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Alcohol/Drug Use and Aggressive Behavior

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Rates of interpersonal violence appear to be increasing in U.S. society. In the United States today, the homicide rate is roughly double what it was in 1960; violence is the leading cause of death for individuals under 45 years of age; a rape, a murder, or an assault occurs every 25 seconds; and six million individuals each year are victims of violent crime (Langan & Innes, 1985). At various times observers have attributed this apparent increase in aggression to many different factors. The breakdown of the nuclear family, the collapse of the moral community structure, and the presence of nonstop violence on television have all been implicated. Currently, however, the number one candidate is drug abuse.

Two questions must be addressed by anyone interested in considering the relationship between drug use and violence. To begin with, how is drug-related violence associated with the distribution of illegal drugs, and/or with the concomitant "war" that has evolved? Nadelmann (1989) lists four possible modes of connection. The pharmacological effect of drugs of abuse is irrelevant, with regards to three of these modes. First, illicit drug users commit crimes, sometimes violent crimes, to gain access to drugs. Second, drug use and violent behavior exist coincidentally because the factors (e.g., antisocial personality) that predispose to both may be similar. Third, those involved in the drug trade necessarily resolve issues/disputes by violent means. This chapter focuses on Nadelmann's fourth connection, the second question: How does the ingestion of certain drugs affect aggression? More specifically, this chapter will concentrate on the relationship between alcohol and aggression.

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as it is in this domain that a substantial body of clinical and experimental knowledge exists. Enough information appears to exist to justify development of theories accounting for the alcohol-intoxication/aggression relationship, and one such theory is described later. This chapter additionally includes a brief discussion concerning the nature of the relationship between use of other drugs and violence.

Another, more specific, way to phrase the immediate question of concern is: How are the neurochemical processes and neurological circuits that control aggressive behavior affected by chemical intoxication? While this question is definitely germane, its utility is limited by a number of factors. First, an extensive body of literature remains conflicted as to the neurochemical and physical bases of aggression. Second, aggression is a behavioral concept, and cannot be regarded solely as a physiological process, except at a very primitive level. Any theory that attempts to account for drug-induced aggression must therefore be able to account for variance introduced by factors of personality, previous learning, and immediate context. Third, and finally, the picture is complicated by the fact that the neurochemical circuits affected by particular drugs are in many cases nonspecific in fact, or are poorly identified for methodological reasons.

Alcohol and Violence

Crime statistics and controlled experiments both demonstrate that heavy drinking and violence co-occur. Studies analyzing crime statistics are unusually unanimous. Alcohol-intoxicated individuals are involved in the majority of violent crimes, including murders, assaults, sexual assaults, and family violence (Brain, 1986; Collins, 1981; Murdoch, Pihl, & Ross, 1990; Pernanen, 1976, 1981). Murdoch et al. (1990) examined 9,304 cases reported in 26 studies from 11 countries. Overall, 62% of violent offenders were drinking at the time of the crime. Comparison between studies revealed that the range of alcohol-related violent crime was 24% to 85%, which contrasted dramatically with the range reported for alcohol-related nonviolent crimes: 12% to 38%. It might be argued additionally that these figures are underestimates as it is an irregular and relatively recent occurrence for police to record physical measures of alcohol use. Typically, in most crime reports, there is merely a statement that the perpetrator of the crime was intoxicated. Those few studies examining physical measures of inebriation have concluded that individuals involved in violence were often drinking heavily, to levels well above those of legal intoxication (Mayfield, 1976; Shupe, 1954).

Murdoch et al.'s (1990) review of 26 studies also demonstrated that 45% of victims of violence were intoxicated when victimized. One of the first studies of victim-precipitated homicide found that 60% of victims had been drinking heavily (Wolfgang & Strohan, 1956). This figure compares with that of the 47% estimated for victims drinking in offender-precipitated cases. The fact that each individual involved in a violent encounter is likely intoxicated might simply be a function of the environment in which aggression is likely to be expressed, rather than an effect of alcohol *per se*. In addition, crime statistics might also be affected by the fact that intoxicated individuals are perhaps both more likely to be apprehended and less likely to provide appropriate aid to their victims. Thus there are sufficient arguments to challenge any notion of causality in regard to the alcohol-aggression relationship, in the context of the real world. Nonetheless controlled laboratory studies also demonstrate that a relationship between alcohol and violence exists, and suggest that intoxication may play a direct role in increasing aggression and victimization.

