HUMAN AGGRESSION

THEORIES, RESEARCH, AND IMPLICATIONS FOR SOCIAL POLICY

EDITED BY

RUSSELL G. GEEN
Department of Psychology
University of Missouri
Columbia, Missouri

EDWARD DONNERSTEIN
Department of Communication
University of California, Santa Barbara
Santa Barbara, California

ACADEMIC PRESS
San Diego London Boston New York Sydney Tokyo Toronto

6

PSYCHOACTIVE DRUGS AND HUMAN AGGRESSION[1]

STUART P. TAYLOR AND MICHAEL R. HULSIZER
Kent State University

A wide variety of drugs are presumed to be related to aggressive behavior. In an earlier review (Taylor & Leonard, 1983), the evidence concerning the effect of alcohol on human aggressive behavior was scrutinized. The authors concluded, "Alcohol does appear to be a potent causal antecedent of aggressive behavior" (p. 97). This conclusion was confirmed in a more recent review (Taylor & Chermack, 1993). The purpose of this chapter is to critically examine the effects of several other commonly used psychoactive drugs on human aggression: marijuana, amphetamines, benzodiazepines, and morphine. This chapter describes the classification of major psychoactive drugs, discusses the traditional empirical and theoretical perspectives concerning the relationship between psychoactive drugs and aggression, reviews the results of a series of experiments designed to examine the instigating effects of psychoactive drugs, and considers the theoretical and policy implications of the empirical evidence.

CLASSIFICATION OF PSYCHOACTIVE DRUGS

One of the most traditional methods of classifying psychoactive drugs is in terms of their characteristic behavioral or clinical effects. Less typical drug classification schemes involve molecular structure and biochemical actions. Some of the major categories of drugs that alter behavior or mood are stimulants, depressants, opiates, hallucinogens/psychedelics, and marijuana.

[1]Correspondence concerning this chapter should be addressed to Stuart P. Taylor, Department of Psychology, Kent State University, Kent, OH 44242, or to Michael R. Hulsizer, who is now at the Department of Behavioral and Social Sciences, Webster University, St. Louis, MO 63119.

Stimulants consist of compounds that excite the central nervous system (CNS). It is widely accepted that while stimulants increase arousal, alertness, and euphoria, they also decrease fatigue and depression. Commonly consumed stimulants are cocaine, amphetamine, caffeine, and nicotine. Stimulants have been used to treat hyperkinetic and affective disorders.

Depressants include a wide variety of drugs with diverse chemical structures that are capable of inducing progressive depression of the CNS. There appears to be a general consensus that the depressant effects vary along a continuum from anxiety relief to sedation, sleep, and, finally, coma and death. The basic category includes alcohol, barbiturates, antihistamines, and benzodiazepines. Depressants have been used as anesthetics and for the treatment of epilepsy, insomnia, and anxiety.

Opiates, often called narcotic analgesics or opioids, refer to natural or synthetic drugs that have morphine-like actions. This category includes drugs such as morphine and codeine, which are purified from crude opium, and compounds such as heroin, derived from alterations of morphine, and synthetic analgesics. The major psychological effects of drugs such as morphine are euphoria and analgesia. Clinically, opiates are mainly used as painkillers. In fact, there appears to be a consensus in the medical literature that there is no class of drugs superior to opiates for analgesia.

The most commonly used products derived from cannabis sativa in this country are marijuana and hashish. Marijuana is a smoking preparation and consists of a mixture of crushed leaves and flowers. The active compound, Δ^9-tetrahydrocannabinol (THC), is concentrated in the resin obtained from the flowers of the plant. While marijuana effects vary widely from person to person, THC users often report enhanced taste, smell, and touch, an alteration in time perception, an increased sense of well-being, relaxation, and mild euphoria. Nonmedicinal use of marijuana is illegal in the United States. However, there is some indication that it is efficacious in the treatment of glaucoma, epilepsy, chronic pain, and nausea.

Hallucinogens, or psychedelics, cause distortions in perception, cognition, and mood. They tend to alter time and space perception, sense of body image, and sensitivity to sounds, shapes, and textures. Some of the compounds typically included in this category are lysergic acid diethylamide (LSD), phencyclidine (PCP), psilocybin, MDNA (also known as "Ecstasy"), and mescaline. Generally, hallucinogens have no recognized clinical use.

Contemporary psychopharmacologists often categorize drugs in terms of their full dose–response curves (Leccese, 1991). This approach allows for the creation of categories that include drugs that produce similar effects despite differences in basic pharmacology. Thus, certain drugs would be categorized as stimulants if they result in increased attention, heart rate, and wakefulness at low doses; insomnia, stereotypical motor movements, tremors, and highly elevated cardiac activity at moderate doses, and confusion, paranoia, and possible convulsions at very high doses. This category would include such drugs as caffeine, nicotine, amphetamine, and cocaine. A number of other drugs would be categorized as depressants if they tended to reduce motor coordination at low doses; produced loss of coordination, sleepiness, and depressed breathing at moderate doses; and caused coma and death at very high doses. This category would include antihistamines, benzodiazepines, opiates, and alcohol. Leccese (1991), who advocates this categorization system, also includes a separate category for hallucinogens. These drugs produce slight alteration in perception at low doses, hallucinations and sympathetic nervous system stimulation at moderate doses, and profound delusions as well as loss of contact with reality at very high doses.

TRADITIONAL PERSPECTIVES

The use, possession, and sale of some psychoactive drugs are illegal. It is a crime in this country to use, possess, buy, or sell such controlled substances as LSD, heroin, cocaine, and marijuana. However, many commonly used psychoactive drugs are legal or can be acquired legally. Psychoactive substances can be found in over-the-counter remedies (e.g., Dexatrim, No-Doz), in legally purchased products (e.g., tea, cola, cigarettes, alcoholic beverages), and in prescription drugs.

It is estimated that in any particular year, 15% of the U.S. population experience some form of psychological disorder (Klerman, 1983). Another 15% of the population seek clinical advice for symptoms that do not meet specific diagnostic criteria for a psychiatric disorder (e.g., anxiety). Psychoactive drugs are often prescribed to alleviate or control the symptoms of these disorders. According to Ray and Ksir (1990), there are over 300,000 physicians legally writing prescriptions in the United States, and 150,000 pharmacists at 60,000 locations selling the prescribed drugs.

There is a paucity of research on the direct effects of illicit as well as licit psychoactive drugs on human aggressive behavior. The medical establishment has certainly not concentrated its attention on studying the effects of psychoactive substances on aggression. Instead, they have been concerned with adverse medical effects. There are the infrequent letters to the editors of medical journals and surveys of drug-related affective states (e.g., Cole & Kando, 1993). However, a perusal of the *Physicians Desk Reference* clearly demonstrates the lack of interest in drug-induced interpersonal conflict.

Psychologists and psychopharmacologists have also devoted little attention to studying the direct relationship between drugs and human aggression. The best illustration of this problem is the relative lack of attention to drug-elicited aggression in the major psychological texts on aggressive behavior. In Baron and Richardson's (1994) comprehensive review of the psychological literature on aggression, literature is cited on the effects of only two drugs: alcohol and marijuana. The effects of marijuana are discussed for approximately two paragraphs. In his text entitled *Aggression: Its Causes, Consequences, and Control,* Berkowitz (1993), a leading authority on aggressive behavior, made only one reference to the

possible instigating effects of drugs. He concluded, ". . . much of the growth in homicides seems to be independent of drugs" (p. 278). Berkowitz adds, "A better case can be made for the role of weapons" (p. 279).

Law enforcement and social science professionals also do not appear to be preoccupied with the possibility that prescription, over-the-counter, or illegal drugs might facilitate aggressive behavior. Instead, the assumption appears to be that violence is an indirect result of the illegal drug trade (e.g., Watters, Reinarman, & Fagan, 1985). A popular view that appears to be advocated by many contemporary social scientists (e.g., Goldstein, Bellucci, Spunt, & Miller, 1991) is that addicts will harm others to acquire drugs ("economic violence") and drug gangs will use violence to acquire and maintain territory ("systemic violence"). In an article concerning substance use in forensic psychiatry, Kermani and Castaneda (1996) argued, "Most violence associated with drugs other than alcohol is related to the business of selling them" (p. 2). Thus, there appears to be considerable skepticism concerning "psychopharmacological violence," i.e., the direct effects of drugs on aggression, among many contemporary investigators in the medical, psychological, psychopharmacological, legal, and sociological fields (Wish & Johnson, 1986).

Not withstanding the skeptics, a modest body of literature pertaining to the relationship between drug consumption and aggressive responding among humans has evolved. This endeavor has been sustained by a small minority of investigators who have steadfastly supported the possibility of drug-induced violence. These investigators have been influenced to a considerable extent by the numerous, impelling case studies and clinical reports of the facilitation of violence following drug consumption. Goode (1993) observed, for example, "To many of us, the linkage seems as clear, as strong, as direct as morning and the rising sun" (p. 120).

