of a set of twins slept at the same time in separate but identical rooms.[12] Each pair of twins was monitored for three or four nights in succession and returned later

for additional study. After extensive investigation, Jouvet concluded that there was a far greater similarity of REM sleep patterns among identical twins than among nontwin siblings. Nontwins, on the other hand, showed considerable variations in their REM sleep patterns.

These results, Jouvet said, "suggest the existence of a 'family factor' of REM occurrence, which may have a genetic basis." In the dream state, he explained, "it has been shown that some kind of generator takes command of the brain, impinging on about eighty percent of its nerve cells and even on the motor system."[13] This "generator," according to the French scientist, is governed by heredity. Dreams uncover genetic programs that are played out on the stage of the subconscious. He referred to this process during sleep as "genetic programming and re-programming."

Clearly, then, the behavior patterns of identical twins have been shown to have a very marked similarity. Just as clearly, it is difficult to avoid the logical conclusion. As identical twins are genetically the same, their consistently proven similarity in behavior must be attributed to heredity. Nature's own laboratory teaches us that much of human behavior—including criminal behavior—is caused by genetic factors.

NOTES

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6

The Defective Brain

Violent behavior has, of course, been present in humans and other animals for millions of years. One form of such behavior is an instinctual reaction in all animals to a life-threatening force. When confronted by a danger such as an invading predator, the animal either flees or reacts with aggression and even violence, that is, "fight or flight." To preserve the species, the animal brain is genetically "programmed" to react in one of these ways; without it, the species would eventually cease to exist due to predatory destruction.

The brain is obviously the "computer" that is programmed to direct appropriate responses to a threat. Because these instinctual reactions of self-preservation originated of necessity in the early stages of evolution, the mechanisms in the brain that initiate violent behavior are understandably found in the most primitive part of the brain system: the *brain stem*. Similarly, the part of the brain that *controls* violent behavior is to be found in the deepest and most primitive part of the cerebrum, or "large brain": the *limbic brain*. This limbic brain developed in animals 400 million years ago and was eventually "inherited" by humans in the evolutionary process—complete with its program for controlling violence.

It is, then, the limbic brain in humans that is primarily responsible for governing violent behavior. Yet because of the increasing complexity in the structure of humans as they evolved, the potential for a malfunction in this critical limbic system also

increased. As Vernon Mark and Frank Ervin of the Harvard Medical School explained, the proper functioning of the limbic system

> depends on the synchronization of many complex and delicately balanced subsystems. Because self-preservation is so often associated with violence, it is easy to see that a disturbance or disorder of brain mechanisms has the potential of releasing violent behavior even when the circumstances do not call for it. Moreover, the chance of such a disturbance or disorder occurring increases with any increase in the complexity of the nervous system, and also with any increase in the variety of environmental stimuli to which an animal must respond in order to survive.[1]

Despite the complexities of the limbic system, it is possible to isolate the normal functions of this part of the brain. One way is to remove parts of the system surgically and record the results. Scientists have done this with monkeys. When the temporal lobes of the monkey's brain are removed, much of the limbic system is thereby destroyed. The result, scientists discovered, is that monkeys that normally attack an approaching person become, instead, placid and easily handled.[2] When attacked by other monkeys, these "lobotomized" monkeys do not respond in the usual aggressive manner.

Since these relatively early experiments, scientists have been able to isolate the "taming" effect as being the result of destruction of the *amygdala*, an almond-shaped structure in the front of the temporal lobe. The amygdala is, apparently, a critical organ in the regulation of emotions in general and of fear, rage and anger in particular. Again, scientists have experimented with various animals by surgically removing this part of the temporal lobe and observing resultant behavior changes. Thus normally vicious wolverines and lynxes have been instantly "tamed" by this surgical procedure, and rats bred for their killer instinct become unwilling to attack even mice.[3]

Another method of determining the existence of a link between physiological structure and violent behavior is to stimulate the brain with electric current through electrodes placed in various parts of the brain. The weak electrical curent acts to excite that portion of the brain into activity.

Thus electrical stimulation of the brain region that controls hearing has been shown to result in a sensation of sound, and stimulating parts of the brain that control arm movement produces, in fact, an involuntary movement of the hand or arm.

Scientists have caused electrical stimulation in various parts of a cat's limbic brain, with the result that the cat's hair bristles, its back arches, its pupils enlarge, its claws extend and it growls and spits.[4] When confronted with another cat, the stimulated cat will viciously attack it. In fact, confronted with a maze or a barrier, the stimulated cat will frantically try to negotiate the maze or climb the barrier to get at a mouse or another cat. Again, the greatest degree of violent behavior was apparent when the amygdala was stimulated, with similar reactions when the middle of the hypothalamus received the electrical current; considerably more restrained aggressive reactions were obtained when the side of the hypothalamus was excited. Interestingly, these violent rages could be quickly stopped by electrically stimulating other parts of the limbic system; when so stimulated, the raging cat would almost instantly calm down and begin to groom itself.

What is true of the brains of mammals generally is true of the human brain. As the two Harvard medical professors noted:

> It seems clear that there exists within the (human) brain a definable neural system which organizes effective and directed attack behavior. This system has a long and honorable evolutionary past; indeed, it was necessary for survival and still exists in mammals of normally peaceful mien. . . .
>
> [T]he mechanisms of violence are there, but they need never be out of control. If they are, one of two things has occurred: either the limbic system has become pathologically hyperactive due to a lesion or stimulation, or its neocortical (control) inputs have become abnormal.[5]

That aggressive and even violent behavior in humans can be directly affected by disorders relating to the limbic brain system has been demonstrated repeatedly. One method is simply to observe the reactions of humans who are suffering from some type of damage to that portion of the brain. Thus children who have received damage to the limbic brain during childbirth due to

oxygen starvation are often later observed to be hyperactive, indiscriminately aggressive and impulsively violent. Yet when these children are operated on and parts of the amygdala are surgically destroyed, most of them thereafter engage in more sociable behavior and even exhibit previously undetected intellectual abilities.[6] Similarly, it has been observed that severe blows to the head causing damage to the limbic region can result in aggressive and combative behavior.[7] Of interest also is the fact that the virus that causes rabies primarily attacks the brain cells in the limbic region, resulting in the well-known symptoms of uncontrollable rage and violence.

Tumors in the limbic brain area should, if this theory is correct, cause antisocial behavior. This has fairly consistently been proven to be true. In one study of eighteen individuals with limbic brain tumors, all were found to have some psychiatric disorder and all exhibited hostile, aggressive and in some cases even assaultive or suicidal behavior.[8]

Perhaps the most interesting research in this area, however, has involved the study of epilepsy. Epilepsy is not a disease but a disorder of the brain that may have hereditary aspects. This disorder is characterized by electrical discharges within the brain and the resultant excessive stimulation of certain areas. The discharge is manifested in a seizure.

Where the electrical discharges occur in the temporal lobe area of the limbic brain, the reaction of the individual can take on a potentially criminal nature. The symptoms of an attack of temporal lobe epilepsy have been described by Mark and Ervin:

> The epileptic may feel severely depressed or he may be overwhelmed by fear to which he may react by running wildly and aimlessly or by becoming aggressive. Indeed, individuals with temporal lobe seizures or generalized seizures involving the temporal lobe very often have difficulty maintaining control over their behavior. . . .[9]

Or as a scientist has concluded after extensive research, "patients with temporal lobe epilepsy are usually prone to mental disorders and amongst these disorders overtly aggressive and rude behavior is the most common single entity."[10]

This has been confirmed in an interesting fashion in a Rus-

sian survey. Scientists in Russia compared the incidence and type of any criminal offenses committed by epileptics and schizophrenics.[11] Individuals suffering from schizophrenia are generally conceded to be high risks for criminal behavior. Yet the Russian scientists contrasted the randomly selected records of eighty-four schizophrenics with those of eighty-four epileptics and found that the epileptics were more often guilty of violent offenses, were more often intoxicated at the time of the offense and were more likely to be repeat offenders.

An interesting study of one individual suffering from temporal lobe epilepsy is recounted by the two Harvard professors.[12] This person was forty-eight years old before she had her first recognizable seizure. She was treated with drugs after having had five attacks and appeared to have recovered. Six months later, though, she began experiencing periods of unconsciousness lasting for fifteen to twenty seconds accompanied by symptoms of temporal lobe epilepsy such as staring, lip smacking, salivation and feelings of anxiety and panic. These new seizures could not be controlled with drugs, and during the next ten years her personality changed noticeably. Whereas she had always been an outgoing, happy, even effervescent person, she now experienced extended periods of depression and surliness. She began to start fires, in many of which she was herself repeatedly burned. If members of her family made critical comments, she would attempt to strike them with her fists. On at least twelve occasions she beat her husband with a broom handle; finally, she tried to stab him with a knife. At last this unfortunate woman submitted to brain surgery. The surgeon destroyed the left amygdala portion of her limbic brain system. Since this operation, her uncontrollable rages have ceased, she has set no more fires and she seems to have resumed once again her role as a wife and mother.

As was probably true with this woman, researchers have discovered that antisocial behavior does not occur only during or immediately preceding an attack, as had previously been thought. Rather, there appear to be much smaller, even undetectable, electrical discharges in the temporal lobe region between these attacks that act to affect profoundly the individual's "normal" behavior. Two scientists at the National Institute of Health at Bethesda have conducted extensive experiments with temporal

lobe epileptics, concentrating on this inter-seizure pattern of behavior.[13] They personally interviewed twenty-seven patients suffering from temporal lobe epilepsy and gave them a battery of psychological tests. As a comparison group, twelve normal individuals comparable in terms of age, education and socioeconomic level were also interviewed and tested. After observing that epilepsy "outside the temporal lobes does not generally present abnormalities of emotion and behavior," the researchers concluded that temporal lobe epileptics exhibit a considerably greater degree of anger, emotionality, depression, obsessive behavior, paranoia and aggression during supposedly "normal" periods between seizures. In fact, these scientists were able to isolate the causes of such feelings further:

> Experiencing objects and events shot through with effective coloration engenders a mystically religious world view; if a patient's immediate actions and thoughts are so [invested with libidinal energy], the result is an augmented sense of personal destiny. A felt significance behind events that others dismiss constitutes a seed bed for paranoia or may confirm the feeling that the patient is a passive pawn in the hands of powerful forces that structure the world. Feeling fervently about rules and laws may lead to action in which the patient "takes the law into his own hands."[14]

This behavioral profile of a temporal lobe epileptic has an ominously familar sound. In fact, one does not have to look very hard to find an example of the condition: Jack Ruby, the killer of presidential assassin Lee Harvey Oswald, was diagnosed as a temporal lobe epileptic.[15] Other famous assassins and murderers have been similarly diagnosed by reputable physicians.[16]

Yet the typical reaction to research concerning brain disease or damage affecting the limbic region is that the problem is relatively rare and thus of no great impact. This reaction is unfortunate. As at least one commentator observed, in the United States there are approximately 2.0 million epileptics, many of whom are temporal lobe epileptics. There are another 2.5 million children who suffer from hyperkinetic behavior disorders possibly related to limbic region damage.[17] It is unknown how many of the roughly 1.5 million American men who were wounded in World Wars I and II, the Korean conflict, and

Vietnam suffered head injuries. It is known, however, that 3.0 million people suffer injuries in car accidents every year; in one-third of all head injuries sustained in these accidents there is some permanent brain damage. Finally, there is mounting evidence that dietary deficiency can have a negative effect on the development of the brain. What other diseases, inherited abnormalities and accidents result in damage to the limbic brain—and, thereby, possibly create violent behavior—is not yet known.

Scientists are still trying to understand the vast complexities of this dark corner of the human brain. Although some epilepsy may be detectable through observation of recognizable brain waves, other dysfunctions of the limbic system are not so discernible.

Mark and Ervin conducted an interesting study. In an attempt to detect the possible existence of limbic brain dysfunction in penal institutions, they compared the symptoms commonly shared by patients at Massachusetts General Hospital known to have limbic brain disease and temporal lobe epilepsy with four hundred violent prisoners in a large penitentiary. The results indicated that many of these violent prisoners displayed the same symptoms exhibited by the patients. The physicians found that approximately 50 percent of the prisoners exhibited epileptic-like phenomena. Interestingly, thirty-eight of these prisoners had obvious and known histories of epilepsy, representing an incidence of more than ten times that found in the general population.

In an attempt to identify this vague group of individuals who act violently as the result of temporal lobe abnormality, damage or disease, the Harvard professors coined the term *dyscontrol syndrome*. Although this undoubtedly covers a variety of types of organic brain problems, the common thread is an improperly functioning limbic brain system, reflected in continuing aggressive and even violent behavior. If the brain dysfunction cannot always be diagnosed, however, the symptoms exhibited in antisocial conduct can perhaps be recognized. As the professors commented:

> The Texas tower tragedy in Austin, in which Charles Whitman shot 41 people, killing 17, and Richard Speck's murder of 8 nurses in Chi-

cago, are examples of preventable catastrophes. Weeks before committing his crime, Whitman told a psychiatrist of having "forced thoughts" about climbing the tower and killing many students with a rifle. And after he was killed, his postmortem examination showed he had a brain cancer. . . . Richard Speck, too, had symptoms of serious brain disease. . . .

Lee Harvey Oswald . . . is another example of someone about whom eventual murders could have been predicted. He had a history of repeated episodes of uncontrolled impulsive assaultive behavior.[18]

If further confirmation of the link between brain physiology and criminal behavior is needed, a recent study has compared the brain waves emitted by normal adults with that of diagnosed aggressive psychopaths.[19] The findings indicate that 10–15 percent of the general adult population reflect abnormal waves on an electroenchephalogram (EEG), and 48–70 percent of aggressive adult psychopaths showed such waves. This would seem to be clear evidence of a physiological source for criminal behavior.

One cannot help wondering, as the symptoms for temporal lobe dysfunctions are reviewed, how many individuals have been convicted in our criminal courts for conduct that was the result of this undiagnosed condition and, having been sentenced, how futile the traditional attempts at punishment, behavioral rehabilitation and future deterrence would prove—premised as they are upon the belief that behavior can be modified through an exercise of the will.

It has been clearly established that genetic abnormality, disease or damage in the limbic regions of the brain can result in uncontrollable violent conduct. This scientific fact is conceptually important to the criminal justice system in a number of ways. First, it indicates that the possibility of brain dysfunctions should be more carefully considered in the disposition of criminal cases. Second, it helps establish the basic premise that at least some antisocial behavior can be caused by biological factors; insofar as it is biologically caused, the traditional concepts of guilt, deterrence and environmental rehabilitation (prison, and so on) lose relevance. Third, once the fact is accepted that crim-

inal behavior *can* have organic rather than environmental roots, it is not unreasonable to recognize that much of our organic makeup is inherited and that, consequently, criminal behavior is capable of being influenced by genetic factors.

The idea that a given type of complex human behavior can fairly reliably be produced by simply stimulating or damaging an identifiable part of the brain is one with which environmental determinists are uncomfortable. This view, they say, would characterize humans as little more than robots, acting and reacting as their organs and hormones dictate. This is not strictly true, and it is also not true that a human being is no more than a uniform and perfect ball of putty at birth, to be given behavioral shape by environmental factors such as family, education and peer group pressures. Humans are complex creatures, and their behavior is usually the result of an extremely complex interaction between biological and environmental factors. To ignore either is to become blind to cause—and to cure.

Most aggravated forms of criminal behavior—acts of violence against others—may have physiological origins. Obviously, these biological drives can, in most individuals, be successfully tempered by socialization. Equally obviously, they often cannot. In either case, it is important to understand the true *origins* of the violent behavior, to understand that it originates in an organic not environmental source. In finally recognizing the true causes of the problem, perhaps the solutions will become more readily accessible.

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7

The Chemistry of Violence

Reflecting the approach of the environmental determinists, Ashley-Montague wrote that "all of man's natural inclinations are toward the development of goodness . . . there is not a shred of evidence that man is born with 'hostile' impulses."[1] This is a simplistic and naive approach to the problem of identifying the causes of criminal behavior. The fact is that there is much more than a "shred of evidence" to indicate that humans are, after all, born with "hostile impulses." As one noted scientist observed, "there is abundant evidence that man has innate neural and endocrine organizations which when activated result in hostile thoughts and behaviors. . . . [T]here are a number of different kinds of aggressive behavior and each of them has a different physiological basis."[2]

Humans are born a biologically complex structure, and perhaps the most complex and difficult to understand aspect of this structure is the brain's relationship with the body's biochemistry. Yet here, in the interplay of the brain and nervous system with hormones and chemicals, are the answers to much of the behavioral puzzle.

The inherited physiological structure of the brain itself appears to be a significant factor in explaining the causes of antisocial behavior. As has been discussed in the preceding chapter, there are identifiable parts of the brain that clearly create or inhibit aggressive behavior. Thus, for example, if the lateral hypothalamus of the brain is stimulated in an animal, that animal

will suddenly become vicious and attack humans or other animals. Conversely, if the medial hypothalamus is stimulated, the animal will become peaceable and even ignore nearby prey.[3] Like most animals, humans react similarly, and numerous studies have documented the creation of aggressive and even violent behavior in men and women who have had specific regions of the brain stimulated chemically or electrically.[4] As an example, one physician reported the case of a woman who was normally calm and mild mannered. When the amygdala portion of her brain received mild electrical stimulation, she suddenly became aggressive and verbally hostile and actually threatened the physician with physical violence. When the stimulation ceased, she resumed her passive behavior and apologized for her uncharacteristic conduct. This woman—and most other individuals—could be changed from a peaceable person into a violent one and back again with the flick of a switch.[5]

Yet another illustration of the obvious effects of brain physiology on criminal behavior is offered by the results of brain surgery on one particularly violent patient. This patient had frequently exhibited aggressive behavior and had repeatedly attacked other people. He had even tried to strangle his own mother and on one occasion attempted to crush his younger brother with his feet. Yet after surgical destruction of a small portion of his brain he became meek and exhibited no further aggressive behavior.[6]

These cases are only examples of commonly encountered reactions. Such experiments and operations have been performed thousands of times and almost always with the same result: The violent behavior is wholly created or completely disappears. The point is not that brain surgery should be a recommended procedure where antisocial behavior recurs in an individual but that these surgical methods serve to identify the mechanisms that create aggressive behavior.

These "aggression systems," developed as a defensive mechanism over the millions of years of evolution, are obviously not active most of the time. As has been discussed, they may be almost constantly active in individuals with damaged or genetically abnormal brain structure. In most individuals, however, the systems must be activated and deactivated to achieve aggressive behavioral patterns. Thus we must examine what it is that

acts as the "trigger" to set off the system, and what acts as the "inhibitor" to deactivate it. Just as the brain itself may be defective, so may that part of the system that stimulates it.

One of the methods by which the human body activates the aggression system is through hormones produced by the endocrine glands. Thus the level of the male hormone testosterone in the blood system has repeatedly been shown to be correlated with aggressive behavior. In the woman, an imbalance of the female hormone progesterone may also result in violent behavior as a part of what has been termed a "premenstrual syndrome." Thus these sex hormones appear to be instrumental in some way to a brain-triggering system of aggression. These biological phenomena are discussed in chapters 8 and 9.

Another interesting biochemical clue to the identity of aggression "triggers" is found in the medical condition known as hypoglycemia. This condition is caused by low sugar (glucose) levels in the blood and has often been associated with attitudes of hostility. San Francisco politician Daniel White presented evidence that he was suffering from this condition in the famous trial involving the murder of Mayor George Moscone. White's lawyers were able to convince the jury that he had experienced a temporary "personality change" as the result of overindulging in high-carbohydrate "junk food" that caused a shortage of sugar in his system. The so-called "Twinkie defense" was successful: The jury returned a verdict of manslaughter, rather than first-degree murder, on the theory that his homicidal act was the result of an impulse caused by a biochemical reaction in his body.

In one study conducted of six hundred patients suffering from hypoglycemia, 89 percent displayed a psychiatrically documented "hyperirritability," and full 45 percent showed "unsocial, asocial or antisocial behavior."[7] Yet when the blood sugar is raised, physicians have noticed that hypoglycemics become "quite civilized."[8] Clearly, something in the fact of low blood sugar, or some biochemical cause of or reaction to that condition, acts as yet one further "trigger" to the aggressive mechanisms in the brain.

As with any discussion of body chemistry and behavior, the causes of unusual conduct cannot always be attributed to a single source. The body consists of a complex interrelation of hor-

mones, chemicals and organs, and a defect in one area can create a defect in another, or a combination of interrelated defects can create the abnormal behavior. Thus, for example, one research physician noted a relationship between antisocial conduct and the related conditions of hypoglycemia and temporal lobe epilepsy:

> Two sorts of limbic system disorder have been found to combine in the causation of aggressive behavior. When recurrent hypoglycemia is added to temporal lobe EEG dysfunction, the result will probably include psychomotor disturbances which resemble temper tantrums. The hypoglycemic episodes can combine to kindle or lower the response threshold of dopamine receptors of the ergotropic sympathetic response system. . . .
>
> [T]emporal lobe EEG disturbance can combine synergistically with hypoglycemia. The limbic system has a very large number of dopamine receptors in its ergotropic neurones. When their response threshold has been lowered by the kindling effect of repeated hypoglycemic episodes, they will become unusually sensitive to relatively minor stimuli.[9]

This has been confirmed by other scientists. Thus two medical researchers reported finding that hypoglycemia and temporal lobe dysfunction combine to cause an individual to become prone to aggressive temper-tantrum behavior as the result of what would normally be insignificant provocation.[10] The result can clearly be a violent criminal act, as, for example, when a person attempts to kill another because of some trifling incident.

