Genetics, Antisocial Personality, and Criminal Responsibility

Stephen H. Dinwiddie, MD

There is now substantial evidence that heritable biological factors play a role in the genesis of repetitive antisocial behavior. The differing conceptual frameworks of behavioral genetics and the law are described, and the implications that current research in behavioral genetics may have for assigning responsibility for unlawful behavior are discussed.

It is incident to physicians, I am afraid, beyond all other men, to mistake subsequence for consequence.—Samuel Johnson

Assignment of moral responsibility in the presence of mental illness remains one of the most conceptually vexing of medico-legal problems. It is generally (although hardly universally) agreed that in the setting of significant impairment of reasoning ability or loss of reality testing, one should not necessarily be held accountable for one’s actions; but even among those who support the concept of an insanity defense there remains room for debate over the nature and severity of impairment required to absolve one from moral responsibility. Such contention endures not least because, for mental health professionals, the domain of “mental illness” properly extends far beyond that which is useful for legal ends. Perhaps nowhere is the conflict more apparent than in the way the two professions approach the problem of repeated antisocial conduct.

For mental health professionals, the observation that certain individuals show a long-standing pattern of disregard for social norms and the rights of others, appear not to experience remorse (at least as it is commonly understood), and tend not to change their behavior in response to social sanction becomes a nosologic issue: How best does one describe and study this abnormal pattern of behavior? While mental health professionals cannot avoid addressing the multitude of moral issues that arise in the course of treatment of such individuals, at the level of description and study, the issue of blameworthiness of the individual is simply not relevant. Rather, what is desired is accurate
and reliable description of a clinical entity. While repeated criminal behavior frequently accompanies this syndrome, criminality is neither necessary nor sufficient to establish its presence. Such classification, therefore, differs in purpose from the needs and ends of the criminal justice system, and extrapolation from research on a clinical syndrome to applications in the justice system may be inappropriate.

At this level of descriptive study, in fact, it is clear that the boundaries of the condition remain somewhat fluid, with agreement between diagnostic systems rather disappointing. Nonetheless, the legitimacy of this syndrome has been acknowledged for decades, and the concept of antisocial personality disorder (ASPD) is as well validated as any psychiatric diagnosis now in common use. That is, using the principles initially set out by Robins and Guze, this psychiatric entity can be shown to be separable on clinical grounds from other psychiatric illness, stable over time, and reliably diagnosed. While no specific laboratory test exists that can establish the presence or absence of ASPD, a number of studies have demonstrated population differences between subjects with and without ASPD on markers such as autonomic activity, electroencephalographic patterns, hormone levels, and cerebrospinal fluid metabolite levels.

Additionally, numerous family, twin, and adoption studies have established the presence of heritable liability factors for antisocial behavior, leaving little doubt that biological, presumably genetic, factors contribute in part to its ultimate expression. Such findings are remarkably consistent despite marked differences in selection of subjects, criteria for diagnosis (e.g., restriction to "criminality" or inclusion of noncriminal behaviors characteristic of ASPD or conduct disorder), and statistical techniques. While many such studies (particularly earlier ones) suffer from significant methodologic shortcomings, there is substantial agreement in the finding that such behaviors aggregate in families.

But while such aggregation cannot be explained solely by environmental factors, genetic factors probably manifest most strikingly in the context of exacerbating environmental conditions. Thus, as for many complex disorders (such as other psychiatric disorders, atherosclerotic heart disease, or many neoplasms), a syndrome of repetitive antisocial behavior is best conceived of as the result of a combination of genetic and nongenetic factors acting in common.

Nonetheless, given the existence of moderate genetic influence on antisocial behavior, and given the rapid progress in our ability to identify specific genetic sequences, it seems likely that researchers will soon begin to explore the possibility of associating genetic markers with ASPD or other persistent dysocial behavior patterns in a manner similar to research efforts currently underway for alcoholism, manic-depressive illness, and schizophrenia.

If such strategies bear fruit, molecular genetic studies will thus prove to be the latest in a long line of investigations showing population differences between antisocial and non-antisocial individuals.
Behavioral Genetics

The finding of genetic markers by which those at high risk for such behaviors could be differentiated from those at lower risk would be a tremendous advance from a research perspective.

