



The Prediction and Explanation of Criminal Violence

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Scientific explanations of behavior involve a specification of both antecedent events or initial conditions and general laws that describe how each situation causes the succeeding one. Thus, laws acting on the current state of a system endlessly produce new states (Simon, 1992). To logical positivists, explanations are deductions about particular phenomena made from universal laws. Thus, in a formal sense, predictions and explanations differ only in that, in the former, future events instead of past events are at issue (cf. Blackburn, 1993).

However, prediction can also be based purely upon induction; in this case, prediction does not entail explanation. For example, we can predict on inductive grounds that the sun will rise in the morning without knowing anything about the celestial mechanics that can explain its apparent rise. Similarly, we can predict on inductive grounds that someone who has committed many assaults on persons will commit another (Walker, Hammond, & Steer, 1967), but not be able to explain why. Explanations, therefore, involve specifying a causal mechanism, whereas predictions may be based on pure induction. Intellectually satisfying explanations must involve a causal mechanism that can be imagined to produce the effect. Theoretical models are explanations of this kind in which the initial conditions and general laws are quantified.

An illuminating discussion of these issues has been provided by Einhorn and Hogarth (1986), who argue that scientists and lay people use similar systematic rules for assessing the causal status of variables. A variable is likely to be perceived as the cause of some effect to the degree to which it (a) stands out as a difference among the variety of other potential causes, (b) covaries with the effect, (c) precedes the effect, (d) is contiguous with the effect in time and space, (e) is similar to the effect in terms of duration, magnitude, and physical characteristics, (f) can be linked to the effect by a causal chain or mechanical

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model, and (g) is more plausible than rival causal candidates. A causal explanation, therefore, involves much more than simple induction (covariation).

A familiar illustration is provided by horse racing. Bookmakers establish the odds that a particular horse will win a given race from its past performance and the past performance of rival horses in the particular race in question.¹ Such laying of odds, similar in principle to the activities of actuaries, is thus based on induction, and is quite different from a prediction based upon a scientific explanation.

A scientific prediction of the outcome of a horse race in terms of Einhorn and Hogarth's scheme involves causal mechanisms. Many real or potential causes, however, provide only background. Those phenomena that are at a physical level, level of abstraction, or temporal order inappropriate to the question that is posed, such as variables having to do with quarks, the evolution of horses, the history of race tracks, and so on, will not be considered causal candidates, although they in principle form part of extended causal chains leading to a particular horse winning a particular race.

A satisfactory scientific explanation would likely focus on the physical characteristics of the horses (such as stride length and aerobic capacity) and training methods, not to mention jockey variables. Obviously, a prediction based upon a scientific explanation is far more difficult than one based upon induction. Nevertheless, one can view the previous winning record of a horse as a proxy for the combined effect of all the variables causally related to the horse's disposition to win races.

Research on the prediction of violent behavior is similar to the activities of bookmakers or insurance actuaries: A group of at-risk individuals are followed, and it is determined which of a variety of likely candidate variables is related to the likelihood with which a person will commit a new violent offense. This paper will discuss what may be learned about the explanation of violent crime from the research of my colleagues from the Oak Ridge Division of the Penetanguishene Mental Health Centre, in particular, Grant Harris and Marnie Rice, and I on its prediction among mentally disordered and non-mentally disordered male offenders.

Prediction

Our efforts to predict violent behavior have proceeded along two lines. The first has used clinical judgment as the predictor. In agreement with a large body of evidence on the prediction of a variety of phenomena, clinical judgment has proven to be a rather poor predictor of future violence (Quinsey & Maguire, 1986). This inaccuracy is a result of (a) low inter-clinician agreement about the dangerousness of particular individuals (Quinsey & Ambtman, 1979; Quinsey & Cyr, 1986; Quinsey & Maguire, 1983), (b) disagreement among clinicians about the dangerousness of populations of offenders, as reflected by their estimates of the base rate or proportion of dangerous persons in a population

¹ Using a similar idea, the Elo method of measuring chess skill is based on wins, losses, and draws in tournament play. It is a very sophisticated system with excellent psychometric and predictive properties (Holding, 1985).

