Jan Volavka,¹ M.D., Ph.D.; Dan Martell,² Ph.D.; and Antonio Convit,³ M.D.

Psychobiology of the Violent Offender

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ABSTRACT: The antecedents of violent crime may include childhood victimization, head injuries, and alcohol and drug abuse. Neuropsychological and neuropsychiatric findings suggest temporal and frontal lobe dysfunctions in violent offenders; these dysfunctions appear to be more pronounced in the dominant hemisphere. Recent studies implicate disturbances of central serotonergic functions in impulsive homicide and arson. These results provide an adequate rationale for larger interdisciplinary studies using neurochemical, neuropsychiatric/neuropsychological, and psychosocial methods on the same subjects.

KEYWORDS: psychiatry, violence, neurobiology, human behavior

It has been ten years since the biology of violence was reviewed in a chapter coauthored by one of the authors of this paper [I]. Much has happened in this field since that time. These new developments, important in themselves, have also led to new insights and permitted reevaluation of the preexisting literature. We have therefore decided to provide an updated review. This review has two main purposes: to inform the reader of recent developments and to address critically what we see as main obstacles to progress in the field.

It is becoming apparent that biological factors may be very important for understanding the etiology and prevention of violent crime. Biological factors have multiple links to psychological and social forces impinging on the individual. There is an increasing realization that violent criminal behavior results from *interactions* among these multiple factors. However, systematic studies of such interactions are not yet common. So far, the literature on the neurobiology of violent crime has been scattered across many fields. A specialized review focusing on electroencephalography (EEG) and aggression has recently appeared elsewhere [2]. In the present report, we will provide a broader summary of the literature.

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¹Chief, Clinical Research Division, Nathan S. Kline Institute for Psychiatric Research, Orangeburg, NY, and professor, Department of Psychiatry, New York University Medical Center, New York, NY.

²Forensic research scientist, Kirby Forensic Psychiatric Center, Nathan S. Kline Institute for Psychiatric Research, Orangeburg, NY and Clinical Instructor, Department of Psychiatry, New York University Medical Center, New York, NY.

³Director of research, Kirby Forensic Psychiatric Center, Nathan S. Kline Institute for Psychiatric Research, Orangeburg, NY, and assistant professor, Department of Psychiatry, New York University Medical Center, New York, NY.

Antecedents of Violent Crime

Childhood Victimization

The hypothesis that abused children will become tomorrow's murderers has been with us for at least 25 years [3]. A history of childhood victimization was given by children who later killed [4] and by prisoners sentenced to death [5].

Similar data relating parental brutality to violent behavior in their offspring have been reported by other workers [6-10]. A large prospective cohort study [11] has demonstrated that males who had been abused and neglected as children had a somewhat higher frequency of arrests for violent offenses as adults than nonabused males.

Widom [12] has reviewed the literature on the intergenerational transmission of violence. It appears that while childhood victimization may contribute to the later development of criminal violence, other factors which add to or interact with victimization may also be involved. Being victimized as a child or just living in an abusive family may contribute to later development of violent behavior through a learning experience (modeling), or through brain injuries which result in neurological damage to the victimized child. However, the majority of abused children do not become violent criminals.

Perhaps the stressful experience of growing up in an abusive family or being victimized can be overcome without the development of later violence by persons with an intact central nervous system, but not by those who are neurologically impaired [13]. We must consider that abused children who have had brain injury develop an especially elevated predisposition for violent behavior. Studying these issues requires interactive models of causation of criminal violence.

An additional difficulty in interpreting the literature on childhood victimization is the possibility that abusive parents may suffer from psychiatric disorders that may be transmitted to the abused children. Some of these disorders may predispose the subject to violence. This genetically transmitted predisposition may explain a part of the increased propensity for violence in the abused children. This possibility has not been examined empirically.

Deviant Rearing Environment

Most studies associating childhood victimization with adult crime fail to consider the possibility that families may have many problems in addition to child abuse. Other factors such as parental substance abuse and psychiatric hospitalizations, or otherwise poor family functioning may account for the relationship seen between child abuse and adult crime. Ratings on a scale measuring the deviancy of the family rearing environment were significantly higher for the assaultive than for the nonassaultive psychiatric inpatients [14, 15]. It may be prudent for those studying the relationship between childhood and adult violence to also consider the potential role of other family problems in this relationship.

One of the more important areas of research developed over the past decade is the role of serotonin in human aggression. We will discuss this research in some detail later in this review. At this point, it is appropriate to mention recent work in nonhuman primates linking a deviant rearing environment to enduring serotonin abnormalities [16]. Is it possible that an abnormality of the serotonin system is involved in the mechanism whereby a deviant rearing environment confers a propensity to adult human violence?

Medical and Neuropyschiatric History

Injuries and illnesses have been linked with later development of criminal violence. Increased use of general medical services was observed to predate the onset of delinquent behavior [17]. Medical and neurological problems such as heart disease, epilepsy, and severe head injury were reported in 36% of killers in Iceland [18]. A history of head injury was reported in most prisoners [19]. Head injury may of course be a consequence rather than an antecedent of criminal violence: victims may fight back, injuring the perpetrator. In addition, perpetrators may have become victims of other crimes during which they have sustained head injuries. In a subsample of epileptic prisoners [19], 47% of head injuries which were related to the development of epilepsy (see below) were classified as "interpersonal"—presumably sustained in fights and assaults in which the person's role (aggressor or victim) remained unreported [19]. Lewis et al. [5] were able to show that, in 12 of their 14 cases (of juveniles on death row), head injuries had occurred at age 12 or earlier. Some of the injuries of the central nervous system may be prenatal or perinatal, as was suggested by a retrospective study of aggressive children [20]. Prospective studies are needed to replicate these findings.

A history of seizures was reported in homicide offenders [4,21]. A large survey showed that the prevalence of epilepsy among British male prisoners was almost twice as high as in the general population [22]. The prevalence of epilepsy among men entering the Illinois prison system was four times higher than that among comparable nonprisoners [19]. However, virtually no crimes are directly attributable to an ictal or postictal confusional state [22,23]. Furthermore, epileptic prisoners are not more likely to have committed violent crimes than nonepileptic ones [19,24]. There is no evidence that epileptics in general have elevated crime rates. Nevertheless, there may be special subtypes of epilepsy that are linked to violent crime, or perhaps even to specific subtypes of violent crime. If such relationships exist, they would be obscured rather than elucidated by studies of unselected large samples. For example, it is conceivable that the relationship between epilepsy and violent crime is only apparent in special populations, such as homicidal juveniles [4]. Alternatively, it is possible that it is specifically temporal lobe epilepsy which is linked to violence [4]. The ongoing discussion of that link is complicated by variations in the definition of the temporal lobe (psychomotor) symptoms. The definition used by Lewis and her group [25] may be too broad [19,26-28].