STIMULANTS

Traditionally, there has been a pronounced expectation, based on meager empirical evidence, that stimulants facilitate aggression. Mayfield (1983) presented this view in a typical fashion: "The amphetamines would seem to be a likely group of drugs to be implicated in aggressive physical assaults" (p. 147). He added, "Not only do these drugs enhance noradrenergic activity and general level of arousal, they produce paranoid psychosis with some regularity" (p. 147). Cohen (1981), the former director of the Division of Narcotic Addiction and Drug Abuse, the National Institute of Mental Health, was even more convinced of the instigating effects of stimulants. He professed, "It can be predicted that stimulants in large doses with their capacity to cause hyperactivity, paranoid suspiciousness and impulsivity will be productive of violence" (p. 362). More dramatically, Cohen described how "instances of interminable stabbing or clubbing of a victim long since dead are well known" (p. 362). Powers and Kutash (1978) argued that "aggression and violence due to amphetamine use are particularly likely in individuals with premorbid aggressive tendencies and problems of impulse control" (p. 327). They also indicated that "many individuals, however, with no apparent personality abnormalities have evidenced aggression or violence during amphetamine use" (p. 327). This perspective tends to be shared by numerous contemporary investigators. For example, Meloy (1987) noted, "Anecdotal clinical experience also suggests that certain psychostimulants, such as methamphetamine and cocaine, may precipitate violence that is characterized by both intense rage and paranoia" (p. 40). In a more recent article, Miller and Gold (1994) argued, "The link between stimulants and criminal activity has been known for some time" (p. 1070).

The belief that amphetamines instigate aggressive behavior may have been derived from two sources: the pervasive assumption that drugs which enhance arousal states increase aggression potential and early clinical reports of the adverse effects of amphetamines. One of the most influential studies on the effects of stimulants on aggression was conducted by Ellinwood (1971). In accordance with traditional assumptions, Ellinwood's rationale for the study was based on the following observation: "Reports from law-enforcement personnel and psychiatrists, as well as from drug abusers themselves, have indicated that amphetamines may also be related to aggressive behavior" (p. 1170). The author added, "Perhaps more specifically than any other group of drugs" (p. 1170). Ellinwood examined case histories of 13 persons who committed homicide, presumably under the influence of large dosages of amphetamines. Ellinwood concluded that "homicide was clearly related to an amphetamine-induced delusional process and/or state of emotional lability" (p. 1175). Of course, given the nature of the study, there is no way to assess the role played by the amphetamine presumably ingested by the subjects. For example, it is difficult, based on the self-report data, to determine the time that elapsed between the amphetamine ingestion and the murderous act, the dosages actually consumed by the subjects, or the independent effects of the many other factors involved in each case. Most telling, the assailants were polydrug users. While noting that, "At the present time, we have no basis for an estimate of the relative importance of amphetamine abuse in violent behavior," Mednick, Pollock, Volavka, and Gabrielli (1982) concluded that "Ellinwood's suggestion that every person arrested for a violent crime have a urine test for drugs of abuse is certainly worthwhile" (p. 60).

A few authors have questioned the widespread belief that stimulants are potent, independent instigators of aggressive behavior. Allen, Safer, and Covi (1975) concluded, following their review of the literature, that stimulants only instigate aggression in doses that produce "amphetamine psychoses." Moss, Salloum, and Fisher (1994) argued that amphetamines may lead to aggressive behavior only in presence of chronic use, paranoid psychosis, or sociopathy.

Allen et al. (1975) pointed out that amphetamines may actually reduce the likelihood of aggressive acts. They noted the efficacy of stimulants to reduce aggressive tendencies in hyperactive children, brain-damaged adults, and delinquent adolescents. A more recent review by Connor and Steingard (1996) confirmed the possibility that stimulants may actually reduce the aggressive acts of children and adolescents referred for psychiatric treatment.

Cocaine, a psychomotor stimulant, has often been associated with aggression in the literature (Honer, Gewirtz, & Turey, 1987; Rivinus & Larimer, 1993; Wetli

& Fishbain, 1985). There are many proposed theoretical explanations for this presumed association. Many investigators assume that the loss of control following cocaine consumption is due to the neuroanatomical locations effected by the drug (Burrows, Hales, & Arington, 1988; Sheard, 1988). It is conjectured that cocaine exerts its influence in the frontal lobe and limbic system, where aggressive motivational states are suspected of being regulated. As with amphetamines, other investigators argue that aggressive behavior only occurs when the consumer in intensely intoxicated (e.g., Brody, 1990).

While there is a great deal of speculation concerning the relationship between aggression and cocaine, there is little empirical evidence regarding cocaine-induced aggression. Miller, Gold, and Mahler (1991) asserted, "While violence has been associated with many drugs of abuse and addiction, no quantitative assessment of violence associated with cocaine use . . . has been reported" (p. 1078). In an attempt to rectify this problem, these authors interviewed, on the telephone, cocaine addicts who were attempting to obtain information about cocaine. They found that 26% of the participants admitted to committing a crime while using crack cocaine, the majority violent. Of course, this study has the limitation of being retrospective and the results are based on telephone interviews conducted with a very selective sample of addicts. Furthermore, a number of correlational studies have not found a relationship between cocaine use and violent behavior (e.g., Kozel & DuPont, 1977).

A common interpretation of the presumed relationship between cocaine and aggression, generally accepted by contemporary drug experts, is that the observed violence is due to systemic or economic influences (Fagan & Ko-Lin, 1990; Goldstein, 1989). In a review article appearing in the *Journal of the American Medical Association,* Hatsukami and Fischman (1996) asserted that "violence and crime associated with cocaine are considered to be in large part related to either the system of drug distribution (systemic crime) and/or economically or financially driven" (p. 1585). The authors went on to say that "in fact, violence directly induced by the pharmacological effects of cocaine hydrochloride or crack is considered uncommon" (p. 1585).

DEPRESSANTS

Depressants, sometimes labeled sedative hypnotics, decrease CNS activity. At low doses, depressants are often called anxiolytics or sedatives, and they are prescribed to reduce anxiety. At higher doses, they are labeled hypnotics and are used to induce sleep. Alcohol is one of the most commonly used sedative drugs. However, it is rarely used therapeutically. Two depressant drugs that have fallen out of favor recently in the medical community due to their addiction potential and adverse side effects are barbiturates and methaqualone. These depressants have been replaced, therapeutically, by benzodiazepines (minor tranquilizers).

Depressant drugs, such as barbiturates and benzodiazepines, are not generally considered to be facilitators of aggressive behavior. In fact, these drugs are commonly presumed to decrease aggression. Corrigan, Yudofsky, and Silver (1993) decided that "the greatest effects of these drugs on aggression is the sedation of patients who are currently assaultive . . ." (p. 127). The authors concluded, "We recommend use of these drugs during periods of current assaultive outbursts" (p. 127).

Gunn (1979) argued that barbiturates and aggression may be associated under only two conditions: when consumed with amphetamines and when used for suicide. While recognizing the remote possibility that benzodiazepines may enhance hostility, Gunn concluded, "Nevertheless, these drugs are useful sedatives and we do use them . . . in the violence clinic" (p. 189). Some clinical literature tends to support the perspective that benzodiazepines have calming, antiaggressive effects (e.g., Bond, Mandos, & Kurtz, 1989; Pilowsky, Ring, Shine, Battersby, & Lader, 1992; Rickels & Downing, 1974).

Although the clinical literature advocates the use of benzodiazepines for the acutely agitated patient, there have been reports of antisocial behavior following benzodiazepine ingestion. Increased anger, hostility, and aggression have been associated with benzodiazepines since they made their commercial appearance with the introduction of chlordiazepoxide in 1960 (DeMascio, Shader, & Giller, 1970; Tobin & Lewis, 1960). Numerous case reports and clinical studies have documented aggressive responses in some patients consuming benzodiazepines (Hall & Zisook, 1981; Rosenbaum, Woods, Groves, & Klerman, 1984; Salzman, Kochansky, Shader, Porrino, Harmatz, & Swett, 1974). The medical community has traditionally downplayed the importance of these case reports, labeling the observed hostile responses to benzodiazepine ingestion "paradoxical rage."

Medical incident reports tend to support the claim that aggression occurs infrequently in patients taking benzodiazepines. Svenson and Hamilton (1966) reported that only .24% of approximately 18,000 patients who had received chlordiazepoxide developed irritability as a side effect. The authors concluded that troublesome reactions were rare and their occurrence was due to excessive dosages. Miller (1973) reported that a combined total of approximately 1% of patients receiving chlordiazepoxide or diazepam developed excitement and agitation reactions. In a study of approximately 11,717 patients who received alprazolam, only 4 patients reported hostile reactions and 13 irritability (Dietch & Jennings, 1988). These authors concluded, "In most surveys the incidence of aggressive dyscontrol after benzodiazepines are administered is quite low (less than 1%) and is comparable to the effects produced by a placebo" (p. 186).

Contemporary investigators have begun to express their concern that benzodiazepines may facilitate aggression. For example, Ratey and Gordon (1993) stated, "The disinhibiting effects of benzodiazepines may lead to the precipitation of hostility or aggression, and are, therefore, a potential adverse effect of their use" (p. 66).

OPIATES

There appears to be minimal controversy concerning the direct effect of opiates on aggressive behavior. Most investigators assume that opiates decrease aggressive behavior. Powers and Kutash (1978) argued that the consumption of an opiate creates "euphoria, relaxation, drowsiness, and lethargy." These effects, according to

the authors, "reduce aggression rather than increase it" (p. 330). Goode (1993) recognized that the traditional view of the aggression-instigating properties of opiates has been based on the assumption that "since heroin sedates and tranquilizes, its effects incline the user *away* from aggressive, violent acts" (p. 137). Blue and Griffith (1995) stated, quite unequivocally, that "heroin intoxication has not been shown to induce violence" (p. 576). Goldstein (1989) concluded that "early reports, which sought to employ a psychopharmacological model to attribute violent behavior to the use of opiates and marijuana, have now been largely discredited" (p. 25). He assumed, instead, that violence associated with heroin is perpetrated to acquire money to secure more drugs and is an inherent risk of the drug trade.