Individuals suffering from low blood sugar can also apparently react violently when they eat certain kinds of food. Although this is another area that is not yet fully understood by modern science, it has been observed that hypoglycemia tends to intensify allergic reactions in such a way that the individual can become abnormally aggressive and even violent.[11] Again, some unknown biochemical reaction is taking place that acts to set off the individual's aggressive mechanism, resulting in antisocial behavior having little or nothing to do with environmental influences.

Depression, too, with its often attendent criminal behavior, is now in the process of being discovered to have not an environ-

mental but a biological source—with genetic origins for the biological causes. Herbert Meltzer, professor of psychiatry at the University of Chicago, recently discovered that certain kinds of depression can be diagnosed by analyzing an individual's blood platelets. Platelets are found in the blood, where they assist in clotting, and in the brain, where they act as transmitters of neural message impulses. On the surface of these tiny cells are uptake sites for serotonin, a substance that has been discovered to exist in insufficient amounts in persons who have clinically been found to suffer from recurrent severe depression. "In some depressed patients," Metzger reported, "the total of serotonin uptake sites will be thirty to forty percent below average."[12]

Researchers at the Karolinska Institute in Sweden confirmed these findings in their studies of the causes of suicide. They found that suicidal tendencies in an individual can be fairly reliably predicted by testing his spinal fluid for 5-HIAA, a metabolic byproduct of serotonin. In one of their surveys, these scientists discovered that more than 65 percent of the people who had attempted to kill themselves possessed insufficient levels of this material in their bodies. "The substance appears to act as a brake," said a member of the Swedish research team. "If you can't apply the brake, you might do things other people wouldn't."[13] Two American scientists found evidence that the physiological causes of depression are genetic, that a predisposition to depressive illnesses are present in the genes and occur in clusters within families.[14]

Yet another example of a genetically transmitted condition that results in antisocial behavior patterns is represented by Tourette's syndrome. This condition, found usually only in males, manifests itself in facial tics, jerking movements, and uncontrollable obscene language and hostile conduct. Scientists at Yale University discovered evidence that defective neurotransmitter systems in the body are to blame. "We take the view that this is the tip of the iceberg," noted one of the investigators. A scientist at Mount Sinai School of Medicine, in commenting on the syndrome and its causes, said, "Recently, the genetic contribution to extreme problem behaviors in young people . . . has been confirmed. And it's a basic tenet in biology that where extremes exist, it's a sure bet a variety of milder forms are responsible for

a wide range of related problems.[15] In other words, other types of inherited biological defects, as yet undetected, can also cause deviant behavior.

The key to the biochemical triggering system—and the causes of defects in that system—has been the subject of extensive research and experimentation. No definitive answers have yet been found to explain the biochemical process by which the critical parts of the brain's aggression program is activated and deactivated. However, it appears that answers are imminent, for scientists are beginning to discover clues—clues that continue to indicate that violent criminal behavior has a genetic factor.

One group of scientists has studied the effects of adenosine monophosphate (AMP), a cellular enzyme in the body whose purpose is to act as a kind of mediator between hormones and nerve-cell transmitters in a manner not entirely understood. Its counterpart is AMP phosphodiesterose, a cellular enzyme whose sole apparent purpose is to destroy AMP. These two enzymes appear to have some important role in the creation of aggression. Thus unusually high levels of AMP have been observed to result in "hyperanxiety" states, fear-motivated aggression, and even mania.[16] Conversely, high levels of AMP phosphodiesterose cause greater destruction of AMP and a resulting decrease in aggression—to the point of causing severe depression.

Similar research has been conducted by a number of different teams on the effects of another chemical found in the brain: norepinephrine. This substance was discovered to be present in reduced amounts when the brains of cats were electrically stimulated to induce violently aggressive behavior.[17] Further experiments indicated that there was, in fact, a correlation between rage and lowered norepinephrine in the brain and in the adrenal glands. It was found that drugs that stimulated the action of norepinephrine when injected into the animal caused violent behavior; drugs that impaired the chemical's action resulted in passive behavior. Furthermore, the amount of norepinephrine in the brain is proportional to the intensity of the aggressive behavior.[18]

Scientists have concluded that the flooding of the nerve synapses in the brain stem and limbic brain with norepinephrine is

a factor in the causation of violent behavior; the chemical is thus used up and found in depleted amounts in the brain.[19]

This theory has been applied in observations of manic-depressive patients at the National Institute of Mental Health. These patients were carefully observed every eight hours and rated by psychiatrists as to their relative levels of manic-depressive symptoms. Every twenty-four hours a urine sample was collected from each patient and analyzed for the presence of norepinephrine and other chemicals. After correlating the data, the research team observed that there was a significant increase in norepinephrine in the brain on the day *before* patients experienced an episode of mania. Conversely, the levels of the chemical decreased on days when the patients experienced a shift back to a depressed state. What does all of this mean? As one scientist concluded:

> The most interesting feature of this study is that the changes in norepinephrine occur prior to any detectable symptom of mania. They may therefore act as biochemical triggers for the switch to mania. . . .
>
> The implications of the work are directly relevant to the understanding of aggression. . . . Manic states are typically linked to overt aggression.[20]

Thus there appears to exist yet another biochemical factor in human behavior—a factor that can, like any other, be subject to malfunctions, and it is these malfunctions that can result in violent criminal behavior. Furthermore, the malfunction of the norepinephrine triggering system would, again, appear to be yet another inherited trait. As the scientists who conducted the study of manic-depressive patients concluded, "manic depressive states are in part genetically determined (certainly the entity appears to have familial trends). Patients with this predisposition are susceptible to certain stresses and chemicals which can trigger the effect."[21]

Yet other substances in the body's intricate biochemical makeup appear to have critical roles in the activation and deactivation of aggressive responses in humans. After isolating norepinephrine, scientists next uncovered the effects of acetylcholine on the brain. When injected into cats and rats, the animals become violently

aggressive and attack humans and other animals.[22] Murderous behavior has been created even in animal species that do not normally engage in killing activity. Conversely, when atropine methyl nitrate, a chemical that acts to inhibit the production of acetylcholine, is injected into a strain of rats bred for their "killer instinct," they become tame and refuse to attack mice.[23]

This biochemical trigger for the brain's aggressive mechanism also has its deactivating counterpart in the body: acetylcholinesterase. This chemical acts to break down acetylcholine and thereby shut down aggressive behavior. Thus when neostigmine, an inhibitor of acetylcholinesterase, is injected into an animal, the deactivation process is blocked and the animal is observed to engage in violent and even murderous conduct.[24]

It would therefore appear that yet another clue to the biochemical activation and deactivation of the brain's aggression system has been identified. Whether each of these various chemicals found in the human brain constitutes a separate triggering system, or whether they are all interrelated in some way, is not yet known. But as scientists have concluded of the acetylcholine-acetylcholinesterase function, "on the balance the research [suggests] . . . an influence of acetylcholine on [homicidal] aggression. . . . [T]here is mounting evidence that [these biochemical] processes do act to modify aggression, and in a very striking manner."[25]

Yet another recent inquiry into the possibility of genetically transmitted biochemical determinants of violent behavior involves the presence of abnormal amounts of trace elements in the body. An analytical chemist at Argonne National Laboratory, William Walsh, theorized that inborn chemical imbalances may be a cause of criminal violence. If an individual's metabolism was genetically defective so that abnormal levels of chemicals were retained by the body, he reasoned, there may be an impact on that individual's patterns of behavior.

Walsh proceeded to analyze twenty-four pairs of brothers between the ages of eight and eighteen. "I selected pairs where there was a very delinquent, violent kid in the same family, eating the same food with an 'all-American boy'—a kid who had never been in trouble, who was an excellent student, and whose incidence of violence was zero," Walsh said.[26] He then attempted to ana-

lyze the levels of various trace elements in the bodies of each of the boys. The use of blood samples was rejected, as such substances have usually been used, eliminated or stored within hours or days of entering the bloodstream. Hair, on the other hand, has about two hundred times the concentration of trace elements in it than blood does, and represents a longer term indication of the presence of these elements.

"The results were quite clear," Walsh observed.[27] Chemical analysis using atomic-absorption mass spectroscopy indicated that all twenty-four of the violent boys had extremely high levels of lead, cadmium, iron and calcium, and extremely low levels of zinc, lithium and cobalt. Yet, none of the nonviolent brothers were found to have unusual levels of trace minerals—despite similar if not identical diets.

Walsh then tested these results with a group of ninety-six adult men with known histories of extreme violence. To ensure that any results were not simply reflective of similar diets, he selected the ninety-six from two different prisons as well as from lists of men who had left prison at least two years earlier and men who were first offenders and had not yet been incarcerated. For comparison purposes, he selected another group of nonviolent men who were matched to the violent group by age, race, socioeconomic status, and urban-rural environment; one-third of each group were blacks, for example, one-third Hispanic, and one-third of European heritage.

The results were again impressive. All but four of the violent men exhibited the trace mineral patterns found by Walsh earlier in the boys. Interestingly, the four not reflecting this pattern were simply very low in *every* trace element. By comparison, the ninety-four nonviolent men all had normal levels of the elements.

Cobalt appeared to be the most effective predictor of violence: The lower the level of cobalt in an individual's hair sample, the more violent the individual's history. "We took a group of violent people and controls," Walsh observed upon concluding his study in 1983, "and found you could practically predict their degree of violence from cobalt concentrations."[28]

Other materials found in the human body are in the process of being studied as to their effects on behavior. A group of brain

proteins called endorphins was discovered at the Salk Institute to cause various kinds of behavior in laboratory animals: One type will cause them to experience anxiety, another will transform angry animals into docile ones and a third will cause the animal to groom himself.[29] Another group of brain proteins, enkephalins, is apparently critical in the learning process. Researchers at Tulane University School of Medicine found that injection of enkephalins into rats enables them to learn how to negotiate a maze much more quickly than would normally be expected.[30] These physicians have already achieved positive results by using the protein in improving attention and learning among both healthy and mentally retarded human beings.

It is clear that human behavior is directly affected by biochemical factors. Applied to criminal behavior, it appears that the brain's biochemistry has a significant role in activating and deactivating the aggressive mechanisms that cause the very antisocial behavior that sociologists insist is the exclusive result of environmental influences. Certainly, environmental influences can be relevant to the causation of hostile conduct. But just as certainly, physiological factors—both inherited and acquired—are at least as important. The answer probably lies somewhere in a complex interreaction of the two. But it is a terrible mistake to ignore the critical role that hereditary biochemistry plays in the causes of criminal conduct. When chemicals normally present in the brain can be manipulated to produce predictable behavior, the relevance of that link cannot be rejected. As one scientist observed:

> It is quite feasible that drugs used in natural settings can be used to dissect social behaviors into their individual components and specify their contextual significance. This approach relies on the genetically programmed neural structures that have evolved for adaptive reasons. Moreover, it can capitalize on hormone-dependent behaviors, so that functional links can be established between hormones, brain biochemistry and behavior. Here, then, is perhaps one of the most significant areas for piecing together the puzzle called aggression.[31]

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8

The Case of the Super Male

One of the most interesting areas of study involving the comparative effects of genetics and environment on criminal behavior concerns the XYY chromosomal deviation, or the so-called *super male syndrome*. Few areas of research have generated such heated debate as have these investigations into the behavioral consequences of a specific genetic abnormality, threatening, as they do, the very heart of our traditional concepts of free will and environmental determinism. Here we have an identifiable genetic aberration that is inherited and that may be directly responsible for antisocial conduct. The implications of such findings are critical to our criminal justice system: If any individual is, in a sense, genetically "programmed" before birth to commit criminal acts, our traditional attitudes toward guilt and punishment must be reassessed. If we are able to identify one such genetic condition that is influential—we must accept the entire concept—that is, that much of crime today may be caused or at least strongly influenced by other, as yet undetected, genetic factors.

The XYY aberration involves a variation in the number and/or sexual characteristics of the chromosomes. Normally, every person carries forty-six chromosomes in every cell of his body, ar-

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ranged in twenty-three pairs. Of these twenty-three pairs, twenty-two are *autosomes*, or genes that contain most of the individual's biological characteristics (for example, color of hair, height, nose shape); the remaining pair of genes are *gonosomes*, which determine remaining traits such as the primary sexual characteristics. In women, these paired sex chromosomes are called X chromosomes; in men, the gonosomes are represented by one of the X chromosomes paired with a much smaller male Y chromosome. They are referred to by geneticists as the XX and XY gonosomes, and their presence in a fertilized egg determines, among other things, whether the child will be a male or a female.[1]

On a rare occasion, however, the process of fertilization by the male sperm of the female ovum malfunctions, and a fetus is created that contains chromosomal abnormalities. If an extra twenty-first chromosome is present, for example, a mental and physical defect known as Down's syndrome (mongolism) results. Similarly, if the gonosomes, or "sex chromosomes," are altered in number or characteristic, certain fairly predictable results will take place. Thus when an extra X chromosome is added to a female XX pair, the result is the XXX or so-called super female syndrome; such women will appear physically normal but will be mentally retarded.

The studies relevant to this discussion involve aberrations in the male XY gonosomes. These abnormalities can occur in a number of ways. If an extra X chromosome occurs, an XXY male is created; also known as the "Klinefelter syndrome," such an individual will be mildly retarded, have some breast enlargement and be sterile. The Klinefelter syndrome occurs approximately once in four hundred male births, and there are indications that besides the obvious defects there may also be present antisocial behavioral patterns such as alcoholism and homosexuality.[2]

Similarly, if the male gonosomes receive an additional Y chromosome, that is, an extra "male" chromosome, the XYY or so-called super male is created. Such individuals apparently tend to be much taller than average and often have an acne condition of the skin. Thus in one study in a British prison, one-half of the twenty-two XYY men were found to be in the tallest

5 percent of the general prison population. Although such men may be of normal or even short stature, the vast majority are six feet tall or over. The incidence of severe acne condition appears to be less widespread among XYY men, but the prevalence rate is still unusually high—probably about 50 percent.[3]

Statistically, it is currently believed that a super male occurs about once in every one thousand male births. This figure is based upon a study where thirty-five hundred consecutive male births in a hospital were observed and the babies chromosome-typed for chromosomal aberrations.[4] In only five was the XYY defect detected, indicating a rate of 0.14 percent, or a little more than one super male per thousand births. This figure has been roughly confirmed in subsequent studies.

A series of scientific surveys beginning in the early 1960s, however, seemed to indicate that there was a much higher rate of sex chromosome deviations in prison populations. The figures were initially dismissed as coincidental, but the studies continued consistently to reflect extraordinarily high percentages of sex chromosome aberrations in penal and mental institutions. Thus in 1963 a survey of 760 patients in three Swedish institutions for criminal and "hard-to-manage" males of subnormal intelligence was conducted. Two percent of these institutionalized men were found to have aberrations in the sex chromosomes.[5] In a study of 942 males in two similar institutions in England, 2.2 percent appeared to have sex chromosome deviations.[6]

Intrigued by these initial indications, a team of scientists in Great Britain decided to attempt to discover whether any specific sex chromosomal deviation was more prevalent than others in prison. If a particular deviation was, in fact, consistently present in unusually high numbers, they wanted to know why: Was there a link between the chromosomal aberration and criminal conduct? As the scientists later wrote, the early studies "led us to wonder whether an extra Y chromosome predisposes its carriers to unusually aggressive behavior. We decided that if this were the case, then we might expect an increased frequency of XYY males among those of a violent nature."[7]

The team conducted a study of inmates at the maximum-security state hospital at Carstairs, Scotland. The hospital housed 342 inmates; 249 of them had been committed by the British

courts, and all but 10 of the 342 had criminal records involving violent conduct. Of the 342, the team was able to obtain blood samples from 197 for purposes of testing for chromosomal abnormalities.

Assuming an XYY incidence rate of one in every 1,000, the researchers could expect to find none or possibly 1 among the 197 inmates tested at Carstairs. Instead, they found 7 carriers of the defect, an incidence of 3.5 percent, or thirty-five times what could statistically be expected. The team later concluded that finding 3.5 percent of the population to be XYY males indicated a much higher frequency than occurs in the general male population.

The implications of the Carstairs study are enormous and the reactions of the scientific community predictable. The data were immediately explained away by the environmental determinists; the research methods used by the team were attacked, the meaning of the figures was questioned, and the need for further studies to "clarify" the issue was stressed.

The further studies were not long in coming. The following year another group of researchers conducted a study of similar maximum-security institutions at Rampton and Moss Side in England.[8] In this study of mentally subnormal men detained because of antisocial behavior, only inmates over six feet tall were *karyotyped*, or checked for genetic aberration. Fifty such men were found. Amazingly, fully twelve of these men were discovered to be so-called super males—an incidence of 24 percent, or 240 times greater than would be expected.

The next significant study took place in 1968 in the United States. Again, scientists studied inmates of four criminal institutions, this time karyotyping only those over five feet eleven inches in height. Of the 129 men who were examined, 5 had the XYY aberration, giving an incidence rate of 4 percent. The American scientists concluded:

> The results of this limited survey appear to confirm British observations that gross chromosomal errors contribute, in small but consistent numbers, to the pool of antisocial, aggressive males who are mentally ill and who become institutionalized for criminal behavior. Our data show, furthermore, that these men are to be found in general prisons as well as in mental hospitals for the "hard to handle."

To this we would add the observation that despite good physical care and much psychiatric attention throughout repeated incarcerations, these individuals are not being identified in the institutions we have surveyed.[9]

In the same year, researchers in Australia conducted a study of thirty-four inmates at a Melbourne prison.[10] Four of them were found to be XYY deviants, giving an incidence rate approximately 125 times what would normally be expected. Interestingly, of the four deviants, one was a convicted murderer, one had been charged with murder but had been found unfit to enter a plea, one was convicted of attempted murder and only the fourth—a thief—had been convicted of an offense not involving at least the attempt to commit murder.

Scientists in Denmark were the next to test this strange phenomenon.[11] Researchers there karyotyped inmates over 180 centimeters (approximately five feet eleven inches) in height at a state hospital during a period of one-and-a-half years. Of 23 inmates who were studied, 3 were discovered to be carrying the XYY deviation, representing an incidence rate of 13.0 percent for men over 180 centimeters and of 0.25 percent of the entire population of the hospital surveyed during the year and a half (1,180 patients); even the 0.25 percent figure is 2.5 times higher than should be expected. Of the 3 XYY men, all had criminal records and all displayed aggressive character traits. Interestingly, none of the 3 had any history of criminality in his family; all had been raised in normal households.

In 1970 scientists in Australia conducted a karyotypic survey of inmates at a maximum-security ward for the male mentally ill.[12] Of fifty-five in the ward, three had gonosomal aberrations, again indicating an incidence rate vastly higher than should occur by chance alone.

In 1976 a team of psychologists from Denmark and the United States tracked down males who had been born in Copenhagen from 1944 to 1947.[13] They identified 4,139 of these men who were still living and were over six feet in height and then set about determining the existence of XYY in their blood. The resulting data indicated a 2.9 percent incidence for the condition. Much more interestingly, however, these men were investigated as to the existence of any criminal histories. Existing statistics

indicated that 9.3 percent of the general over-six-foot male population had suffered at least one criminal conviction. Among the "super males," however, this incidence of criminal convictions soared to fully 42 percent.

One year after this study, British researchers surveyed eighteen hospitals for the mentally handicapped in England for chromosomal aberration.[14] They found that the highest incidence (1.3 percent) was found in the hospital that had the highest proportion of patients with behavioral disorders. After studying the YXX men, the English researchers concluded that they were considered the "black sheep" of their families relatively early in their lives, that they showed little forethought and acted impulsively, and that there was a marked tendency to engage in "unusual or abnormal" behavior in the teenage years or earlier.

There have been other studies as well, usually reflecting similar indications of an abnormally high rate of criminal behavior among men carrying the XYY defect. In fact, one scientist collected all of the known studies—thirty-five in all—and collated their results into one huge data pool.[15] He began by eliminating the studies involving only tall men so that the figures would be consistent and then computed the composite frequency with which super males were found in criminal institutions. Finally, he collated all of the figures available for the incidence of XYY in the general population.

The results indicated that the composite average of all data for incidence in the general population was 1 in 975, or about 0.10 percent. The average of all of the studies of incidence in criminal institutions, on the other hand, was 2.05 percent, or more than twenty times what it is among the general population. Representing as it does the collective work of thirty-five teams of scientists from around the world—some with admitted biases against the idea of inherited behavior—the figure must be given considerable credence.

Can the findings be explained in a way acceptable to the environmental determinists? This was attempted in the original study of the Scottish inmates (the so-called Carstairs study). Batteries of psychological tests were given the XYY aberrants, but surprisingly there appeared to be no significant differences between that group and a control group of randomly selected inmates.

No significant physical differences were noted, other than the obvious high incidence of height among XYY males. However, a number of findings tended directly to refute the environmentalists' arguments that family or social factors were the basis for the antisocial conduct of the XYY aberrants. First, unlike the general institutional population, the XYY inmates were found to have no significant family history of mental illness or crime (of 9 XYY subjects investigated, only 1 had an immediate family member who had been convicted, and that member had been convicted only once; by contrast, of 18 control subjects at Carstairs, there were 139 convictions recorded in the immediate families). Second, the XYY inmates had histories of crime much earlier in their lives than did the control group (3 of the 9 XYY men had been convicted before reaching 10 years of age, and the group's mean age for the first conviction was 13.1; none of the randomly selected control group had been convicted before 10, and their mean age for the first conviction was 18). Third, it appeared from comparative studies that the XYY inmates were considerably more resistant to rehabilitation than were other inmates.