The putative specific link between temporal lobe epilepsy and violence was not supported by Gunn and Bonn [29], who reported that it was generalized idiopathic epilepsy rather than temporal lobe epilepsy (psychomotor) which was associated with convictions for violent crime. Relationships between seizure disorders and violence were discussed in a companion review of EEG findings and aggression [2].

Alcohol

Drinking has been associated with violent crime [30-32], but the nature of this association remains unclear. The percentage of prison inmates who drank daily prior to their offense was three times as high as that rate in the general population [33], but this relationship was not specific for violent offenses. Another study [34] examined the use of alcohol and drugs at the time of the offense, comparing homicide and nonviolent offenders. More homicide than nonviolent offenders used alcohol and drugs at the time of the offenders used alcohol and drugs at the time of the offense set of the offense [34].

Clinical observations suggest that alcohol may have qualitatively different and specific effects on mood in violent than in nonviolent individuals: dysphoric effects may prevail over relaxing, euphoric effects [35,36]. These clinical observations were not dose-controlled, however, so it is possible that the violent individuals simply drank more and that this dose discrepancy accounted for the differing effects on mood. On the other hand, it is also possible that organic brain dysfunction present in some violent individuals distorts the usual dose-response curve for alcohol. Certain cases of pathological intoxication may represent an extreme example of this effect. Brain damage reduces tolerance to alcohol [37]. Thus, it is possible that an interaction between acute alcohol effects and

underlying brain dysfunction accounts for a portion of the variance in criminal violence. Such an interaction has been proposed [35] but not tested.

Abram [38] studied the effect of co-occurring antisocial personality, alcoholism, and drug disorders on criminal careers. She found that it was drinking during or immediately prior to the crime (rather than the lifetime prevalence of alcoholism per se) that was associated with criminal behavior. Furthermore, she pointed out that studies demonstrating an alcohol-crime relationship have not controlled for associated psychopathology: the observed link may be an artifact of the association between alcohol and a third variable such as antisocial personality or drug abuse.

Drug Abuse

There is a clear association between drug abuse and crime in general [39-41]. A large percentage of drug users have been involved in non-drug-related criminal acts [42, 43]. Addiction patterns covary with criminality: involvement in crime is higher during periods of active addiction than during periods of infrequent use or abstinence [44]. The majority (53 to 79%) of the men arrested in twelve major cities in the United States tested positive for illicit drugs, the most frequently detected being marijuana, cocaine, heroin, phencyclidine, and amphetamines [41].

The specific relationship between drug use and violent crime is less well documented. In a large multistate prison survey, Innes [45] noted that 35% of state prison inmates reported being under the influence of drugs at the time of their offense: 33% for violent offenders, 39% for property offenders, and 43% of drug offenders. Daily drug use in the month preceding their offense was elevated for all groups.

Abram [38] found a relationship between drug disorders and property crimes independent of antisocial personality disorder. As in the case of lifetime alcoholism measures, lifetime drug disorders were not related to violent crime. She explained the discrepancy with other reports by hypothesizing that the effect of drugs on violent crime is due to drug use around the time of the crime, and that this effect may be confounded by antisocial personality disorder, which was not controlled for in previous studies.

Violent behavior was reported in abusers of amphetamine [46], phencyclidine [47], and cocaine [48]. However, these observations were not controlled for the potential effects of antisocial personality disorder; this disorder is known to be associated with drug abuse and violent acts.

Psychosis

Careful interviews of violent offenders frequently reveal a history of symptoms suggestive of psychosis, including depression with or without suicidal ideation, delusional ideas of reference or persecution, or hallucinations [4,5,13]. These symptoms may be subthreshold in the sense that they are insufficient for a rigorous diagnosis of a distinct disorder. It is also possible that homicide offenders simply may not differ on psychiatric diagnosis from nonviolent offenders or community controls [49]. Reports of the psychiatric assessment of such offenders give widely divergent results [50-54]. The divergence may be explained by different (and frequently undisclosed) sampling methods and by the failure to use explicit diagnostic criteria.

Mood disorders may be implicated in violent criminal behavior, either directly or in combination with other factors [55]. Surveys of prisoner mental health suggest that lifetime and current prevalence of major depressive disorder may be elevated [56].⁴ Findings of

⁴Bean, J., "The Prevalence of Mental Illness Among Inmates in the Ohio Prison System," draft report, Ohio Department of Mental Health, 1987.

current depression may, however, represent a response to incarceration, since another survey focusing on newly arrived inmates failed to find the elevation of depressive symptoms [57].

Lifetime prevalence of schizophrenia in prisoners may be similar to that of the general population [57],⁴ or it may be somewhat elevated as reviewed by Jemelka et al. [58]. All these surveys included prisoners of all types (violent and nonviolent). Not surprisingly, the surveys found a very elevated prevalence of antisocial personality disorder and substance abuse or dependence in prisoners.

Antisocial Personality Disorder

This disorder is clearly related to crime, but its diagnostic criteria (DSM III-R) [59] rely heavily on lawbreaking behavior. Many offenders, by definition, fulfill these criteria and thus the use of this diagnosis in prisoners is not very helpful. Hare has been using the older term "psychopathy," which he rigorously redefined by a research checklist (Psychopathy Checklist, PCL) designed for use in criminal populations [60]. Using the PCL, Hare and his group determined that psychopaths commit a disproportionately large number of crimes (compared with other male criminals) [61]. Furthermore, the crimes of psychopaths are more violent than those of other criminals [62]. Psychopaths seldom commit crimes of passion, and their victims are most frequently strangers [63]. The PCL score predicted (with the effects of age and criminal variables controlled) the violation of conditional release [64]. There is some evidence suggesting that the effect of psychopathy on crime may be due to its interaction with other variables such as brain dysfunction [65], drug abuse [38], or intelligence [66]. Using an empirical measure of psychopathy, Heilbrun [66] determined that psychopathy was related to crime only in less intelligent prisoners. Thus, intelligence could be a variable which modulates the relationship between personality disorder and violent crime. In a follow-up paper [67], Heilbrun introduced additional modulating variables: cognitive control, empathy, and self-reinforcement. The interactions between these modulating variables and psychopathy yielded three models of violent crime. This very interesting heuristic work has not yet been replicated.