The traditional perspective concerning the antiaggressive effects of opiates was based on the informal reports of opiate addicts. However, more controlled questionnaire studies have found evidence that opiates may facilitate aggressive behavior. For example, opiate-experienced participants often evidence higher, rather than lower, hostility scores on self-report or observer-reported measures (Babor, Meyer, Mirin, Davies, Valentine, & Rawlins, 1976; Lindquist, Lindsay, & White, 1979).

Miczek (1987) observed that "the evidence on opiates and human aggression ranges from the earlier practice of using acute morphine as an antiaggressive drug to the increasing concern with the high incidence of aggression and criminal behavior in narcotics addicts" (p. 253). He concluded, "It is surprising that the effects of acute and chronic opiates on aggression and violent behavior in humans have not been directly assessed in controlled experiments" (p. 253).

MARIJUANA

There has been a great deal of controversy concerning the relationship between marijuana and aggressive behavior. In the past, marijuana was suspected of instigating a wide variety of aggressive behaviors. In fact, these allegations had been used to support the position that marijuana should be prohibited. In the 1930s and 1940s, Harry Anslinger, the Commissioner of Narcotics, observed, "How many murders, suicides, robberies, criminal assaults, holdups, burglaries, and deeds of maniacal insanity it causes . . . can only be conjectured" (Kaplan, 1970, p. 89).

During the mid-1960s and 1970s the image of marijuana as the "killer weed" was modified. Investigators began to argue that, if anything, marijuana inhibits aggression. The National Commission on Marihuana and Drug Abuse, formed in response to the Comprehensive Drug Abuse Prevention and Control Act of 1970, conducted a study to determine whether marijuana caused violence. The survey of marijuana users suggested that marijuana was unlikely to cause violent crime (Goode, 1972). In 1977, Abel determined, after an extensive review of the literature, that marijuana was not an antecedent of aggression. Following a review of the literature, Mednick et al. (1982) argued, "Feelings of hostility or overt aggression are not caused by marijuana under either experimental or 'real-life' conditions" (p. 61). Mayfield (1983) observed, "It is generally agreed that cannabis use is associated with quiescence and passivity more than vigor and aggressivity" (p. 148).

In a study of 268 incarcerated participants, Spunt, Goldstein, Brownstein, and Fendrich (1994) found that a large percentage of homicide offenders had used marijuana in their lifetime (86%) and a number (32%) even used it on the day of the killing. However, the authors concluded that "marijuana rarely played a determining role in the homicides that were committed" (p. 209). Thus, there appears to be a consensus among investigators that marijuana is not an antecedent of aggression.

HALLUCINOGENS

The two drugs most commonly referred to as hallucinogens or psychedelics are LSD and PCP. LSD is one of the most common hallucinogenic drugs used in the United States. It appears to induce visual hallucinations and feelings of depersonalization. Consumers can suffer a number of untoward reactions, including panic reactions or "bad trips," over psychotic reactions and "flashbacks," or the reappearance of drug symptoms without further consumption. PCP tends to be mistakenly classified as a hallucinogen because it occasionally elicits hallucinations. However, following PCP ingestion, consumers do not experience vivid or unusual colors in their hallucinations or many of the other symptoms characteristic of LSD use. In fact, many of the subjective reactions are similar to those produced by sedative hypnotics. At large doses, however, some users have been observed to experience psychotic episodes, hallucinations, and convulsions.

From the early 1940s until the early 1960s, LSD was used extensively in psychotherapy. Few adverse reactions were reported. A survey was conducted by Cohen and Ditman (1963) of 44 therapists who provided LSD or mescaline to approximately 5000 patients. The most common problems associated with the use of LSD were psychotic reactions and suicide attempts. The authors reported that aggression was rare and consisted mainly of paranoid reactions. During the late 1960s, there was a rapid growth in the illicit use of LSD. Many instances of "bad trips" were documented, in which users experienced panic reactions.

There are many reports in the literature of aggressive responding associated with hallucinogen use (e.g., Fauman & Fauman, 1980; Reid, 1986; Siegel, 1980). However, the consensus in the literature appears to be that LSD is not a potent antecedent of aggressive behavior. It is assumed that aggression may occur as a result of disorganized responding during a panic reaction (Hurlbut, 1991). Mayfield (1983) concluded that hallucinogen-instigated aggression occurs "in the context of 'bad trips,' and the amount of assaultiveness relative to the use of these drugs is difficult to ascertain but is probably low" (p. 148). Powers and Kutash (1978) observed, "It is relatively uncommon for aggression and violence to occur under the influence of LSD . . ." (p. 337). According to Cohen (1981), "The most frequent cause of lethality during an LSD experience is accidental death" (p. 362).

There appears to be greater concordance concerning the instigating affects of PCP. Cohen (1981), for example, commented that "with the exception of phencyclidine, violent or criminal activities during the hallucinogenic state are infrequent" (p. 362). Blue and Griffith (1995) concluded that PCP has been "associated

with increased aggression" (p. 576). There is controversy, however, concerning the nature of the relationship between PCP and aggression. Some investigators contend that the violence is due to psychotic reactions (Fauman, Aldinger, & Fauman, 1976; Luisada & Brown, 1976). Others assert that violence associated with PCP is economically motivated. Wish (1986) examined a sample of 4847 arrestees and found that "many PCP users are apprehended for goal-oriented, income-generating crimes" (p. 187). The author indicated, "We did not find a preponderance of the types of offenses one might expect from persons committing the bizarre, irrational acts ascribed to PCP users" (p. 187).

The literature that suggests a relationship between hallucinogens and violence has been criticized methodologically. Sbordone, Gorelick, and Elliott (1981) observed, "Phencyclidine has recently been associated with pathological aggressive behavior in humans." The authors charged, "Its reputation, however, is based largely on anecdotal case reports of the bizarre behavior induced in PCP users, rather than on epidemiological or experimental data." Miczek (1987) also observed that "dramatic episodes of seemingly inexplicable violent behavior in hallucinogen-using individuals have led to frequent allegations that these drugs provoke violence" (p. 261). The author acknowledged, however, that "the empirical evidence is mostly limited to statistics from apprehended delinquents, clients of drug abuse clinics, case reports, or uncontrolled, open trials" (p. 261). Furthermore, he correctly recognized that the reports are based on the behaviors of polydrug users. Wilkens (1989) suggested that "despite reports of violence associated with PCP use by humans . . . a causal relationship between PCP and aggressive behavior remains unclear" (p. 277).

EXPERIMENTAL INVESTIGATION

Enormous quantities of licit as well as illicit drugs are consumed in our society. It has been speculated that one presumed consequence of this consumption is aggressive behavior, yet there is relatively little experimental evidence concerning the direct effects of acute drug use on human aggression. Experimentalists within the scientific community have devoted attention to the influence of psychoactive drugs on addiction and adverse medical effects. However, they have paid minimal attention to the possible disruptive influence of drugs on complex human social behavior. The American Psychological Association has intermittently expressed concern about the apparent relationship between substance abuse and aggression. For example, they conducted a "mini-convention" on the effects of substance abuse on aggressive behavior at the 1990 American Psychological Convention. Although many hours of programming were devoted to this critical issue, there was a paucity of controlled, experimental research on drug-elicited aggression in humans. The American Association for the Advancement of Science held a conference entitled, "Drugs, Crime and Violence: What Do We Know?" Once again, few researchers at the conference acknowledged utilizing experimental methodology to investigate the influence of drug consumption on human aggressive responding. In fact, there appeared to be a startling animosity toward the use of experimental methodology in the investigation of the relationship between drugs and human aggression.

There are conventional methods used to study the effects of drugs on aggression that are considered to be "acceptable." These methods rarely provoke hostile criticism. One popular method involves correlating subjects' descriptions of their drug use with self-reported aggressive behaviors. While potentially providing useful information, there are significant problems associated with this fashionable methodology. Many people are reluctant to reveal such personal and potentially incriminating information. Furthermore, it is difficult to ascertain causal relationships from such data. A second method that has been fiercely promoted by critics of experimental methodology involves naturalistic observation. This approach has been especially advocated by a small group of animal researchers (ethopharmacologists). This research approach is especially difficult with human subjects, as aggression has a low base rate in natural settings and it is a laborious task to control such important variables as dose in a natural environment. For example, it is difficult to assess how much of a drug a subject had ingested prior to a "natural" observation. The use of crime statistics, another common approach, can be troublesome due to sampling biases, unreliable police observations, and underreporting.

Our laboratory has been involved for a number of years in a program of research designed to experimentally investigate the effects of commonly used drugs on human aggressive behavior. The paradigm used in these controlled, laboratory experiments provides a subject with the opportunity to aggress against a bogus opponent while competing in a reaction time task. Prior to each competitive trial, the subject receives a signal to select the intensity of shock he wishes to administer to his competitor. The subject and his competitor then compete on a reaction time trial. The person with the slower reaction time receives the shock that had presumably been selected by the competitor. The person with a faster reaction time does not receive a shock. However, he is informed, by means of feedback lights, of the intensity of shock his competitor had set for him. Thus, the subject realizes that either he or his opponent will receive a shock, depending on the outcome of the trial, and that each can select the intensity of shock the other will receive. The measure of aggression used in this paradigm is the intensity of electric shock the subject selects for his opponent.

In comparison to case study and correctional designs, the experimental paradigm randomly assigns subjects to conditions, disguises the true nature of the experiments by telling the subjects that the purpose of the study is to examine the effects of drugs on performance variables, controls the dosage of the drug, obtains a direct, behavioral measure of the propensity to harm under controlled conditions, uses a normal, nondrug-using sample, and assesses the effects of an acute dose.