Laboratory Studies and Theories of Intoxicated Behavior

A recent meta-analytic review of 30 experimental studies concluded that "alcohol does indeed cause aggression" (Bushman & Cooper, 1990, p. 341). Nonetheless the alcohol-aggression relationship is hardly direct. Numerous variables act as strong modifiers, as Bushman and Cooper (1990) and the authors of several additional alcohol-aggression reviews have noted (Evans, 1980; Pihl, 1983; Pihl & Ross, 1987; Taylor, 1983). Alcohol intoxication by no means provides the necessary and sufficient precondition for aggressive behavior. Anthropological studies (reviewed in MacAndrew & Edgerton, 1969) illustrate this fact, demonstrating large cultural variations in aggression-linked intoxicated behavior. MacAndrew and Edgerton (1969) detail the consequences of extreme drunkenness among members of numerous cultures, and note little change in social behavior when the drinking occurs in certain well-defined contexts. In addition, the behavior of intoxicated women in general is not congruent with that of intoxicated men, and there is of course marked variability in aggression among drunk males. In fact, the experimental studies on alcohol and aggression appear to demonstrate that various cognitions, social pressures, and dispositions toward aggressive behavior both increase and decrease alcohol-related aggression.

Theorists of intoxicated aggression (reviewed by Bushman & Cooper, 1990; Graham, 1980) posit that alcohol produces aggression because of disinhibition, stimulation-arousal, and/or because of social expectancies.

Theories of disinhibition postulate that normal physiological and/or cognitive processes are reduced or rendered dysfunctional during intoxication. These theories are predicated on the assumption that aggression results from an absence or diminution of normal behavioral braking, which occurs either because relevant brain areas are reduced in function and/or because relevant cognitive processing is obviated. Arousal theorists adopt the opposite position, hypothesizing that those physical mechanisms involved in aggressive behavior are directly stimulated by the ingestion of alcohol. The accelerator is depressed, so to speak, and relevant brain areas are ignited. Finally, social-expectancy theorists posit psychological reasons for aggression and argue that alcohol simply provides a rationale for unacceptable behavior—they blame the bottle. A variation of this position is that the problem is situational: Drinking occurs in provocative environments, likely in themselves to increase aggression. These theoretical views are neither mutually exclusive nor collectively exhaustive. Recent experiments conducted in the authors' laboratory have produced results that would support or rebut each of these general theoretical positions, if considered individually.

An Interactive Process

Figure 14.1 is based upon the obvious conclusion that any explanation of the alcohol-aggression relationship cannot be predicated upon purely pharmacological, cognitive, or social considerations. Each factor modifies the other in turn and is further modified by the resultant behavior. This conclusion is not posited to negate theory-building, as the presence of each of these factors contributes to the final consequence. Indeed, the nature of each of these factors must first be assessed, and specific predictive statements generated and tested before modes of interaction can finally be identified. Prior to discussing the role of alcohol per se in producing aggression (and briefly commenting upon the contributing role of each additional factor), some basic issues of methodology and definition must be addressed.

It has proved difficult to determine precisely how the central nervous system is affected by ethanol. Alcohol flows through the body like water, and easily penetrates the blood-brain barrier. It bathes all of the body's tissues. Ethanol's effects are dependent upon dose, rate of administration, time passed postconsumption, and subject characteristics that are determined by genetic factors and by previous drinking experience. Doses below 0.5 mL 95% USP alcohol/kg of body weight produce relatively insignificant effects on the cognitive, psychophysiological, and motor functioning of experienced social drinking nonalcoholic young men (Peterson,