Thus the evidence seems to point very clearly to the simple fact that the criminal behavior of the super males was genetically caused, with relatively little effect from social or familial influences. Despite this landslide of scientific evidence, there is still no shortage of critics who question the studies. Thus those who advocate environmental determinism point to the fact that XYY males tend to be the products of lower socioeconomic groups. Because they tend to be generally less intelligent and less attractive, so the argument goes, a kind of "reverse natural selection" takes place that relegates adult XYY males to poorer socioeconomic status. Since the trait is genetically passed on, the children are more likely to have it than are children in other socioeconomic classes. Therefore, proportionately more XYY children will be subjected to poorer environmental influences and will accordingly be more represented in penal institutions. Thus, say the sociologists and psychologists, socioeconomic conditions—not genetics—are after all shown to be the primary factor in determining behavior.

This interpretation of the XYY results was tested by two sci-

entists in 1975.[16] The men studied the records of 14,206 consecutively born males in one hospital and obtained the karyotypes of each. They then obtained information about the educational and occupational status of as many of the fathers as possible. When all of the information was in, the two men had complete data on 10,348 of the males. The results clearly indicated that there was an even distribution of the incidence of XYY; there was absolutely no correlation between incidence and socioeconomic group.

These findings were confirmed in other studies that also found that XYY men do not appear more likely to come from families of lower socioeconomic status.[17] In fact, at least one such study indicated the contrary, that a slightly *higher* incidence of XYY was found in upper socioeconomic classes.[18]

Undaunted, the environmental determinists next pointed to the prevalence of height among XYYs. Personality patterns developing in children, they pointed out, are affected by their size: Larger children are treated differently from those of normal height, and thus their adult personalities and behavior patterns will not be "normal." Even after reaching adulthood, taller men are perceived by others as more "threatening," thus resulting in a "channeling" of behavior patterns.

The obvious answer to this was pointed out in an article by a research pediatrician.[19] If larger size is a major factor in the personality development of XYY males, the physician observed, it should also be a major factor in the development of non-XYY males of large size. Yet a review of studies concerning the incidence of tall men in prisons indicates that there is no correlation: The percentage of tall men in prisons is no different from the percentage of tall men in the general population.[20]

Very well, reply the antigenetics crowd, but the personalities of XYY men have been adversely affected by the fact that they tend to have severe acne. Again, children with acne conditions are treated differently from other children, with a resulting negative environmental effect on the developing behavioral patterns.

Again, however, existing data on the subject indicates that the incidence of severe acne (nodulocystic acne) among prisoners is approximately 5 percent, no different from the rate in the gen-

eral population.[21] Furthermore, the incidence of acne among XYY males is less than 50 percent; as at least two scientists have independently noted, such an incidence would not begin to be high enough to account for the vastly higher prevalence of XYY men in penal institutions.[22]

The conclusions that must be drawn from the XYY studies are difficult to avoid. As Mary Telfer of the Elwyn Institute in Philadelphia observed after conducting her own research in the area, the results are sufficiently definite to define an "XYY syndrome," with the symptoms described as "extremely tall stature, long limbs, with strikingly long arm span, facial acne, mild mental retardation, severe mental illness (including psychosis) and aggressive, antisocial behavior involving a long history of arrests, frequently beginning at an early age."[23]

The inferences of these studies are, of course, disturbing. That there appears to be a genetic "type" that is inherently disposed to criminal conduct appears to be a distinct possibility.

NOTES

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17. See, for example, W. M. Court-Brown, 5 *Journal of Medical Genetics* 341 (1968).

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22. Hook, *supra* note 3.

23. Telfer, *supra* note 9 at p. 1250.

9

The Chemical Woman

Janet is a thirty-year-old housewife living in London, England. She is in most respects a normal individual, with the exception that seven days before the beginning of her menstrual period she begins to experience irritability and tension, which accelerate into aggressive and finally violent behavior toward her husband, her children and even herself. This reaction every month began when she was a teenager, and in the intervening years she has attempted suicide by taking an overdose of aspirins, taking an overdose of Valium, slashing her wrists, stabbing herself and jumping off a train. Each of these suicide attempts occurred during her premenstrual cycle. Yet on the first day of menstruation all symptoms cease and she becomes calm, friendly and rational. Most recently, she requested authorities to take her five-year-old daughter into protective custody after physically attacking her during the violent phase of the cycle.[1]

Margaret, a thirty-five-year-old teacher married to a headmaster in England, described her monthly reaction to the approach of menstruation:

For seven days during the premenstruum I became tense, shouting, irritable, weepy, tired, bloated with swelling of legs and ankles and with

The author is indebted to Dr. Katharina Dalton, with whom he co-authored an article upon which this chapter is largely based. The article appeared in the December 1982 issue of the *California Western Law Review*.

headaches over the eyes. I have had two children and at those times when I am in an uncontrollable temper I have hit them really hard. . . .

I would never have believed that an intelligent woman like me, with high morals and good education, could ever lose control of herself to such an extent that she would batter her children, for I love my children dearly. How utterly illogical it is that I personally should cause them permanent damage.[2]

Anne, yet another sufferer from premenstrual effects, described her monthly change as one "from Dr. Jekyll to Mr. Hyde. I become subject to depression, feel emotionally unstable, hysterical and miserable. Sometimes I get so keyed up that I can't sleep for 2 to 3 days before a period starts. I feel pressure across the base of my skull as if my brain was swollen."[3]

These three Englishwomen suffer from a common malaise, one that has only recently begun to be recognized and designated as the "premenstrual syndrome," or PMS. In a 1981 article in the *American Journal of Obstetrics and Gynecology*, Robert L. Reid, M.D., and S.S.C. Yen, M.D., and chairman of the Department of Reproductive Medicine, School of Medicine, University of California (San Diego), recognized the phenomenon as "a major clinical entity affecting a large segment of the female population."[4] In another 1981 article, in *Medicine, Science and the Law*, a lecturer in forensic medicine at the London Hospital Medical College concluded that "the premenstrual syndrome is well established as an entity and its complications are now largely appreciated from the clinical point of view."[5] Richard E. Shader, professor and chairman of the Department of Psychiatry, Tufts University School of Medicine, discussed various treatments for the syndrome in an even more recent article in the *Journal of the American Medical Association* and estimated that severe PMS probably affects about 20 percent of the female population. The author of that same article reported that "Premenstrual syndrome (PMS) may be the newest women's health issue in the United States. Some severely affected women are now going public with their stories and are demanding treatment. . . . The small minority of women whose lives are seriously disrupted by the syndrome reputedly may experience exacerbations of chronic medical illnesses, abuse their children or

commit violent crimes. Some are said to be suicidal."[6] In 1982 the first "premenstrual clinic" in the United States was opened in Reading, Massachusetts, by Harvard psychiatrist-neuroendocrinologist Ronald V. Norris.

What is this newly emerging phenomenon called premenstrual syndrome? Although PMS is only now beginning to be recognized by medical experts around the world, general symptoms of menstrual distress have been around for some time. In the sixth century B.C., Semonides described the emotional condition of women he observed: "Often it lies calm and innocent and still . . . then it will go wild and turbulent . . . this woman's disposition is just like the sea's." More recently, the effects of the menstruation—although not necessarily of the syndrome itself—were recognized in female aviators. In a 1934 article that appeared in the *Journal of Aviation Medicine*, the causes of air accidents involving women pilots were studied. It was found that in a significant number of accidents where a specific cause for the accident could not be found, the pilot had been in the menstrual phase of her cycle.[7]

Recognition of the syndrome itself, however, as well as its criminological effects, has been a very recent development. Yet it has been the subject of intensive study by one physician for some time. Katharina Dalton, head of the Premenstrual Syndrome Clinic, University College Hospital, London, first discussed the phenomenon in 1953 in the *British Medical Journal*.[8] Since then she has written two books and fifty-one articles on the subject and is generally recognized as the pioneer in this area. Dalton defined the syndrome medically as "the presence of recurrent symptoms in the premenstruum or early menstruation with complete freedom of symptoms in the postmenstruum."[9] It is a hormone deficiency disease, created by the lack of the female hormone *progesterone*. It should not be confused with the much more common *menstrual distress*, which can be defined as "the presence of intermittent or continuous symptoms present throughout the menstrual cycle, which become worse at menstruation, either before, during or after."[10]

The syndrome includes a wide variety of symptoms that regularly recur in the same phase of each menstrual cycle, followed by a symptom-free phase. The average menstrual cycle

consists of about twenty-eight days, and although most women experience certain symptoms such as fatigue, depression or a bloated feeling, a significant minority of the female population suffers from much more severe symptoms, which begin a few days before the onset of menstruation and which reach a peak during the last four days of the *premenstruum* (premenstruation period) or the first four days of actual menstruation; this eight-day period is referred to as the *paramenstruum* (the phrase *premenstrual syndrome* was coined before the phenomenon was better understood; actually, the symptoms occur during the paramenstruum). Usually, they peak in a cluster about the time of menstruation, either before or during the onset, with the remaining days free from all symptoms; of course, timing as well as specific symptoms vary considerably from individual to individual.

Symptoms that can occur in this syndrome are of extraordinary diversity. One characteristic is the tendency for the patient to have a series of symptoms, with an increasing accumulation of these symptoms reaching a crescendo on the final day of the premenstruum. Frequent combinations of symptoms are tension, headache and mastitis and depression, backache and nausea. The symptoms do not necessarily all start at the same time. A woman may wake up one morning feeling that the world is against her and feeling tired, yet make an effort to get up and carry on with her normal routine. A couple of days later she may be conscious of painfully engorged breasts and realize that she is irritable with her children. Gradually, she develops a headache, which increases in severity over the next twelve to twenty-four hours until she is prostrated with photophobia, vomiting and a throbbing hemicranial headache. In contrast, the end of an attack is often abrupt, coinciding with the onset of the full menstrual flow, which may be described in phrases such as "a cloud lifts," "like a switch it's gone" or "suddenly my head clears and I know I will be all right."

The psychological symptoms of premenstrual tension, with its depression, irritability and lethargy, are undoubtedly the most common. As with all hormonal diseases, at times of stress the symptoms of the premenstrual syndrome are increased in severity and number. The mild and easily controlled tension or headache, otherwise relieved by a simple analgesic, may be sud-

denly exacerbated and additional symptoms become manifest at times of sorrow, anger, financial reversals, or whenever the life situation becomes intolerable. Stress can also alter the length of cycle and the severity of symptoms. This was illustrated in an analysis of the days of menstruation of ninety-one girls sixteen years of age at a boarding school in England who were all taking critical scholastic examinations at the same time.[11] Whereas an average of sixteen girls were menstruating on any one day during the previous month, on one day during the stress of examination week as many as thirty-six girls were menstruating.

The immediate biological causes of the psychological symptoms appear to result from a combination of the following biochemical effects of premenstruation: water retention, potassium depletion and sodium retention, hypoglycemia, allergic reactions, lowered resistance to infections and inflammatory reactions. The localization of water retention may be manifested in migraine headaches, epilepsy, sinus headaches and vertigo. The imbalance of potassium and sodium appears to be responsible for the tension symptoms of lethargy, muscle weakness, irritability and depression. Lowered blood sugar levels and lowered glucose tolerance during the premenstrual cycle may account for symptoms such as fainting, panic attacks, aggressive outbursts, headaches and nausea.

The immediate causes of PMS symptoms are often further exacerbated by consumption of alcohol. In a survey by two psychiatrists, 67 percent of menstruating female alcoholics related their drinking bouts to their menstrual cycle, and 100 percent indicated that drinking had begun or increased during the premenstruum.[12] Furthermore, alcohol appears to be more intoxicating during the paramenstruum when there is water retention present. This, together with a premenstrual lack of self-control and depression, causes many women to have a monthly drinking bout—often in secret. During an investigation in a prison, several women were noted to have a record of imprisonment at monthly intervals for being drunk and disorderly; one prisoner described how she always seemed to start menstruation in the jail cell shortly after her arrest.[13]

How widespread is PMS? As has already been indicated, Shader estimated that probably about 20 percent of the female popula-

tion suffer from "severe" PMS. Based upon her observations and studies, Dalton estimated that the incidence may be as high as 40 percent. In a 1954 survey of 825 women in north London, 27 percent indicated having recurrent premenstrual symptoms severe enough to demand medical attention or cause loss of work.[14]

The premenstrual syndrome is usually not a problem in women under 20 years of age but becomes more troublesome after the age of thirty. In fact, many women who have previously been unaffected by PMS begin to experience the symptoms for the first time in their thirties, leading some to suggest the term "mid-thirties syndrome." In a study of one hundred consecutive patients referred to the PMS clinic at University College Hospital in London by their general practitioners because of severe PMS symptoms, forty-four were between the ages of 30 and 40, thirty-four were between 41 and 50, nineteen between 21 and 30, three over fifty and none under twenty.[15]

Interestingly, the familial incidence is high, not only for the existence of PMS but also for individual symptoms. Thus premenstrual syndrome appears to be genetically transmitted: If the mother suffers from the condition, the chances of the daughter also having it are greatly enhanced. Furthermore, several members of two or three generations of a family may all experience the same combination of symptoms within the syndrome, for example, epilepsy, depression and migraine.[16]

What is the relevance of all of this to the criminal justice system? There appears to be a *direct correlation* between the premenstrual syndrome and the incidence of criminal behavior. As early as 1894, Italian scientists were observing the general effects of menstruation on criminal conduct.[17] They studied eighty women who had been arrested for "resistance to public officials," and found that fully seventy-one were menstruating at the time of the offense. Similar results were obtained in a study of women arrested for shoplifting in Paris in the 1890s.[18]

These early findings, primitive though they may be, are consistent with modern studies. One such study, for example, indicates a similarity in the delinquent behavior of boys and girls before they reach puberty. It is only after reaching puberty—after the appearance of the effects of testosterone in the male and

progesterone in the female—that this behavior becomes distinct according to sex, with young women tending to commit certain types of crimes and young men other types.[19] This difference in type of crime being committed can certainly be easily explained in terms of social roles that men and women are expected to conform to. What cannot be so easily explained, however, is the marked difference in the *ages* of men and women where criminal conduct is most prevalent. The evidence clearly suggests that older women constitute a much larger percentage of female criminal offenders than would be expected.[20] In one study involving criminal convictions in England, for example, women who were over the age of thirty were responsible for nearly *half* of all crimes committed by women; men over the age of thirty, on the other hand, made up less than one-fourth of the total number of male criminals.[21]

The statistics were found roughly applicable as well to cases where criminal offenders were committed to state mental hospitals. In a 1981 article in the *Bulletin of the American Academy of Psychiatry and the Law*, women ordered by the criminal courts of Missouri to undergo psychiatric evaluation during the years 1974 and 1975 were carefully studied. It was found that older female offenders formed an unusually higher proportion of referrals. "It is of interest to note," the authors commented, "that one out of four women committed was above the age of 40 compared to one out of every eight men." After concluding their research, the authors observed that "although it is generally assumed that criminal propensity declines over advancing years, female criminality may show significant increase during the menopausal period. . . . It appears that medical conditions play an important role in female criminality."[22]

These statistics, which indicate a much higher incidence of criminal conduct among women in their thirties and forties than among men, assume particular importance in view of one of the medical findings concerning premenstrual syndrome: PMS appears to be most common among women after reaching the age of thirty. Is there a correlation between PMS, then, and the high incidence of crime among women past thirty? More specifically, is this genetically transmitted syndrome a cause of criminal behavior?

A series of studies conducted by Dalton and reported in the *British Medical Journal* is of considerable interest.[23] In that study, boarding-school girls in England were medically observed during a period in which their disciplinary problems were carefully recorded. Over a twenty-eight-day period, 272 offenses were committed by girls that required punishment. It was discovered that fully 29 percent of these offenses were committed during the first four days of menstruation—more than twice the statistically expected incidence of 14 percent had there been an even distribution of offenses committed during the twenty-eight days.

An interesting collateral phenomenon was observed during the study. In the boarding school, female students aged sixteen to eighteen are permitted to punish the younger girls for misbehavior. The data gathered indicated that these older students gave significantly more punishments during their *own* menstruation; their standards of discipline tended to rise at each menstruation and then gradually fall during the cycle. The same phenomenon was observed to be true of the teachers.

In another and more disturbing article for the *British Medical Journal*, Dalton conducted a study in an English woman's prison that attempted to correlate the timing of an inmate's criminal offense with her menstruation cycle.[24] It was found that among these new inmates who had committed their offense during the previous twenty-eight days, 49 percent of 156 newly admitted prisoners had been sentenced for crimes committed during the eight-day paramenstruum—or about twice as many as would statistically be expected. The premenstrual syndrome was present in an incapacitating severity in 27 percent of these 156 prisoners, and it was discovered that 67 percent of them had committed their crimes during the paramenstruum. Clearly, PMS was a significant—perhaps determinative—factor in the reasons why these women had committed criminal acts.

The study went on to comment that during their stay in prison those who became disorderly were reported daily to the prison warden. Again, a high correlation between menstruation and antisocial behavior was noted. Among those whose misbehavior caused them to be reported more than once, fully 70 percent of the offenses had been committed during the paramenstruum.

In a subsequent intensive study and treatment of three iso-

lated female convicts, Dalton carefully kept menstrual charts.[25] All three women had been imprisoned for repeated criminal acts ranging from theft to murder. Menstrual records clearly showed that the three women broke the law *only* during their premenstrual days.

As with criminal conduct, so psychiatric disabilities have been documented as being accelerated by PMS. Dalton examined acute admissions for psychiatric illnesses in a hospital.[26] She found that 46 percent of admissions of female patients occurred during the paramenstruum; in fact, those suffering from PMS constituted 53 percent of the attempted suicides, 47 percent of those admitted for acute depression and 47 percent of schizophrenic admittees. These findings have since been corroborated by four physicians in an article for the *American Journal of Obstetric Gynecology*.[27]

A suicidal urge resulting from PMS has also been documented. In one survey of attempted suicides, scientists discovered that 21.0 of 95.0 attempts were made during the menstrual week and 35.0 attempts during the premenstrual week. Thus 56.0 suicide attempts were made during the menstrual and premenstrual fourteen days, compared with a statistically expected 47.5 attempts had there been an even distribution. When the menstrual cycle is divided into phases, it is seen that 39.0 attempts were made during the critical paramenstruum, compared with 27.1 attempts that could be expected on an even distribution.

This correlation was confirmed in yet another study reported in the *British Medical Journal*. Using a completely different approach, scientists carefully reviewed 102 consecutive autopsies performed upon women and examined the condition of the uterine tissue to determine whether the individual had been in the menstrual or premenstrual phase at the time of death.[28] Of the 102 bodies examined, 38 had been suicides, and 68 percent of these suicides, it was found, had taken place during the critical paramenstruum. In other words, a random sampling indicated that there had been more than twice as many suicides during this critical part of the menstrual cycle as would be expected by pure chance.

The premenstrual syndrome can be observed in noncriminal settings as well, with the criminal implications nevertheless ap-

parent. Thus in the wards of four London teaching hospitals it has been observed that 52 percent of admissions of females for accidents occurred during the paramenstruum.[29] This figure has since been confirmed by the United States Center for Safety Education, which pinpointed the forty-eight hours immediately before the onset of menstruation as the time when accidents are most likely to occur.[30] In a study conducted by Texas Industries, it was noted that among women employed for the asembly of electrical components, a female worker's normal production rate of one hundred components an hour was reduced to around seventy-five during the paramenstruum. In England the Industrial Tribunal ruled that it is unfair to dismiss a female employee who has a premenstrual tantrum.

Although the syndrome and its causes are still not fully understood, some success has been achieved in its treatment. Dalton has been able to relieve the symptoms of PMS in many cases through the administration of the hormone progesterone. The hormone is naturally secreted in the corpus luteum and passes from the ovary to the endometrium in increasing amounts from the time of ovulation. It reaches a peak about five to seven days before menstruation and then the level falls off until the onset of menstruation. Thus the time of premenstrual symptoms coincides with the presence of progesterone in the blood.

Georgeanna Seegan Jones of the Eastern Virginal Medical School, however, administered progesterone and claimed it did not improve PMS symptoms.[31] Richard Shader, chairman of the Department of Psychiatry at Tufts School of Medicine (mentioned earlier), claimed that treatment with lithium carbonate produced dramatic relief of symptoms.[32] Samuel Gershon, chairman of psychiatry at Wayne State University School of Medicine, thought that lithium carbonate might be helpful only for treating those suffering from severe psychological symptoms from the syndrome.[33] Most recently, physicians in the Department of Obstetrics and Gynecology at King's College Hospital Medical School in London have produced evidence that suggests that Dalton's progesterone treatment is, after all, feasible.[34]

Whatever the possibilities for treatment, premenstrual syndrome now appears to be a medical reality. It is a condition that

exists in a relatively large percentage of the female population and one that is apparently the result of hereditary factors. Most importantly, the syndrome is increasingly being isolated as a primary factor in the incidence of criminal behavior among women. As with the "XYY syndrome," then, the sufferer from PMS who commits antisocial acts may simply be acting out a genetically determined role.

NOTES

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10

The Chemical Man

If criminal behavior in women is influenced by inherited hormonal factors, is there a corresponding biochemical activity in men? Is there some hormone present in the male of the species that has a strong, perhaps even deterministic, influence on the existence of antisocial conduct?