The reaction time paradigm provides a valid measure of aggressive behavior. The paradigm discriminates between groups of subjects theoretically expected to differ in aggression (e.g., Dengerink, 1971; Genthner & Taylor, 1973) and is

sensitive to situational variables expected to influence aggressive responding, such as social pressure and gender of target. In their review of contemporary paradigms, Baron and Richardson (1994) concluded, "The Taylor procedures do in fact yield a useful and valid measure of physical aggression" (p. 81). Bernstein, Richardson, and Hammock (1987) examined the convergent and discriminate validity of the Taylor paradigm and reported that the procedure provided a valid measure of aggressive behavior.

In a review of the literature on "drugs and violent crime," Goldstein (1989) concluded that "the need for better data to elaborate on these relationships is clear and pressing" (p. 41). He added, "It is important that we move beyond simple correlations between drug use and violent crime to achieve a real understanding of how drugs contribute to the process of violence" (p. 41). It is the authors' belief that the utilization of methodologies that actually manipulate the ingestion of a variety of drugs and monitor acts with a potential for physical harm or injury can most optimally contribute to this objective.

During the first stage of the research program, attention was focused on the potential instigative effects of alcohol on aggression. Prior to the initiation of this research, there was a paucity of controlled, experimental research concerning the effects of alcohol consumption on aggressive interactions. The research involved administering varying doses of alcohol to subjects in the reaction time paradigm. The results indicated that alcohol was a potent causal antecedent of aggressive behavior (Taylor & Leonard, 1983).

MARIJUANA

Following the successful attempt to study the effects of alcohol on aggression, an effort was made to investigate the effects of other commonly used psychoactive drugs. Studies were initially conducted to explore the effects of marijuana on aggressive responding. There appeared to be a consensus among investigators that marijuana was not a potent antecedent of aggression. The research that had been done, however, relied on retrospective case studies, anecdotal evidence, and correlational studies of the relation between marijuana and violent crime. These methodologies rely on unreliable self-reports of witnesses and cannot substantiate the presence of marijuana in an aggressor. Our research provided empirical support for the conviction that marijuana did not instigate aggressive responding. In the first study (Taylor, Vordaris, Rawtich, Gammon, Cranston, & Lubetkin, 1976), subjects were provoked by the confederate following their ingestion of high or low dosages of either alcohol or THC. As expected, aggression was found to be related to the quantity of alcohol ingested. The high dose of alcohol facilitated more intense aggression (higher shock settings) than the low dose. The high dose of THC, however, did not increase aggressive responding. In fact, it tended to suppress aggressive behavior. In the second study, conducted by Myerscough and Taylor (1985), subjects received intense provocation following their ingestion of one of three doses of THC. Subjects in the low dose condition responded in a more aggressive manner than subjects in the moderate and high dose conditions. Subjects in the high dose condition tended to respond in a relatively nonaggressive manner throughout the experimental session. The authors concluded that the results were "congruent with a growing consensus among drug investigators that marijuana does not instigate, precipitate, or enhance aggressive behavior" (p. 1555).

Cherek, Roache, Egli, Davis, Spiga, and Cowan (1993) challenged the revisionist position that marijuana decreases aggression. These authors investigated the effects of smoking marijuana on a behavioral measure of aggression. The participants in this study were eight "inner-city males with extensive drug use histories and self-reported "anti-social" behavior patterns" (p. 167). The study demonstrated that aggressive responding increased during the first hour after marijuana smoking. Given the small number and characteristics of the participants, the authors included the following caveat: "Determining the effects of marijuana smoking on aggressive responding among marijuana smokers with no other drug use history and not meeting criteria for anti-social personality disorder would be essential in interpreting the present data" (p. 167).

STIMULANTS

Our research, using an experimental approach, provided support for the traditional assumptions concerning the effects of both alcohol and marijuana on aggressive behavior. We next turned our attention to the possible instigating effects of CNS stimulants. As discussed earlier, there has been considerable disagreement concerning the instigating effects of stimulants. While arousal theories assume that acute amphetamine consumption may enhance aggression, there has been little evidence to support this perspective. In fact, there is some indication that amphetamines may actually decrease aggressive tendencies.

Three studies were conducted in our laboratory in an attempt to examine the direct effects of stimulants on aggressive behavior (Beezley, Gantner, Bailey, & Taylor, 1987). In these studies, subjects consumed varying dosages of dextroamphetamine prior to competing in the reaction time task. The results of the three studies were very consistent. While the high doses of amphetamine increased systolic and diastolic blood pressure, aggressive behavior was not found to increase as a function of amphetamine dosage. These findings are congruent with clinical evidence and demonstrate that amphetamines in acute, moderate doses do not appear to facilitate aggressive behavior. Following a review of the experimental literature, Bushman (1993) concluded that "CNS stimulants do not consistently facilitate or inhibit aggression" (p. 150).

Findings on the relationship between amphetamines and aggression have important theoretical as well as practical implications. It has traditionally been assumed that "arousal" is one of the major facilitators of aggression. Although amphetamines enhanced physiological arousal, they had a minimal impact on aggression. These findings indicate that researchers must reconsider the role of arousal in regulating aggressive behavior.

Cocaine has become a major drug of abuse. It has been estimated that approximately 30 million people have used cocaine in the United States. Many researchers believe that cocaine facilitates aggressive behavior (Honer et al., 1987; Miller et al., 1991; Siegal, 1982), whereas others tend to question this belief (Kozel & DuPont, 1977).

Licata, Taylor, Berman, and Cranston (1993) attempted to experimentally investigate the propensity of cocaine to facilitate aggression. Participants received a placebo, a low dose (1 mg/kg), or a high dose (2 mg/kg) of orally administered cocaine prior to competing in the competitive reaction-time task. Results indicated that subjects in the high dose condition behaved more aggressively than placebo subjects under all levels of provocation.

It has been suggested that the route of administration may influence cocaine-induced violence (Giannini, Miller, Loiselle, & Turner, 1993). One proposal is that the route which provides the quickest onset of intoxication would be most likely to enhance aggression. Findings reported by Licata et al. (1993) are not congruent with this proposition. Orally ingested cocaine does not provide direct entry of cocaine into the central nervous system, yet subjects who received the high dose of orally ingested cocaine selected higher levels of shock than the placebo subjects during each block of trials.

Cocaine and dextroamphetamine are central nervous system stimulants. However, research cited earlier suggests that moderate doses of cocaine may enhance aggression, whereas moderate doses of dextroamphetamine may have no appreciable influence on aggressive behavior. Future research must determine whether the variation in aggression observed was due to pharmacological differences between these drugs or the particular dosages used in the studies. The findings do underscore the possibility that two drugs within the same drug category may produce very different levels of aggressive responding.

DEPRESSANTS

There appears to be a consensus that opiates reduce aggressive responding. It is assumed that opiates decrease aggressive behavior by inducing a positive affective experience (Khantzian, 1974, 1985). Unfortunately, these beliefs of the antiaggressive properties of opiates tend to be based on informal, self-reports of opiate addicts.

As discussed earlier, Powers and Kutash (1978), who argued that opiates reduced aggression, believed that opiates produced "euphoria, relaxation, drowsiness, and lethargy" (p. 330). These adjectives quite accurately describe the subjective effects of other commonly used depressants. Given the fact that the effects of opiates such as morphine are similar to other depressants and that depressants such as alcohol tend to facilitate aggression, it is not unreasonable to argue that morphine might also increase aggressive behavior.

Berman, Taylor, and Marged (1993) attempted to examine the effects of an acute dose of morphine, a prototypical opiate, on a behavioral measure of aggression. Subjects were randomly assigned to either a morphine or an inactive placebo condition. They were then given the opportunity to aggress against an increasingly provocative opponent in the competitive reaction time task. Subjects in the morphine condition received 45 mg of immediate-release oral morphine sulfate placed in a gelatin capsule. Subjects in the placebo condition received an inactive placebo.

Subjects in the morphine condition initiated attacks against the opponent prior to receiving information about the opponent's aggressive intentions and responded more aggressively than the subjects in the placebo condition under all provocation levels. The results suggest the possibility that violent acts of opiate users might not be solely determined by economics (acquisition of drugs) and involvement in drug trafficking. It is very plausible to assume that the consumption of opiates itself facilitates aggressive behaviors. The authors concluded, "The results of this study suggest that the traditional view that opiates reduce aggressive behavior requires re-examination" (p. 267).

A similar controversy exists concerning the effect of benzodiazepines, the drug class of choice for the treatment of anxiety, on aggressive behavior. Since their introduction, numerous case studies have reported that benzodiazepines, such as diazepam and chlordiazepoxide, may facilitate hostility in psychiatric patients (e.g., Gardos, DiMascio, Salzman, & Shader, 1968). However, the medical community has continued to label the instances of aggressive behavior following benzodiazepine ingestion as "paradoxical rage reactions" as the response is contrary to their expectation of how a patient should respond to these agents. For example, in an article on the use of benzodiazepines in the treatment of anxiety disorders, Shader and Greenblatt (1993) concluded, "There is no evidence that benzodiazepines directly impair impulse control or conscience or lead to aggressive or self-destructive acts" (p. 1402).