(Quinsey, 1981), (c) reliance on highly salient information that has relatively little predictive value, such as history of institutional violence (Quinsey, 1979; Quinsey & Ambtman, 1979; Quinsey & Maguire, 1986), and (d) reliance on certain variables, such as a diagnosis of schizophrenia or degree of victim injury, as predictors of violence that are in fact related to lower, as opposed to higher, risk levels among serious offenders (Quinsey, 1979; Rice, Harris, Lang, & Bell, 1990).

Although unaided clinical judgment is not an efficient predictor of violence, clinical judgments themselves can be well predicted from simpler linear models (Quinsey & Ambtman, 1979; Quinsey & Maguire, 1986). Fortunately, the unreliability of clinical predictions of dangerousness can be substantially ameliorated by averaging independent appraisals of different clinicians. Increasing the validity of these appraisals, however, requires the use of an expert system or actuarial aid.

The second, and more practically profitable, line of research has examined the accuracy of actuarial prediction. Harris, Rice, and Quinsey (1993) followed 618 male offenders who had been treated in the maximum-security Oak Ridge psychiatric facility or briefly assessed there prior to imprisonment. Over an average time at risk of 81.5 months, 31% of these men committed a new violent (including sexual) offense against persons.

Multivariate statistical procedures were used to select 12 variables for use in a prediction instrument. In descending order of the size of their relationship to violent recidivism, the 12 variables were: the Psychopathy Checklist or PCL-R, a 20-item checklist scored from file information that measures characteristics such as lack of empathy, proneness to boredom, impulsivity, and irresponsibility (Hare, 1991); elementary school maladjustment; age at index offense (older offenders less likely to commit a violent offense); DSM-III diagnosis of personality disorder; separated from parents under age 16; failure on prior conditional release; criminal history of property offenses; never married or lived common-law; DSM-III diagnosis of schizophrenia (schizophrenics less likely to commit a violent offense); victim injury in index offense (less likely to commit a violent offense); history of alcohol abuse; and male victim in index offense.

Following Nuffield (1982), the violent recidivism rate for each value or range of values for each variable was determined. For every difference of more than 5% from the mean overall violent recidivism rate, a weighting of plus or minus one was added, depending upon whether the value was associated with an increase or a decrease in recidivism from the mean. Each subject could then be scored on each variable, and the scores could be added to form a single risk score for each offender.

As can be seen in Figure 1, there was a linear relationship between risk score and likelihood of violent recidivism. The correlation between violent recidivism and risk score was .44. Choosing the 80th percentile of risk scores as a cutoff yielded 74% correct classification; the proportion of violent recidivists correctly identified (sensitivity) was .41 and specificity, the proportion of successes correctly identified, was .88.

These results show that dangerousness, the disposition to commit violent offenses is linearly related to risk score. Similarly, Quinsey, Rice, and Harris

(in press) have found that sexual recidivism among child molesters and rapists is also linearly related to the actuarial risk scores of an instrument designed for sex offenders. Such linear relationships can be used in determining the amount of supervision and/or intervention resources required to reduce the likelihood of violent recidivism of an individual offender to an acceptable level.

There are a variety of factors that determine the precision with which a probability of violent recidivism can be assigned to an offender using the above system. These include the degree of resemblance of the population from which the new case is sampled to the population upon which the instrument was developed, the accuracy with which the variables in the instrument are measured, and so on. However, for our present purposes, the relevant point is that the variables included in the instrument predict future violent offending. What, if anything, does this predictive relationship mean for an explanatory theory of violent offending?