There is. The primary sex hormone in the woman is, as we saw in chapter 9, progesterone. The corresponding sex hormone in the male is *testosterone*. Both hormones belong to a class of chemicals known as steroids, and both appear to be instrumental in the origins of aggressive and antisocial behavior within the individual. Like progesterone, testosterone is produced by the sex glands. Using radioactive "tagging," scientists have been able to trace the flow of the steroid from the gonads into the blood and on into the brain.[1] There, testosterone concentrates in specific areas of the brain, acting to lower the threshold of nerve fibers in the route taken by "messages" going to the hypothalamus. By lowering the firing threshold, less stimulus is required for conduct to be initiated by the male. Thus testosterone acts to excite the brain to direct the body to respond in a way consistent with the function of the areas in which testosterone is present. As expected, these areas of the brain are critical in the creation of sexual behavior toward the female. Interestingly, however, they have been discovered also to be critical to the formation of violent conduct. Testosterone, then, has an excitatory influence on both sexual and aggressive behavior in the human male.

Research in this area began, as it usually does, with experiments on animals. The pioneering work was done in 1966 with the injection of testosterone into newborn female rats.[2] Scientists quickly discovered that the normal female sexual behavior of the rats disappeared, replaced with a tendency toward male patterns of aggressive behavior. When newborn male rats, on the other hand, were castrated, that is, when the production of testosterone was eliminated, they exhibited femalelike patterns of behavior.

Experiments were then conducted with monkeys in an attempt to deal with animals that were more humanlike in their physiology and behavior patterns. In a 1971 study by two psychiatrists at the Walter Reed Army Institute of Research, thirty-four adult male rhesus monkeys were placed on a one-third-acre outdoor compound and observed over a period of nine months.[3] During this period the behavior of the monkeys was regularly recorded using a standard psychological inventory describing seventy-three activities. After nine months, the observations of approximately eight thousand behavioral responses by the monkeys were recorded and analyzed for levels of testosterone.

The results were clear: A consistent relationship emerged between testosterone and aggression. The researchers observed a consistent correlation between such aggressive behavior as threat and chase and testosterone concentration. In fact, the five monkeys who were eventually judged to be the most aggressive in the group all had considerably more testosterone in the blood than the average for the entire group; the single most aggressive monkey consistently had the second highest levels of the hormone and was found to be regularly attacking other monkeys.

Interestingly, however, the most aggressive monkeys were not necessarily the most dominant, nor were the least aggressive the least dominant. The studies showed that submissive, that is, nondominant, monkeys did not necessarily have low levels in their blood; nonaggressive monkeys, on the other hand, did reflect lower levels of the hormone.

Another interesting fact that was observed was the correlation between testosterone and levels of tension in the monkeys. Again, the psychiatrists observed higher states of tension in high-testos-

terone monkeys, reflected in conduct such as teeth grinding and shaking or banging objects.

Very well for monkeys, but what about human beings? The pioneer work in the area of the effects of testosterone on human behavior was done in 1971 by a team of scientists at the Albert Einstein Medical Center in Philadelphia.[4] The scientists obtained two groups of men to study: The first group consisted of eighteen young men from local colleges; the second was comprised of fifteen older men, averaging forty-five years in age. Each subject was given a battery of psychological tests, including the IPAT Anxiety Scale, the Manifest Anxiety Scale and Depression Scale from the Minnesota Multiphasic Personality Inventory (MMPI), and the Boss-Burkee Hostility Inventory. The tests were administered to each subject during one-hour periods in which blood was withdrawn from the subject and analyzed for testosterone; thus the testosterone levels, which can fluctuate, were recorded at the time of psychological testing.

The results were impressive. The researchers discovered a "highly significant relationship obtained between aggressive feelings and testosterone production rate" in younger men. No effort was made to observe actual hostile behavior of the subjects, but only to determine their attitudes, that is, their feelings of aggressiveness, anxiety and hostility. Yet the findings clearly indicated that the presence of elevated levels of testosterone had a direct effect on the inclination of younger men to harbor potentially antisocial feelings.

Reviewing their gathered data, the scientists offered a theory consistent with their observations: "The capacity to experience *aggressive* feelings is associated with an ability of the male gonad to produce testosterone, while the manifest expression of *hostile* feeling is associated with the circulating hormone level bathing the peripheral tissues."[5] Thus the amount of testosterone produced appeared to relate to an individual's aggressive attitudes, and the amount actually in the brain and central nervous system affected his feelings of hostility.

The findings were different, however, with the group of older men. These subjects exhibited levels of testosterone roughly half that of the younger men. Yet aggression and hostility test scores

differed only slightly between the younger and older men. There was little correlation between aggression indicators and levels of testosterone in the older men. Why the difference? Again, the scientists offered an explanation that reflected other findings in the study:

> With the onset of puberty, the peripheral tissues, including the central nervous system, are suddenly and with increasing intensity bathed by rapidly rising concentrations of hormones possessing potent physiologic properties. As aggressions engendered in puberty (or earlier) are mastered or subside, age exerts its retrogressive actions to their inexorable outcome: senescence (aging) of the target cells and reduction of testosterone secretion to minimal levels.[6]

Thus feelings of aggression and hostility appear to be directly affected by the male sex hormone in younger men. Among older men, however, the decreasing levels of testosterone and the aging of the brain and central nervous system cause the correlation to disappear: Aggression and hostility appear to exist in older men as the result of other—possibly environmental—factors.

The next significant research in this area took place in 1972.[7] Two psychiatrists attempted a study of young male prisoners at the Patuxent Institute at Jessup, Maryland. This penal institution was designed for the indefinite confinement of habitual criminals who were considered to be a clear danger to society. The psychiatrists focused the study on 89 of the total of 564 prisoners at the institution by eliminating all who were not Caucasian, between the ages of eighteen and thirty-five, and with an IQ over ninety (the latter to facilitate psychological testing). This list was further pared down to a final group of twenty-one prisoners. Of these twenty-one men, four had been convicted of murder as the basis for their commitment, eight for armed robbery, three for assault, four for burglary and larceny, one for rape, and one for destruction of property. None of these prisoners was diagnosed as psychotic.

The prisoners were then given psychological tests, and their criminal records were obtained from the FBI and reviewed for the frequency, type and age of commission of past criminal offenses.

Once again, the results proved interesting—and disturbing. "In the total study group of 21, the 10 men with histories of the more violent and aggressive offenses during adolescence had a significantly higher testosterone [level] compared to [that] for the 11 men without such a history," the researchers found. In fact, "*all* of the men with the highest levels of plasma testosterone had committed one or more of the more aggressive or violent crimes of armed robbery, escape from prison, assault, murder and attempted murder during adolescence." By contrast, however, "*none* of the men with the lowest levels of plasma testosterone had engaged in these more aggressive crimes during adolescence."[8]

The facts appeared very clear. As the researchers concluded, "those prisoners who committed more aggressive and violent crimes during adolescence have significantly higher levels of plasma testosterone than those prisoners who did not commit more aggressive crimes." The apparent reason for this, the psychiatrists continued, was that:

> the individuals with higher levels of plasma testosterone may have experienced earlier onset of testosterone rise and more rapid increase in levels, as well as having attained higher levels, and were therefore placed at increased risk to commit more violent and aggressive acts during adolescence.[9]

The evidence was building that the male hormone was a direct factor in the incidence of criminal behavior.

In 1974 yet another study added further evidence to this theory.[10] A team of psychiatrists from the Yale University School of Medicine and the University of Utah Medical School initiated research involving thirty-six prisoners at the Connecticut Correctional Institute at Somers, Connecticut. These thirty-six men, ranging in age from eighteen to forty-five, were selected according to their "fit" as to one of three groupings of characteristics and background. Twelve of the men were chosen for Group 1, consisting of men who were in prison for violent crimes such as aggravated assault or murder and who continued to exhibit violently aggressive behavior in prison. Another twelve were selected because they fulfilled the requirements for Group 2—

men who had been imprisoned for nonviolent crimes such as theft and narcotics, but who exhibited social dominance within the prison's social structure. Finally, the third twelve chosen for Group 3 consisted of men who had been convicted of nonviolent offenses, who were nonaggressive, and who were not socially dominant.

As with the previous studies, here each of the men was subjected to periodic blood sampling, and each was given a battery of psychological tests. The results were familiar. "The aggressive group had a significantly higher mean testosterone level than the nonaggressive group," the psychiatrists concluded. Similarly, "the socially dominant group had a significantly higher mean level of testosterone than the nonaggressive group." Furthermore, "the aggressive group had a significantly higher level of testosterone than the two other groups combined."[11]

Although these conclusions were based upon psychological tests, there was a direct correlation as well between testosterone and the length of a prisoner's sentence, reflecting more violent crimes. The average length of the sentence of the high-testosterone Group 1 was more than twice that of the other two groups combined; the actual time served was *three* times as long, reflecting increased antisocial behavior on the part of the high-testosterone Group 1 while in prison, resulting in extended sentences and/or fewer grants of parole.

In attempting to contrast the psychological character of the aggressive high-testosterone prisoners with those of the other two groups, the researchers said the former group had "less responsibility, were less socialized, less tolerant. They strove less for achievement through conformity or via independence. They were less flexible and reported themselves less feminine. On the other hand, they were autonomous and more aggressive."[12]

Confronted with the growing body of evidence that testosterone appeared to be a significant factor in violent behavior, the sociologists quickly pointed out that the studies had an environmental explanation. They argued that high testosterone levels result in a stronger physical build and a more masculine appearance, characteristics that would lend themselves to learning a "role" of physical and social domination. Addressing this argument, the Yale and Utah psychiatrists pointed out that:

there was no significant difference in the heights or weights of the subjects in the three groups, nor did outward appearance suggest obvious differences in masculinity. Rather, the quality of toughness is apprehended by more subtle clues stemming in large part from intensity and/or malignity of gaze and general posture.[13]

So far, each of the approaches to the problem of testosterone and violent behavior involved a cross-sectional study of a group of men selected from a general prison or nonprison population. A slightly different approach to the problem was attempted by three psychiatrists in 1976.[14] Rather than compare general populations, these men studied individuals who had been convicted of one of the most violent of crimes: rape. Unlike most other offenses, rape involves both sexual *and* violent behavior. Unlike other studies, this study would attempt to focus on the contributing causes of a specific type of aggressive criminal conduct.

The researchers selected fifty-two convicted rapists incarcerated at California's Atascadero State Hospital, where mentally disordered sex offenders are treated. All but two of these fifty-two prisoners were known to have committed multiple rapes. The men were classified into one of four groups, depending on the degree of violence used in the course of the rape: Group 1—use of verbal threats only (thirteen men); Group 2—possession of a weapon, but without its actual use (twelve men); Group 3—physically forcing the victim but without personal injury (twenty-two men); Group 4—violently inflicted physical injury on the victim (five men). Each of the men was classified according to information gathered from the police records, hospital records, and personal interviews with the prisoners. Each was also tested for testosterone levels, and each was given the usual array of psychological tests. For purposes of comparison, a control group of twelve prisoners convicted of child molesting was also tested.

Once again, the findings indicated a definite aggressive trait linked with elevated amounts of the male sex hormone. There proved little difference in levels of the hormone between Groups 1, 2 and 3, the control group of child molesters, and the average level in the nonpenal population. However, the prisoners in Group 4 were an entirely different matter. The group of the most

violent rapists had a significantly higher average level of testosterone than normal individuals, child molesters, and less violent rapists. Thus testosterone was not a critical factor in the causes of rape per se, but it *was* critical in the degree of *violence* committed during a rape. In fact, the highest testosterone level encountered occurred in the only rapist *murderer* in the study: The highest level of the hormone corresponded with the most violent of the rapes.

Why this correlation? "Higher levels of testosterone," the research team theorized, "might either facilitate a more intense state of hostility under certain conditions or might facilitate the transition from the affective state of hostility to the actual aggressive violent act."[15]

The next significant study of the effects of testosterone took place in 1980 in Sweden. There, a group of scientists from Norway, Sweden and the United States conducted intensive research into the behavior patterns, personality characteristics and testosterone levels of forty juvenile delinquents.[16] These young men, varying in age from fourteen to nineteen years, were incarcerated in a Swedish institution for repeat criminal offenders. A battery of six psychological tests was given to each juvenile, including this time the Olweus Multifaceted Aggression Inventory for Boys (OMAI), which measures physical aggression against peers and verbal aggression against adults; the Thurstone Temperament Scale; the Multicomponent Anxiety Inventory (MCI); and the Impulsivity Scale, assessing an individual's tendency to act impulsively and make quick decisions without deliberation and planning. In addition, each subject was personally interviewed by a staff member and rated as to aggressive attitudes on the Peterson-Quay Behavior Problem Checklist. Finally, a psychiatrist conducted at least two interviews with each juvenile, and his family and criminal background was reviewed; based on this interview and background research, each subject was subjectively rated by the psychiatrist as to his propensity for physical fighting, verbal quarrels, escapes from institutions, explosiveness, assertiveness, social dominance, degree of violence used in criminal behavior, and the existence of any psychiatric disabilities such as neurosis, psychopathic-antisocial behavior, or brain disfunctions.

The subjects were then put into one of three groups: Group 1—juveniles who had committed only property offenses, such as theft or burglary (nineteen subjects); Group 2—juveniles who had committed both property offenses and an occasional assault and battery (fourteen subjects); Group 3—juveniles known to have committed repeated assaults, at least one armed robbery, and who were considered the most violent inmates while in the institution (seven subjects). Finally, each subject was given a blood test and his testosterone level determined.

The data were collected, collated, and compared to information obtained from identical tests given to a sample group of fifty-eight "normal" high school juveniles.

The team of scientists confirmed what other researchers had already discovered. There was little correlation between an individual's testosterone level and his size or other physical characteristics. However, the "study showed a trend toward higher mean testosterone levels in the group of 40 institutionalized delinquent males when compared to the group of 58 high school boys." Furthermore, the researchers observed, "testosterone levels showed a positive correlation with the scales of verbal aggression and aggressive attitude and impulses." Thus, the scientists concluded, "the testosterone levels correlated with those personality traits that have been described as typical for anti-social personality and for one type of psychopathic individual."[17]

The team then took a closer look at the subjects in Group 3—that is, the most violent of the young criminals. Again confirming previous findings, they determined that the hormone's level in the violent juveniles was considerably higher than that found in the other groups of inmates.

There was a clear link between the male sex hormone and violent criminal behavior. But the team of Scandinavian and American scientists wanted to take a closer look at the fifty-eight normal Swedish high school boys: Were their attitudes toward violence and aggressive behavior also shaped by testosterone levels? In a separately published article on this group, the men found that they were.[18]

The fifty-eight boys ranged in age from fifteen to seventeen and had been randomly selected from the total male population of three hundred at a Swedish high school. As with the criminal

offenders, each had been given extensive tests, each had been the subject of interviews, and each had had his testosterone level determined.

Again, the amount of the hormone in the system had no correlation with the individual's physical size, nor did it relate to his body type (ectomorph, endomorph or mesomorph—or, simplistically, thin, muscular or fleshy). But, again, there was a definite correlation between the testosterone level of a youth and his attitudes toward both verbal and physical aggression. Interestingly, however, the scientists discovered that this correlation was most noticeable when the individual was responding "to provocation, including threat or unfair treatment." A greater hormone level was apparently a critical factor in whether a subject acted aggressively or even violently when provoked or treated unfairly.

The case for testosterone and antisocial behavior appears strong. The studies have consistently pointed to a direct relationship between the male steroid and aggressive—even violent—conduct in the male. But is there a way to isolate the effect of the hormone more completely? Is there, in other words, a way to duplicate the experiments where testosterone was injected into female rats? If so, it would certainly appear to be fairly conclusive proof of the influence of the chemical on behavior in humans.

Of course, experimentation on human females with testosterone would be inhumane. But somewhat similar research *has* been conducted. In 1980 three scientists from the Salk Institute in San Diego and the Neurobehavioral Clinic at UCLA conducted a study of female psychiatric outpatients.[19] Although testosterone is a male hormone, it is found to exist in relatively insignificant amounts in females. If there is a correlation between the male hormone and aggressiveness, the scientists theorized, elevated levels of the hormone might appear in females who have exhibited antisocial behavior.

The theory proved valid. In tests on a comparison group of normal women, the researchers found the amounts of testosterone to be fairly consistently at a low level. The same tests conducted on a group of women who exhibited aggressive and even violent behavior, however, reflected significantly higher levels of the male hormone. The scientists concluded that a dysfunction

of the female endocrine glands caused the elevated levels of testosterone. But whatever the cause, the results were unarguably evident: Testosterone and antisocial behavior were again found to be related—this time independent of any social factors that could arguably have resulted in learned male role playing.

The accumulated evidence, then, on the effects of testosterone on antisocial conduct appears to be clear. Once again, a biochemical cause for criminal behavior has been identified. Since testosterone production has been conclusively shown to be an inherited trait, the conclusion must again be drawn: Genetics, not environment, is the primary culprit in the attempt to explain the causes of criminal behavior.

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11

The Born Alcoholic

Alcoholism is one of the major causes of criminal behavior today. Obviously, it is central to problems such as public drunkenness and driving under the influence of alcohol. Less obviously, however, the problem is directly reflected in the statistics of more serious, seemingly unrelated, criminal offenses.

After an extensive study on the effects of drinking within the criminal justice system, the President's Commission on Law Enforcement and Administration of Justice concluded:

> The basic question is, is alcohol related to crime? The basic answer is "yes." . . .
>
> In 1961, 55 percent of all arrests in the United States reported to and by the FBI were for alcohol-related offenses—drunkenness, liquor law violation and drunk driving—or for offenses which often involve drinking—disorderly conduct and vagrancy. In 1963 the proportion out of total offenses was the same.
>
> In addition to the 55 percent of arrests that are for alcohol use offenses *per se*, a considerable number of other offenses are committed by persons who have been drinking just prior to the commission of the offense. Some crimes show a high frequency of alcohol involvement. Homicide for example is an alcohol-related crime.[1]

A number of studies have confirmed the correlation between alcohol and murder. Thus in one study in Ohio, 43 percent of all persons convicted of homicide in a given period had been drinking at the time of the killing.[2] In a study of 588 murders

committed in Philadelphia, it was found that alcohol was present in *both* the victim and the offender in 44 percent of the cases.[3] "On the basis of the present data," the President's Commission observed after reviewing many such studies, "one can say that there is a strong link between alcohol and homicide and that the presumption is that alcohol plays a causal role as one of the necessary and precipitating elements for violence." The commission went on to note that "such a role is in keeping with the most probable effects of alcohol as a depressant of inhibition control centers in the brain—leading to release of impulses."[4]

Murder is not the only crime affected by heavy drinking, however. In a survey of eighty-six individuals convicted of sex-related offenses in England, nearly half were intoxicated at the time of the offense.[5] In another study of one hundred randomly selected cases of sex offenses, 35 percent were found to be drinking at the time of commission and 8 percent were diagnosed as chronic alcoholics; many of the offenders indicated that they *needed* alcohol to commit the rape or other sex crime.[6]

Whatever the crime committed under the influence of alcohol, it is more likely to be one of violence. In a group of 882 men arrested in Columbus, Ohio, during or shortly after the commission of felony, blood and urine samples had been quickly taken. It was discovered that alcohol was present more often in crimes of violence (92 percent of all knife slashings, for example) and less often in property crimes such as burglary or forgery.[7]

Perhaps the most exhaustive examination of the connection between heavy drinking and criminal behavior was conducted in the various prisons of California. During a certain period, questionnaires were obtained from 2,325 newly incarcerated men.[8] These questionnaires reflected some very disturbing statistics: Of these convicted felons, 29.0 percent indicated that alcohol was a major problem in their lives, with 6.4 percent having received medical treatment for the problem. Of the problem drinkers, 23.0 percent had a record of criminal convictions, compared with a 14.0 percent figure among nonproblem drinkers. Fully 28.0 percent of those surveyed in the study admitted that they had been drunk at the time they committed the offense for which they were imprisoned.

These figures have been borne out by other studies as well. Thus, for example, the chief psychiatrist at Sing Sing Prison in New York reviewed all admissions at that institution over a two-year period and concluded that alcoholism was either directly responsible or closely related to the commission of the crime for which the subject was imprisoned in 25 percent of the cases.[9] In the study of the arrest records of 286 male patients admitted to a psychiatric unit in Wyoming during a ten-year period, it was found that 25 percent of the men diagnosed as alcoholics had been arrested for violent crimes.[10] A survey of 242 males in the Mississippi State Penitentiary revealed that 82 percent were "regular drinkers" and that 60 percent had been drinking at the time of committing the crime for which they had been incarcerated; the researchers concluded further that there was strong evidence linking drinking habits and the likelihood of repeated offenses.[11] In yet another study of 307 male prisoners convicted of serious crimes involving assault (80 percent of which involved murder), 36 percent of the subjects were problem drinkers, roughly a third of whom had received treatment for alcoholism; of the problem drinkers, fully half had criminal records reflecting previous serious assaults, which, in the opinion of the researchers conducting the study, would in most cases not have occurred in the absence of drinking.

After conducting his own exhaustive research in the area, one widely recognized expert (a psychiatrist) estimated that:

> The average city policeman spends one-half of his time dealing with alcohol-related offenses. Nearly half of the men and women in prisons are alcoholic or, at any rate, heavy drinkers. Most murderers are drinking at the time they commit a murder, and so are most of the victims. Between 20 and 30 percent of male psychiatric admissions are alcoholic or have alcohol-related problems . . . about one out of four suicides in the United States is an alcoholic.[12]

The findings, then, are clear: One of the primary causes of violent crime is alcohol abuse. But why? Consumption of alcohol is supposed to "relax" an individual, give him a feeling of euphoria and generally result in a greater degree of sociability. How is this consistent with the apparently violent reactions that

are so often encountered among the criminal population? Why is alcoholism such a dominant factor in serious crimes? Much of the answer lies in the fact that the popular conception of alcohol as a sociable tension reducer is simply false, at least when found in large amounts and among certain individuals. Scientists have relatively recently discovered that alcohol does *not* alleviate anxiety and depression; rather, it appears to *aggravate* these conditions, and the severity of the depression and anxiety tends to increase with the volume of alcohol consumed, at least as to alcoholics.[13] An odd effect of heavy drinking, however, perhaps explains the misconceptions: During sober periods, alcoholics usually report that they experienced positive effects during intoxication, despite the fact that they exhibited distinctly negative feelings during the same drinking "binge" they are so fondly recalling.