Earlier reports of aggressive behavior following the ingestion of benzodiazepines were based on clinical descriptions using single-case designs, uncontrolled studies, and self-report measures of aggression. The first attempt to study the effect of a benzodiazepine on a direct measure of physical aggressive behavior, in a controlled laboratory setting, occurred in the early 1980s. Pagano (1981) monitored the aggressive behavior of male subjects in the reaction time paradigm after they had ingested a placebo, a 5-mg, or a 10-mg dose of diazepam. The subjects in the 10-mg condition were observed to display significantly higher levels of aggression as compared to subjects in the other groups. Furthermore, under high provocation, when the bogus opponent was setting high shocks, the 5-mg diazepam group behaved as aggressively as the 10-mg group.

The results of the Pagano study were quite surprising. We did not anticipate that a tranquilizer would increase the subjects' tendencies to administer intense noxious stimuli to peers, especially when the peers could retaliate. We have naively accepted the conventional belief that a "tranquilizer" produces a calm, peaceful state. The findings instigated a series of studies designed to explore the relationship between benzodiazepine consumption and aggressive behavior.

In a subsequent study, Wilkinson (1985) assessed the acute effects of diazepam on the aggressive behavior of subjects varying in trait anxiety. High, moderate, and low trait anxiety groups were administered either 10 mg of diazepam or a placebo capsule. Wilkinson reported that under conditions of high provocation, all anxiety trait groups who received diazepam had significantly higher levels of aggressive behavior than those who received a placebo. However, she also found that under minimal provocation conditions, in which the opponent set low intensity shocks, low anxious subjects administered diazepam were more aggressive than highly anxious subjects who received the drug. Thus, subjects who came into the laboratory with low anxiety levels were more aggressive under nonthreatening conditions than subjects who were anxious. Wilkinson concluded, "The results are consistent with reports of the ability of antianxiety drugs to disinhibit suppressed behaviors" (p. 101).

There is considerable evidence that a larger proportion of females than males use benzodiazepines. Yet there is a paucity of research investigating the interactive effects of diazepam and the sex of the aggressor on aggressive responding. Gantner and Taylor (1988) randomly administered diazepam (10 mg) or a placebo to male and female subjects prior to their participating in the paradigm. Results were similar to earlier studies: the aggression-enhancing effects of diazepam occurred for both male and female competitors.

A number of studies have related triazolam (Halcion), a benzodiazepine used to treat insomnia, with acts of aggression. For example, van der Kroef (1979) observed hostile responses in several patients who received triazolam. Regestein and Reich (1985) reported a series of cases in which agitation and anger were associated with triazolam. These behaviors were observed in patients with and without psychiatric histories.

To investigate the effects of triazolam on aggressive behavior, Berman and Taylor (1995) provided subjects with either a placebo or 0.25 mg of triazolam prior to their participation in the reaction time paradigm. The 0.25-mg dose was used in the study because it is the highest dose marketed in the United States and has been reported to produce greater cognitive effects than the only other available dose, 0.125 mg (Greenblatt, 1992).

The results of the study demonstrated, very convincingly, that triazolam enhanced aggressive behavior. Participants who received triazolam set more intense shock for the opponent than participants who received a placebo, prior to knowledge of the opponent's aggressive intent as well as during the competitive trials and selected the most extreme shock response for the opponent more frequently than the placebo subjects.

Bond and Lader (1988) examined the differential effects of two other benzodiazepines, oxazepam and lorazepam, on aggressive behavior. Subjects were administered these drugs or a placebo in a modification of the Taylor competitive paradigm (noise intensity was used instead of electric shock). The higher dose of lorazepam was reported to have increased aggressive behavior more than the oxazepam or placebo. Weisman (1991) conducted a study to compare the effects of

diazepam and oxazepam on aggression using the traditional paradigm. He observed that diazepam produced a higher level of aggression than oxazepam. Thus, in neither study did oxazepam, a less potent benzodiazepine, facilitate aggressive responding.

A major implication of this series of experiments is that the ingestion of depressants, such as benzodiazepines, may result in an escalation in aggressive behavior. Following a meta-analysis of the available evidence, Bushman (1993) concluded that "there is strong evidence to suggest that low doses of CNS depressants cause aggressive behavior in humans" (p. 150).

Depressant drugs that sedate but do not have potent anxiolytic properties may be less apt to instigate aggressive responding. Chermack and Taylor (1993) reported that neither secobarbital nor pentobarbital, potent sedatives with relatively weak anxiolytic attributes, had an appreciable impact on aggressive responding.

DISCUSSION

There is a great deal of controversy in the literature concerning the direct effects of psychoactive drugs on the expression of aggressive behavior. Traditional literature suggests that stimulants instigate and depressants inhibit aggressive acts. Recent experimental evidence suggests that these conventional assumptions are in need of revision.

The dissension in the field is partly due to the traditional methodologies used to study the relationships between drugs and aggression. Due to the multiple threats to validity inherent in these methodologies, the evidence generated by traditional research strategies must be interpreted with considerable caution.

In an attempt to rectify this problem, we have been conducting a program of research that involves administering a variety of psychoactive drugs to nonaddicted subjects in a controlled laboratory situation designed to monitor interpersonal physical aggression. The methodology assures the random assignment of subjects, provides placebo comparisons, disguises the true purpose of the experiments, and enables investigators to manipulate important experimental conditions.

The results of these experiments are consistent with traditional perspectives in three major respects. First, alcohol is a potent antecedent of aggressive responding. Second, marijuana does not appear to facilitate aggressive behavior. Finally, there is tentative evidence of a relationship between cocaine consumption and physical aggression. Many investigators have suggested that violence by cocaine users may simply be the by-product of the activities necessary to procure the drugs. Results of the study conducted by Licata et al. (1993) suggest that this perspective needs to be revised. One may recall that the authors found that subjects in the high dose condition reacted more aggressively than placebo subjects under all levels of provocation.

Our research findings are not congruent with conventional "wisdom" pertaining to the instigating effects of amphetamines, opiates, and benzodiazepines. First,

no appreciable evidence has been found that amphetamine consumption, at least in moderate doses, enhances aggression. Subjects who consumed moderate doses of dextroamphetamine were no more willing to harm their opponent than subjects who consumed a low dose of dextroamphetamine or a placebo. Subjects who received dextroamphetamine evidenced no affective or behavioral tendency that could be interpreted as offensive. This was decidedly the case in the experimental paradigm as well as in their interactions with the experimenters. Of course, future studies must investigate the effects of higher doses, chronic use, and divergent subject populations. Furthermore, researchers must attempt to delineate the factors responsible for the differential effects of amphetamine and cocaine on aggressive responding. Second, there is strong evidence of a relationship between morphine consumption and aggressive responding. Many studies have demonstrated a significant relationship between opiate use and violence. However, they have not been able to explain the nature of the relationship. Usually, the violence is attributed to the procurement of the drug. The study conducted by Berman et al. (1993) suggests that aggression can result from the psychopharmacological effects of morphine. Finally, traditional literature suggests that benzodiazepines reduce aggressive behavior. Our research has clearly and convincingly demonstrated that benzodiazepines can increase aggressive responding.

Given our understanding of the traditional literature, the experimenters were surprised to observe the aggressive responding of the subjects who consumed morphine and benzodiazepines and the nonaggressiveness of the subjects who consumed amphetamine. How can we account for the apparent discrepancies between our observations and the traditional literature on drugs and aggression? One possibility is that drug investigators and practitioners have been biased by traditional theories and opinions concerning the effects of CNS activity on affective processes.

One of the most pervasive beliefs in the biomedical and psychological communities is that a direct relationship exists between the activity of the CNS and both anxiety and aggression. Presumably, as the activity of the CNS increases, the probability of the occurrence of fear and aggression is elevated. Kirov (1989), a representative proponent of this established perspective, argues that anxiety can turn to fighting and vice versa due to the fact that both affective states have a similar origin: the "basic activity of the nervous system," the "normal functioning of the neuron itself" (p. 846). Due to the presumed parallel relationship between anxiety and aggression, it is posited that a drug which decreases anxiety would also decrease aggression and a drug which decreases aggression would also decrease anxiety. Thus, Kirov advocates a "rule of thumb" for medical professionals concerning the dispensing of drugs. He proposes that any drug which stimulates the CNS, such as amphetamine, would be expected to facilitate both aggression and anxiety and any drug which depresses the CNS would be expected to decrease both aggression and anxiety. Although fear and aggression would appear to be very different symptoms, they could both be treated with the same psychoactive substances. For example, if benzodiazepines decreased anxiety, they would also

be expected to decrease aggression. What if this anticipated relationship is not observed and a patient experienced an increase in aggression after being treated for anxiety with a benzodiazepine? Advocates of this "rule of thumb" would argue that this "rare" observation is due to an exceptionally high dosage of the substance or an "idiosyncratic" characteristic of the consumer. Anxiety and aggression are assumed to have a common origin and are positively correlated. Thus, aggressive behavior would not be expected from a drug that is suppose to decrease anxiety. The presence of aggression subsequent to the ingestion of a moderate dose of a tranquilizer would be considered "paradoxical." The resultant affective state would be interpreted as being due to some unspecified characteristic of the consumer, not the drug.

One major consequence of this pervasive view is that adverse clinical or behavioral drug reactions, such as hostility, may not be monitored or reported by physicians and drug investigators. Expectancies concerning drug effects may strongly bias the medical community against observing disruptive behavior. Manufacturers rely on physicians' reports of adverse drug reactions. Spontaneous reporting occurs when a physician reports that a particular patient has suffered an adverse reaction following the consumption of a drug. It is recognized that only a small proportion of adverse drug reactions are actually reported by doctors (Inman, 1972). Given the small proportion of serious side effects reported, it is not unreasonable to assume that an even smaller proportion of aggressive acts would be reported. Because of prevalent expectancies, a physician may fail to recognize that aggressive behaviors are drug related, a physician might not question patients about aggressive events, a patient may be reluctant to report interpersonal behaviors, and reports of incidents of aggression would most likely be interpreted as "idiosyncratic."