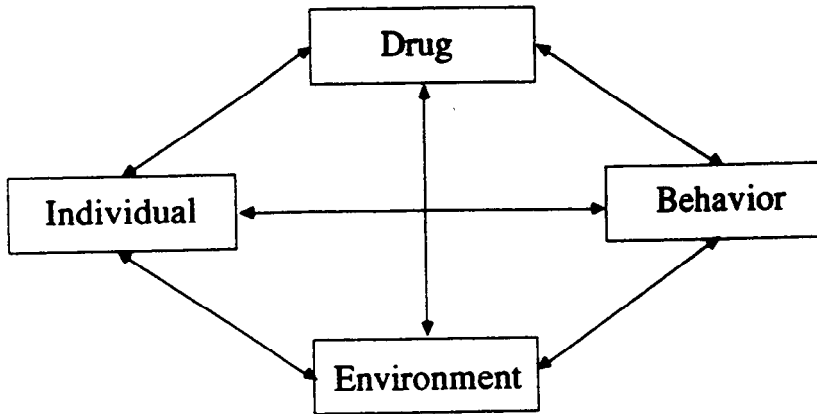


Figure 14.1. A Priori Factors Affecting the Drug-Behavior Relationship

Rothfleisch, Zelazo, & Pihl, 1990; Pihl, 1983). Extremely high doses of ethanol are capable of producing severe motor impairment and unconsciousness. Most laboratory studies of ethanol effects examine the consequences of intoxication produced by doses that are at the lower end of the range defined by these two extremes. It is the effect of alcohol, at doses high enough to have a measurable effect (but too low to produce excessive loss of coordination or unconsciousness), that provides the specific focus of this discussion.

The nature of aggression itself must also be considered. Aggressive behavior takes place in a variety of contexts, but most experimental work has been conducted within the confines of the Buss-Taylor paradigm. Within this paradigm, two subjects, one real, one confederate, participate in an experimental task. The experimental subject is required to exchange electric shocks, or an alternative punishing stimuli, with the confederate, for reasons that vary from experiment to experiment. The intensity and duration of this voluntarily administered punishment varies with subject choice, within certain confines established by the experimenter. *Aggression* is defined as intensity and duration of stimulus administered. There is evidence that aggression can be validly and reliably measured in this fashion (Bernstein, Richardson, & Hammock, 1987). Buss-Taylor procedure scores correlate positively with peer-rated aggression (Williams, Meyerson, Eron, & Semler, 1967) and self-rated aggression (Shemberg, Leventhal, & Allman, 1968), and with degree of previous antisocial behavior (Hartman, 1969).

Aggression (or active avoidance) can be reliably elicited by at least two classes of stimuli, punishment and frustrative nonreward (Gray, 1982, 1987). The Buss-Taylor task essentially punishes the experimental subject by exposure to electric shock. Most modifications of this procedure alter the context within which that shock is received, by manipulating the expectancies of the experimental subject, or by frustrating him or her, in addition to the punishment. In normal, sober human beings (and in experimental animals) the context within which any stimulus is received modifies the behavioral response that stimulus elicits. This is especially obvious in the case of *frustrative nonreward*, which is defined as the absence of a reward that the subject expects. Without expectancy, which is the context within which the experimental manipulation takes place, frustration is impossible. In addition, the consequences of a behavioral response should modify that response. Any behavior has certain benefits and certain costs, which should in turn come to alter the expression of that behavior, in an organism capable of benefiting from experience. Consideration of these methodological and theoretical issues further limits the topic of this chapter to the effects of moderate doses of alcohol on aggression elicited by punishment and frustration, presented within a variety of different experimental and ecological contexts.

Three Effects of Alcohol

Three alcohol effects appear to heighten significantly the likelihood of aggression: increased pain sensitivity, reduced response to cues of punishment and cues of frustrative nonreward, and decrease in response flexibility. Figure 14.2 illustrates how each of these putative effects independently or collectively might increase aggression.

Increased Pain Sensitivity

Pain-elicited aggressive behavior is the most consistent stimulus for aggression among most animals. Fight or flight only partially summarizes a phenomenon that occurs in response to "psychological" pain (like frustration) and that appears unconditioned, nonappetitive, and displaceable (Berkowitz, 1983; Moyer, 1976). The idea that alcohol may actually increase pain sensitivity in some individuals might seem counterintuitive, given that the pain-reducing effects of alcohol are well known and that alcohol was one of the first anesthetics ever used (Mullin & Luckhardt, 1934; Wolff, Hardy, & Goodell, 1942). However, a pharmacological agent that is anesthetic at very high doses is not necessarily analgesic at lower doses. Furthermore, it is well