But scientific debate may be radically changed when such findings are applied to issues of social policy. Highly complex, multifactorial constructs risk becoming oversimplified and their scientific worth thereby diminished because of misunderstanding and misapplication in practice. Especially in the current political climate, the risk of such distortion is perhaps nowhere greater than in the application of research findings to the social problems caused by antisocial behavior.

To demonstrate the complexity of the problem, the phenotype of violent behavior, as a frequently studied aspect of antisocial behavior, will be used as an example. Violence is known to be a multiply determined phenomenon, its expression depending on characteristics of the victim and the setting in which the confrontation occurs, as well as the qualities of the aggressor. Characteristics of the aggressor at the time of the act, in turn, include both state variables (recent alcohol intake, current emotional status, access to weapons, etc.), predisposing trait variables (e.g., personality characteristics, neurological factors), and prior experiential factors such as history of head trauma or exposure to physical abuse in childhood. Genetic factors may still operate at a variety of levels, with varying degrees of specificity in relation to expression of the particular phenotype, influencing such disparate factors as, for example, neural development, temperament, amount of alcohol use and risk of alcoholism. Along the path leading to expression of violent behavior, these factors may interact with one another and with experiential factors, and as well may possibly influence exposure to environmental factors, which in turn may influence risk of violence. Finally, some genetic factors may be identified that seem to be very closely associated with risk for violent behavior, but because of low population frequency, contribute little to the total amount of violence in the community.

Other criminal or antisocial behaviors, although not as intensively studied, are if anything more likely to be influenced by genetic factors, with at least two large adoption studies indicating a heritable component to property, but not violent, crime. Nonetheless, the same complexities as found in studying the causes of violent behavior remain.

Thus, while population differences between those who exhibit antisocial behavior and those who do not are very likely to exist at the genetic level, just as they do for other biological (or environmental) characters, it is unlikely that identification of genetic liability factors would by itself lead to accurate prediction of who might commit antisocial acts, violent or otherwise. At best, genetic factors are likely to be part of a multitude of influences affecting the likelihood of antisocial behavior, none of them either necessary or sufficient causes. Thus, in a manner analogous to current ability to predict violent behavior based on clinical characteristics, in any population with a
low base rate of antisocial behavior, classification of risk on genetic grounds alone is likely to be in error much more often than not. Such errors would occur both by misclassifying those with genetic risk factors but who, because of ameliorating characteristics, do not behave in an antisocial fashion ("false positives"), as well as by misclassifying those without the genetic factors who exhibit antisocial behavior ("false negatives").

Unfortunately, this lack of specificity may not be appreciated when cases are ascertained after the fact, as is often the situation when forensic mental health practitioners are involved. Retrospective bias may inflate the apparent importance of predictive factors, causing the evaluator to impute an inevitable, cause-and-effect relationship between a moderately powerful predictor (such as a genetic marker) and subsequent antisocial behavior. Currently, results of neuropsychological testing or sophisticated neuroimaging studies are more and more commonly used to bolster opinions regarding mental states of interest to the courts. Typically such tests demonstrate the presence of organic findings uncommon in the general population; it is then suggested that the behavior in question was due to the organic anomaly, and therefore the defendant did not form a culpable mental state. It seems inevitable that as genetic factors involved in antisocial behavior become better characterized, experts will soon be asked to give similar opinions regarding criminal responsibility in defendants who are putatively genetically predisposed to such conduct.

### Cause and Choice in Law and Psychiatry

The usual justification for the existence of criminal law is that some mechanism for ensuring public order and safety is needed and that society is justified in punishing those whose conduct harms individual or public interests. But the moral justification for imposing punishment is the presumption that the individual had the ability to opt otherwise, and therefore actors may be excused because of compulsion, law enforcement, self-defense, or necessity. One may also be excused if, because of involuntary intoxication or autonomism, one lacked the ability to perform a purposeful act.

Profound mental disturbance may also exculpate. In this case, the actor is conscious, but exculpation is usually justified by considering the illness to either irresistibly impel the wrongful behavior or to prevent the actor from truly understanding its nature.

From the standpoint of the law, the ability to refrain from acting wrongly is therefore necessary to justify punishment, and thus, for both philosophical and policy reasons, the role of choice is emphasized. Some choices may be hard ones, but if the actor can be presumed to have had the ability, although unexercised, to do otherwise, he may be considered blameworthy.