This emotional despondency—and the deceptive memory that follows—is not limited to alcoholics. Individuals who are merely "social drinkers" have been shown to react to ingestion of alcohol with fits of depression and anxiety as well. Again, the severity of this emotional state appears to be linked to the amount of alcohol consumed.[14]

Depression and anxiety can, of course, have a direct impact on the antisocial nature of the behavior of an individual. What is particularly interesting, however, is the effect of alcohol in directly creating aggressive behavior. Again, the popular conception is that alcohol soothes aggressive tendencies and results in more sociable conduct. Again, scientists are now finding that the opposite is true: At least in large amounts, or among alcoholics, the consumption of alcohol has been shown to cause aggressive behavior.[15] As one researcher at the Harvard Medical School asked:

> If alcohol is an effective antianxiety and antidepressant compound, why are alcohol abusers at such high risk of committing suicide when they are heavily intoxicated? If alcohol is such a good social lubricant and acts to enhance conviviality, why do so many intoxicated alcohol abusers become recklessly aggressive and hostile on the highway?[16]

Alcohol, then, appears to have a direct effect on an individual's state of depression, anxiety and on aggressive attitudes—

and, thereby, an indirect effect in the creation of criminal behavior. These effects appear most pronounced in those individuals who exhibit a chronic dependence on alcohol, that is, alcoholics.

Given these widely documented facts, the criminal justice system must take a very close look at the alcoholic in our society. If alcohol is involved in a majority of crimes committed today, and if a significant percentage of violent crimes are being committed by individuals who exhibit a chronic need for alcohol, the question must be asked: what *causes* alcoholism? What is the process by which alcoholics are created? As with other sources of criminal behavior, if the causes here are understood, the discovery of cures becomes more possible.

The traditional—and current—view is that alcoholism is (once again) the result of environmental influences. Thus sociologists and psychologists point to the family and society in general as the causative culprits in the creation of an alcoholic. Children learn from their parents; holding them up as role models, they, too, adopt chronic alcohol abuse as a learned pattern of behavior. Confronted with the fact that many children never touch alcohol because they are disgusted with the abuses of their parents, these social scientists quickly point out that such reactions are equally the result of family influences, that is, negatively influenced behavior.

Society is perhaps the greatest culprit in the eyes of the environmental determinists. Alcoholics, they say, drink to escape. They are lonely, frustrated, insecure people who must constantly obliterate the harshness of the real world with the fuzzy unreality of the inebriated state. They experience anxiety and depression in facing the daily complexities and uncertainties of life, and alcohol represents the answer. As one psychiatrist replied, however, "the fallacy in this reasoning is a simple one: most people are frustrated, unhappy, insecure, or lonely much of the time but do not become alcoholic. Thirty percent of adults do not drink at all."[17] The proper question would appear to be not whether anxiety and depression due to environmental influences are causes of alcoholism but why certain individuals become alcoholics and others do not.

Then there are those environmentalists in the psychoanalytic school. The causes of alcoholism to these scientists lay in the

deep-rooted psyche: The alcoholic is simply attempting to escape from his neurotic discomfort and return to the pleasures and security of infancy. Thus drinking is at once a Freudian oral gratification and an escape to the warmth of maternal contact and a full tummy.[18] One psychiatrist has even labeled drinking as "alimentary orgasm" in the alcoholic's blurring of mind and body as he or she regresses to infantile bliss.[19]

Yet others interpret alcoholism as a reaction to a premature weaning from the breast and the later realization that alcohol "supplies the oral love symbolically, in the form of precious liquor taken by mouth, the 'mother's milk' which was so much craved."[20]

Whatever the conflicting theories advanced by the various behavioral sciences for alcoholism, they are united on one point: It is not caused by physiological factors, and it is *certainly* not caused by any physiological factors that are inherited. To put it as succintly as possible, alcoholism is the result of psychological compulsion, not physiological craving.

Despite this prevalent attitude, there are increasing numbers of scientists who are beginning to look within the body itself for the answers to the problem posed by alcoholism. Having looked inside and discovered identifiable causes, many of these same scientists are asking the next logical question: Are the biological causes inherited?

The view that alcoholism may be an inherited trait, although currently unpopular, is not new. Aristotle wrote that drunken women "bring forth children like themselves," and Plutarch commented that "one drunkard begets another."[21] But it has only been in very recent years that scientists have been able to isolate the physiological origins of alcoholism and offer explanations for how the condition may, after all, be inherited.

This new genetic view of alcoholism is not one held by a small group of unknown pseudoscientists. Rather, it is one being adopted by increasingly large numbers of our most respected thinkers. Thus David D. Rutstein of the Harvard Medical School wrote that "in contrast to the generally accepted view that alcoholism may result from cultural, environmental and psychological influences, newer epidemiological evidence suggests that in addition to environmental factors there is probably a strong ge-

netic component in the disease."[22] Two other physicians at the Harvard Medical School observed that:

> During the past decade there has been substantial progress toward understanding the causation and natural history of alcoholism. . . . Alcohol problems are now being examined by many disciplines, including medical genetics, endocrinology, biochemistry and clinical and behavioral pharmacology. . . .
>
> There is increasing evidence that genetic factors are associated with alcoholism. Moreover, genetic factors may also influence alcohol metabolism and enzymatic mechanisms, which in turn may determine the expression of tolerance and physical dependence on alcohol.[23]

What is the scientific body of evidence that is swaying ever greater numbers of scientists to embrace the concept that individuals may have genetic predispositions toward alcoholism?

The first type of evidence involves the comparison of alcohol sensitivity among members of different ethnic groups. If the human body is biologically uniform in the way it reacts to alcohol, the differences between alcoholics and nonalcoholics is more arguably due to environmental causes. If, however, there appear to be biochemical differences between ethnic groups in their biological reactions to alcohol, a strong bit of evidence will be available to the heredity camp.

This ethnic approach was first used in 1972 in a study of the comparative effects of alcohol on men and women in Japan, Taiwan, Korea and the United States.[24] Interested by the lower rate of alcoholism among Orientals, an American physician selected thirty-eight Japanese, twenty-four Taiwanese, twenty Koreans and thirty-four Americans as subjects (all between the ages of twenty-five and thirty-five). He fed each subject measured amounts of beer, with Americans (that is, Caucasians) receiving more than twice as much per pound of body weight as the Orientals. He then measured the body's reaction to the alcohol by recording the flushing of the earlobe with an optical densitometer, as well as increases in pulse pressure. If there were no genetic differences in reactions to alcohol, the physician could expect to find that flushing (an indication of vessel dilation) and pulse pressure—both under the control of the autonomic ner-

vous system—would be consistent among the various ethnic groups.

The results, however, clearly indicated a genetic factor in the reaction to alcohol. Fully 83 percent of the Oriental subjects responded to the measured amounts of alcohol with a marked flush, but only 6 percent of the Caucasians did, despite the latter having received larger doses. Similarly, increases in pulse pressures were observed in 74 percent of the Orientals, with only 3 percent (one adult) of the Caucasians demonstrating such a reaction.

To insure against any possible cultural effects on alcohol consumption, the physician next duplicated the experiment with Japanese, Taiwanese and American infants, giving them small amounts of port wine in a glucose solution. Again, the results showed that heredity rather than environment dictated the body's automatic reaction to alcohol: Of the oriental babies, 74 percent responded with flushing, but of the Caucasian babies, only 5 percent (one baby) so reacted. Clearly, the alcohol-induced changes in blood flow were not learned or conditioned responses.

The physician concluded that the often-observed fact that Orientals rarely became alcoholics may be attributable to the simple fact that their bodies are unable to accept even moderate amounts of alcohol without experiencing symptoms of intoxication. In any event, the first step was taken in the genetic explanation for alcoholism: The physiological reaction to alcohol itself appeared to be genetically determined.

These experiments were repeated by a team of scientists two years later, this time with twenty-four Chinese and twenty-four European subjects.[25] The results proved to be the same: Skin flushing, increased heart rate and decreased blood pressure in response to alcohol were much more noticeable in the Orientals. The scientists concluded that physiological rather than cultural factors determined the relatively low rate of alcoholism in Orientals.

In 1978 yet another ethnic comparison contrasted the relative abilities of forty-seven Japanese, thirty-nine Chinese and sixty-eight European subjects to metabolize alcohol in their bodies.[26] Again, the Japanese and Chinese were found to be similar in

their ability to convert and eliminate alcohol, but the Europeans exhibited a distinctly slower metabolism.

The ethnic studies clearly indicated that the body's mechanisms in dealing with alcohol were determined by hereditary factors. But could it be shown that alcoholism itself was the result of genetic predisposition? In other words, is alcoholism hereditary?

The initial evidence for the inheritance of alcoholism is the incidence of the condition within families. If alcoholics could fairly consistently be shown to have parents, siblings and children who also suffer from the problem, an argument could certainly be made that heredity is the common factor.

This familial incidence has been repeatedly demonstrated. As early as 1929 researchers in Germany conducted a survey of nearly 1,000 male alcoholics and 166 female alcoholics.[27] They discovered that alcoholism existed in *half* of the fathers of these individuals and in 30 percent of the brothers. Subsequent studies have reflected varying rates of incidence.[28] But in none was there less than a 25 percent figure for fathers and brothers, a figure that is itself five times that for alcoholism among the general male population.[29]

Of course, such familial incidence can easily be explained in terms of environmental determinism: Members of families are subjected to similar parental and social influences and can be expected to have similar reactions such as exhibited by alcoholism. Also, alcoholism can simply be explained in terms of role learning: The child imitates the alcoholism of the parent.

The focus of the study, then, must be on children who were raised by individuals other than their blood parents, that is, by adopted parents. If such children are subsequently studied in their adulthood, the results should be fairly conclusive of the heredity-environment argument about alcoholism. If children of alcoholic parents are raised by nonalcoholic parents and do not become alcoholics themselves, the arguments of the environmentalists would appear valid. By the same token, if such children still show a tendency toward alcoholism despite the absence of a family environment reinforcing the condition, genetic causes would appear to be primary.

In 1973 a team of psychiatrists from the Washington Univer-

sity School of Medicine undertook a study of all patients who had been admitted to the alcoholic unit of a state hospital during an eighteen-month period.[30] Only those patients who had half-brothers or half-sisters were selected for examination. Each of the resulting sixty-nine patients was interviewed, and the incidence of alcoholism was recorded as to his or her biological parents, adopted parents, full-siblings and half-siblings; many of these relatives or adopted parents were also interviewed. If the results indicated a link of alcoholism between these patients and their *adopted* families but not with their *biological* relatives, the researchers reasoned, the causes of the condition must be environmental.

The results, however, indicated the contrary. The psychiatrists concluded that alcoholism in the half-siblings was not related to being raised by an alcoholic parent; there was, however, a clear link with alcoholism in the biological parents. Furthermore, living with alcoholic parents did not noticeably increase the likelihood of alcoholism in the offspring. In other words, the biological children of alcoholics were much more likely also to become alcoholics—and this was not influenced at all by whether they were raised by alcoholic parents or by nonalcoholic adopted parents. Conversely, children of nonalcoholic parents were not likely to become alcoholics when they were raised by alcoholic adopted parents. In every situation, the team concluded, the predictability of alcoholism in full and half-siblings was much more reliable by considering alcoholism in the biological parent than any environmental factors.

A similar study was conducted the next year in Denmark.[31] There, a sample of 133 men who had been separated from their biological parents within a few weeks of birth and then adopted were gathered and interviewed by psychiatrists. Of the 133, 55 had a biological parent who was alcoholic; the remaining 78 represented a control group of men who had nonalcoholic parents. The researchers soon observed the familiar pattern: The rate of alcoholism in the group with an alcoholic biological parent was almost four times that in the control group. In other words, despite similarity in environmental influences of the 133 men, those who had a genetic link to an alcoholic were considerably more likely to become alcoholics themselves.

In 1978 a psychiatrist at the University of Umea in Sweden

initiated further research on the incidence of alcoholism among adopted individuals. He obtained official Swedish records concerning alcohol abuse among two thousand men who had been raised by adopted parents. Again, there appeared to be a significant correlation between alcoholism in the biological parent and alcoholism in the adopted son. "The results suggest that there is a genetic determinant for alcoholism," the psychiatrist concluded. "There is a genetic predisposition to misuse alcohol."[32]

Reviewing this area of research, two Harvard Medical School physicians concluded that "The finding that the risk of alcoholism is increased in men with a biologic alcoholic parent has now been confirmed in several studies. . . . Alcoholism in the biologic parent appears to be a more reliable predictor of alcoholism in the children than any other environmental factor examined."[33]

The evidence that alcoholism is inherited becomes even stronger, however. Additional studies have been conducted with identical twins. As explained in an earlier chapter, identical (monozygotic) twins are genetically "carbon copies," having come from the same fertilized ovum. Thus if alcoholism is, in fact, largely the result of inherited traits, one would expect to find the condition in one identical twin when it is present in the other more often than one would expect to find the correlation among fraternal twins.

The studies in this area have reflected exactly such a difference. In another Swedish study, identical twins exhibited a significantly higher degree of similarity in the presence of alcoholism than did fraternal twins.[34] Furthermore, the more severe the alcoholism, the greater the difference between rates in identical versus fraternal twins. In a Finnish study, identical twins were again found to have a much higher correlation of alcoholism than were fraternal twins.[35] Similar results were obtained in the United States.[36] Most recently, in 1981, two scientists from Princeton University and the National Academy of Sciences conducted a survey of 15,924 pairs of male twins. The scientists found that an identical twin was almost four times more likely to be an alcoholic if his brother was than was a fraternal twin with an alcoholic brother. This data, the men concluded, "lend some support to the hypothesis of genetic predisposition."[37]

The composite evidence, then, appears strongly to indicate that

alcoholism is, at the very least, strongly influenced by genetic factors: Alcoholism is, to a considerable degree, inherited. But what exactly is the mechanism that is inherited? Is alcoholism a pattern of behavior that is encoded in the individual's chromosomes? Or is alcoholism secondarily caused by some physiological condition that is the inherited trait? Recent evidence suggests that the latter is the correct explanation: Alcoholism derives from a biochemical condition in the body. It is this biochemical condition that is inherited.

In 1977 a psychologist at Purdue University conducted some interesting experiments with laboratory rats, injecting them with various chemicals and enzymes.[38] Normally, animals have an aversion to alcohol and will reject any liquid containing it. He discovered, however, that injections of a certain chemical caused the rats to drink alcohol. In fact, given a choice between alcohol and water, the rats consistently showed an uncharacteristic preference for the alcohol. Within three to six days of injections of the chemical into the brain, the rats were drinking increasingly large amounts and exhibiting clear signs of intoxication; when deprived of the alcohol, they would show indications of withdrawal usually present in alcoholics.

The substance injected was Tetrahydropapaveroline (THP), a condensation product of dopamine. As will be explained in chapter twelve, dopamine is a substance that is critical to the passage of messages within parts of the brain and that has recently been found to be instrumental in the causes of schizophrenia. Also of interest is the fact that THP is a biosynthetic substance from which the narcotic morphine is formed.

The psychologist conducted further experiments with rats and THP, theorizing that there was a "strangely powerful, but missing, link between the neurochemical mechanism underlying the addictive process and alcohol drinking."[39] He found that the amount of THP necessary to cause alcohol addiction in the rats was infinitesimal, so minute that its presence in an animal (or a human) would be very difficult to detect. He also found that once injected with the chemical, the rats continued for months to crave alcohol without further injections, a clear indication, he concluded, that the action of THP in the rat's brain is perhaps irreversible.

The scientist then turned to the critical question: Why do some humans drink alcohol without becoming addicted while others do become addicted? How is this predisposition to addiction (that is, alcoholism) inherited? In view of the alcoholism induced in rats by the simple injection into the brain of microscopic amounts of THP, he suggested a number of theories. One is that the chemical is present in some individuals as an inherited trait, thus causing alcoholism. Another is that the alcoholic may genetically possess neurochemical machinery that favors the synthesis of minute amounts of the chemical in the brain itself. Yet another theory is that certain individuals inherit lesions or "holes" in the brain tissue that normally keep THP from coming into contact with nerve receptors.

Whatever the causal link, the experiments provided one further step toward understanding the causes of alcoholism. If the behavioral patterns of alcohol addiction could be "created" with a single chemical, the key to the problem would appear to be biochemical, not environmental.

Another clue to the genetic origins of alcoholism was suggested by reports that alcoholics were found to produce unusually high levels of the chemical acetaldehyde, believed to be involved in the body's metabolization of alcohol. When alcoholics and nonalcoholics were given single doses of alcohol, the acetaldehyde levels in the alcoholics were considerably higher than in the nonalcoholics.[40]

Interested by this increased presence of acetaldehyde in alcoholics, two scientists from the University of Washington in 1979 attempted to determine whether presence of the amounts of the chemical itself might be inherited. They located twenty healthy young college students, none of whom had yet exhibited any signs of alcoholism; each, however, had at least one parent who was an alcoholic. These students were then given blood tests. The results were disturbing: The levels of acetaldehyde in the students were much higher than would normally be expected. Yet they had drunk no alcohol.

"Our results indicate," the scientists later wrote, "that young, healthy men with family histories of alcoholism who might be predisposed to alcoholism themselves also demonstrate increased acetaldehyde levels."[41] The researchers then theorized

that the presence of a higher level of acetaldehyde in the body might cause the production of enzymes in the body, resulting in the production of addicting morphine-like alkaloids. One of these addicting alkaloids, it should be remembered, is THP—the same chemical that caused alcoholism in rats.

The scientists duplicated the experiment in 1980 with a new group of nonalcoholic young men with family histories of alcohol.[42] The results were the same. Despite the lack of alcohol, these young men also exhibited high levels of acetaldehyde.

One further clue in the biochemical puzzle of alcoholism has surfaced. In the course of autopsies conducted by scientists from Japan and Germany in 1980, the livers of forty Japanese were examined.[43] The livers were found to contain a coenzyme, alcohol dehydrogenase (ADH), not normally present—at least, in non-Orientals. Usually, the acetaldehyde formed in the liver during the metabolization of alcohol is quickly oxidized. But the scientists believe that the presence of ADH in the liver may alter the metabolism, resulting in greater amounts of acetaldehyde and therefore greater sensitivity of the individual to alcohol. Thus the presence of ADH in the liver may explain why Orientals are so sensitive to alcohol and why they so rarely become alcoholics.

ADH, then, may represent one more bit of evidence that alcoholism is primarily caused by inherited characteristics, not by social influences. With this gradual unlocking of the origins of alcoholism is coming the increasing realization that much of criminal behavior is genetically influenced.

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12

The Programmed Assassin

On Sunday, March 29, 1981, a twenty-five-year old man returned to his hotel room in Washington, D.C., and sat down to write a short letter. "Dear Jodie," it began, "There is a definite possibility that I will be killed in my attempt to get Reagan." He wrote four more paragraphs, then concluded, "This letter is being written an hour before I leave for the Hilton Hotel. Jodie, I'm asking you to please at least give me the chance with this historical deed to gain your respect and love. I love you forever." He signed it "John Hinckley." He sealed the letter intended for the famous young actress, a woman whom he had never met, but failed to mail it.[1]

One hour later, the young man stood outside of the Hilton Hotel. Inside, the president of the United States was addressing an audience of labor union representatives. The young man was observed by a nearby Pinkerton employee. "He looked fidgety, agitated, a little strange," the employee later remarked.[2]

As the president stepped out of the hotel and approached his waiting limousine, the young man reached into his raincoat and pulled out a .22 caliber "Saturday-night special" and pointed it at him. He shot twice at the president, paused, then fired four more rounds—all in a period of two seconds. One of the "Devastator" bullets, designed to explode on impact, ricocheted off the limousine and struck the president in his side. One Secret Service agent threw himself against the president and knocked him into the car, while another stepped protectively into the line

of fire. The second agent was immediately struck in the stomach by one of the "Devastator" bullets. A third bullet hit a local police officer in the neck, and a fourth lodged in the brain of presidential Press Secretary Jim Brady.

John W. Hinckley, Jr., was subsequently tried by a District of Columbia jury for attempted assassination. That jury's verdict created a popular wave of indignation and cries of reform in the criminal justice system: Hinckley was found not guilty by reason of insanity.

While the trial was pending, Hinckley wrote a letter explaining why he tried to kill President Reagan and forwarded it to a *Time* correspondent. It read, in part:

> The most important thing in my life is Jodie Foster's love and admiration. If I can't have them, neither can anybody else. We are a historical couple, like Napoleon and Josephine, and a romantic couple like Romeo and Juliet. . . .
>
> I only hope Yale [University] doesn't destroy Jodie. Four years at that place is enough to ravage anyone. I tried to rescue her once, and it looks like I may have to do it again.[3]

Who was John W. Hinckley, Jr.? What terrible environmental influences acted to twist his mind into a labyrinth of dark delusions?