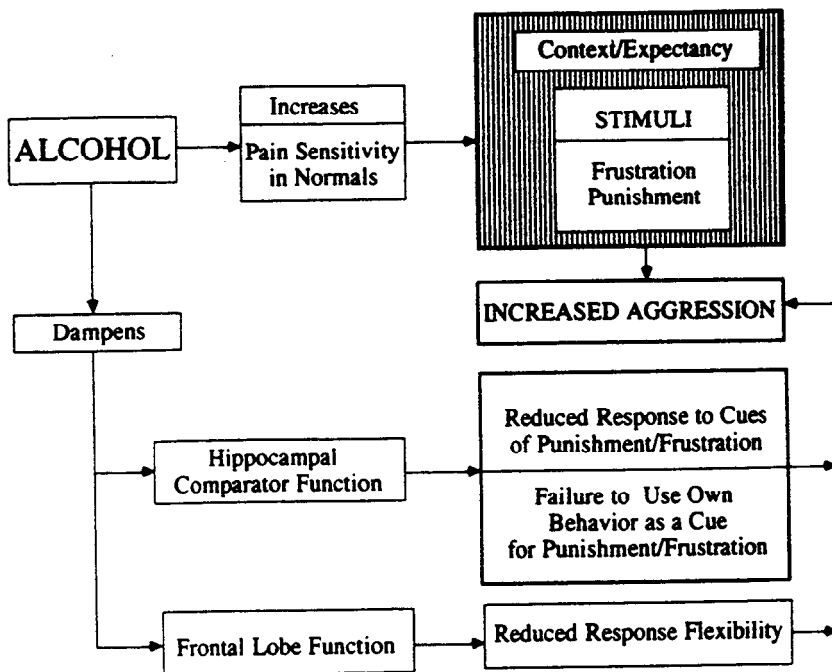


Figure 14.2. The Effect of Alcohol on Aggression

known that there is great individual variability in sober pain sensitivity and in alcohol-intoxicated pain response. Gustafson (1986) has demonstrated that subjects heightened their rating of pain-sensitivity to electric shocks experienced while participating in the Buss-Taylor task when given alcohol as opposed to placebo. In addition, Gray (1982) summarized a series of studies demonstrating that alcohol-intoxicated rats are characterized by reduced flinch and jump thresholds to electric shock.

A study recently completed by Stewart, Finn, and Pihl (1991) does not precisely replicate this phenomenon, but may help explain some of the variability in response. This study specifically demonstrated that men at heightened genetic risk for the development of alcoholism rate electric shocks as more uncomfortable and painful than low-risk control subjects at no or low doses of alcohol, but not at clearly intoxicating doses. Previous work on young men from a multigenerational family history (MGH) of high risk for alcoholism has demonstrated their susceptibility