The psychological establishment envisions the dynamics of aggression in a similar manner. The most common conceptualization concerning the etiology of aggression in the psychological literature is that aggression is mediated by enhanced arousal. In a review of the aggression literature, Baron and Richardson (1994) stated, "Several formal theories of aggression (Berkowitz, 1981, 1988; Zillman, 1988) and some explanations for effects of other variables (e.g., noise) are based on the notion that aggression and arousal are closely related" (p. 263). Most traditional explanations of aggression theorize that there are certain antecedent conditions, such as frustration or noxious cues, that reliably elevate arousal states. The heightened arousal increases the probability that aggressive behavior will eventuate. An important implication of this pervasive model is that any drug that enhances arousal states facilitates hostile acts and any drug that decreases arousal states reduces aggression. Thus, stimulants, such as amphetamine, would be expected to increase aggressive behavior, whereas sedative hypnotics, such as minor tranquilizers, would be expected to decrease aggression. The prototypical impressions in the psychological literature are that of the crazed amphetamine addict or agitated patient who is subdued with a sedative. This picture fits the traditional conceptualization of a direct relationship between aggression and CNS arousal.

A second related reason for the failure to appreciate the aggression-enhancing potential of certain psychoactive drugs is justifiable apprehension concerning their potentially life-threatening medical side effects. The biomedical community concedes that psychoactive substances, licit and illicit, can produce adverse drug reactions. Most drugs affect many neurotransmitter systems and their effects are not localized to the CNS. The use of psychoactive substances, including psychotherapeutic drugs, can result in a wide range of adverse effects. A large number of drugs antagonize dopaminergic, adrenergic, acetylcholine, and histaminergic neuronal systems. Therefore, medical practitioners routinely monitor such symptoms as postural hypotension, weight gain, allergic reactions, cardiac conduction abnormalities, seizures, vomiting, menstrual dysfunction, dystonia, blurred vision, and hyperthermia. More specifically, there is serious concern in the medical community that stimulants, such as cocaine, could result in heart failure or a convulsion-induced halt in breathing. Depressants, such as ethyl alcohol and barbiturates, are acknowledged to produce life-threatening emergencies, such as respiratory depression, as well as vomiting, blurred vision, muscle weakness, anterograde amnesia, and circulatory collapse. Adverse medical effects of opiates, such as morphine and methadone, include respiratory and circulatory depression, leading to possible cardiac arrest as well as seizures, delirium, pruritus, and urticaria.

Because of the obvious seriousness of the aforementioned problems, the medical/pharmacological community tends to limit its focus to serious medical complications resulting from the pharmacodynamic effects of drugs. Insufficient attention is paid to adverse clinical or behavioral events. When describing the psychological side effects of a drug, there is a tendency to concentrate on the drug's immediate impact on the CNS (e.g., memory deficits) rather than on complex, interpersonal behavior.

Pre- and postmarketing surveillance of most drugs focuses on identifying adverse drug reactions that involve serious medical complications. It has been argued that by focusing attention on adverse drug reactions, the medical/pharmacological community may fail to observe important adverse clinical events (e.g., aggressive behavior). Fisher (1995) has argued that the types of postmarketing surveillance used by the FDA may compromise its ability to delineate and resolve side effects of psychoactive drugs. The system used by the FDA relies almost exclusively on judgments of physicians to identify serious medical reactions with little focus on clinical reactions. The FDA appears to have little interest in gathering information directly from patients who consume drugs. The approaches currently used to detect adverse drug reactions involve spontaneous physician reporting, physician case reports, consolidation of medical records on particular patients, and postmarketing studies initiated by drug manufacturers. Although these approaches may be useful for detection of medical events, they are relatively ineffective in detecting less serious drug reactions (e.g., alterations in interpersonal behavior).

There is a third possible reason for the failure of drug researchers to detect the instigating effects of widely used psychoactive drugs. Given the traditional training of experimental psychologists and psychopharmacologists, they have felt more comfortable utilizing animal models. They have been reluctant to use controlled, experimental methods to examine the effects of drugs on human aggressive behavior. This has led to many complications. For example, these researchers sometimes fail to appreciate that the effects of drugs on human aggressive behavior are mediated by complex social and cognitive processes. Furthermore, because of their inherent biases, they have a tendency to disregard the experimental evidence concerning the relationship between psychoactive substances and human aggression. These biases are clearly manifested in the material selected for inclusion in contemporary biopsychology textbooks (Carlson, 1994, 1995; Kalat, 1995; Pinel, 1997). These textbooks are devoid of experimental research pertaining to the relationship between psychoactive drugs and human aggression.

POLICY IMPLICATIONS

The Panel on the Understanding and Control of Violent Behavior was established, in 1989, in response to requests from a number of federal agencies (e.g., the National Science Foundation) to conduct a comprehensive analysis of violent behavior. The panel admitted that it was not possible to design a comprehensive national policy for preventing drug-induced aggression. This was based on the realistic appraisal that our understanding of the instigating effects of drugs was deficient. The panel, nevertheless, recommended the following policies: developing tactics to disrupt illegal drug market, monitoring drug usage of pretrial releases, implementing drug abuse treatment for criminals, designing drug abuse prevention projects, and the development of pharmacological therapies to reduce the craving for psychoactive drugs (Reiss & Roth, 1993).

Based on the evidence presented in this chapter, a very different list of recommendations could be generated. First of all, we should not be influenced by the artificial distinction between licit and illicit drugs. A wide variety of drugs, some provided by means of prescriptions, have the potential to facilitate aggressive behavior. There is mounting evidence that drugs which depress the CNS, such as minor tranquilizers, alcohol, and morphine, have the potential to enhance aggression. Some of these drugs are obtained legally, others are acquired illegally. Texts and journal articles on the relationship between drug use and aggression all too often deal exclusively with illicitly acquired drugs.

Second, we must stimulate and promote experimental as well as correlational research on the effects of psychoactive drugs on human aggression. This will require financial support from federal and state agencies, interdisciplinary cooperation among a wide variety of disciplines, and the development of alternative experimental methodologies. Of even greater importance is the need for supportive, enlightened attitudes in the scientific establishment. It is acceptable to study the instigating effects of drugs on animal aggression by means of experimental paradigms. However, due to ethical concerns, intransigent negative perspectives concerning social psychological research, and the misguided belief that the only

viable means of studying human behavior is through self-report, some investigators question the credibility of experimental paradigms. Thus, Leccese (1991) argued that "controlled experiments may be difficult to conduct in an ethical manner . . . [because] . . . it may be that violent behavior can only be induced by doses that are sufficient to cause organ damage or other behavioral toxicities" (p. 200). This position is unfortunate as controlled laboratory experiments are not only feasible, but crucial for delineating the complex relationships between drug use and aggression. The National Institute on Drug Abuse published a monograph entitled, "Drugs and Violence: Causes, Correlates, and Consequences." The manuscript, edited by De La Rosa, Lambert, and Gropper (1990), covered such topics as distribution of crack, gangs, mental illness, and drug sales. Not one paper even alluded to human experimental research.

Third, the biomedical establishment must appreciate that psychoactive drugs have interpersonal consequences. This requires pre- and postmarketing assessments of drug-induced behavioral dysfunction, routine monitoring by physicians of the aggressive behaviors of patients consuming psychoactive drugs, enhanced interest by the FDA in gathering behavioral information directly from patients who consume drugs, and educational programs to inform the public of the behavioral consequences of drug use.

Fourth, while recognizing the importance of social, economic, and developmental factors, it is essential that we acknowledge that psychopharmacological factors play a decisive role in determining whether a particular drug will facilitate aggressive behavior. Efforts to understand the role of these underlying biological mechanisms will eventually provide us with the knowledge to prevent and control drug-induced aggressive behavior.

Following a review of the literature concerning the effects of alcohol on aggression, Taylor and Leonard (1983) concluded that alcohol was a potent antecedent of aggression. The authors recommended that researchers attempt to delineate the variables that interact with alcohol to produce aggressive responding and strive to understand the processes that mediate the relationship. In the years following this review, researchers have expended considerable energy investigating the dynamics of the alcohol–aggression relationship. In 1993, Taylor and Chermack reviewed the evidence concerning the relationship between alcohol/drugs and aggression. They confirmed the earlier conclusion that aggressive behavior is related to alcohol consumption. The authors also indicated that there was sufficient evidence to suggest that certain prescription medications could facilitate hostile behavior. Taylor and Chermack counseled, "While depressants such as diazepam may reduce anxiety and be helpful in the treatment of insomnia, they may also result in impaired judgment and a propensity to behave aggressively" (p. 80). The current authors reaffirm this position and recommend that contemporary investigators reconsider their beliefs concerning the potential instigating effects of licit as well as illicit psychoactive drugs. Collins (1991) concluded, "There is virtually no evidence that the pharmacological effects of drugs (alcohol excepted) account for a substantial proportion of drug-related violence" (p. 265). Due to the lack of epidemiological data, it is impossible to currently estimate the degree of violence instigated by the consumption of prescription drugs. However, there is sufficient anecdotal, case study, and experimental evidence to contradict this unsophisticated and intransigent position. Given the current empirical evidence suggesting the presence of pharmacological-induced aggression, it is sobering to reflect on the fact that "during the last 25 years it has been estimated that over 500 million people worldwide have taken a course of benzodiazepine treatment" (Leonard, 1993, p. 99).