Neurobiologic Findings in Violent Offenders

Neuropsychiatric and Neuropsychological Findings

Diffuse (or Multisite) Brain Dysfunction

Most reports of diffuse brain dysfunction are based on empirical observations obtained as part of forensic assessments of criminal defendants. Results of *neurological examinations* are sometimes briefly mentioned [49], but it appears that such examinations are often done perfunctorily or not at all. However, one group performing detailed neurological examinations of juvenile homicide offenders described highly abnormal results [4,5]. The abnormalities were diverse, many of them consistent with consequences of head injuries reported by these prisoners. We have constructed a quantitative neurological examination scale [15] which showed high abnormality scores in assaultive psychiatric inpatients. These patients had a 35% rate of conviction for violent crime [14]. Among the assaultive patients, the subgroup of *persistent* repetitive assaulters appeared more neurologically impaired than the assaultive patients who responded to treatment by stopping their assaults [15].

Neuropsychological tests in violent offenders suggest multiple functional impairments; unfortunately there are few studies in this area. The Halstead-Reitan and Luria-Nebraska batteries showed more abnormality in a violent than in a nonviolent offender group;

however, the groups were small and the differences were not significant [35]. Clear impairment on the Halstead-Reitan battery was demonstrated in an uncontrolled study of juveniles on death row [5]. A specially constructed neuropsychological test battery discriminated between violent and nonviolent offenders [68].

Intellectual impairment has been reported in violent juvenile offenders [5]; however, test intelligence in adult violent offenders does not appear to be grossly impaired [35]. Violent offenders were reported to have lower intelligence quotient (IQ) scores than nonviolent offenders [68, 69]. However, full-scale IQ scores in a sample of 243 indicted homicide offenders ranged from 53 to 140, with an average of 96 [70], which approximates the distribution in the normal population. As mentioned above, some studies have suggested that intelligence may modulate the relationship between personality disorder and violence [66, 67].

Localized Brain Dysfunction

Temporal Lobes—Links between temporal lobe dysfunction and violence have been reported since the early days of clinical EEG [21,71]; these reports represent the first modern attempts at a biological explanation of human aggression. Electrical discharges in the deep structures of the temporal lobes were demonstrably associated with violent outbursts [72]. These valuable observations in a relatively small group of patients have enhanced our understanding of certain mechanisms of aggression in humans [72]. However, the contribution of these mechanisms to violent crime in general is probably quite small. The literature on temporal lobe epilepsy and violence has been discussed above in the section on medical and neuropsychiatric history and in a specialized review focusing on EEG [2].

Frontal Lobes—Frontal lobe functioning involves the "executive control" and regulation of behavior [73,74]. However, the current state of knowledge on the role of the frontal cortex in violent behavior is limited. Several authors offer theoretical perspectives on the role of the frontal lobes in the control, regulation, inhibition, or suppression of violent impulses [75–77]. There is little scientific evidence, however, to support or refute their formulations.

Volkow and Tancredi [78] employed positron emission tomography (PET) to examine the brain function of four violent psychiatric inpatients. Dysfunction of the frontal cortex was observed in two of the four patients, and temporal lobe dysfunction was present in all of them. Pontius and Yudowitz [79] employed a clinical narrative technique and a neuropsychological test (Trail Making, Part B) to study frontal lobe dysfunction in 30 young adult criminals. In their view, a subgroup (33%) of these men demonstrated "specifically immature action behavior indicative of (frontal lobe system) dysfunctioning." This conclusion, we feel, was more specific than warranted by the data. In another experiment, Pontius [80] studied eight white males who had committed violent acts (murder, attempted murder, or rape), allegedly evoked by specific stimuli (certain words, specific objects, and so forth) postulated to be iodiosyncratic triggers of aggression. She hypothesized a "seizure-like imbalance between frontal lobe and limbic systems," although no empirical measures of frontal lobe function were employed.

Yeudall [81] studied 25 aggressive psychopaths and 25 depressed criminal patients to examine the lateralization of neuropsychological impairments in psychiatric and criminal disorders. His findings suggested that the neuropsychological impairments of both groups were localized in the anterior regions of the brain, with dysfunction in the aggressive psychopaths more frequently lateralized to the dominant hemisphere. More recently, Heinrichs [82] reported that focal frontal cerebral lesions (confirmed through computerized tomography) were associated with violence in chronic neuropsychiatric patients.

Additional evidence of frontal dysfunction in habitually aggressive patients comes from EEG studies. Williams [83] has clearly demonstrated that persistent violent behavior was associated with EEG slowing over the frontal (and temporal) areas in a large sample of offenders. Frontocentral EEG slowing was also associated with habitual aggressivity in male adult drug abusers [84]. Gorenstein [65] provided data supporting a link between psychopathy and frontal lobe dysfunction. However, subsequent research [62,85] has failed to replicate this association.

Taken together, these studies suggest that frontal lobe dysfunction may play a role in violent behavior. However, the small sample sizes, lack of reliable and valid measures of frontal lobe functions, absent control or comparison groups, and confounded research designs limit the interpretation of these results.

Lateralized Hemispheric Dysfunction—Yeudall [81] reported that adult aggressive psychopaths had neuropsychologic impairments lateralized to the dominant hemisphere (predominantly anterior). However, neuropsychiatric impairment was predominantly lateralized to the nondominant hemisphere in the violent adolescents [86]. Tentative evidence for the lateralization of dysfunction to the left hemisphere was obtained by studies of dichotic listening in adult prison inmates [87]. The inmates were categorized (somewhat confusingly) as either murderers, violent offenders, or other offenders. The violent offenders (but not murderers) demonstrated a left ear advantage on one of the three dichotic listening tests; this can be intepreted as a left hemisphere dysfunction. Additional indirect support for a dysfunction of the dominant hemisphere comes from the observation that offenders tend to be left-handed more frequently than controls [88].

Taken together, these reports suggest that certain violent offenders may exhibit dysfunctions of the dominant hemisphere. This lateralization may depend on the offender's age. A summary of earlier literature on hemispheric dysfunction in antisocial behavior has been published [89].

Neurochemical Findings

Serotonin—Links between serotenergic transmission and aggression in animals have been known for many years [90]. In 1976, Asberg et al. [91] reported low levels of 5hydroxyindoleacetic acid (5-HIAA), a principal serotonine metabolite, in the cerebrospinal fluid (CSF) of depressed patients who had committed violent (but not nonviolent) suicide attempts. Psychological links between aggression turned inwards and outwards have been proposed at least since Freud. An important biological link between these two phenomena was discovered by Brown et al. [92], who reported that aggressiveness (measured by questionnaires) was significantly and negatively related to CSF levels of 5-HIAA. Brown's subjects were military men. Aggression against others was associated with a history of suicidal behavior [92]. This trivariate relationship between aggression, suicide, and low CSF 5-HIAA levels has been confirmed in another sample [93].