Before proceeding to discuss the theoretical implications of these predictive findings, it may be useful to consider exactly what is being predicted. Obviously, not the prediction of individual violent events (i.e., one cannot make statements such as patient X will assault person Y on October 10th, 1994). Rather, this research allows one to assess a male offender's propensity to commit a violent offense (cf. Blackburn, 1993), the likelihood with which he will commit at least one violent offense in a given period of time at risk. Thus, the usefulness of the predictive instrument depends upon differences in dangerousness among persons. Clearly, these variations are substantial.

But how much of this predictive efficiency is simply due to induction or, as a lawyer might put it, similar fact evidence? Strictly speaking, in terms of a past history of violent crime, almost none. Two of the predictors refer to the index offense, victim injury, and female victim, and both are *negatively* related to violent recidivism. From the PCL-R, only criminal versatility (variety of offending), juvenile delinquency, and early behavior problems can be said to be clearly, if only partially, related to violent offending. Similarly, a DSM-III diagnosis of antisocial personality disorder, the weakest predictor, contains 21 substantive criteria of which only two refer specifically to violence.

There would be a stronger element of induction if one were to predict general (both violent and nonviolent) recidivism because a number of predictors refer to criminal history. In addition to those mentioned in the above paragraph, these are: failure on prior conditional release, property crimes, and, from the PCL-R, juvenile delinquency and revocation of conditional release. Four of the antisocial personality diagnosis criteria refer to criminal behaviors. Even so, the ability of the instrument to predict violent recidivism is greater than that based purely on criminal history: The best single predictor of violent recidivism in the actuarial instrument; the PCL-R, for example, improves the prediction of violent recidivism even after the influence of criminal history (previous violent offense, number of admissions to corrections, previous criminal convictions, and convictions history) has been statistically removed (Harris, Rice, & Cormier, 1991).

As mentioned above, the accuracy of the assessment of dangerousness achieved with this instrument is dependent upon the assessed person being a serious offender. The type of followup research leading to the development of

the prediction instrument is ill suited to discovering variables related to a person becoming or not becoming a violent offender in the first place. This question is best dealt with in longitudinal studies of particular birth cohorts. In the context of follow-up studies of serious offenders, variables leading to the initiation of a violent criminal career can only form part of the causal background. To illustrate, Stattin and Klackenber-Larsson (1993) found that language development at 6, 18, and 24 months was negatively correlated with adult criminality among males. It is possible, however, that this relationship might not hold among a group of offenders, because all might be expected to have had poor verbal ability.

The nature of the predictors provides some information that can inform a theory of dangerousness. All of the predictors are based upon information available at the time of the index or admission offense. This means that the amount of predictive variance that can be accounted for by institutional treatments, post-release supervision, and situational factors is limited by the degree of accuracy of the predictive instrument. This limitation, however, only pertains to the treatment and supervision practices in effect at the time of the follow-up research. To the extent that more powerful or better implemented interventions are employed, the predictive accuracy of static or historical predictors declines. For example, Paul and Lentz (1977) demonstrated that no historical or patient variables were related to the outcome of a social learning program for chronic schizophrenics because of the extremely high success rate associated with their program.

Surprisingly, some of the predictors of violent recidivism in the Oak Ridge followup studies pertain to childhood and teenage adjustment: separation from parents before age 16, elementary school maladjustment, and, from the PCL-R, early behavior problems and juvenile delinquency. This result is surprising, because these childhood variables are good predictors among adults who are already serious offenders.

Although the predictive ability of childhood antisocial history variables is surprising in a study of serious adult offenders, it is highly reminiscent of early work on the development of psychopathy. Robins (1966) described the characteristics of adult "sociopaths" identified in a follow-up study of persons referred to a child guidance clinic.