By contrast, mental health clinicians tend to deemphasize choice, concentrating instead on the role of explanatory, putatively causal, factors. Historically, two of the leading paradigms in the field have been psychodynamic theory and
Behavioral Genetics

biological psychiatry.\textsuperscript{50} Both are fundamentally determinist, although the hypothesized causes of behavior are radically different: unconscious intrapsychic processes or (as yet) incompletely characterized functional neural systems. According to these theories, behavior ultimately may be seen as caused by biological or psychological factors that can, at least in theory, be identified by experts.*

In scientific investigation, it is not uncommon to try to find how much change in one variable may be predicted or “explained” by another; such explanation by itself does not speak to causation. But when the practitioner applies such research to treatment ends, he or she often does so in the belief that the intervention will correct the underlying problem (i.e., the cause of the disorder) so that the patient will no longer suffer from the symptoms. Approaches may be radically different—depth psychotherapy to correct underlying narcissistic traits or antipsychotic medication to restore optimal dopaminergic system functioning—but in either case the practitioner frequently imputes a “cause” of the disorder and attempts to reverse or counteract it.

Just as the law’s philosophical approach stresses the role of “free will,” the scientific basis and therapeutic orientation of the mental health professions thus often combine to emphasize the role of deterministic factors. Unfortunately, for virtually any behavior of interest, many factors of roughly equivalent causal efficacy can be found, and when rigorously tested, the field’s success in establishing predictable, cause-and-effect relationships between any specific factors, experiential or biological, and subsequent behaviors has proven to be generally disappointing.\textsuperscript{51–54}

Thus, following Guze,\textsuperscript{55} it seems sounder to assert that few, if any, causes in psychiatry (or any other branch of medicine) are both necessary and sufficient; it is more reasonable simply to define cause for our purposes as “…any event (A) that increases the likelihood of another event (B).”\textsuperscript{56} Fundamentally, this view of cause requires only correlation and temporal ordering. It requires neither that the presumed cause must always be found, given the event, nor that the event must always ensue, given the presence of the causal factor.

Such a relationship is the basis for what has been called “conditional free will.” According to Fishbein\textsuperscript{57}:

\begin{quote}
In accordance with probability theory, social human behavior is contingent on a countless number of possible decisions from among which the individual may choose. Not all of those decisions are feasible, however, nor are the resources available that are required to act on them. Choosing a course of action, therefore, is limited by preset boundaries, which narrows the range of possibilities substantially. Decision-limiting factors include current circumstances and opportunities, learning experiences, physiological abilities, and genetic predispositions … The behavioral result is thus restricted to options available within these guidelines, yet it is “indeterminable” and cannot be precisely predicted. (p 30–31)
\end{quote}

Behaviors are the result, in other words, of interactions between a variety
of state and trait factors and are contingent not only on attributes of the individual, but on the situation in which he finds himself. “Free will,” consequently, if it is to have any meaning at all, must mean behavior that is in accord with (i.e., determined by) the weight of the individual’s values, experiences, goals, and so forth, in a particular setting. Since some of these factors are not easily accessible to conscious reflection and critical evaluation, this is not to say that “free will” in this sense is a purely rational, logic-driven phenomenon. But if this meaning is accepted, the subjective experience of “free will” becomes one in which the individual’s choice stems from this probabilistic interaction between factors, some of which are more available to conscious reflection and modification, and some of which—including heritable factors—are not.

Cause and Moral Responsibility

Knowledge of how some behaviors are caused, even if they can be shown to be under substantial genetic control, therefore is not equivalent to compulsion or lack of free will. Although involuntariness—the judgement that the accused, by reason of his mental disorder, could not have acted differently—is often cited as a justification for the insanity defense, two objections immediately come to mind. First, it is obvious that this is a post hoc judgment; a behavioral outcome that is apparent, given the luxury of hindsight, might not be as predictable beforehand. Such retrospective bias can lend a spurious sense of inevitability to hypothesized causal factors, either biological or psychodynamic, thereby minimizing the role of volition.

More importantly, as noted by Morse, framing the issue of criminal responsibility in terms of being caused by irresistible or overpowering force, such as a mental illness or unalterable genetic factors, may be simply misleading. When an act is excused either because the actor’s reasoning was illogical (for example, believing that sacrificing his child would promote world peace) or when he acted quite logically on an irrational belief (“knowing,” on delusional grounds, that a neighbor intended to kill him and using deadly force in self-defense), the actor is excused not necessarily because he was powerless to act otherwise—there is no reason to assume that irrational choices are necessarily harder to make than rational ones—but because the act was based on beliefs or reasoning processes so profoundly illogical that holding him responsible would be morally unjustifiable.