It is clear that the study of serotonergic function in the central nervous system has great potential value for research into violent crime. Unfortunately, the only available measure of the central serotonergic function—CSF 5-HIAA—requires a spinal tap. This procedure is routine and very safe, but the current constraints on research using prisoners as subjects make it virtually impossible to obtain CSF from violent offenders in the United States. Thus, much of the recent work in this area was done by a Finnish-American team (led by Virkkunen and Linnoila), using voluntarily consenting Finnish homicide offenders [93] and fire setters [94] as subjects.

The homicide offenders [93] were categorized as "impulsive" if the victim was unknown to the offender, if there was no or only minor provocation, and the attack did not represent an attempt to rob the victim. The "nonimpulsive" offenders knew the victim, and there was evidence of premeditation. The principal finding was that the impulsive offenders

had lower CSF 5-HIAA levels than the nonimpulsive ones. Furthermore, the recidivistic offenders had lower CSF 5-HIAA than those who committed only one violent crime. Finally, the impulsive offenders with a history of suicide attempts had lower CSF 5-HIAA than the other offenders.

The fire setters in the second study [94] were all deemed impulsive, but no criteria for impulsivity in these offenders were provided (except that the arson was not committed for profit). The principal finding of this study was that the fire setters had lower CSF 5-HIAA levels than either of the two control groups: violent offenders and normal non-offenders. The violent offender control group was drawn from a sample reported earlier [93]; it is not clear how many of these controls were deemed impulsive. Furthermore, the majority of the fire setters showed an abnormally low blood glucose nadir after an oral glucose challenge.

The subjects of these studies [93,94] were followed up after their release from prison to study recidivistic offending [95]. The blood glucose nadir after an oral glucose challenge and the CSF 5-HIAA (both measured before the subjects' release from prison) were related to recidivism. Linear discriminant analysis using these two measures as independent variables correctly classified 95% of the nonrecidivists, and 46% of the recidivists (6 of 13). This must be considered relatively good classification compared with other methods used to predict violent crime [96]. The results are particularly interesting since the discriminant function used only two variables, and both of them were selected *a priori* on a theoretical basis.

The findings of these three studies [93-95] must be interpreted with some caution. The numbers of subjects were relatively small, and the method of their selection somewhat unclear. Almost all the subjects were alcoholics committing their offenses under the influence of alcohol. It may not be possible to generalize the interesting biochemical findings to nonalcoholics or to offenses commited while sober.

Given these caveats, the results [93-95] suggest that serotonergic transmission may be impaired in certain violent offenders. This impairment may result in a reduction of impulse control. The serotonergic hypothesis is eminently testable; it provides a theoretical rationale for attempts at prevention and treatment of certain types of violence with serotonergic drugs and perhaps precursors.

Links between the recently reported putative impairment of the serotonin system and the earlier findings of frontotemporal damage in violent individuals remain nebulous. Patients with frontotemporal contusions, but not with diffuse cerebral contusions, showed decreased levels of CSF 5-HIAA soon after the injury [97]. If confirmed, this finding might provide such a link.

Serum Glucose

Hypoglycemia may elicit epileptiform and other EEG abnormalities [98], and it was used (with success) as a defense against a murder charge as early as 1943 [71]. The behavioral effects of hypoglycemia may include irritability and aggression, sometimes with amnesia; these effects may be partly mediated by an epileptiform mechanism.

Virkkunen and his colleagues have administered glucose tolerance tests to violent offenders with antisocial personality [99] and to impulsive fire setters [94]. They found a low blood glucose nadir in both offender groups (compared with controls). Insulin secretion in response to glucose load is reportedly enhanced in patients with antisocial personality [100], and this enhancement is probably related to the pronounced hypoglycemic responses to glucose in violent offenders. Furthermore, brain serotonin content is known to increase after the ingestion of carbohydrates [101]. Additional links between the metabolism of carbohydrates, insulin secretion, and brain serotonin are discussed elsewhere [102].

Critique of the Literature

The literature on psychobiology of the violent offender appears to lack a comprehensive theoretical base and has serious methodological difficulties. We will now discuss certain problems shared by many studies in this area.

Violent Crime as the Dependent Variable

Most psychobiological researchers studying violent offenders give surprisingly little thought to their principal dependent variable. In general, the definition of violent crime (and violent criminal) is left to the state without much consideration of the massive constraints imposed by this choice (Ref 103, p. 61). Typically, the subjects are classified as violent (or nonviolent) on the basis of a single (most recent) offense. This offense is important since it usually led to the current incarceration, but it is insufficient as a classification criterion for research purposes. The criminal history is very important, but it is usually ignored. The problem can be illustrated by an example. A study [87] compared three prisoner groups: "murderers," "violent offenders," and "nonviolent" controls; the classification was based on the "present" (most recent) offense in 15% of the subjects in the nonviolent group. The official record usually underestimates the number and severity of offenses actually committed; thus, the 15% of violent offenders in the nonviolent group was probably a very conservative estimate, which raises questions regarding the appropriateness of the control group.

In addition to the conceptual problems involving the offense description, we also have to consider how the information is actually obtained. In the United States, the source of official information closest to the actual offense is the police report. The conviction is the final result of complicated forces which involve plea bargaining and many other factors; the original information about the offense may not be fully reflected in the conviction record. Nevertheless, the conviction record is typically used by researchers for subject classification.

Impulsivity is emerging as a central concept linking violent suicide, assaultiveness, and serotonergic dysfunction. It is unfortunate that the term is not very clear, particularly when applied to criminal acts. In the important paper by Linnoila et al. [93], the definition of impulsivity was based in part on the relationship between the offender and the victim: if the offender did not know the victim, the crime met one of the criteria for impulsiveness. If the offender and the victim knew each other, the data were not used since the reports on premeditation appeared to be unreliable in these cases (Linnoila, personal communication, 1991). It is important to realize that offenses involving domestic violence were excluded; this exclusion represents a potential limitation on the ability to generalize from Linnoila's data.

We noted that impulsivity remained undefined in the paper on fire setters [94]. Operationalizing the concept of impulsivity would clearly be of great importance. A tenitem rating scale of impulsiveness and premeditation has been constructed and applied to the records of 251 criminals [66]. The interrater reliability of this scale was high [66]. Other authors attempted to use a single-item scale to measure the impulsiveness of crimes, but their interrater agreement was lower [29].