REFERENCES

Abel, E. (1977). The relationship between cannabis and violence: A review. *Psychological Bulletin, 84,* 193–211.

Allen, R. P., Safer, D., & Covi, L. (1975). Effects of psychostimulants on aggression. *The Journal of Nervous and Mental Diseases, 160(2),* 138–145.

Babor, T. F., Meyer, R. E., Mirin, S. M., Davies, M., Valentine, N., & Rawlins, M. (1976). Interpersonal behavior in a small group setting during the heroin addiction cycle. *The International Journal of the Addictions, 11,* 513–523.

Baron, R. A., & Richardson, D. R. (1994). *Human aggression* (2nd ed.). New York: Plenum Press.

Beezley, D. A., Gantner, A. B., Bailey, D. S., & Taylor, S. P. (1987). Amphetamines and human physical aggression. *Journal of Research in Personality, 21,* 52–60.

Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control.* New York: McGraw-Hill.

Berman, M., & Taylor, S. (1995). The effects of triazolam on aggression in men. *Experimental and Clinical Psychopharmacology, 3,* 411–416.

Berman, M., Taylor, S., & Marged, B. (1993). Morphine and human aggression. *Addictive Behaviors, 18,* 263–268.

Bernstein, S., Richardson, D., & Hammock, G. (1987). Convergent and discriminate validity of the Taylor and Buss measures of physical aggression. *Aggressive Behavior, 13,* 15–24.

Blue, H. C., & Griffith, E. E. H. (1995). Sociocultural and therapeutic perspectives on violence. *The Psychiatric Clinics of North America, 18,* 571–587.

Bond, A., & Lader, M. (1988). Differential effects of oxazepam and lorazepam on aggressive responding. *Psychopharmacology, 95,* 369–373.

Bond, W. S., Mandos, L. A., & Kurtz, M. B. (1989). Midazolam for aggressivity and violence in three mentally retarded patients. *American Journal of Psychiatry, 146,* 925–926.

Brody, S. L. (1990). Violence associated with acute cocaine use in patients admitted to a medical emergency department. In M. De La Rosa, E. Y. Lambert, & B. Gropper (Eds.), *Drugs and violence: Causes, correlates, and consequences* (NIDA Research Monograph No. 103, pp. 44–59). Washington, DC: U.S. Government Printing Office.

Burrows, K. L., Hales, R. E., & Arington, E. (1988). Research on the biological aspects of violence. *The Psychiatric Clinics of North America, 11,* 499–509.

Bushman, B. (1993). Human aggression while under the influence of alcohol and other drugs: An integrative research review. *Current Directions in Psychological Science, 2,* 148–152.

Carlson, N. R. (1994). *Physiology of behavior* (5th ed.). Boston: Allyn and Bacon.

Carlson, N. R. (1995). *Foundations of Physiological Psychology* (3rd ed.). Boston: Allyn and Bacon.

Cherek, D. R., Roache, J. D., Egli, M., Davis, C., Spiga, R., & Cowan, K. (1993). Acute effects of marijuana smoking on aggressive, escape and point-maintained responding of male drug users. *Psychopharmacology, 111,* 163–168.

Chermack, S., & Taylor, S. P. (1993). Barbiturates and human physical aggression. *Journal of Research in Personality, 27,* 315–327.

Cohen, S. (1981). *The substance abuse problems.* New York: The Hawthorne Press.

Cohen, S., and Ditman, K. (1963). Prolonged adverse reactions to lysergic acid diethylamide. *Archives of General Psychiatry, 8,* 475–480.

Cole, J. O., & Kando, J. C. (1993). Adverse behavioral events reported in patients taking alprazolam and other benzodiazepines. *Journal of Clinical Psychiatry, 54(Suppl. 10),* 49–61.

Collins, J. J. (1990). Summary thoughts about drugs and violence. In M. De La Rosa, E. Y. Lambert, & B. Gropper (Eds.), *Drugs and violence: Causes, correlates, and consequences* (NIDA Research Monograph No. 103, pp. 265–275). Washington, DC: U.S. Government Printing Office.

Connor, D. F., & Steingard, R. J. (1996). A clinical approach to the pharmacotherapy of aggression in children and adolescents. *Annals of the New York Academy of Sciences, 794,* 290–307.

Corrigan, P. W., Yudofsky, S. C., and Silver, J. M. (1993). Pharmacological and behavioral treatments for aggressive psychiatric inpatients. *Hospital and Community Psychiatry, 44,* 125–133.

De La Rosa, M., Lambert, E. Y., & Gropper, B. (Eds.) (1990). *Drugs and violence: Causes, correlates, and consequences* (NIDA Research Monograph No. 103). Washington, DC: U.S. Government Printing Office.

Dengerink, H. A. (1971). Anxiety, aggression, and physiological arousal. *Journal of Experimental Research in Personality, 5,*223–232.

Dietch, J. T., & Jennings, R. K. (1988). Aggressive dyscontrol in patients treated with benzodiazepines. *Journal of Clinical Psychiatry, 49,* 184–188.

DiMascio, A., Shader, R. I., and Giller, D. R. (1970). Behavior toxicity. In R. I. Shader & A. DiMascio (Eds.), *Psychotropic drug side effects: Clinical and theoretical perspectives* (pp. 132–141). Baltimore: Williams & Wilkins Co.

Ellinwood, E. H. (1971). Assault and homicide associated with amphetamine abuse. *American Journal of Psychiatry, 127,* 1170–1175.

Fagan, J., & Ko-lin, C. (1990). Violence as regulation and social control in the distribution of crack. In M. De La Rosa, E. Y. Lambert, & B. Gropper (Eds.), *Drugs and violence: Causes, correlates, and consequences* (NIDA Research Monograph No. 103, pp. 8–43). Washington, DC: U.S. Government Printing Office.

Fauman, B. A., Aldinger, G., & Fauman, M. (1976). Psychiatric sequelae of phencyclidine abuse. *Clinical Toxicology, 9,* 529–538.

Fauman, M. A., & Fauman, B. J. (1980). Chronic phencyclidine (PCP) abuse: A psychiatric perspective. *Psychopharmacology Bulletin, 16,* 70–72.

Fisher, S. (1995). *Patient self-monitoring: A challenging approach to pharmacoepidemiology. Pharmacoepidemiology and Drug Safety, 4,* 359–378.

Gantner, A. B., & Taylor, S. P. (1988). Human physical aggression as a function of diazepam. *Personality and Social Psychology Bulletin, 14,* 479–484.

Gardos, G., DiMascio, A., Salzman, C., & Shader, R. (1968). Differential actions of chlordiazepoxide and oxazepam on hostility. *Archives of General Psychiatry, 18,* 757–760.

Genthner, R. W., & Taylor, S. P. (1973). Physical aggression as a function of racial prejudice and the race of the target. *Journal of Personality and Social Psychology, 27,* 207–210.

Giannini, A. J., Miller, N. S., Loiselle, R. H., & Turner, C. E. (1993). Cocaine-associated violence and relationship to route of administration. *Journal of Substance Abuse Treatment, 10,* 67–69.

Goldstein, P. J. (1989). Drugs and violent crime. In N. A. Weiner & M. E. Wolfgang (Eds.), *Pathways to criminal violence* (pp. 16–48). Newbury Park, CA: Sage.

Goldstein, P. J., Bellucci, P. A., Spunt, B. J., & Miller, T. (1991). In S. Schober & C. Schade (Eds.), *The epidemiology of cocaine use and abuse* (NIDA Research Monograph No. 110, pp. 113–138). Washington, DC: U.S. Government Printing Office.

Goode, E. (1972). Marijuana use and crime. In National Commission on Marihuana and Drug Abuse, *Marihuana: A signal of misunderstanding* (Vol. 1., pp. 447–469). Washington, DC: U.S. Government Printing Office.

Goode, E. (1993). *Drugs in american society* (4th ed.). New York: McGraw-Hill.

Greenblatt, D. (1992). Pharmacology of benzodiazepine hypnotics. *Journal of Clinical Psychiatry, 56(Suppl. 6),* 7–13.

Gunn, J. (1979). Drugs in the violence clinic. In M. Sandler (Ed.), *Psychopharmacology of aggression* (pp. 183–195). New York: Raven Press.

Hall, R. C. W., & Zisook, S. (1981). Paradoxical reactions to benzodiazepines. *British Journal of Clinical Pharmacology, 11,* 995–1045.

Hatsukami, D. K., & Fischman, M. W. (1996). Crack cocaine and cocaine hydrochloride: Are the differences myth or reality? *Journal of the American Medical Association, 276,* 1580–1588.

Honer, W. G., Gewirtz, G., & Turey, M. (1987). Psychosis and violence in cocaine smokers. *Lancet, 2,* 451.

Hurlbut, K. M. (1991). Drug-induced psychoses. *Emergency Medicine Clinics of North America, 9,* 31–52.

Inman, W. (1972). Monitoring by voluntary reporting at national level. In D. Richards & R. Rondell (Eds.), *Adverse drug reactions.* Baltimore: Williams & Wilkins, Co.

Kalat, J. W. (1995). *Biological Psychology* (5th ed.). Pacific Grove, CA: Brooks/Cole.

Kaplan, J. (1970). *Marijuana: The new prohibition.* New York: World.

Kermani, E. J., & Castaneda, R. (1996). Psychoactive substance use in forensic psychiatry. *American Journal of Drug and Alcohol Abuse, 22,* 1–27.