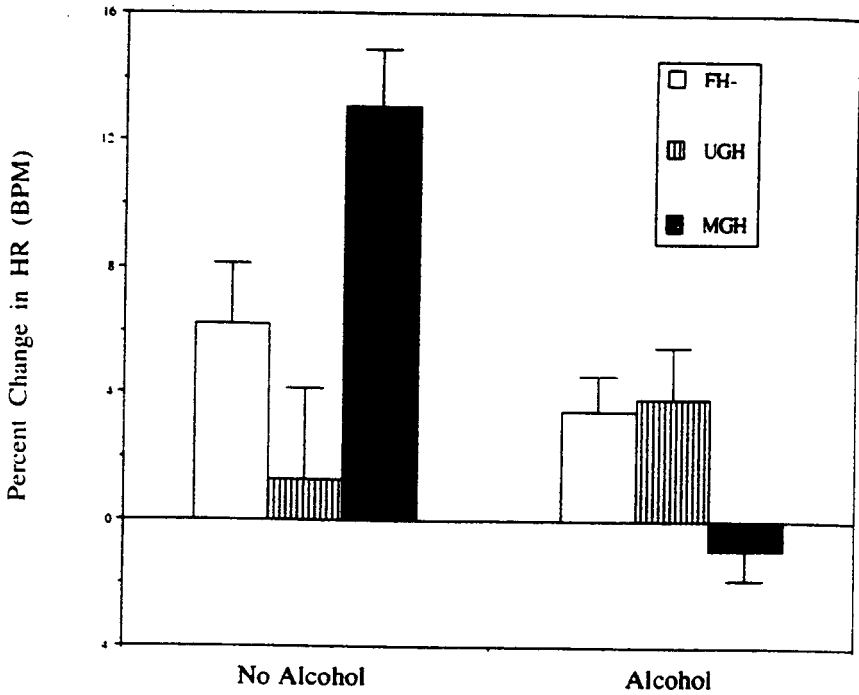


Figure 14.3. Heart Rate Reactivity to a Signaled Electric Shock Among Males With No (FH-), Unigenerational (UGH), and Multigenerational (MGH) Family History Male Alcoholism

to alcohol dampening of cardiovascular response to electric shock. Figure 14.3 presents cumulative data from four studies that illustrate this response pattern, and contrasts this pattern with that characteristic of young men with alcoholic fathers only (unigenerational family history [UGH]) and of normal controls (Pihl & Peterson, 1991). If this stress-dampening response represents an alcohol-induced decrease in pain sensitivity, like that characteristic of chronic alcoholics (Pihl, Peterson, & Finn, 1990), these MGH men should be less aggressive when intoxicated. That is in fact what appears to happen. Multigenerational family history positive individuals deliver higher-level electric shocks while competing in the Buss-Taylor task while sober, and deliver lower levels while intoxicated (with 1.32 mL 95% USP alcohol/kg of body weight), when compared to controls (Figure 14.4).