Apparently unaware of the literature on serotonin and impulsive crime, criminologists Gottfredson and Hirschi [103] have developed a general theory purporting to explain all crime on the basis of offenders' insufficient "self-control." Although these authors do not clearly define the concept of self-control, they include impulsiveness as one of its dimensions (Ref 103, p. 95). The other dimensions are insensitivity and lack of intelligence. Thus, the dimensions of self-control are "factors affecting calculation of one's own acts" (Ref 103, p. 95). Thus Gottfredson and Hirschi provide an independently derived theoretical support for the importance of impulsiveness in crime.

Subject Selection

One can study the entire population of known homicide offenders in a geographic area, such as Iceland [18], Northern Sweden [34], or a county in California [53]. Such methods avoid sampling bias, but these retrospective studies do not permit reliable assessment of neuropsychiatric impairment. A more typical source of subjects is a referral for forensic pretrial evaluation [49,50,52,54]. Individuals may be referred by the police, judges, prosecutors, or defense lawyers. The biases involved in these referrals are unknown and probably vary greatly among different jurisdictions; they also probably depend on the availability of forensic science services and the experience and motivation of the referring officials. Other researchers select their subjects among inmates of prisons [5,51], mental hospitals [51], or institutions whose characteristics were not disclosed.

The methods of subject selection from these sources are variable. Some of the workers reported on consecutive referrals within a specified period of time [50,54]. Others, reporting on convicted offenders, selected them by the sentence received [5]. Finally, the subject selection criteria for some studies are not clearly defined [4,51].

Thus, most studies used samples of opportunity; the sample selection was based more on subject availability than on theoretical considerations of the ability to generalize to a population. This is a basic common flaw which makes the results difficult to interpret or replicate.

Unfortunately, appropriate epidemiological methods are particularly difficult to implement in psychobiological studies of violent offenders. Such studies cannot be done without informed and voluntary consent of the subjects. The requirement of consent is of course firmly based on the general principles of human rights, and everybody supports it. However, it is important to realize that the subjects' decision to consent is determined by many factors which may ultimately result in biasing the selection process. The literature on consent bias in samples of psychiatric patients is beyond the scope of this review, but we note that paranoid and hostile patients are less likely to consent than others.

To complicate matters further, it is not clear whether the decision to consent to participate in research can ever be truly voluntary in prisoners. Some ethicists feel that incarceration makes prisoners so vulnerable to real or imagined coercion that they would consent against their own will. This view appears to be more popular in the United States than in Europe.

Thus, the subject selection method is a crucial step in the study design. The principal factors to consider include the following: the ability to generalize from the sample, the resources available, and ethical as well as political issues.

Lack of Controls

Most reports on neurobiology of violent crime lack control or comparison groups. Those researchers who use a control group make no provisions for "blinding" their interviewers and testers: these researchers are invariably aware of the subjects' group membership (for example violent versus nonviolent prisoners). This is a serious problem with research involving interviews, evaluation of EEG recordings by visual inspection, and any other procedures calling for subjective judgment on the part of the tester. We have not found any cross-sectional studies dealing adequately with this problem. Prospective study designs build in protection against such biases.

Lack of Prospective Studies

With some exceptions [4,13,95], neurobiological research into violent crime has been cross-sectional and retrospective. The major inherent weakness of the retrospective studies is the inability of this design to reliably distinguish between the antecedents and consequences of violent crime.

Lack of Studies of Interactions

Most studies report one or two variables related to violent crime (for example, the psychiatric diagnosis of the offender). Those researchers who study more variables at a time only rarely attempt to develop an explanatory multivariate model [13,38,77]. Violent crime appears to have complex causation, and a multiple-variable model generally provides more explanatory power than studying one variable at a time. We feel that substantial progress in this field will be impossible without integrative multidisciplinary studies.

References

- [1] Mednick, S. A., Pollock, V., Volavka, J., and Gabrielli, W. F., Jr., "Biology and Violence," Criminal Violence, M. E. Wolfgang and N. A. Weiner, Eds., Sage Publications, Beverly Hills, CA, 1982, pp. 21-80.
- [2] Volavka, J., "Aggression, Electroencephalography, and Evoked Potentials: A Critical Review," Neuropsychiatry, Neuropsychology, and Behavioral Neurology, Vol. 3, 1990, pp. 249-259.
- [3] Curtis, C. G., "Violence Breeds Violence-Perhaps?" American Journal of Psychiatry, Vol. 120, 1963, pp. 386-387.
- [4] Lewis, D. O., Moy, E., Jackson, L. D., Aaronson, R., Restifo, N., Serra, S., and Simos, A., "Biopsychosocial Characteristics of Children Who Later Murder: A Prospective Study," American Journal of Psychiatry, Vol. 142, 1985, pp. 1161-1167.
- [5] Lewis, D. O., Pincus, J. H., Bard, B., Richardson, E., Prichep, L. S., Feldman, M., and Yeager, C., "Neuropsychiatric, Psychoeducational, and Family Characteristics of 14 Juveniles Condemned to Death in the United States," American Journal of Psychiatry, Vol. 145, 1988, pp. 584-589.
- [6] Duncan, J. W. and Duncan, G. M., "Murder in the Family: A Study of Some Homicidal Adolescents," American Journal of Psychiatry, Vol. 127, 1971, pp. 1498-1502.
- [7] Corder, B. F., Ball, B. C., and Haizhip, T. M., "Adolescent Parricide: A Comparison with Other Adolescent Murder," American Journal of Psychiatry, Vol. 133, 1976, pp. 957-961.
- [8] King, C., "The Ego and the Integration of Violence in Homicidal Youth," American Journal of Orthopsychiatry, Vol. 45, 1975, pp. 134-145.
- [9] Ressler, R. and Burgess, A., "The Men Who Murdered," FBI Law Enforcement Bulletin, Vol. 540, 1985, pp. 2-6.
- [10] Sorells, J., "Kids Who Kill," Crime and Delinquency, Vol. 312, 1977, p. 320. [11] Widom, C., "Child Abuse, Neglect and Violent Criminal Behavior," Criminology, Vol. 27, 1989, pp. 251-271.
- [12] Widom, C. S., "The Cycle of Violence," Science, Vol. 244, 1989, pp. 160-166.
- [13] Lewis, D. O., Lovely, R., Yaeager, C., and Della Femina, D., "Toward a Theory of the Genesis of Violence: A Follow-up Study of Delinquents," Journal of the American Academy of Child Psychiatry, Vol. 28, 1989, pp. 431-436.
- [14] Convit, A., Jaeger, J., Lin, S. P., Meisner, M., and Volavka, J., "Prediction of Assaultiveness in Psychiatric Inpatients: A Pilot Study," Hospital and Community Psychiatry, Vol. 39, 1988, pp. 429-434.
- [15] Krakowski, M., Convit, A., and Volavka, J., "Patterns of Inpatient Assaultiveness: Effect of Neurological Impairment and Deviant Family Environment on Response to Treatment," Neuropsychiatry, Neuropsychology and Behavioral Neurology, Vol. 1, 1988, pp. 21-29.
- [16] Higley, J. D., Suomi, S. J., and Linnoila, M., "Parallels in Aggression and Serotonin: Consideration of Development, Rearing History, and Sex Differences," Violence and Suicidality: Perspectives in Clinical and Psychobiological Research, H. M. Van Praag, R. Plutchik, and A. Apter, Eds., Brunner/Mazel, New York, 1990, pp. 245-246.