Hinckley was the son of a wealthy oil company executive and the product of an apparently happy and well-adjusted family enjoying all of the benefits of a luxurious home in a prestigious neighborhood. His brother was an engineering major at Vanderbilt University and his sister the head cheerleader, a homecoming queen candidate and a member of the National Honor Society at the local high school.

Hinckley himself seemed to fit into this mold during his early days. His elementary school basketball coach recalled that "he was a beautiful-looking little boy, a wonderful athlete, really a leader." Later, in junior high school, he was twice elected president of his homeroom and became manager of the basketball team. "No one rooted louder than Hinckley for the Highland Park Raiders," a friend recalled. A high school acquaintance later remembered Hinckley as being "so normal he appeared to fade into the woodwork."[4]

Yet sometime after his graduation from high school he began unaccountably to change. He spent seven interrupted years at Texas Tech, yet apparently made no friends. The few people who knew of him recall him as "an expressionless blank." Said one, "I only saw him with another human being one time."[5] Eventually, he dropped out and went to California, flirted with the Hollywood subculture and then returned to Texas Tech where he suddenly joined the American Nazi party; he was kicked out because, as a party member said, "When somebody comes to us and starts advocating shooting people, it's a natural reaction: the guy's either a nut or a federal agent."[6]

Looking back at the young man's life, a friend of the family observed, "Something happened to that boy in the last six to eight years to break him from the family tradition and the family lifestyle."[7] What *did* happen?

After the verdict of not guilty by reason of insanity, Hinckley was sent for psychiatric evaluation to St. Elizabeth's Hospital. There he was questioned and physically examined and tested; his personal history was studied; and the results were evaluated by a team of psychiatrists. He was then returned to court along with a psychiatric summary and recommendations to the judge who would decide what treatment, if any, Hinckley would receive.

The doctor's findings were clear. Hinckley was suffering from the most common of serious mental disorders: schizophrenia. What is schizophrenia? What causes a young man such as John Hinckley, reared in the best of environmental conditions, to attempt to assassinate the president of the United States?

There is some confusion among psychiatrists about what schizophrenia consists of. Some medical experts consider it a disease, others a syndrome of personality disorders; still others deny the existence of such a concept entirely. Most, however, recognize a relatively common condition to which the diagnostic phrase is attached—so common, in fact, that it is estimated that over 2 million individuals in the United States suffer from schizophrenia to some degree. That condition has been described in the "Diagnostic and Statistical Manual" of mental disorders commonly used by American psychiatrists in diagnosing patients:

This large category includes a group of disorders manifested by characteristic disturbances of thinking, mood and behavior. Disturbances in thinking are marked by alteration of concept formation which may lead to misinterpretation of reality and sometimes to delusions and hallucinations, which frequently appear psychologically self-protective. Corollary mood changes include ambivalent, constricted and inappropriate emotional responsiveness and loss of empathy with others. Behavior may be withdrawn, regressive and bizarre.[8]

The vague nature of this definition, however, resulted in an attempt to be more specific in the third edition of the manual. In the new definition, various symptoms are simply listed as being characteristic of the condition. For example, the existence of any of the following symptoms could be indicative of schizophrenia:

Delusions of being controlled: Experiences his thoughts, actions, or feelings as imposed upon him by some external force.

Somatic, grandiose, religious, nihilistic or other delusions without persecutory or jealous content.

Delusions of any type if accompanied by hallucinations of any type.

Thought withdrawal: Belief that thoughts have been removed from his head, resulting in a diminished number of thoughts remaining.

Auditory hallucinations in which either a voice keeps up a running commentary on the individual's behavior or thoughts as they occur, or two or more voices converse with each other.[9]

Merely giving examples of schizophrenic symptoms, however, has not resulted in any clear pictures of the condition. Yet most experts recognize that the condition exists, that it is a *psychotic* (out of touch with reality) condition, and that it is commonly present in most cultures.

The fact that schizophrenia exists in widely differing types of cultures provides, perhaps, the first indication that this mental illness—and possibly others—is not determined by environment as has been popularly thought for so long. The fact is that schizophrenia appears to have existed in every known culture throughout recorded history. Although the exact term *schizophrenia* was not applied until 1911, the symptoms making up the condition have been described in the literature and art of every known period. The ancient Greeks and Romans, in fact,

specifically recognized and described emotional illnesses such as dementia, paranoia, melancholia, manic-depression and hysteria.

But we do not have to rely upon secondary sources in determining the geographic and cultural extent of schizophrenia. We can simply study primitive cultures that exist today, looking for the symptoms of the condition. If primitive tribesmen suffer from a mental aberration similar to that suffered by modern city dwellers, the environmental premise for that aberration fails and we must look to other causes of the condition.

Such studies of primitive cultures exist. The work of Harvard anthropology professor Jane Murphy is perhaps most notable.[10] Murphy studied two very different but relatively primitive groups, the Eskimos of Northwest Alaska and the Yorubos of rural Nigeria, in an attempt to determine whether mental illness was a result of modern sociological factors as is commonly believed. Or do such primitive groups contain members who exhibit familiar symptoms of insanity? If so, do these groups recognize the condition, or do they have labels for psychological differences that resemble our definitions of mental illness?

Murphy discovered that a few members of the Eskimo village exhibited a complex pattern of behavior that was interpreted by the villagers to mean that "something inside the person—the soul, the spirit, the mind—is out of order." These affected individuals would engage in conduct such as talking to oneself, screaming at oneself, screaming at an imagined person, believing oneself to be an animal, hiding in strange places, refusing to talk, making strange faces, becoming violent, threatening others and exhibiting abnormal strength. The Eskimos have a word for this: *nuthkavihak*. They translate it as "being crazy."

The primitive Yorubos, too, had their share of tribal members who displayed behavioral disorders indicating mental illness. The behavior included hearing voices, talking constantly or refusing to talk, setting fires, hitting others with weapons and exhibiting abnormal strength. They also had a word for this condition: *were*. This was translated to Murphy as "insanity."

After completing her studies and reviewing other research conducted on the subject, Murphy concluded that "The expanding ethnographic literature on this topic indicates that . . .

phenomenal processes of disturbed thought and behavior similar to schizophrenia are found in most cultures." She then compared the prevalence of schizophrenic symptoms among the Eskimos and the Yorubos with that found in Western civilization. Comparing studies that had been done of Canadian and Swedish populations, she determined that the rate of schizophrenia in the four cultures was almost identical.

"The non-Western way of life does not offer protection against mental illness," she concluded.

> The rates of mental illness patterns are much more striking for similarity from culture to culture than for difference. . . . Rather than being simply violations of the social norms of particular groups, symptoms of mental illness are manifestations of a type of affliction shared by virtually all mankind.[11]

If schizophrenia is, after all, a universally found condition, there must be universally found causes. This does not preclude environment as a cause: The argument can still be made that all cultures have similar pressures operating on its members—pressures that create the schizophrenic condition. But, certainly, the universality of the condition strengthens the view that it is inherited rather than acquired.

Are there any other means of determining whether schizophrenia is determined predominantly by genetic or sociological factors? There are. In fact, there have been three types of studies that scientists have engaged in over the years to resolve this heated issue.

The first line of research has involved the consanguinity factor. In these studies, there is an attempt to determine the prevalence of schizophrenia among related individuals. In other words, is a schizophrenic patient likely to have more relatives who also suffer from the condition than would statistically be expected? If not, then certainly the environmental position would appear secure.

Robert Cancro, professor of psychiatry at the University of Connecticut School of Medicine, summarized the research in this field succinctly:

> The consanguinity studies consistently show that the prevalence of schizophrenia is significantly higher in the genetic relatives of schizo-

phrenic patients than it is in the general population. This has been found in every such study published, including those done by investigators with a markedly environmental bias. The relative stability of the prevalence rate in the general population both within and between many cultures argues, in itself, for a genetic factor. More important than the simple finding is the significant positive relationship between the frequency of the disorder and the degree of kinship. The closer the genetic relationship to the patient, the more likely it is that the relative will show the disorder.[12]

In fact, these consanguinity studies have consistently indicated a schizophrenic prevalence rate of 10–15 percent among the parents, siblings and children of those suffering from the condition, compared with a prevalence rate of less than 1 percent among the general population.

There are problems with this line of research, however. As those advocating the sociological-environmental causation of mental illness are quick to point out, the closer the degree of kinship, the more similar will be the environmental experience: The very fact of kinship means the individuals are likely to have similar family environments, similar values, and so on. Furthermore, closer kinship usually signifies closer and more intense relationships—again, resulting in greater similarity in influences among close relatives.

The second approach to the problem has been to study the presence of schizophrenia in sets of twins. As has been explained more fully in chapter 5, twins are of two types: monozygotic, or identical, and dizygotic, or fraternal. Unlike the fraternal twins, identical twins come from the splitting of a single egg that has been fertilized by a single sperm. The resulting two embryos are genetic mirror-images of each other. If, then, the prevalence rate of schizophrenia among identical twins could be compared with that among fraternal twins, the results should be conclusive. Since the environmental factors should be the same, that is, each identical or fraternal twin is subjected to the same environmental influences as his or her sibling, any significant variation in prevalence between identical and fraternal twins would have to be attributed to genetic factors.

There have been a number of such studies, starting as early as 1928. In that year, Hans Luxenburger of Germany located all fraternal and identical twins where at least one of the twins

had been treated at either the Basle Clinic or the Kaiser Wilhelm Institute for Psychiatric Research in Munich.[13] From among 16,382 patients, a total of 211 individuals were identified as twins. The results of this early study indicate that a fraternal twin had a 2.1 percent chance of suffering from schizophrenia if his twin also had the condition; an identical twin's chances, however, were 55.0 percent.

The next significant study was conducted by the American scientist Franz J. Kallman in 1946.[14] Kallman received regular reports from twenty New York mental hospitals over a period of nine years as to admittees who were twins. During that period he was able to identify 691 sets of twins where at least one of the set was diagnosed as schizophrenic. Kallman's resulting data indicated that the schizophrenic expectancy rate for a fraternal twin was 12.2 percent and for an identical twin, 73.0 percent.

In 1953 the Englishman Eliot Slater mailed questionnaires to 16,632 individuals who had been patients at one of ten psychiatric hospitals in London.[15] From the replies, as well as from other sources, he was able to identify 147 sets of twins where schizophrenia had been diagnosed with at least one of the twins. When the information had been completed, Slater's findings were almost identical to those of Kallman: a 12.3 percent concordance rate for fraternal twins, with a 70.0 percent rate for identical twins.

Clearly, the scientific studies were consistently reflecting a much higher incidence of schizophrenia in an identical twin where his or her counterpart suffered from the condition than in a fraternal twin in a similar situation. Just as clearly, the obvious conclusions were difficult to avoid: Heredity was much more important than environment in determining the presence of schizophrenic disorders.

But the studies continued. Perhaps unwilling to accept the fact that a mental illness could be genetically caused, other scientists and medical experts conducted their own research using the twins method, always with the same result. Thus in 1961 the Japanese psychiatrist Eiji Inouye of the University of Tokyo read a paper to the World Congress of Psychiatry in Montreal in which he described his study of 55 identical and 72 fraternal twins taken from Japanese mental hospitals and psychiatric clinics.[16] His conclusions: a 9.0 percent expectation among fraternal twins, a

48.0 percent rate among identical twins. In 1966 the British scientists Irving Gottesman and James Shields conducted yet further research in the area based upon a review of all patients treated at two English hospitals during a period of sixteen years.[17] From the resulting 392 twins, the men identified 47 who had been officially diagnosed as schizophrenics. Again, the results were familiar: a 9.1 percent concordance rate for fraternal twins, a 59.0 percent rate for identical. The two men concluded their research with the observation that it seemed the specific nature of most of the schizophrenias was largely caused by genetic factors.

Perhaps the most striking—and fascinating—single study conducted in the area was of the famous Genain quadruplets. Conducted by David Rosenthal, a research psychologist with the National Institute of Mental Health, the study involved a family of four identical quadruplets—*each* suffering from schizophrenia. The odds against such an occurrence have been computed at roughly 1.5 billion to one. As Gottesman and Shields later commented, "One would have thought that no further proof of a genetic basis for schizophrenia would be needed after finding a set of monozygotic quadruplets all affected with schizophrenia."[18]

Faced with this landslide of evidence, proponents of the environmental determinism school have pointed to the special relationship that exists between identical twins. Unlike fraternal twins, they say, identical twins look alike and so are treated more alike than are fraternal twins. With resulting greater similarity in environmental influences, a greater correlation in existence of schizophrenic disorders can be expected.

Although it is difficult to accept this explanation in view of the wide statistical difference in concordance between the two types of twins, it can be laid to rest in view of a study by Slater of sixteen cases where identical twins had been raised separately from each other.[19] In each of the sets of twins, at least one of the twins had been diagnosed as schizophrenic. Despite their separate upbringing, the correlation was very similar to that of previous studies involving identical twins raised together: 62.5 percent. Clearly, any arguable environmental influences had no apparent effect.

If further evidence is required, however, perhaps the most

compelling comes from a third line of research. In this type of study, biological parents and children who have been separated so the child is raised by an adoptive parent are examined. The most respected research of this type has been conducted by Seymour Kety, professor of psychiatry at Harvard Medical School, and his associates.[20] Kety reviewed the official records of all adopted individuals in Denmark between the ages of twenty-five and fifty; of the resulting 14,500 adoptees, 5,500 were selected for study on a geographical basis. From governmental medical-care records, 500 of them were found to have been admitted at some time to a psychiatric facility. The hospital records of each of these 500 adopted individuals was then read by the researchers. Those whom all of the researchers agreed were diagnosable as schizophrenic were then selected—33 in all. To provide a control group, that is, a group used for comparison, another 33 were selected from the remaining 5,000 adoptees; none of them had any history of mental illness, and they matched the 33 schizophrenics in age, sex, socioeconomic class and so on.

The biological and adoptive close relatives—parents and siblings—were then identified and the resulting 512 relatives investigated for any mental disorders, particularly schizophrenic illness. At the time of investigation, the researchers did not know which relatives were associated with which adoptees. When diagnosis of each of these relatives was completed, the data were collated and the relatives were designated as biological or adoptive; each was then identified with either schizophrenic or non-schizophrenic adoptees.

The biological relatives of the schizophrenic adoptees showed more than four times as great an incidence of past treatment for schizophrenia than did the biological relatives of the normal adoptees. In other words, the genetic relatives of identified schizophrenics were much more likely to have received treatment for the same disorder, even though there had been no similarity in parental influence or of environment. Furthermore, the relatives who had adopted the schizophrenics showed the same low incidence of the defect as did the adoptive and blood relatives of the normal group.

The 512 relatives were then individually interviewed by the researchers, and psychological tests administered, in an attempt

to detect mental illnesses for which they had not received formal treatment. Again, Kety and his team discovered a clear difference between the biological relatives of adopted schizophrenics and biological relatives of normal adoptees: Eight percent of the former were definitely diagnosed as also suffering from the condition, but only 1 percent of the biological relatives of normal adoptees exhibited schizophrenic symptoms.

Thus the arguments of the environmental determinists were met. Although it could always be tenuously argued that the adoptive environments might affect the incidence of schizophrenia in the adopted children, the already existing incidence of schizophrenia in their genetic relatives could not be affected by those environments. Furthermore, despite the wide disparity in incidence between the two groups of adopted children, the clear fact is that the genetic relatives of both adopted and nonadopted schizophrenic children had the same high incidence of the condition.

It is difficult to avoid concluding with Cancro that the consanguinity and twins studies obviously indicate a prevalent genetic factor in the causes of schizophrenia.

Yet the vast majority of psychologists and sociologists continue to refuse to believe that mental illness of any kind can be inherited: Schizophrenia, like any other mental disease, *must* be the result of familial and societal influences. As the psychologists and sociologists go, so go the lawyers, legislatures and penologists. The criminal justice system will continue to treat individuals such as John Hinckley as products of their environment.

But what if the causes of schizophrenia could be physically isolated and identified? Rather than use field studies with conclusions that can always be debated, what if we can actually point to a chemical or organ and prove that *that* is the source of the illness? There would certainly be little room left for the argument that environment is the dominant factor in the existence of schizophrenia.

It now appears that such an isolation of the organic cause of schizophrenia is taking place. For a number of years biochemists and neurologists have known that the ingestion of amphetamines by schizophrenics can dramatically worsen their existing symptoms. Yet this effect is not found in neurotics,

manic-depressives or individuals with other mental illnesses—or, for that matter, in mentally healthy individuals. In fact, the reaction is so specific that physicians not certain of a diagnosis will administer amphetamines and observe the reaction: If the patient's condition quickly worsens, he or she is deemed suffering from schizophrenia.[21]

However, when individuals without any schizophrenic symptoms are given very *large* amounts of amphetamines, they often begin exhibiting traits that are indistinguishable from schizophrenia. How, then, is it possible to accelerate schizophrenic symptoms with a drug? How is it possible to duplicate those symptoms in a normal person with large doses of the same drug?

To understand the phenomenon and the reasons for it, one must start with the basic physiology of the brain. The brain receives and transmits messages by electrical impulses through its brain cells. These transmissions of impulses are facilitated by the presence of "neurotransmitters," biogenic amines such as dopamine, tryptamine and acetylcholine. One of these neurotransmitters, dopamine, is found in relatively high concentrations in two critical portions of the brain—the *caudata nucleus* (the part that controls and coordinates voluntary movement, development of motor skills and complex movement of the muscles), and the *limbic region* (which is responsible for emotional thinking). The dopamine is secreted by nerve fibers in a nearby area, the *sustantia nigra*; it is manufactured by enzymes in each cell from the synthesis of two natural amino acids found in food, tyrosine and phenylalenine.

Amphetamines, it has fairly recently been discovered, act on the human system to stimulate the secretion of dopamine at these nerve endings. A similar effect can be attained by the administration of LSD and certain other drugs that suppress antidopamine substances. The ingestion of any of these chemicals will result in an excess of dopamine in the brain and, thus, the existence of a kind of "superconductivity" for transmission of messages. The end result is that the brain is receiving "messages" that simply are not there: hallucinations, delusions and so on—all indicative of schizophrenia. Certain other chemicals, on the other hand, have the ability to block dopamine receptors. Thus

phenothiazines and butyopherones have been used as antipsychotic drugs to inhibit schizophrenic symptoms.

The result of these studies of biochemical phenomena has been the theory that psychosis in general, and schizophrenia in particular, is nothing more than the effects of an increase in the level of dopamine in the brain—either as the result of greater secretion of the substance or of inhibition of antidopamine agents.[22]

The theory is given further credence by the fact that dopamine is structurally very similar to the hallucinogenic substance mescaline. Mescaline, of course, can temporarily produce many of the same delusions and hallucinations that are characteristic of the schizophrenic. Consequently, some scientists have theorized further that the normal metabolism of dopamine and other neurotransmitters may be faulty in schizophrenics, with the result that they are broken down into hallucinogenlike substances.

Most recently, Philip Seeman and Tyrone Lee of the University of Toronto have presented evidence for a new approach in this area. In a paper presented to the Society for Neuroscience in November 1981, the two men suggested a slight revision of this theory of dopamine production and inhibition.[23] In studies coordinated among scientists at Toronto, Cambridge University and the University of Vienna, twenty brains of deceased schizophrenics were physiologically compared with twenty-eight normal brains. Seeman and his colleagues mixed radioactively tagged antipsychotic drugs that compete with dopamine for access to the receptors in a test tube with pieces of a subject's brain membrane. The radioactive molecules of the drugs that bonded to the dopamine receptors were then detected and subsequently counted.

The expectation is that there would be approximately one thousand dopamine receptors on each nerve cell in the caudate and the limbic region. This is what was found in counting the receptors in the normal brains. But, surprisingly, the nerve cells of the schizophrenic brains contained about two thousand receptors, or twice as many as would be normal. Thus by devising a method of counting the infinitesimal receptors, Seeman and Lee have been able to establish that it is not simply the amount

of dopamine itself that determines the psychotic symptoms of schizophrenics but the increased number of dopamine receptors that schizophrenics appear to have in their brains.

The evidence appears to indicate two clear facts: Schizophrenia (and very possibly other mental illnesses) is physiologically caused, and it is primarily determined by genetic rather than environmental factors. It is, of course, not a coincidence that the results of these two independent fields of study are interrelated. If schizophrenia is, in fact, genetically transmitted, one would expect to be able to identify some physiological object—a chemical or a structure—that would be capable of being inherited along with the color of eyes or size of feet. That a physiological cause has been found for the mental condition is consistent with the evidence that the condition appears to be inherited.

The complex phenomenon of schizophrenia, then, long thought to be the result of a confusing interplay of the influences of family and society, may be caused by nothing more than an unusually large clump of chemical receptors.

NOTES

1. *Time*, April 13, 1981, p. 25.
2. *Id.*
3. *Time*, October 12, 1981, p. 25.
4. *Time*, *supra* note 1 at p. 25.
5. *Id.*
6. *Id.*
7. *Id.*
8. *Diagnostic and Statistical Manual of Mental Disorders*, 2nd ed. (Washington, D.C.: American Psychiatric Association, 1968).
9. *Diagnostic and Statistical Manufal of Mental Disorders*, 3rd ed. (Washington, D.C.: American Psychiatric Association, 1980).
10. J. M. Murphy, "Psychiatric labeling in cross-cultural perspective," 191 *Science* 1019 (March 1976).
11. *Id.*
12. R. Cancro, "Genetic considerations in the etiology and prevention of schizophrenia," in *Schizophrenia: Biological and Psychological Perspectives*, ed. G. Usdin (New York: Brunner/Mazel, 1975).