Mental illness may, in addition, impair one’s ability to keep from doing what one knows is wrong, but such inability to refrain is only one aspect of the legal conception of insanity, and one which some jurisdictions exclude completely. If all acts are caused, by whatever factors, as a matter of practice, we are reduced to excusing the actor if some classes of cause are invoked, but not others. From the standpoint of attributing blame, there is no reason to suppose that having more complete knowledge of the pathway leading to criminal behavior should mean that such individuals need not be held to the same standard of conduct.
Behavioral Genetics

Nonetheless, as more and more biological factors, particularly heritable ones, are found to be associated with antisocial behavior, it is likely that exculpatory arguments will be framed in terms of cause. Particularly if it is shown that an offender has a genetic marker associated with higher likelihood of committing certain acts, it may be reasoned that the actor in some way cannot help himself, because his acts were somehow genetically predestined.

Such an argument rests on several questionable assumptions. First, it assumes that the hypothesized genetic marker could be shown to have a direct causal relationship to antisocial behavior: observing a correlation between gene and behavior is far easier than tracing the pathway from gene product to behavior. Second, such arguments imply that genetic factors are in some way inescapable, that no protective or ameliorating factors (biological or social) could counteract a genetic impetus toward crime. Certainly this is not true of other syndromes with a heritable component; atherosclerotic heart disease comes to mind as a syndrome whose risk can be altered by environmental factors, including behavioral interventions. There is nothing inherent in the nature of genetic factors that makes them inaccessible to postnatal modification and correction. Moreover, given what is known regarding the heritability of ASPD, it seems clear that nongenetic factors also play a substantial role in its development, thus undermining any argument that genetic factors would be necessary or sufficient to cause antisocial behavior.

Indeed, it is not clear why biological correlates should have primacy over other factors associated with antisocial behavior, for example poverty or unstable living situations. Such factors have been known for many years to be at least statistically correlated with criminality (and arguably are as causally important as biological factors), yet are not considered excuses. Again, the issue is not causation as such, but whether the actor reasonably can be said to have a choice of actions under the particular circumstances in which the crime occurs.

Finally, a fundamental objection to any such statistical association is that such an observation by itself is poor evidence of the existence of some inexorable mechanism. Even in a group in which the probability of offending is high, some members will not commit crimes, thus vitiating any necessary and direct connection between gene and behavior, or for that matter environment and behavior. Moreover, such statistical association merely implies that ultimately some antisocial or criminal behavior is likely to occur, not that any specific act is somehow genetically foreordained. Although the elevated probability of an antisocial individual committing a specified unlawful act is a reflection of an intrinsically greater predisposition toward disregarding social norms, he or she will have the subjective experience of choice, weighing the risks and benefits of a number of unlawful behaviors before acting. In cases of exculpatory mental illness, either the logic of choosing or the ends desired are so disordered that moral responsibility is negated. In the case of the antisocial
individual, both the process and the ends are understandable: it is the values and weights accorded them that go into such choices that are felt worthy of condemnation.

Behavior Genetics and Insanity Tests

This line of reasoning therefore falls prey to the same objections leveled at the Durham “product” rule. Originally adopted in order to overcome the perceived overreliance on cognition exemplified by the M’Naghten test, the Durham decision adopted a broader test: “The accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect.” “Product” was further defined as “but for” causation: but for the mental disease or defect, would the defendant have committed the crime? In retrospect, it is apparent that such a question would be difficult if not impossible to answer on scientific grounds, and indeed, in practice, testimony on the issue was so unproductive that experts were ultimately forbidden to speak to the issue at all—precisely the opposite of the original intent, and Durham was replaced by the American Law Institute (ALI) test in 1972.

Undoubtedly part of the failure of the Durham rule was an overreliance on psychodynamic explanations and a simplistic view of causation. But substitute “genes causing antisocial behavior” for “mental disease or mental defect,” and identical problems arise. It is apparent that in the long etiologic chain stretching from genes to behavior there are many causes of criminal behavior, potentially operative at many levels. Expert testimony to decide whether or not a given criminal act was the result of genetic influence would be to run far in advance of scientific foundation. How could one even attempt to determine whether or not “but for” the presence of those genetic factors a different behavior might have resulted?