Khantzian, E. J. (1974). Opiate addiction: A critique of theory and some implications for treatment. *American Journal of Psychotherapy, 28,* 59–71.

Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: Focus on heroin and cocaine dependence. *The American Journal of Psychiatry, 142,* 1259–1264.

Kirov, K. (1989). Aggressiveness, anxiety and drugs. *British Journal of Psychiatry, 155,* 846.

Klerman, G. (1983). The prevalence and impact of mental illness on society. In R. L. Habig (Ed.), *The brain, biochemistry, and behavior.* Washington, DC: American Association for Clinical Chemistry.

Kozel, N. J., & DuPont, R. L. (1977). Trends in drug use and crime and their relationship in an arrested population. *American Journal of Drug and Alcohol Abuse, 4,* 413–429.

Leccese, A. P. (1991). *Drugs and society: Behavioral medicines and abusable drugs.* Englewood Cliffs, NJ: Prentice Hall.

Leonard, B. E. (1992). *Fundamentals of psychopharmacology.* New York: John Wiley & Sons.

Licata, A., Taylor, S., Berman, M., & Cranston, J. (1993). Effects of cocaine on human aggression. *Pharmacology, Biochemistry and Behavior, 45,* 549–552.

Lindquist, C. U., Lindsay, J. S., & White, G. D. (1979). Assessment of assertiveness in drug abusers. *Journal of Clinical Psychology, 35,* 676–679.

Luisada, P. C., & Brown, B. I. (1976). Clinical management of the phencyclidine psychosis. *Clinical Toxicology, 9,* 539–545.

Mayfield, D. (1983). Substance abuse and aggression: A psychopharmacological perspective. In E. Gottheil, K. Druley, T. Skoloda, & H. Waxman (Eds.), *Alcohol, drug abuse and aggression* (pp. 139–149). Springfield: Charles Thomas.

Mednick, S., Pollock, V., Volavka, J., & Gabrielli, W., Jr. (1982). Biology and violence. In M. Wolfgang & N. Weiner (Eds.), *Criminal violence.* Beverly Hills, CA: Sage.

Meloy, J. R. (1987). The prediction of violence in outpatient psychotherapy. *American Journal of Psychotherapy, 41,* 38–45.

Miczek, K. (1987). The psychopharmacology of aggression. In L. Iversen, S. Iversen, & S. Snyder (Eds.), *Handbook of psychopharmacology* (pp. 183–328). New York: Plenum Press.

Miller, N. S., & Gold, M. S. (1994). Criminal activity and crack addiction. *The International Journal of the Addictions, 29,* 1069–1078.

Miller, N. S., Gold, M. S., & Mahler, J. C. (1991). Violent behaviors associated with cocaine use: Possible pharmacological mechanisms. *The International Journal of the Addictions, 26,* 1077–1088.

Miller, R. R. (1973). Drug surveillance utilizing epidemiologic methods: A report from the Boston Collaborative Surveillance Program. *American Journal of Hospital Pharmacology, 30,* 584–592.

Moss, H. B., Salloum, I. M., & Fisher, B. (1994). Psychoactive substance abuse. In M. Hershen, R. T. Ammerman, & L. A. Sisson (Eds.), *Handbook of aggressive and destructive behavior in psychiatric patients* (pp. 175–201). New York: Plenum Press.

Myerscough, R., & Taylor, S. P. (1985). The effects of marijuana on human physical aggression. *Journal of Personality and Social Psychology, 49,* 1541–1546.

Pagano, M. R. (1981). *The effects of diazepam (Valium) on human physical aggression.* Unpublished doctoral dissertation, Kent State University, Kent, OH.

Pilowsky, L. S., Ring, H., Shine, P. J., Battersby, M., & Lader, M. (1992). Rapid tranquillisation: A survey of emergency prescribing in a general psychiatric hospital. *British Journal of Psychiatry, 160,* 831–835.

Pinel, J. P. J. (1997). *Biopsychology* (3rd ed.). Boston: Allyn and Bacon.

Powers, R., & Kutash, I. (1978). Substance-induced aggression. In I. Kutash, S. Kutash, & L. Schlesinger (Eds.), *Violence: Perspectives on murder and aggression.* San Francisco: Jossey-Bass.

Ratey, J. J., & Gorden, A. (1993). The psychopharmacology of aggression: Toward a new day. *Psychopharmacology Bulletin, 29,* 65–73.

Ray, S., & Ksir, C. (1990). *Drugs, society and human behavior* (5th ed.). St. Louis: Times Mirror/Mosby.

Regestein, Q. R., & Reich, P. (1985). Agitation observed during treatment with newer hypnotic drugs. *Journal of Clinical Psychiatry, 46,* 280–283.

Reid, G. W. (1986). Acute care for the violent patient. *The Journal of the Arkansas Medical Society, 82,* 541–543.

Reiss, A. J., Jr., & Roth, J. A. (Eds.) (1993). *Understanding and preventing violence.* Washington, DC: National Academy Press.

Rickels, K., & Downing, R. W. (1974). Chlordiazepoxide and hostility in anxious outpatients. *American Journal of Psychiatry, 131,* 442–444.

Rivinus, T. M., & Larimer, M. E. (1993). Violence, alcohol, other drugs, and the college student. *Journal of College Student Psychotherapy, 8,* 71–119.

Rosenbaum, J. F., Woods, S. W., Groves, J. E., & Klerman, G. L. (1984). Emergence of hostility during alprazolam treatment. *American Journal of Psychiatry, 141,* 792–793.

Salzman, C., Kochansky, G. E., Shader, R. I., Porrino, L. J., Harmatz, J. S., & Swett, C. P., Jr. (1974). Chlordiazepoxide-induced hostility in a small group setting. *Archives of General Psychiatry, 31,* 401–405.

Sbordone, R., Gorelick, D., & Elliott, M. (1981). An ethological analysis of drug-induced pathological aggression. In P. Brain & D. Benton (Eds.), *Multidisciplinary approaches to aggression research.* New York: Elsevier/North-Holland Biomedical Press.

Shader, R. I., & Greenblatt, D. J. (1993). Use of benzodiazepines in anxiety disorders. *The New England Journal of Medicine, 328,* 1398–1405.

Sheard, M. H. (1988). Clinical pharmacology of aggressive behavior. *Clinical Neuropharmacology, 11,* 483–492.

Siegal, R. K. (1980). PCP and violent crime: The people vs. peace. *Journal of Psychedelic Drugs, 12,* 317–330.

Siegal, R. K. (1982). Cocaine smoking. *Journal of Psychoactive Drugs, 14,* 272–341.

Spunt, B., Goldstein, P., Brownstein, H., & Fendrich, M. (1994). The role of marijuana in homicide. *The International Journal of the Addictions, 29,* 195–213.

Svenson, S. E., & Hamilton, R. G. (1966). A critique of overemphasis on side effects with the psychotropic drugs: An analysis of 18,000 chlordiazepoxide-treated cases. *Current Therapeutic Research, 8,* 455–464.

Taylor, S. P., & Chermack, S. T. (1993). Alcohol, drugs and human physical aggression. *Journal of Studies on Alcohol (Suppl. 11),* 78–88.

Taylor, S. P., & Leonard, K. E. (1983). Alcohol and human physical aggression. In R. G. Geen & E. I. Donnerstein (Eds.), *Aggression: Theoretical and empirical reviews* (2nd ed., pp. 77–101). San Diego, CA: Academic Press.

Taylor, S. P., Vardaris, R. M., Rawtich, A. B., Gammon, C. B., Cranston, J. W., & Lubetkin, A. I. (1976). The effects of alcohol and delta-9-tetrahydrocannabinol on human physical aggression. *Aggressive Behavior, 2,* 153–161.

Tobin, J. M., & Lewis, N. D. C. (1960). New psychotherapeutic agent, chlordiazepoxide: Use in treatment of anxiety states and related symptoms. *Journal of the American Medical Association, 174,* 1242–1249.

van der Kroef, C. (1979). Reactions to triazolam. *Lancet, 2,* 526.

Watters, J. K., Reinarman, C., & Fagan, J. (1985). Causality, context and contingency: Relationships between drug use and delinquency. *Contemporary Drug Problems, 15,* 351–373.

Weisman, A. M. (1991). *The effect of diazepam, clorazepate, and oxazepam on human physical aggression.* Unpublished doctoral dissertation, Kent State University, Kent, OH.

Wetli, C. V., & Fishbain, D. A. (1985). Cocaine-induced psychosis and sudden death in recreational cocaine users. *Journal of Forensic Science, 30,* 873–880.

Wilkens, J. (1989). Clinical implications of PCP, NMDA and opiate receptors. In L. S. Harris (Ed.), *Problems of drug dependence 1989: Proceedings of the 51st annual meeting of the Committee on Problems of Drug Dependence* (NIDA Research Monograph No. 95, pp. 275–281). Washington, DC: U.S. Government Printing Office.

Wilkinson, C. J. (1985). Effects of diazepam (Valium) and trait anxiety on human physical aggression and emotional state. *Journal of Behavioral Medicine, 8,* 101–114.

Wish, E. D. (1986). PCP and crime: Just another illicit drug? In D. H. Clovet (Ed.), *Phencyclidine: An update* (NIDA Research Monograph No. 64, pp. 174–189). Washington, DC: U.S. Government Printing Office.

Wish, E. D., & Johnson, B. D. (1986). The impact of substance abuse on criminal careers. In A. Blumstein, J. Cohen, J. A. Roth, & C. A. Visher (Eds.), *Criminal careers and career criminals* (pp. 52–88). Washington, DC: National Academy Press.