13. I. I. Gottesman and J. Shields, "Contributions of twin studies to perspectives in schizophrenia," in *Contributions to the Psychopathology of Schizophrenia* (New York: Academic Press, 1977).

14. F. J. Kallman, "The genetic theory of schizophrenia," 103 *The American Journal of Psychiatry* 309 (1946).

15. E. Slater, "Psychotic and neurotic illnesses in twins," in *Medical Research Council Special Report Series, No. 278* (London: Her Majesty's Stationery Office, 1953).

16. E. Inouye, "Similarity and dissimilarity of schizophrenia in twins," in *Proceedings, Third World Congress of Psychiatry*, vol. 1 (Toronto: University of Toronto Press, 1963).

17. Gottesman and Shields, *supra* note 13.

18. D. Rosenthal, *The Genain Quadruplets* (New York and London: Basic Books, 1963).

19. E. Slater, "A review of earlier evidence on genetic factors," in *The Transmission of Schizophrenia*, ed. D. Rosenthal and S. Kety (Oxford: Pergamon Press, 1980).

20. B. Davis and F. Flaherty, eds., *Human Diversity: Its Causes and Social Significance* (Cambridge: Ballinger, 1976).

21. I. Creese and L. Snyder, "Biochemical investigation," in *Schizophrenia: Science and Practice*, ed. J. C. Shershow (Cambridge, Mass., and London: Harvard University Press, 1978).

22. A. Clare, *Psychiatry in Dissent* (Philadelphia: Institute for the Study of Human Issues, 1979).

23. "Chemical clues to schizophrenia," 112 *Science News* 342 (November 1981). See also *Los Angeles Times*, November 12, 1981, at p. 1.

13

Rethinking Criminal Justice

The arguments between the geneticists and the sociologists will, of course, continue. But it does appear that, at the very least, there is strong and continually emerging evidence that genetic factors are influential in determining an individual's conduct throughout his or her life. As science is able to dig even deeper into the mysterious miniuniverse of DNA, further disturbing discoveries about genetically influenced behavior will probably emerge.

It appears, then, that the results of continuing genetic research are increasingly pointing to a disturbing conclusion: Much of our conduct is determined to varying degrees by genetic factors. Much to the delight of the philosophical "determinists," our lives are programmed to a considerable extent long before we are even brought into this world.

As unsettling as these scientific discoveries are, however, the issues posed to our system of criminal justice are even more ominous. For once the concept of genetic determination is accepted and applied to antisocial conduct, a number of questions present themselves to our system of criminal justice for resolution—questions that cannot be ignored. First, how should society deal with the offender whose crime was at least genetically influenced? Second, and more disturbingly, what steps can—or should—society take to protect itself from genetically identifiable *potential* offenders?

Assuming future acceptance by the legal system of the con-

cept that antisocial conduct in certain individuals is at least strongly influenced—if not actually determined—by genetic considerations, the question remains of how to deal with the phenomenon. This question involves two further issues: Is genetic "aberration" to be considered in mitigation of, or a defense to, a criminal charge? What type of punishment, if any, should be administered?

Addressing first the question of genetic aberration as a complete defense, the clearest application is to the defense of insanity. Society has decided that no individual should be held accountable for acts performed when he or she is mentally incapacitated. However, the application of the concept of genetic defect to that of legal insanity appears to depend upon the standard of insanity employed. The one applied in some form today by the majority of jurisdictions is the *McNaghten* rule:

> [T]o establish a defense on the ground of insanity, it must be clearly proved that, at the time of the committing of the act, the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong.[1]

The term *disease* has been widely interpreted to include congenital defects and can thus be applied to genetic abnormalities such as the XYY syndrome.[2] A second standard that has been recognized by a number of jurisdictions is that of the Model Penal Code, also known as the "substantial capacity"standard:

> A person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality (wrongfulness) of his conduct or to conform his conduct to the requirements of the law.[3]

The third most well-known, but today universally rejected, standard is the *Durham* or "product" rule: "The rule . . . is simply that an accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect."[4]

The *McNaghten* test clearly takes the position that insanity is a cognitive rather than a behavioral disorder. The concern of

the criminal justice system is not with the defendant's ability to *control* his or her conduct but the ability to appreciate its significance. Since having a genetic aberration such as the XYY syndrome does not seem to affect the ability to appreciate the "nature and quality" of criminal conduct, or to understand whether it is right or wrong, it appears that the possession of an extra Y chromosome would constitute no grounds for an insanity defense.

However, the defense could at least arguably be available under other legal definitions of insanity. Applying the Model Penal Code, a genetic deviant may satisfy the description of an accused who "lacks substantial capacity . . . to conform his or her conduct to the requirements of the law." This is similar to the *irresistible impulse test*, which is usually defined as an impulse that the individual is unable to resist due to mental disease or defect. Under either test, persons carrying a defective gene that can be shown to inhibit strongly their capacity to control antisocial acts would seem to have a complete defense.

Similarly, a genetic aberrant could be found "insane" by using the *Durham* rule. Applying that standard, scientific evidence may be produced that indicates that the genetically influenced defendant's criminal conduct was "the product of mental . . . defect."

Much time can be spent in argument over the exact interpretation of each of these standards and their relative merits, but for the purposes of this discussion, it is readily recognized that society's position can be viewed as a willingness to exculpate offenders for their antisocial conduct where, as a result of mental defect, they could not understand the nature of their acts or of their wrongfulness or, understanding them, nevertheless could not control themselves. In other words, we are unwilling to punish a person for conduct that was largely the result of a defect over which the person has little or no control.

The parallel is clear: The policy behind the insanity defense seems to apply with equal validity to genetic aberration. Mental aberration caused by congenital defects, it has already been noted, is universally recognized to be within the scope of the insanity defense. Is it a great step to include genetic aberration within that scope—assuming that such aberration resulted in the indi-

vidual's being unable to appreciate the wrongfulness of his or her acts or to conform his or her conduct to avoid that wrongfulness? In fact, is it *any* step at all? Do we even need to create another category of "genetic determination" to rest alongside insanity as a complete defense? The causes and effects of such genetic aberrations at least arguably fit well within the definitions given for insanity. Would not the following fit both the genetic aberrant's conduct and the insanity standard (here, using a variation on the Model Penal Code)?

> A person is not responsible for criminal conduct if as a result of genetic defect he lacks substantial capacity either to appreciate the criminality (wrongfulness) of his conduct or to conform his conduct to the requirements of the law.

Again, in assessing the suitability of an insanity-type defense to a genetically influenced crime, the reasons behind society's providing that defense must be analyzed and measured for fit. A policy decision has been made that no individual should suffer for conduct that is the result of a defect in reason or will. Assuming scientific evidence that structural aberrations in genetic structure can cause such a defect in one's ability to perceive or control oneself, would not the very same policy considerations apply?

An alternative method of dealing with criminal conduct determined or influenced by genetic factors is to consider such matters in mitigation of the offense charged or proven. Most jurisdictions today recognize some variation of the concept of "diminished capacity" or, as it is sometimes known, "partial insanity." In those jurisdictions the law provides that there be a mental disorder of such a nature as to lessen the degree of guilt without completely establishing innocence. The usual example is, of course, homicide: The existence of such a mental disorder can negate elements necessary to establish the offense of murder in the first or possibly second degrees. If, for example, the mentally disordered individual is deemed unable to formulate deliberation or premeditation, the offense of first-degree murder will be reduced to second degree. If such an individual is not capa-

ble of freely formulating malice, the charge may be reduced further.

There is no reason why this approach could not be applied to the genetic-aberration case. The reasoning behind the "substantial capacity" and "diminished-capacity" doctrines is simply an awareness that mental disease or defect is not an all-or-nothing proposition: There is no clear separation between those who are deemed not criminally responsible due to mental defect and those whose defects are not sufficient to avoid responsibility. In other words, there is a recognition that an individual may not be completely incapacitated by his or her mental problem, yet still suffer an impairment sufficient to warrant some limitation on accountability. For this individual, society may decide to adopt a doctrine of partial responsibility—in reality, a compromise, representing discomfort with the notion of complete exoneration, yet realization that mental impairment should somehow lessen the harshness of punishment.

For those, then, who are uncomfortable with the idea of, in effect, granting a license to commit crimes to those who have genetic aberrations, this approach has much to recommend it. Science may not be able to say for some time—if ever—that a given criminal act was actually *determined* by the individual's genetic structure rather than merely *influenced* by it. If influenced, to what *degree* was the conduct influenced by "genetic disposition?" Again, we find ourselves getting into the uncomfortably grey areas of mental defect that caused many courts and legislatures to embrace the diminished-capacity compromise.

Related to the question of mitigation of degree is that of whether an individual has the ability to form a requisite specific intent. To summarize briefly once more, our law requires of certain criminal offenses that a specific intent be shown before criminal liability attaches. Thus, for example, the crime of burglary requires proof not just that the defendant entered a structure without permission, but that he or she did so with the specific intention of stealing something or committing a felony once within that structure. Our laws recognize that an individual may be in such a mental condition that he or she is capable of knowingly entering the structure, yet incapable of forming the plan or in-

tent of stealing, burning and so on necessary to establish the *corpus* of burglary. It is at least arguable that a genetic deviant may be capable of controlling his or her decision to enter a building without permission (to continue with the example), but incapable of controlling a continuing urge to set fire to that building once inside. In such a case, evidence of the accused's genetic structure should be relevant to the question of whether he or she was guilty of burglary (here, entry with intent to commit arson) or of mere trespass.

To a certain degree we play word games in discussing what type of crime is involved and in what degree. These games are important only in that they lead to a given punishment or range of punishments. What, then, does the criminal justice system *do* with an individual who has been convicted of an offense and who has a genetic impairment?

In answering this, we should initially analyze the primary theories behind punishment. It is generally recognized that there are four purposes for punishing someone who has committed an antisocial act. The first of them is simple retribution or revenge, based upon ancient concepts of "an eye for an eye." Although there is perhaps some value to granting the victim of the antisocial conduct a degree of satisfaction in seeing the offender punished, there are very few legal scholars or penal experts who would accept revenge as a valid consideration in determining the punishment to be meted out. In any event, applying the revenge theory to the genetic aberrant makes little sense: Are we going to exact retribution from a person because of a birth defect?

The second recognized basis for punishment is deterrence. Under this theory, we punish an offender not so much for the effect it has on that person as for the effect it will have on others. Nothing can be done to correct the harm that has been done by this offender, the reasoning goes, but we can set an example by punishing that person and thus inhibit him and others from committing similar acts. Although this is a valid theory generally, the flaw when applied to the genetic deviant is obvious: How does one deter individuals who have little or no control over their conduct? If an individual commits an antisocial act because of a complex interplay of biological factors beyond his or her un-

derstanding or control, how would the threat of punishment have any effect? Once we accept scientific evidence that conduct can be determined or at least strongly influenced by a person's "programming," deterrence as a factor in punishment ceases to exist. Of course, the *extent* to which an individual's conduct is influenced may well be relevant to the question of deterrence—if that extent can ever be scientifically or medically determined.

The third premise of punishment is rehabilitation. Currently, the most fashionable among penal experts and sociologists, this theory takes the approach that the offender should not be punished per se, or at least that punishment should not be the primary objective in dealing with the criminal offender. Rather, both society and the offender will receive greater benefit if he or she is rehabilitated, that is, changed through counseling, education and so on, into a more productive and cooperative member of society. Although this is a commendable goal, its application to the individual carrying abnormal genes is probably pointless. Assuming that a person convicted of a crime committed that crime because of genetic programming, how can he or she be rehabilitated? Rehabilitation proceeds on the assumption that conduct is environmentally caused, and that attitudes and behavior patterns that lead to criminal conduct can be modified. But when the conduct is biologically caused, the theory behind rehabilitation fails.

There is the possibility that the concept can be expanded to include biological rehabilitation. Science may, for example, some day learn to alter genetic structure or body chemistry in a manner sufficient to neutralize a person's congenital genetic defects. However, this opens whole new worlds—dark worlds—reminiscent of involuntary sterilization, prefrontal lobotomies and electrical and chemical shock therapy.

The fourth and final reason behind punishment is simply one of isolation. The primary concern here is not for the offender but for the protection of members of society by removal of the offender from that society—either by imprisonment or by some other process such as deportation. Imprisoning or deporting an individual because of a birth defect goes against the grain of our sense of justice, however, and it would be difficult to justify such action. Yet the interests of society in being safe from the future

criminal acts of a genetically aberrant convicted offender must be weighed against that offender's rights and our sense of justice. For here, alone among the four penal theories, may be the only realistic method of dealing with the genetic deviant: isolation.

What is done with individuals found not guilty by reason of insanity? They are certainly not returned to the streets to commit another antisocial act. Rather, they are usually committed to a mental institution for treatment until they are cured; if they cannot be cured, at least theoretically they may be institutionalized for the rest of their lives. Although this type of commitment may vary little realistically from imprisonment in a penal institution, the process is easier on society's sense of justice. Granted that the incurably insane will, in effect, be imprisoned for the rest of their lives because of a medical condition not their "fault," the interest of society in being safe from them nevertheless outweighs the personal injustice.

Perhaps so, too, with the genetically impaired individual—if such persons have committed antisocial acts as a direct result of that impairment. It would prove fruitless to seek retribution against them, attempt to rehabilitate them or offer them as a warning to other genetically impaired members of the community. But society can protect itself by committing such individuals to a facility should it appear likely that antisocial conduct will recur.

NOTES

1. *McNaghten's Case*, 10 Clark & F. 200, 211, 8 Eng. Rep. 718, 722 (1843).
2. Rollin Perkins, *Criminal Law*, 2nd ed. (Mineola, N.Y.: Foundation Press, 1969) at p. 859.
3. Model Penal Code, sec. 4.01.
4. *Durham v. United States*, 214 F.2d 862, 874–875 (1954).

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Prophylaxis

Some medical authorities have suggested that science will some day enable us to analyze an individual's DNA or other genetic structure and predict with considerable accuracy the likelihood of violent or otherwise antisocial conduct in the future. We may, for example, be able to take a blood sample from a three-year-old child and after analyzing it determine that, based upon extensive data from millions of other children, there is a 92 percent chance of that child's growing up to become a habitual thief, a 78 percent chance that he will become a compulsive rapist or a 61 percent chance that he will eventually take at least one human life violently and without justification. What then may—must?—society do to protect itself from this three-year-old child?

This is not so improbable as might be initially believed. Certainly, the trend of genetic research clearly points in that direction; aided by computerization, increasingly efficient information-gathering methods and statistical analysis, relatively accurate predictions of probable future criminal conduct seem not so far-fetched.

Nor is the idea so extreme of identifying and isolating individuals who can be statistically shown to be a potential threat to society, regardless of the fact that those individuals may never before have committed an antisocial act. Consider, for example, a suggestion by Hyman Gross of New York University's Law School and formerly Arthur Goodheart Professor of Legal Science at Cambridge University:

[T]he possibility of a radically different system deserves consideration, for we might abandon much of criminal justice as we know it now and might instead adopt procedures designed to identify, sequester and correct criminally dangerous persons. Just as the automotive industry calls back for correction models found to be defective and a danger on the road, we might require persons who have shown signs of being a threat to social safety to submit first to diagnostic examination and then to a regime of corrective therapy if the existence of dangerous tendencies is confirmed.

In such a system, rules of conduct in the law would be replaced by specifications of suspicious behavior indicating possible dangerous tendencies. . . . There would no longer be room for excuses and other claims to avoid blame that would leave dangerous persons at large simply because they could establish that under the circumstances it would be unjust to condemn them.[1]

Gross was not referring to genetic aberrations in this discussion but to individuals who could be identified by other means:

Though examination of personal traits will generally prove disappointing, there is other information that is much more promising as a way of predicting crime and making its prevention possible. Social statistics make it clear that often there is a heavy concentration of crime among the part of the population that can be identified by reference to such things as education, race, occupational history, sex, economic status and age. If a target group is defined with sufficient statistical precision, an astoundingly high proportion of its members can be expected to commit crimes, and often just the types of crimes that concern other members of the community most urgently.[2]

Yet when compared to the potential accuracy of genetic prediction, these means appear relatively imprecise. The concept, however, is the same: Identification of probable future criminals through the application of statistical analysis to reliably observable data.

Of course, this all has the ominous ring of Orwell's "1984" and causes a dark discomfort somewhere deep inside the gut. Yet the scientific data on genetics *will* be available in the near future, and when it is available, the criminal justice system will have to deal with the problems posed. The problem will not simply be one of how to deal with a defendant who committed

an offense because of his or her genetic makeup: It will also be one of whether to seek out persons with "dangerous" genes and forcefully isolate them from society. It will be difficult to ignore the fact that society now has the ability to identify many of those who will kill, maim or rape. Faced with the prospect of saving innocent future victims, how heavily will the "rights" of a genetic aberrant weigh?

Yet the cost of such a decision would be great. As Gross recognized concerning his own theory of preventive isolation-treatment:

> Though such a program for control of dangerous persons would be based on a rational assessment of dangerousness, it would shock the conscience of a society committed to egalitarian ideals and to rights of self-determination. Citizens would be deprived of their liberty because of their statistical misfortunes.[3]

Obviously, it is unjust to punish someone for being genetically different—just as it would be wrong to punish a person for being mentally ill. But this is not the answer, for we *do* "punish" the mentally ill, and, furthermore, we punish them long before they have committed any criminal acts. Our society has already weighed the countervailing considerations involved and determined that a person who is found to be potentially dangerous because of a mental abnormality may be committed to an institution through noncriminal procedures. Granted that most such cases in our society today involve persons who have previously committed antisocial or even dangerous acts, the fact remains that we have already weighed the considerations and made a collective decision that it is the *potential* dangerousness of mental aberrants and not the present existence of any criminality in their conduct that justifies taking away their freedom.

The concept, then, has already been adopted; it only awaits application to the genetically impaired. The difficult decisions, however, do not end there. Once science has opened Pandora's box for the law, dilemmas abound. For example, just how far are we to go in identifying individuals with deviant genes? Certainly, science presently has the capability through the process of "karyotyping" of testing an individual and determining whether

his or her genetic structure contains a "flaw." Is everyone, then, to submit to genetic testing upon reaching the age of eighteen? Will it be a felony to avoid such testing, much as it now is to fail to register for the draft?

There are difficult moral questions to be considered in facing such decisions. Yet what is morally correct in this context? What is ethically justifiable? Ethics and morality are merely reflections of both the state of knowledge of our universe and the existing needs of our society. As one commentator noted:

> Science and technology are the precipitators of most of our moral questions. As they add to our knowledge and thus to our control and power of choice, they frequently pose new issues about right and wrong, good and evil, desirable and undesirable. . . .
>
> With the weakening of authoritarian ethics morality is being based more and more pragmatically on human need and less and less on alleged revelations of the divine will or arguments based on such revelations.[4]

It must be recognized, as has one noted physician, that "man exercises ever more certain and effective control" over the quality of human life. "It will become necessary and acceptable to place relative rather than absolute values on such things as human lives, the use of scarce resources, and the various elements which are to make up the quality of life or of living which is to be sought." Thus, he concluded, "a new ethic" must be recognized in "what is almost certain to be a biologically oriented world society."[5]

Given the future ability of science to identify genetic factors that can be linked statistically or otherwise to criminal behavior, what specific means will society have at its disposal to prevent that behavior? Given that the right of society to protect innocent potential victims will be considered superior to traditional concepts of "fairness" and the right of the individual, are there *any* standards other than those of efficacy?

Essentially, there are two preventive or prophylactic approaches to the problem of genetically identifiable criminal behavior. The first is to deal with the individual carrying the genetic trait. The second is to prevent his or her existence to begin with.

Where society is able to identify an individual who suffers from a genetic abnormality that manifests itself in dangerous antisocial conduct, there are a number of steps that can be taken to eliminate the risk of harm this person represents. The first possibility is to execute that individual. If, in fact, it can be shown that the individual is almost certain to cause death or great suffering to others, recognition of the utility of prophylactic execution is unavoidable. But this is a drastic step and one that will undoubtedly be unnecessary in view of the alternatives that science will make available. Certainly, looking at it from current concepts of morality and human rights, it appears indefensible. The idea of executing someone who has not yet done anything wrong but is merely a *potential* source of danger is abhorrent. Even were we able today to establish clearly that a given individual would kill an innocent victim at some time in the future, we would draw back from taking that individual's life as a preventive measure. Our present morality requires that the individual be permitted to complete his or her lethal act before being "punished." Our morality is based upon free will and fault, not upon genetic determination and social utility.

Another prophylactic approach would be to isolate the individual, that is, to incarcerate this person before he or she commits the statistically probable crime. This represents an economically burdensome procedure to society: The costs of preventively incarcerating—possibly for life—thousands of individuals, at a price tag of perhaps a million dollars each, would be staggering. However, the cost to society of *not* isolating these future criminals would also be great.

Again, the idea of locking up someone who has not yet done anything wrong rubs against our concepts of justice. Those concepts will change in time as do all moral standards. But is it really all that unacceptable by even present standards? The fact is that for centuries we have preventively imprisoned persons because of their *potential* for criminal conduct. For example, the existence of vagrancy laws is almost universal. Such laws make it illegal to wander about without visible means of support and/or without apparent business. Certainly, no harm is done by such conduct itself. Yet under these laws, "vagrants" are either thrown in jail or the threat of being thrown in jail is used to force the

individual to "move on." This is done because such individuals represent a *statistically* significant *future* risk to the community: Vagrants are known to be generally more likely to commit crimes than are other people.