Another potential difficulty with this test is that if genetic causes of behavior were potentially excusable but other causes were not, the result might be a continuous, rather than “all-or-none,” assessment of responsibility: if genetic factors were operative at all, how much of the accused’s behavior could be attributed to them? To what degree should the accused by held responsible? Such an approach would risk transferring the task of assigning blameworthiness from the finders of fact to the expert.

Under other tests of insanity, genetic factors, if invoked, might play a smaller role. As a matter of definition under the ALI test, a psychiatric condition “manifested only by repeated criminal or otherwise anti-social conduct” cannot be considered a “mental disease or defect.” Thus, questions as to whether genetic factors related solely to antisocial behavior might have prevented the defendant from having an awareness of the wrongfulness of his behavior or might have impaired

1(1) 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. (2) As used in this Article, the terms “mental disease or defect” do not include an abnormality manifested only by repeated criminal or otherwise antisocial conduct (Model Penal Code, Section 4.01).
Behavioral Genetics

his ability to refrain from wrongful behavior apparently would not be meaningful under this test. Such resolution by definition underscores a fundamental difference between medicine and law, since ASPD is no less strongly heritable than other psychiatric disorders such as schizophrenia or manic-depressive illness, which are potentially exculpatory.

Under the M’Naghten standard, where it must be shown 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,” it is possible that genetic factors might be considered relevant. Much hangs on how “wrongfulness” and “knowingness” might be interpreted, as well as which mental disorders would qualify as “disease of the mind.”

To take the latter point first, historically ASPD (in its earlier diagnostic incarnations, such as “psychopathy”) has generally, but not always, been considered not to constitute a “disease of the mind” for this purpose. But there is certainly no legal necessity that this be so; ultimately, the decision of how medical and legal terms equate in a particular case is the province of the finder of fact, and there is evidence to suggest that a number of insanity acquittees may in fact have primary diagnoses of personality disorder. Evidence indicating genetic cause could conceivably strengthen arguments in favor of considering ASPD a mental illness, by placing it on a par with other psychiatric disorders such as schizophrenia or manic-depressive illness, as a mental disorder with demonstrated biological liability factors. But it should be kept in mind that even the most complete elucidation of biological cause of any disorder, including ASPD, is not necessarily a reason for the law to accept it as a mental illness “for its purposes.” As pointed out by Slovenko:

In the final analysis, what is or what is not mental illness in the test of criminal responsibility depends on our sense of both justice and protection of society. Whether we label a cluster of characteristics “mental illness” or exclude certain categories rests on policy decisions . . . (p 117).

Even if a given diagnosis is accepted by the court as qualifying as a “disease of the mind,” it remains to be shown how that disease might affect moral judgment in the case at hand. Under the M’Naghten test, “wrongfulness” has proved to be an ambiguous term. Under the most narrow reading, it is sufficient to demonstrate that the accused was aware that the act was illegal. In jurisdictions where this “illegality standard” holds, awareness of unlawfulness (whether or not the actor believed the act to have been morally justified) is sufficient to negate the insanity defense.

A second meaning which has been attached to the term, referred to as the “objective moral standard,” is that the issue is whether or not the actor was aware that the act would be held as wrong by society—regardless of the actor’s personal belief.

Clinical experience with sociopathic individuals suggests that they are acutely aware of both of these issues. However, a
third meaning of wrongfulness, called the “subjective moral standard,” is based on whether or not the accused, as a result of mental illness, believed he was morally justified in acting as he did.

If ASPD were accepted as falling within the legal parameters of mental illness, this would raise a troublesome point. It could certainly be argued that the life histories of many sociopathic individuals provide behavioral evidence that they either operate on moral codes markedly different from society’s or have a fundamentally impaired understanding of morality; it is precisely that point that provokes moral condemnation. It is not so much that the sociopath lacks intellectual understanding of conventional morality as that he lacks the motivation and social judgement to conform. However, court decisions emphasize that this subjective standard is based on more than simply following strongly held convictions or holding the belief that one is above the law; the implication is that the belief must be irrational.