- [17] Lewis, D. O. and Shanok, S. S., "A Comparison of the Medical Histories of Incarcerated Delinquent Children and a Matched Sample of Non-delinquent Children," Child Psychiatry and Human Development, Vol. 9, 1979, pp. 210–214. [18] Petursson, H. and Gudjonsson, G. H., "Psychiatric Aspects of Homicide," Acta Psychiatrica
- Scandinavica, Vol. 64, 1981, pp. 363-372.
- [19] Whitman, S., Coleman, T. E., Patmon, C., Desai, B. T., Cohen, R., and King, L. N., "Epilepsy in Prison: Elevated Prevalence and No Relationship to Violence," Neurology, Vol. 34, 1984, pp. 775-782.
- [20] Lewis, D. O., Shanok, S. S., Grant, M., and Ritvo, E., "Homicidally Aggressive Young Children: Neuropsychiatric and Experiential Correlates," *American Journal of Psychiatry*, Vol. 140, 1983, pp. 148-153.
- [21] Hill, D. and Pond, D. A., "Reflections on One Hundred Capital Cases Submitted to Electroencephalography," Journal of Mental Science, Vol. 98, 1952, pp. 23-43.
- [22] Gunn, J. C. and Fenton, G. W., "Epilepsy in Prisons: A Diagnostic Survey," British Medical Journal, Vol. iv, 1969, pp. 326-329.
- [23] Delgado-Escueta, A. V., Mattson, R. H., King, L. Goldensohn, E. S., Spiegel, H., Madsen, J., Crandall, P., Dreifuss, F., and Porter, R. J., "The Nature of Aggression During Epileptic Seizures," New England Journal of Medicine, Vol. 305, 1981, pp. 711-716.
- [24] Gunn, J. C. and Fenton, G. W., "Epilepsy, Automatism and Crime," The Lancet, Vol. 1, 1971, pp. 1173-1176.
- [25] Lewis, D. O., Shanok, S. S., Pincus, J. H., and Glaser, G. H., "Violent Juvenile Delinquents: Psychiatric, Neurological, Psychological, and Abuse Factors," Journal of the American Academy of Child Psychiatry, Vol. 18, 1979, pp. 307-319.
- [26] Virkkunen, M., Letter to the Editor, American Journal of Psychiatry, Vol. 140, 1983, pp. 646-647.
- [27] Ehrenberg, B. L., Hardy, P. M., and Zifkin, B. G., Letter to the Editor, American Journal of Psychiatry, Vol. 140, 1983, p. 647.
- [28] Stevens, J. R. and Hermann, B. P., "Temporal Lobe Epilepsy, Psychopathology, and Violence: the State of the Evidence," Neurology, Vol. 31, 1981, pp. 1127-1132.
- [29] Gunn, J. and Bonn, J., "Criminality and Violence in Epileptic Prisoners," British Journal of Psychiatry, Vol. 118, 1971, pp. 337-343.
- [30] Gerson, L. W. and Preston, D. A., "Alcohol Consumption and the Incidence of Violent Crime," Journal of Studies on Alcohol, Vol. 40, 1979, pp. 307-312.
- [31] Goodwin, D. W., "Alcohol in Suicide and Homicide," Quarterly Journal of Studies on Alcohol, Vol. 34, 1973, pp. 144-156.
- [32] Pernanen, K., "Alcohol and Crimes of Violence," in Kissin, B., Begleiter, H., eds. The Biology of Alcoholism, Plenum Press, New York, 1976, pp. 351-444.
- [33] "Prisoners and Alcohol," BJS Bulletin, Jan. 1983, pp. 1-4.
- [34] Lindqvist, P., "Criminal Homicide in Northern Sweden 1970-1981: Alcohol Intoxication, Alcohol Abuse and Mental Disease," International Journal of Law and Psychiatry, Vol. 8, 1986, pp. 19-37.
- [35] Langevin, R., Ben-Aron, M., Wortzman, G., Dickey, R., and Handy, L., "Brain Damage, Diagnosis, and Substance Abuse Among Violent Offenders," Behavioral Sciences and the Law, Vol. 5, 1987, pp. 77-94.
- [36] Yesavage, J. A. and Zarcone, V., "History of Drug Abuse and Dangerous Behavior in Inpatient Schizophrenics," Journal of Clinical Psychiatry, Vol. 44, 1983, pp. 259-261.
- 37] Finger, S. and Stein, D. G., Brain Damage and Recovery, Academic Press, New York, 1982.
- [38] Abram, K., "The Effect of Co-occurring Disorders on Criminal Careers: Interaction of Antisocial Personality, Alcoholism, and Drug Disorders," International Journal of Law and Psychiatry, Vol. 12, 1989, pp. 133-148.
- [39] Gropper, B., "Probing the Links Between Drugs and Crime," NIJ Briefs, U.S. Department of Justice, Washington, DC, 1985.
- [40] Nurco, D. N., Ball, J. C., Shaffer, J. W., and Hanlon, T. E., "The Criminality of Narcotic Addicts," Journal of Mental and Nervous Disease, Vol. 173, 1985, pp. 94-102.
- [41] National Institute of Justice Report 208, National Institute of Justice, Washington, DC, 1988, pp. 8-9.
- [42] Johnson, B. D., Goldstein, P., Preble, E., Schmeider, J., Lipton, D. S., Sprunt, B., and Miller, T., Taking Care of Business: The Economics of Crime by Heroin Abusers, Lexington Books, Lexington, KY, 1985.
- [43] Stephens, R. C. and Ellis, R. D., "Narcotic Addicts and Crime: Analysis of Recent Trends," Criminology, Vol. 12, No. 4, 1975, pp. 474-488.
- [44] Nurco, D. N., Hanlon, T. E., Kinlock, T. W., and Duszynski, K. R., "The Consistency of Types of Criminal Behavior over Preaddiction, Addiction, and Nonaddiction Status Periods," Comprehensive Psychiatry, Vol. 30, 1989, pp. 391-402.