“. . . almost every sociopath had a poor work history, had been financially dependent on social agencies or relatives, and had marital problems. Three-quarters of them had multiple arrests leading to prison terms. They drank excessively, were impulsive, sexually promiscuous, had been vagrant, were belligerent, delinquent in paying their debts, and socially isolated. . . . The symptoms that best distinguished them from all other diagnostic groups were their poor marital histories, their impulsiveness, vagrancy, and use of aliases. . . . Sociopaths had a higher rate of injuries and deaths by violence than had other subjects. Children resulting from their unions had a high rate of problem behavior and failure to graduate from high school.”
(p. 296)

Robins found that the best predictor of sociopathic personality was the variety, number of episodes, and seriousness of childhood antisocial behaviors. Future sociopathy was also strongly related to having an alcoholic or sociopathic father, but not to social class, the stigma of childhood mental health clinic involvement, or parental divorce, rejection, or disciplinary practices. Disciplinary practices were, however, related to adult criminality among non-sociopaths. These findings are in accord with a very large literature linking adulthood criminality to childhood delinquency (e.g., Loeber & Stouthamer-Loeber, 1987). Other aspects of these results have been confirmed as well; in particular, Frick et al. (1992) found in a study of clinic-referred boys that paternal antisocial personality was strongly associated with a diagnosis of conduct disorder in the sons, but that maternal disciplinary practices were not.

Harris, Rice, and Quinsey (1994) have presented evidence that psychopathy as measured by the PCL-R is a taxon (i.e., a categorical variable like biological sex). In essence, this means that higher scores on the PCL-R do not indicate increasing amounts of the trait, but rather a greater likelihood of taxon membership. The PCL-R items most closely correlated with the PCL-R total and membership in the psychopathy taxon were: proneness to boredom, manipulativeness, callousness, parasitic lifestyle, early behavior problems, lack of realistic goals, impulsiveness, and irresponsibility.

Although PCL-R items provided evidence of a taxon, it is important to note that adult criminal history variables in themselves provided no evidence of a taxon. Another set of non-PCL-R variables, however, did provide evidence of a taxon and could predict which subjects would be assigned to the psychopathy taxon. These variables pertained to childhood history specifically: elementary school maladjustment, teen alcohol abuse, childhood aggression, suspended or expelled, arrested under age 16, separation from parents, parental alcoholism, and childhood behavior problems.

Many of the predictors of violent recidivism, therefore, appear to be indicators of an offender's membership in the psychopathy taxon. Harris, Rice, and Quinsey (1994) observed that about 65 of the offenders in their sample appeared to be prototypical psychopaths, in that they were identified as taxon members using each of the methods employed in that study. Many of these individuals were responsible for particularly heinous reoffenses (see Figure 1).

Of course, not all violent offenders are psychopaths and not all psychopathic children grow up to be serious violent offenders, but those who do become the most recidivistic. Among the clearest implications this work has for predictive accuracy is that different predictors are likely to be related to violent recidivism among non-psychopathic offenders who, of course, will have a lower incidence of violent recidivism. One such example has already been identified: Alcohol abuse is a predictor of violent recidivism only among non-psychopathic, mentally disordered offenders (Rice & Harris, 1992). For a further discussion of these issues, see Belmore and Quinsey (1994), Harris, Rice, and Quinsey (1994), and Stattin and Magnusson (1989).

Explanation

So far, this paper has associated the dangerousness of serious criminal offenders with a psychopathy category, the members of which manifest a stable