Nor is vagrancy an isolated example. Our laws are replete with examples of situations where an individual will be imprisoned because of the *potential* for harm that he or she represents. Thus, for example, we do not wait until a drunk driver injures someone on a highway: We punish that person for driving while intoxicated because of the possibility of future harm—and despite the fact that the act of drunk driving per se involves no harm to anyone. As another example, we have the crime of burglary. This offense consists of the mere entry into a structure with the *intent* to commit a theft or felony. Again, then, we do not wait for the person to commit the theft or felony: The *potential* for theft or felony constitutes the offense. In fact, we do not even wait for the burglar to enter the house. If this person is carrying burglar tools, we imprison him or her for that, despite the fact that carrying screwdrivers and crowbars is not in itself a harmful act.

Perhaps the best example of society's present willingness to preventively imprison persons is represented by our laws concerning solicitation, conspiracy and attempt. We make it a serious offense to solicit another to engage in a criminal act, even though no criminal act ever, in fact, occurs. We impose prison terms for conspiring with another to commit an offense, again, even if nothing further ever takes place. We recognize that individuals should be thrown in jail for unsuccessfully attempting to commit a crime, despite the fact that, by the very nature of this crime, no harm is actually caused. Each of these common legal doctrines represents a view of society that preventive detention is appropriate, for in each case no actual "crime" was ever committed by the person thrown into jail. As the eminent legal scholar Herbert L. Packer observed:

> The law of attempts represents an uneasy compromise with the pressure for preventive detention. It is a compromise that falls so far short of what would be demanded if the idea were given full sway in the criminal law, as those holding the behavioral view would like to see

done, that we can see in it a paradigm of the criminal law's essentially self-denying ordinance: we will concern ourselves with prevention of future antisocial conduct, but we will do so only when we can anchor the necessary prediction to something specific and concrete that the actor has done in the past.[6]

Thus, as Packer noted, this aspect of our existing criminal law represents a compromise between the need for requiring *some* type of concrete conduct before imposing punishment and the need to preventively imprison individuals who have demonstrated that they represent risks of *future* criminal behavior. The concept, then, is not unique: Prophylactic detention is already accepted in theory by our existing laws.

Yet another way of dealing with individuals who are genetically identifiable as future criminals is to treat them medically. Chemical or hormonal therapy may be a possibility. Another, mentioned in chapter 6, is brain surgery: Destruction of portions of the amygdala or prefrontal lobes, for example, have been shown reliably to eliminate violent behavior without precluding the leading of a productive life. Still another medical procedure that will undoubtedly be available in the future is genetic surgery.

Scientists in the field of genetics are making incredible advances. As one observer noted, "Science in general has been doubling its information every ten years, biology every five, and genetics every *two* years."[7] Already, geneticists are in the process of creating new life forms; the concept of genetically altering existing life is almost "old hat." One of the methods for altering an individual's genetic structure surgically is called "gene splicing."

The crucial factor is the biologist's increasing ability to snip out specific genes and move them from one cell to another. This bit of cut-and-sew work was regarded as impossible two decades ago; today it is fast becoming routine. . . .

In theory, the road to treating genetic disease is wide open. To cure a condition caused by an enzyme deficiency [for example] one would simply put the gene for the missing enzyme into human cells and then put those cells into the body of the patient. The cells would produce the enzyme, thus treating the disease.[8]

The concepts of genetic surgery apply not only to treatment of medical diseases but to "treatment" of genetically influenced criminal behavior. Given an individual with a genetic abnormality that is likely to manifest itself in future violent conduct, there is apparently no medical reason why a "bit of cut-and-sew work" could not be employed some day to correct that condition.

Yet another possible prophylactic approach to the problem would be the treatment not of just isolated genetic aberrants but of the general population. Just as many populations are today vaccinated against smallpox or have flouride placed in their drinking water to prevent dental problems, so some day may people routinely be administered some substance that will counter criminal tendencies. As a professor at Carnegie-Mellon University wrote:

> Most of the milk available today is pasteurized and homogenized and has vitamin D added. Will the milk available tomorrow have an antihostility drug added or will such a drug be added to the water supply? Will this make for a peaceful population, and if so, will it be worth it? [Another scientist] has suggested that by year 2000 we will have a drug-controlled society or a society that will self-destruct.[9]

Again, the specter of mass drugging of the public causes uncomfortable feelings—even outrage—in most of us. Again, however, the precedent has already been set, and it becomes only a question, perhaps, of degree. Again, the alternatives of violence and injury may eventually cause a shift in current attitudes toward such "Brave New World" practices.

Dealing with the individual who is carrying a "dangerous" genetic trait is only the first of two possible approaches to the problem. The other is to insure that no such individuals are ever born in the first place.

One method of preventing their existence is through genetic screening. This procedure simply involves the identification of persons carrying genes that cause criminal behavior and prohibiting them from having children if there is a likelihood that their offspring will also carry the trait. If, for example, science determines that a genetic aberration, which we will call the "TZ Syndrome," results in predictably violent behavior, and that both

the father and the mother must carry the defect, a man with TZ genes would not be permitted to have children with a TZ woman.

Again, there are obvious moral questions involved here. The Nuremburg Code, for example, specifically outlaws any genetic screening programs in the absence of the subject's informed consent.

As with other procedures for dealing with potentially dangerous genetic abnormalities, the precedent for genetic screening has already been established. Between 1963 and 1968, for example, the legislatures of forty-three states passed laws requiring the testing of children for the genetic disease known as phenylketonuria. Phenylketonuria, or PKU as it is called, inevitably results in mental retardation; treated, however, a child might be cured. These laws typically require the physician attending at the time of a child's birth to take a blood sample and to have it analyzed for indicators of PKU. Incredibly, there was fierce resistance to such legislation, and the laws were challenged in the courts. One noted critic expressed the fear that the mandatory PKU tests "are being exhibited as examples of what should be done legislatively in regard to all other diseases allegedly testable in one way or another at birth."[10]

This type of genetic screening is intended to identify individuals who carried the defect so that they may be treated. Other types of genetic-screening programs, however, have been established for the purpose of identifying persons carrying a genetic abnormality so that they may be advised of the dangers of having children.

One such program was instituted by the Italian government in 1962 to screen out individuals genetically who were carrying the gene for beta-thalassemia, or Cooley's anemia. This is a recessive gene that can be carried by persons not suffering from the blood disease caused by it, which is unique to southern Mediterraneans. Under the program, sixteen regional centers were established throughout Italy to identify carriers and to counsel them concerning having children. In fact, Pope Pius XII publicly spoke in favor of such preventive measures, stating that it was permissible to dissuade members of the Catholic Church from getting married or having children if they carried the genetic trait.[11]

Another genetic screening program that has been required by law deals with sickle cell anemia. This disease, which occurs primarily in individuals of African descent, has been a serious problem among the black population in the United States. Approximately 10 percent of the American black population (roughly 2 million people) carry the trait; about fifty thousand actually suffer from the disease. As a result, thirteen states passed laws between 1970 and 1973 designed to identify carriers of the trait and counsel them concerning the risks of getting married and having children. Most of these laws made it mandatory that all black children be tested in school, or that black individuals applying for a marriage license must be tested for the genetic condition. In 1972 the Congress of the United States passed the National Sickle Cell Anemia Act. Despite attacks from critics that these laws were "genetically discriminatory," most of the laws survive.

Yet another genetic abnormality, found among persons of Jewish descent, is Tay-Sachs disease. This condition has also been the subject of state and federal genetic-screening legislation, with the purpose being to identify carriers and counsel them concerning the advisability of marrying and having children.

Other genetic-screening laws have also been passed, and, certainly, more will be in the future as science identifies more and more diseases and medical conditions that are genetically transmitted. Much of the technology is already here. For example, the Nikon Corporation ran the following advertisement in various periodicals:

> Nikon Looks into a Future Baby
>
> The L-Ke microscope plays the role of scientific crystal ball in helping predict possible defects in as yet unconceived children.
>
> All samples are obtained from prospective parents, prepared for karyotyping procedures, and photographed with the Nikon L-Ke. Resulting photomicrographs are then studied for possible chromosome aberrations such as inversion, deletion and translocation.
>
> This data provides the physician with information for advising patients of danger signs which could lead to genetic imbalance.[12]

Attitudes of both lawmakers and the public toward genetic screening are changing. As one noted authority observed:

Mass genetic screening, despite its controversial history and the uncertain size of its therapeutic dividends, is gaining acceptance among public health authorities in the United States. . . .

The rapid proliferation of screening laws . . . suggests that the state has an abiding interest in the reduction of genetic disease. It is reasonable to assume that future detection technologies will continue to receive government support.[13]

Clearly, the concept of genetic screening has been accepted and is being expanded. Is it unreasonable to expect that this procedure may, at some time in the future, be expanded yet further—beyond the detection of genetic disease to include the detection of genetic abnormalities that cause dangerous criminal behavior? As one observer argued:

We issue driver's licenses, even though the cars of some will become lethal weapons; it is the price we pay for motor transport. If we could tell which applicants for a license will be killers we would not license them. It used to be that we had no way of knowing which couples were carrying a common gene defect or which pregnancies were positive for it. But now we *can* know.[14]

Perhaps more serious problems than whether individuals *should* be genetically screened are posed when we try to decide *when* in an individual's life he or she is to be tested for genetic aberration and what steps should be taken thereafter. Should children be subjected to such tests? If a six-year-old child is found to carry a dangerous genetic characteristic, is he or she to be taken away immediately and committed? Or is the child to be permitted to stay with his parents until the age of, say, sixteen, always with the sword of Damocles hanging over the family's head?

Do we go further back and test newborn babies, requiring by law that the infants not be permitted to live, much as is done in many more primitive cultures with babies displaying defects such as Down's syndrome or malformed limbs?

Will we go even further back and test the unborn fetus in every pregnancy? If the fetus is found to have "bad" genes, are we to require an abortion by law? Again, science has already thoughtfully provided us with the process of "amniocentesis" with which

to perform this task. Yet another balancing of the relative interests of society and the individual must be conducted: Amniocentesis involves a very definite risk to the embryo, and in the process of attempting to detect a defective embryo a perfectly normal one may be damaged or even killed. Undoubtedly, however, science will eventually discover new, safer methods for determining whether a fetus is carrying a dangerous genetic trait. Also, undoubtedly, requiring the abortion of such a fetus will be an available option to the legislators of tomorrow.

Will we go all of the way back to the origins of life itself and order sterilization of any person genetically capable of transmitting aberrant genes to offspring? Once again, the concept of sterilization is not as new and shocking as might be originally supposed. There is already considerable precedent for ordering sterilization for genetic purposes. In 1907 Indiana became the first state to enact a sterilization law. Within five years, fourteen other states followed suit, many of them following the so-called Model Eugenics Act, which listed as subjects for sterilization any individual institutionalized for syphilis, leprosy or tuberculosis, as well as deaf, blind and deformed persons, chronic alcoholics and persons who were "dependent on the State." This was justified in a very influential book of the time as "a practical, merciful, and inevitable solution of the whole problem [which] can be applied to an ever widening circle of social discards, beginning always with the criminal, the diseased, and the insane."[15]

Despite the drastic nature of sterilization, these statutes were not challenged in the courts for many years. In fact, it was not until 1925 that a mandatory sterilization law was reviewed by the Supreme Court of the United States. Under attack was such a law in Virginia that had been applied to a mentally deficient woman who, the state court had decided, was "the probable potential parent of socially inadequate offspring likewise afflicted." Speaking for the majority of the Court, the great Justice Oliver Wendell Holmes wrote:

> We have seen more than once that the public welfare may call upon the best citizens for their lives. It would be strange if it could not call upon those who already sap the strength of the state for these lesser sacrifices, often not felt to be such by those concerned, in order to prevent our being swamped with incompetence.[16]

Holmes then found the sterilization statute constitutionally *valid*, relying for precedent upon a case that permitted compulsory vaccination for smallpox.

Within the next ten years, twenty more states adopted compulsory sterilization laws. By 1928 at least twenty thousand individuals had been sterilized under the authority of such statutes; by 1935 the figure had reached forty-five thousand.

Perhaps the most significant legal challenge to the mandatory sterilization laws came in 1942, when the U.S. Supreme Court considered Oklahoma's Habitual Criminal Sterilization Act. That statute provided that any individual who had been convicted of three separate felonies would be "rendered sexually sterile without detriment to his or her general health." The Court overturned the statute, but only because it violated the Equal Protection Clause by exempting certain kinds of felonies, such as embezzlement, prohibition and political crimes. However, the Supreme Court did *not* find compulsory sterilization for criminals to be invalid. In fact, Chief Justice Harlan Stone specifically recognized in his concurring opinion that "science has found and the law has recognized that there are certain types of mental deficiency associated with delinquency which are inheritable." In confirming that government *can* force such individuals to be sterilized, Stone wrote that "the state may protect itself from the demonstrably inheritable tendencies of the individual which are injurious to society."[17]

Approximately another twenty-five thousand persons were sterilized under such laws after this decision. It was not until the late 1950s and the emergence of a civil rights-oriented Supreme Court that mandatory sterilization began to taper off and finally all but disappear. Yet until very recently few states had repealed their sterilization laws; as late as 1966 at least twenty-three states still had such statutes in effect.

Today the few statutes that remain have been reworded or judicially interpreted so that it is nearly impossible to obtain compulsory sterilization for any reason. The Supreme Court has generally taken a position that the individual has a "right of privacy" in the general area of childbearing. Nevertheless, the precedent is there. If a majority of the states—not to mention the U.S. Supreme Court—has endorsed mandatory sterilization for genetic defects before, is there any reason to believe that they

will not do so again? Sterilization of individuals on the theory that they may transmit criminal tendencies is not new. What will be the likely public and political reaction in future years when science can clearly demonstrate the inheritance of such tendencies?

Preventive isolation, genetic surgery, prefrontal lobotomies, genetic screening, abortion, sterilization—all of this has an ominous and terrifying ring to it. There are cold, logical reasons why such methods for dealing with criminality should *not* be adopted. As one religious leader wrote:

> Society does not have very good methods of setting its goals. Those who decide societal goals are actually the powerful members of society, and they tend to decide for their own advantage. Even when they are most idealistic, they project their partisan ideals. . . .
>
> The record of the present generation of mankind is not so impressive as to convince me that it should become the guardian and director of the human genetic future. Genetic planning, if it were to be highly effective, might well perpetuate certain racial or cultural stereotypes so as to force humanity into a cul-de-sac of obsolescence. . . .
>
> Genetic engineering has in it the makings of a totalitarianism the like of which this world has never seen.[18]

An opposing view has been expressed by another observer:

> All alleged human rights cease to be right, become unjust, when their exercise would victimize innocent third parties and bystanders. All rights are "imperfect," not absolute or uncontingent. We might say this particularly of the so-called "right to privacy" as it bears on propagating at will and inordinately. The social welfare and protection of third parties has a prior claim. The "right" to reproduce, like all others, is—morally weighed—really only a privilege. . . .
>
> Not to control, and not to weigh one thing against another, would be subhuman. A mature ethics is social, not egocentric. Call it what you will—mathematical morality, ethical arithmetic, moral calculus—we are obliged in conscience to think of benefits relative to costs.[19]

Yet another perspective is presented by John Stuart Mill in his essay "On Liberty":

> The fact itself, of causing the existence of a human being, is one of the most responsible actions in the range of human life. To undertake

the responsibility—to bestow a life which may be either a curse or a blessing—unless the being on whom it is bestowed will have at least the ordinary chances of a desirable existence, is a crime against that being.[20]

The trend of current genetic research is clear. Assuming that the legal system is eventually presented with the scientific capability of "reading" DNA and the statistical ability of predicting with substantial accuracy the probable future consequences of aberrant genetic structure, some very difficult questions will be posed. How should the criminal justice system deal with a criminal offender whose conduct was caused by a genetic aberration? Does society have the right to seek out such individuals before they cause harm and remove them from the community? Can society prevent such individuals from being born in the first place? As always, it will be up to our legal system to attempt to realize advances made possible by science at the least cost to human liberty. The decisions cannot be avoided: Society will simply not ignore the reality of being able to predict and thereby prevent harm to its innocent members.

NOTES

1. H. Gross, *A Theory of Criminal Justice* (Oxford: Oxford University Press, 1979) at pp. 41–42.
2. *Id.* at p. 43.
3. *Id.* at pp. 43–44.
4. J. Fletcher, *The Ethics of Genetic Control* (New York: Anchor Press/Doubleday, 1974) at pp. xviii–xix.
5. M. Watts, "A New Ethic for Medicine and Society," 113 *California Medicine* 67–68 (September 1970).
6. H. L. Packer, *The Limits of the Criminal Sanction* (Stanford, Calif.: Stanford University Press, 1968) at p. 101.
7. Fletcher, *supra* note 4 at p. xvii.
8. E. Edelson, "Genetic engineering: Where will it lead?" 11 *Family Health* 31 (May 1979).
9. K. E. Moyer, "The physiology of aggression and the implications for aggression control," in *Personality and Psychopathology*, ed. J. L. Singer (New York and London: Academic Press, 1971) at p. 61.
10. S. P. Bessman, "Legislation and advances in medical knowledge: Acceleration or inhibition?" 69 *Journal of Pediatrics* 334 (1966).

11. P. Reilly, *Genetics, Law and Social Policy* (Cambridge, Mass., and London: Harvard University Press, 1977) at p. 63.
12. "Nikon looks into a future baby," *Science* 190 (November 21, 1975).
13. Reilly, *supra* note 11 at pp. 106,121.
14. Fletcher, *supra* note 4 at p. 160.
15. M. Grant, *The Passing of the Great Race* (New York: Arno, 1916).
16. *Buck v. Bell*, 274 U.S. 200 (1927).
17. *Skinner v. Oklahoma*, 316 U.S. 535 (1942).
18. R. L. Shinn, "Ethical issues in genetic choices," in *Genetic Responsibility*, ed. M. Lipkin and P. T. Rowley (New York and London: Plenum Press, 1974) at p. 115.
19. Fletcher, *supra* note 4 at pp. 179–180, 159–160.
20. J. S. Mill, "On Liberty," in *The Essential Works of John Stuart Mill* (New York: Bantam Books, 1970).

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Reflections

For many years, society has been told that behavior and personality were shaped almost exclusively by environmental factors. The occasional whispers from the scientific community that perhaps genetics may have some influence have been shouted down by a united front of social scientists, educators, and politicians. Yet the evidence seems increasingly to indicate that hereditary factors strongly influence an individual's personality and behavior. Such evidence would seem critically important not only to the criminal justice system, but to those concerned as well with education, employment, poverty and mental illness, to name but a few fields that have based their efforts on a mistaken belief that changing environmental conditions will always result in a change in the individual.

Why do we continue burying our heads in the sand and pretending genetics does not exist? We do so because the very concept of genetically determined conduct runs contrary to our democratic society's standards and beliefs. "All men are created equal," after all, and America is the land of opportunity. People can make of themselves what they wish, limited only by their own efforts. Any child can grow up to be president.

It is unlikely, however, that the founding fathers had biological sameness in mind when they declared all persons to be equal. Obviously, the intent was to express *political* equality—the right of each individual to the same liberty and opportunity. Unfortunately, the difference has been increasingly obliterated, per-

haps in the belief that existence of biological inequality begets political inequality. Certainly, the American sensitivity to racial problems does not help generate a dispassionate approach to questions of genetics.

What we have done, then, is to subvert truth in the name of "social good." As with every civilization that has ever existed, we have buried fact to achieve political goals. The sun revolved around the earth for centuries, because such a scheme suited the social order of the times. Humans were divinely created in the image of God, because such a scheme suited the social order of the times. Human behavior is determined by environment, because such a scheme now suits the social order of our times.

Yet as history has repeatedly shown, the denial of truth and the blind acceptance of the convenient does far more damage in the long run than simply facing reality. As long as we ignore, we impair the ability to assess problems and find answers. If we choose to accept cancer as being divinely caused, we are unlikely to find a cure for that disease. If we choose to believe that crime is caused by environmental conditions because that theory is socially acceptable, we are unlikely to discover ways to prevent criminal conduct or rehabilitate criminals.

Many believe that if the concept of environmental determination is rejected, the basis for our social order—equality—is threatened. This is a misconception. To pervert the democratic concept of political equality into a new meaning of genetic equality, and then to reject all scientific data that refutes it, is to duplicate the history that resulted in Galileo's imprisonment and Socrates's death.

We are *not* all born equal. Insisting that we are—that we are clay at birth, waiting to be formed by the environment—accomplishes nothing but a postponement of the ability to deal effectively with the problem of crime in our society.

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(CONTINUED FROM FRONT FLAP)

scientific knowledge. He suggests that these concepts will eventually be replaced by a preventive rather than punitive approach to criminal behavior.

Born to Crime presents scientific data and legal and ethical beliefs in a vital and timely work. Lawrence Taylor has limited his presentation to the emerging scientific evidence that genetics plays a key role in the origins of criminal behavior. He makes no conclusions and offers no theories concerning the moral issues arising from the application of this evidence, leaving those avenues for the reader to explore. **Born to Crime** is important reading for practitioners in legal, scientific, and social service professions, for students and researchers in these fields, and for those interested in contemporary concepts of crime and punishment.

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LAWRENCE TAYLOR is Associate Professor of Law, Gonzaga University, Washington. He has served as Deputy County Counsel, Deputy Public Defender, and Deputy District Attorney for the county of Los Angeles, Special Prosecutor for the Attorney General of Montana, and has maintained a private criminal defense practice. He is the author of nine books, including **Trail of the Fox**, **Eyewitness Identification**, **A Trial of Generals**, and **Scientific Interrogation**.

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