- [45] Innes, C. A., "State Prison Inmate Survey, 1986: Drug Use and Crime," BJS special report U.S. Department of Justice, Washington, DC, 1988, pp. 1–8.
- [46] Ellinwood, E. H., "Assault and Homicide Associated with Amphetamine Abuse," American Journal of Psychiatry, Vol. 127, 1971, pp. 1170–1175.
- [47] Convit, A., Nemes, Z. C., and Volavka, J., "History of Phencyclidine Use and Repeated Assaults in Newly Admitted Young Schizophrenic Men," *American Journal of Psychiatry*, Vol. 145, 1988, p. 1176.
- [48] Busch, K. A. and Schnoll, S. H., "Cocaine—Review of Current Literature and Interface with the Law," *Behavioral Sciences and the Law*, Vol. 3, 1985, pp. 283–298.
- [49] Langevin, R., Paitich, D., Orchard, B., Handy, L., and Russon, A., "Diagnosis of Killers Seen for Psychiatric Assessment," Acta Psychiatrica Scandinavica, Vol. 66, 1982, pp. 216– 228.
- [50] Gillies, H., "Homicide in the West of Scotland," British Journal of Psychiatry, Vol. 128, 1976, pp. 105-127.
- [51] Okasha, A., Sadek, A. O., and Moneim, S. A., "Psychosocial and Electroencephalographic Studies of Egyptian Murderers," *British Journal of Psychiatry*, Vol. 126, 1975, pp. 34-40.
- [52] Malmquist, C. P., "Premonitory Signs of Homicidal Aggression in Juveniles," American Journal of Psychiatry, Vol. 128, 1971, pp. 93-97.
- [53] Wilcox, D. E., "The Relationships of Mental Illness to Homicide," American Journal of Forensic Psychiatry, Vol. 6, 1985, pp. 3-14.
- [54] Cornell, D. G., Benedek, E. P., and Benedek, D. M., "Juvenile Homicide: Prior Adjustment and a Proposed Typology," American Journal of Orthopsychiatry, Vol. 57, 1987, pp. 383– 393.
- [55] Rosenbaum, M. and Bennett, B., "Homicide and Depression," American Journal of Psychiatry, Vol. 143, 1986, pp. 367–370.
- [56] Herrman, H., McGorry, P., Mills, J., and Singh, B., "Hidden Severe Psychiatric Morbidity in Sentenced Prisoners: An Australian Study," *American Journal of Psychiatry*, Vol. 148, 1991, pp. 236–239.
- [57] Collins, J. J. and Schlenger, W. E., "The Prevalence of Psychiatric Disorders Among Admissions to Prison," Abstract, *Proceedings*, 35th Annual Meeting of the American Society of Criminology, Denver, CO, 1983, pp. 9-15.
- [58] Jemelka, R., Trupin, E., and Chiles, J., "The Mentally III in Prisons: A Review," Hospital and Community Psychiatry, Vol. 40, 1989, pp. 481-491.
- [59] American Psychiatric Association, Dignostic and Statistical Manual of Mental Disorders, 3rd ed., revised, American Psychiatric Association, Washington, DC, 1987.
- [60] Hare, R. D., "Psychopathy and Violence," Violence and the Violent Individual, J. R. Hays, K. Roberts, and K. Solway, Eds., Spectrum, New York, 1980.
- [61] Hare, R. D. and Jutai, J., "Criminal History of the Male Psychopath: Some Preliminary Data," Prospective Studies of Crime and Delinquency, K. Van Dusen and S. Mednick, Eds., Kluwer-Nijhoff, Boston, MA, 1983.
- [62] Hare, R. D. and McPherson, L., "Violent and Aggressive Behavior by Criminal Psychopaths," International Journal of Law and Psychiatry, Vol. 7, 1984, pp. 35-50.
- [63] Williamson, S., Hare, R., and Wong, S., "Violence: Criminal Psychopaths and Their Victims," Canadian Journal of Behavioral Science, Vol. 19, 1987, pp. 454-462.
- [64] Hart, S., Kropp, P., and Hare, R., "Performance of Male Psychopaths Following Conditional Release from Prison," *Journal of Clinical and Consulting Psychology*, Vol. 56, 1988, pp. 227– 232.
- [65] Gorenstein, E., "Frontal Lobe Function in Psychopaths," Journal of Abnormal Psychology, Vol. 91, 1982, pp. 368-379.
- [66] Heilbrun, A. B., "Psychopathy and Violent Crime," Journal of Consulting and Clinical Psychology, Vol. 47, 1979, pp. 509-516.
- [67] Heilbrun, A. B., "Cognitive Models of Criminal Violence Based upon Intelligence and Psychopathy Levels," *Journal of Consulting and Clinical Psychology*, Vol. 50, 1982, pp. 546– 557.
- [68] Spellacy, F., "Neuropsychological Discrimination Between Violent and Non-violent Men," Journal of Clinical Psychology, Vol. 34, 1978, pp. 49-52.
- [69] Kahn, M. W., "A Comparison of Personality, Intelligence, and Social History of Two Criminal Groups," Journal of Social Psychology, Vol. 49, 1959, pp. 33-40.
- [70] Deiker, T. E., "WAIS Characteristics of Indicted Male Murders," Psychological Reports, Vol. 32, 1973, p. 1066.
- [71] Hill, D. and Sargant, W., "A Case of Matricide," The Lancet, Vol. 244, No. 1, 1943, pp. 526-527.
- [72] Mark, V. H. and Ervin, F. R., Violence and the Brain, Harper and Row, New York, 1970.