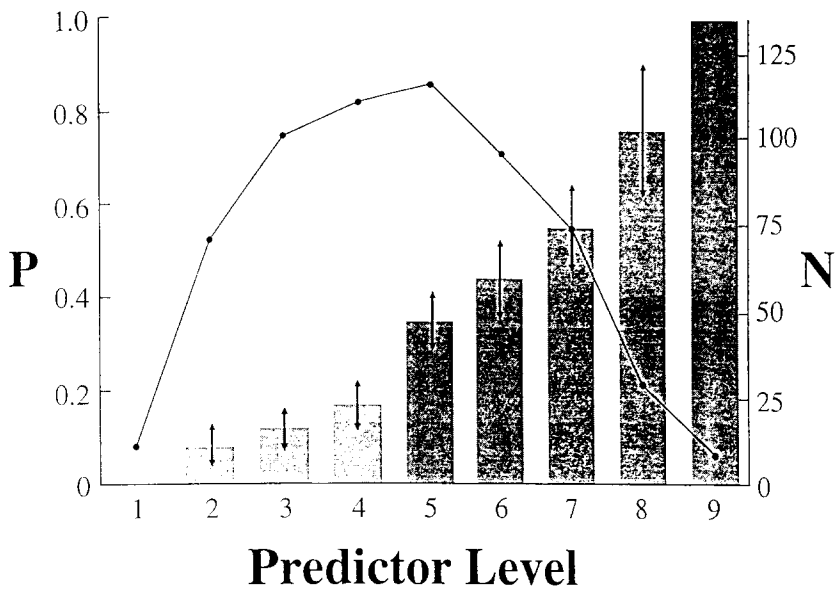


FIGURE 1. The performance of the statistical risk instrument. Bars indicate the probability of violent recidivism (P) for subjects at each of nine equal size steps. The solid line indicates the number of subjects (N) in each step. Arrows indicate the 95% confidence intervals for the violent recidivism rates for subjects in each step. Adapted from Harris, Rice, and Quinsey (1993).

constellation of predatory, exploitative, and impulsive characteristics. Can we provide a causal explanation of psychopathy and its relation to violent crime?

Broadly speaking, explanations of such categorical individual differences involve either pathological or nonpathological causes. In support of the former, Robins (1966) has argued that sociopathy, or psychopathy in the present context, is a psychiatric disease because "It occurs in children whose fathers have a high incidence of the disease and whose siblings and offspring also appear to have an elevated incidence. The symptoms follow a predictable course, beginning early in childhood with illegal behavior and school discipline problems and continuing into adulthood as illegal behavior, marital instability, social isolation, poor work history, and excessive drinking." (pp. 302-303)

However, not all patterns of behavior that are heritable and follow a predictable course are diseases or disorders. Wakefield (1992a, b) has usefully conceptualized a disorder as a harmful dysfunction. Thus, the concept of a disorder is at the interface of the given natural world and the constructed social world. In this view, a person is considered to have a disorder when a failure of the person's internal mechanisms to perform their natural function impinges harmfully on the person's well being as defined by social values and meanings.

In Wakefield's view, a condition is a mental disorder, therefore, if and only if:

- the condition causes some harm or deprivation of benefit to the person as judged by the standards of the person's culture (the value criterion), and
- the condition results from the inability of some mental mechanism to

perform its natural function, wherein a natural function is an effect that is part of the evolutionary explanation of the existence and structure of the mental mechanism (the explanatory criterion).

Daly & Wilson (1988) have captured part of this idea when they note that persons who act against their own inclusive fitness interests are likely to be considered mentally ill. Thus, for example, persons who murder their consanguineous kin are more likely to be perceived as mentally ill than those who murder victims not biologically related to them.

The alternative and nonpathological explanation for psychopathy is that it is an adaptation. Adaptations are characteristics of organisms that arose because of their natural or sexual selection benefits in ancestral environments. Thus, adaptations have increased the relative inclusive fitness of organisms that possessed them. Of course, not all characteristics of organisms are adaptations and there are sometimes difficulties in determining whether a given characteristic represents an adaptation or something else.

A nonpathological or adaptive interpretation of psychopathy starts with the observation that the characteristics associated with the taxon could easily be imagined to contribute to an individual's inclusive fitness under a wide variety of conditions, such as those in which life is "nasty, brutish and short"; obviously, such conditions recur in the historical record, and they undoubtedly occurred earlier as well.

Frank (1988) has developed an interesting evolutionary model, which predicts that the proportion of opportunists and cooperators in a population will stabilize at a value determined by the costs and accuracy of identifying opportunists. Because psychopathic characteristics are likely to be increasingly costly for an individual over time in stable social situations, psychopaths are likely to maximize their outcomes through geographic and social mobility (Harpending & Sobus, 1987). Non-opportunists are likely to do better in interacting with the same people over time. As Frank (1988, p. 237) puts it: ". . . people who love, who feel guilty when they cheat, vengeful when they are wronged, or envious when they get less than their full share will often behave in ways that reduce their material payoffs. But precisely because of this, they will also enjoy opportunities that would not be available to a purely opportunistic person."