A related issue involves the meaning of “knowingness” in the legal setting. Under M’Naghten, this is generally considered to refer only to intellectual appreciation, although before at least to the mid-1960s, many courts actually favored a broader interpretation, comparable to the use of the term “appreciate” in the cognitive arm of the ALI test (i.e., that “the accused is able to evaluate his conduct in terms of its actual impact upon himself and others and . . . is able to appreciate the total setting in which he is acting”) (p 49). As in the discussion above, as the legal interpretation moves from a narrower, more intellectual standard to a more inclusive one requiring emotional appreciation of the consequences of the act, the sociopath’s judgmental defects would seem to be more salient and might be more legitimized if biological factors were seen as directly causative.

**Mitigation**

The insanity defense is, of course, not the only way in which mental state can influence punishment. Depending on jurisdiction, evidence that a specific culpable mental state (such as purposefulness or knowingness) was negated by presence of a mental illness may be presented; in such cases, the defendant may still be convicted of a lesser included offense (e.g., manslaughter instead of murder). In cases where the mental disorder relates primarily to antisocial behavior (such as ASPD), this consideration rarely arises.

Genetic factors could, on the other hand, be considered in mitigation after guilt has been assessed. For example, if predisposing genetic factors were found to be relevant, it might be argued that the concept of “diminished responsibility” should be applied; that is, while the defendant had a culpable state of mind, the mental disorder nonetheless contributed to the defendant’s decision to commit the crime. Similarly, evidence supporting genetic influence on criminal behavior might be used in mitigation of sentence, for example in deciding whether or not to impose the death penalty.

Given the above discussion of causation, such arguments may be more appealing than those advanced for the in-
Behavioral Genetics

sanity defense, since genetic liability would be seen as only one of many interacting factors. Furthermore, mitigation need not require identification of specific genetic factors or markers; given current knowledge of the heritability of antisocial behavior, presence of family history, for example, might be sufficient.

But consideration of genetic liability factors could cut both ways. There is considerable evidence to suggest that such individuals are less likely to learn from experience and more likely to commit further crimes. Thus, from the standpoints of individual deterrence and protection of society, such evidence might justify punishing such individuals more, rather than less, harshly. Furthermore, the observation that such individuals functionally have a poorer grasp of moral principles and exhibit an apparent lack of remorse for their transgressions might be seen as aggravating, rather than mitigating, factors. Indeed, there is infamous testimony (running far in advance of what is scientifically justifiable) as to lack of remorse and likelihood of reoffense addressing just this point.76, 77

Conclusion

Forensic psychiatry and psychology occupy the interface between two disciplines that differ tremendously in their organizing philosophies, approaches to problem-solving, and roles in society. The forensic mental health professional is asked to bring to bear his or her knowledge of psychopathology and human behavior on questions formulated in legal terms, for legal rather than medical purposes.78 Thus, while advances in psychiatric knowledge carry with them the potential for change in the way such questions are analyzed, the answers must ultimately address social and legal, rather than medical, ends, and these objectives may not change even when their scientific underpinnings do. The growing attention to genetic influences on complex, long-standing patterns of behavior such as alcoholism or criminality may radically alter clinicians’ understanding of how such behaviors arise and are maintained, but should not necessarily modify society’s views of whether and to what extent individuals should be held morally responsible for their acts.

In part this is because genetic reductionism is not only overly simplistic in terms of its explanatory power but, by emphasizing deterministic factors, fails to capture important moral aspects of behavior. Such an approach ignores the multiplicity of behavioral causes operating and interacting at many different levels and, by minimizing the individual’s autonomy, risks devaluing his or her status as a citizen and human being—and by extension, the status of all citizens.

New techniques in genetic research, by characterizing important genetic contributors, are likely to deepen our understanding of the developmental process leading to repetitive antisocial behavior; ultimately such understanding may lead to tremendous advances in prevention and treatment of these behavioral problems. However, on the level of assigning moral responsibility, deeper understanding of cause should not necessarily alter our judgment of blameworthiness.
Acknowledgments

The author thanks Jules B. Gerard, JD and Sean H. Yutzy, MD for their comments on an earlier version of this article.

References

28. Raine A, Brennan P, Mednick SA: Birth complications combined with early maternal rejection at age 1 year predispose to violent...
Behavioral Genetics

crime at age 18 years. Arch Gen Psychiatry 51:984–8, 1994
47. U.S. v. Brunner, 471 F.2d 969 (DC Cir 1972)
64. Carter v. U.S., 252 F.2d 608 (DC Cir 1957)
66. M’Naghten’s Case, 8 Eng Rep 718 (1843)