- [73] Goldberg, G., "From Intent to Action: Evolution and Function of the Premotor Systems of the Frontal Lobe," The Frontal Lobes Revisited, E. Perecman, IRBN Press, New York, 1987.
- [74] Stuss, D. T. and Benson, D. F., The Frontal Lobes, Raven Press, New York, 1986.
- [75] Valzelli, L., Psychobiology of Aggression and Violence, Raven Press, New York, 1981.
- [76] Tancredi, L. R. and Volkow, N., "Neural Substrates of Violent Behavior: Implications for Law and Public Policy," International Journal of Law and Psychiatry, Vol. 11, 1988, pp. 13-49
- [77] Yeudall, L. T., Fedora, O., and Fromm, D., "A Neuropsychosocial Theory of Persistent Criminality: Implications for Assessment and Treatment," Advances in Forensic Psychology and Psychiatry, R. W. Rieber, Ed., Ablex Publishing, Norwood, NJ, 1987, pp. 119-191.
- [78] Volkow, N. D. and Tancredi, L., "Neural Substrates of Violent Behavior," ' British Journal of Psychiatry, Vol. 151, 1987, pp. 668-673.
- [79] Pontius, A. A. and Yudowitz, B. S., "Frontal Lobe Dysfunction in Some Criminal Actions Shown in the Narratives Test," Journal of Nervous and Mental Disease, Vol. 168, 1980, pp. 111-117.
- [80] Pontius, A. A., "Specific Stimulus-Evoked Violent Action in Psychotic Trigger Reaction: A Seizure-like Imbalance Between Frontal Lobe and Limbic Systems?" Perceptual and Motor Skills, Vol. 59, 1984, pp. 299-333.
- [81] Yeudall, L. T., "Neuropsychological Assessment of Forensic Disorder," Canada's Mental Health, Vol. 25, 1977, pp. 7-15.
- [82] Heinrichs, R. W., "Frontal Cerebral Lesions and Violent Incidents in Chronic Neuropsychiatric Patients," Biological Psychiatry, Vol. 25, 1989, pp. 174-178.
- [83] Williams, D., "Neural Factors Related to Habitual Aggression," Brain, Vol. 95, 1969, pp. 503 - 520
- [84] Fishbein, D., Herning, R., Pickworth, W. B., Haertzen, C. A., Hickey, J. E., and Jaffe, J. H., "EEG and Brainstem Auditory Evoked Response Potentials in Adult Male Drug Abusers with Self-Reported Histories of Aggressive Behavior," Biological Psychiatry, Vol. 26, 1989, pp. 595-611.
- [85] Hoffman, J. J., Hall, R. W., and Bartsch, T. W., "On the Relative Importance of 'Psychopathic' Personality and Alcoholism on Neuropsychological Measures of Frontal Lobe Dysfunction," Journal of Abnormal Psychology, Vol. 96, No. 2, 1987, pp. 158-160.
- [86] Yeudall, L. T., Fromm-Auch, D., and Davies, P., "Neuropsychological Impairment of Persistent Delinquency," Journal of Nervous and Mental Disease, Vol. 170, 1982, pp. 257-265.
- [87] Nachshon, I., "Hemisphere Function in Violent Offenders," Biological Contributions to Crime Causation, T. E. Moffitt and S. A. Mednick, Eds., Martinus Nijhoff, Dordrecht, The Netherlands, 1988, pp. 55-67.
- [88] Gabrielli, W. F. and Mednick, S. A., "Sinistrality and Delinquency," Journal of Abnormal Psychology, Vol. 89, 1980, pp. 654-661.
- [89] Nachshon, I., "Hemisphere Dysfunction in Psychopathy and Behavior Disorders," Hemisyndromes: Psychobiology, Neurology, Psychiatry, M. Myslobodsky, Ed., Academic Press, New York, 1983, pp. 389-414.
 [90] Valzelli, L., "The Isolation Syndrome in Mice," *Psychopharmacologia* (Berlin), Vol. 31, 1973,
- pp. 305-320.
- [91] Asberg, M., Traskman, L., and Thoren, P., "5-HIAA in the Cerebrospinal Fluid: A Biochemical Suicide Predictor?" Archives of General Psychiatry, Vol. 33, 1976, pp. 1193-1197.
- [92] Brown, G. L., Goodwin, F. K., Ballenger, J. C., Goyer, P. F., and Major, L. F., "Aggression in Humans Correlates with Cerebrospinal Fluid Amine Metabolites," Psychiatry Research, Vol. 1, 1979, pp. 131–139.
- [93] Linnoila, M., Virkkunen, M., Scheinin, M., Nuutila, A., Rimon R., and Goodwin, F. K., "Low Cerebrospinal Fluid 5-Hydroxyindole Acetic Acid Concentration Differentiates Impulsive from Non-impulsive Violent Behavior," Life Sciences, Vol. 33, 1983, pp. 2609-2614.
- [94] Virkkunen, M., Nuutila, A., Goodwin, F. K., and Linnoila, M., "Cerebrospinal Fluid Monoamine Metabolites in Male Arsonists," Archives of General Psychiatry, Vol. 44, 1987, pp. 241-247.
- [95] Virkkunen, M., De Jong, J., Bartko, J., Goodwin, F. K., and Linnoila, M., "Relationship of Psychobiological Variables to Recidivism in Violent Offenders and Impulsive Fire Setters: A Follow-up Study," Archives of General Psychiatry, Vol. 46, 1989, pp. 600-603.
- [96] Monahan, J., "The Prediction of Violent Behavior: Toward a Second Generation of Theory and Policy," American Journal of Psychiatry, Vol. 141, 1984, pp. 10-15.
- [97] Van Woerkom, T. C. A. M., Teelken, A. W., and Minderhoud, J. M., "Difference in Neurotransmitter Metabolism in Frontotemporal-Lobe Contusion and Diffuse Cerebral Contusion," The Lancet, Vol. I, 1977, pp. 812-813.

- [98] Dawson, M. and Greville, G. D., "Biochemistry," Electroencephalography, J. D. N. Hill and G. Parr, Eds., MacDonald, London, 1963, pp. 158-161.
- [99] Virkkunen, M. and Huttunen, M. O., "Evidence for Abnormal Glucose Tolerance Test Among Violent Offenders," Neuropsychobiology, Vol. 8, 1982, pp. 30-34.
- [100] Virkkunen, M., "Insulin Secretion During the Glucose Tolerance Test in Antisocial Personality," British Journal of Psychiatry, Vol. 142, 1983, pp. 598-604. [101] Fernstrom, J. D. and Wurtman, R. J., "Brain Serotonin Content: Increase Following Ingestion
- of Carbohydrate Diet," Science, Vol. 178, 1971, pp. 1023-1025.
- [102] Virkkunen, M., "Cerebrospinal Fluid: Monoamine Metabolites Among Habitually Violent and Impulsive Offenders," Biological Contributions to Crime Causation, T. E. Moffitt and S. A. Mednick, Eds., Martinus Nijhoff, Dordrecht, The Netherlands, 1988.
- [103] Gottfredson, M. R. and Hirschi, T. A., A General Theory of Crime, Stanford University Press, Stanford, CA, 1990.

Address requests for reprints or additional information to Jan Volavka, M.D., Ph.D. Nathan S. Kline Institute for Psychiatric Research Orangeburg, NY 10962