Belsky, Steinberg, and Draper (1991) have argued that a reproductive strategy emphasizing mating effort as opposed to parental investment arises out of an early environment of unpredictable resources and social relationships, although they do not discount the possibility of genetic polymorphism or genetic induced differential susceptibility to a particular rearing environment. Belsky et al. believe that this reproductive strategy is triggered by a stressful early environment, in turn leading to insecure attachment to parents and subsequent behavior problems; followed by early puberty and precocious sexual behavior, and, in adulthood, unstable pair bonding and limited parental investment.

Is psychopathy then the result of a mental disorder or an adaptation? There are a variety of possible etiologies of this taxon, and each has implications for this question.

- (a) The behaviors are the direct effect of some gene or set of genes.
- (b) The capacity to exhibit psychopathy is present in everyone, but is only made manifest under certain prenatal, perinatal, or early childhood conditions.
 - (1) Environmental conditions (e.g., failure of attachment, poor supervision).
 - (2) Biological conditions (e.g., brain damage incurred during birth).
- (c) The individual behaviors are the consequence of some general rule learned in childhood.
- (d) The behaviors in the taxon are each learned independently during childhood, and environmental characteristics are responsible for their tight covariation (cf. Meehl, 1992).

It is, of course, necessary that psychopathy be genetically related for it to be considered an adaptation. This does not imply, however, that it would necessarily be found to be heritable in behavior genetic analyses, because such analyses determine the amount of variation among persons that is attributable to genetic influence. If there is no variation among persons in a genetically determined trait (as, for example, in the number of eyes people have), a behavior genetic analysis will show zero heritability. Thus, if psychopathy was a facultative adaptation (i.e., a genetically controlled response to environmental conditions) as in alternative *b1* above, there would be no genetic variance, and psychopathy would not appear to be heritable in behavior genetic studies.

A definitive choice among the etiological alternatives is at present impossible, not least for the reason that there have been no behavior genetic studies of psychopathy per se, and very few of violent crime (Mednick, Gabrielli, & Hutchings, 1983). With respect to antisocial behavior more generally, there are at present data supporting genetic (e.g., Mednick et al., 1983), environmental (Patterson, 1982), and an interaction of genetic and environmental contributions (as in alternative (b), Cadoret, Cain, & Crowe, 1983).

The taxonicity of psychopathy, the early onset of antisocial behaviors and their persistence (e.g., Patterson, 1992), the evidence for a genetic influence on criminality, the possibility that psychopathic characteristics may have been adaptive in our environment of evolutionary adaptation, the failure to find disciplinary practices related to the subsequent behavior of "sociopaths," the predictability of violent and general recidivism among psychopaths, and the failure to demonstrate treatment-induced reductions in psychopaths' rates of general and violent recidivism (Harris & Rice, in press; Ogloff, Wong, & Greenwood, 1990) collectively encourage therapeutic pessimism and belief in a genetic diathesis.

We must, however, be mindful of the "deterministic fallacy," the belief that characteristics produced by evolution or genetically related are unamenable to change, in particular, environmental change. The familiar example of phenylketonuria illustrates the falsity of this belief. Phenylketonuria is a genetic disorder that causes severe mental retardation, but that can be prevented by a phenylalanine-restricted diet.

The speed with which scientific knowledge about psychopathy has accumulated in recent years raises the possibility that future interventions to reduce

their likelihood of violent recidivism could be based upon a causal explanation of psychopathy. The implementation of a successful intervention would destroy the validity of the actuarial predictive scheme and, with it, the criminal destiny of psychopaths. A successful explanation, therefore, can fundamentally alter predictions founded in the natural course of events.

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