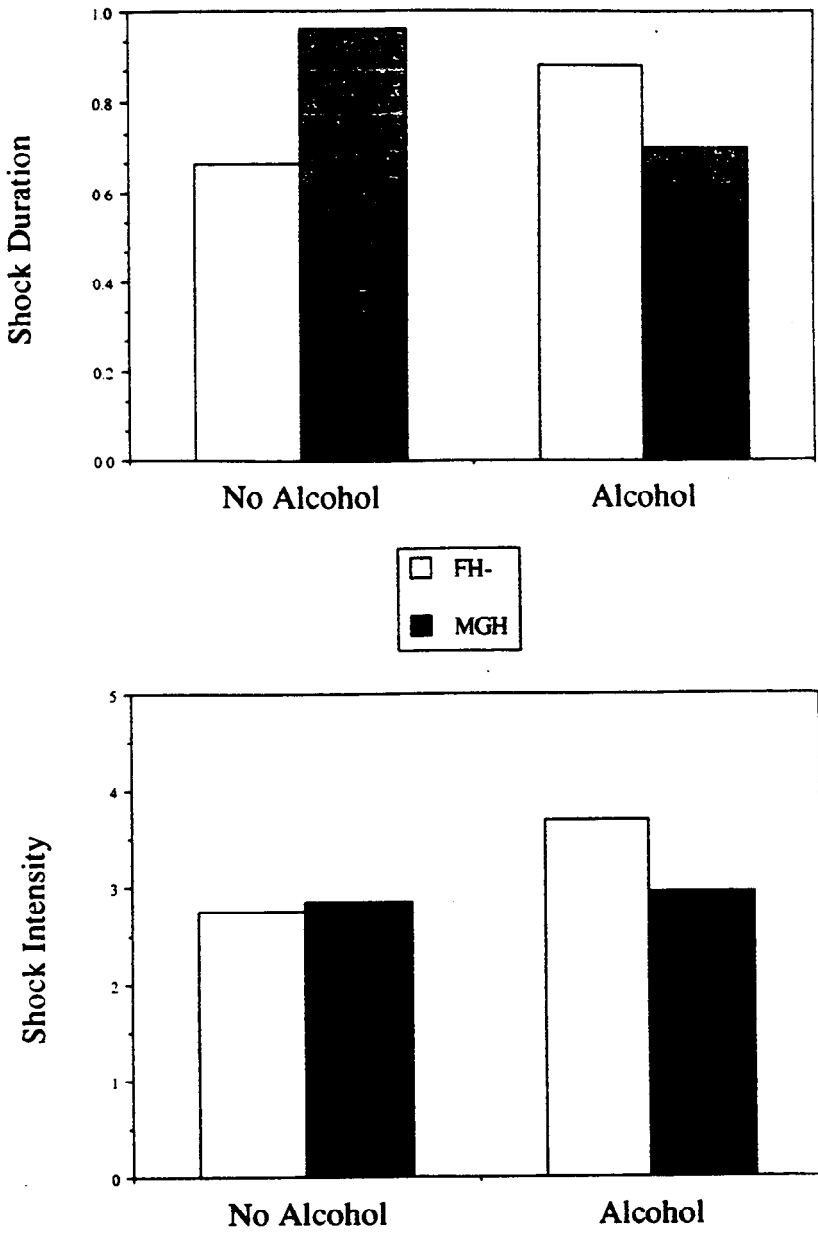


Figure 14.4. Shock Intensity and Duration Delivered by Males With No (FH-) and Multigenerational (MGH) Family Histories of Male Alcoholism, While Sober and While Alcohol-Intoxicated

Reduced Response to Cues of Punishment and Cues of Frustrative Nonreward

Exhaustive analysis of the relevant animal and human literature led Jeffrey Gray (1982, 1987) to propose that the behavioral effects of benzodiazepine, barbiturate, and ethanol were similar. These drugs appear to interfere with the operation of a complex limbically based neurological system responsible for governing and integrating organismal response to cues of threat, punishment, frustrative nonreward, and novelty.

This neurological circuit, the behavioral-inhibition system, is composed primarily of the septum, the hippocampus, their interconnections, and the afferent/efferent pathways connecting them with other cortical structures (Gray, 1982). In the face of threat, the behavioral inhibition system brings ongoing activity (instrumental, classically conditioned, or innate) to a halt, and initiates sensory (Sokolov, 1969) or motor/cognitive processes (Peterson & Pihl, in press) directed toward detailed analysis of current events or stimuli (Gray, 1982).

The effects of alcohol and the other antianxiety agents appear specific, within a range of moderate doses. They do not reduce (and may even enhance) response to punishment or frustration. However, they do reduce fear or anxiety generated to cues associated with these two classes of stimuli. The combination of these two facts make consideration of Gray's general neuropsychological model particularly interesting with regard to the alcohol-aggression relationship.

Aggression can be reliably elicited by punishing or frustrating an organism. The same can be said of active avoidance or escape, however. Both of these strategies, designed to reduce the probability of further punishment or frustration, have their attendant limitations. These limitations are the costs of engaging in either behavior. Active avoidance entails abandoning the search for information in a given situation. This might be considered its primary cost. Aggressive action, by contrast, has as its primary drawback high potential for structural damage. This means that the act of engaging in aggression should logically come to serve as a cue for punishment.

This line of reasoning is very fruitful when it is applied to the problem of human socialization. It might be said that the entire process of socialization revolves around attempts to teach children to regard their own participation in or initiation of certain types of behavior as a threat to their own well-being and to the well-being of the social group to which they belong, and on whose beneficence they ultimately depend. Individuals who have failed to learn such connections, for whatever reason, pose a

serious threat to the safety of those around them and to the overall integrity of a given social group.

Sober, reasonable, well-socialized persons participate in aggressive acts under extremely restricted conditions. These conditions are carefully defined according to the social canon as justifiable. A justifiably aggressive act by a given individual involves threat, punishment, or frustration directed at another, who had been previously and unjustifiably threatening, punishing, or frustrating to himself or herself or others. A stable society, and the well-socialized members of that society, ensure that aggressive action outside of such extremely restricted conditions is followed by punishment or frustration. This is the threat that society ensures is attendant upon the expression of nonjustified aggression.

One of the pharmacological properties of alcohol, however, is the reduction of fear or anxiety produced to threat. This means that alcohol disinhibits all behaviors that are under the general inhibitory control of fear. Fear appropriately retards many acts of aggression, which in themselves expose the aggressor to potential damage, and which more generally might expose him or her to the revenge of society at large. The tendency for drunks to become victims can also be profitably considered in this light. Fear produced in response to threat cues helps most people avoid physical harm. If the anxiety these cues produce is reduced, then the chances for harm logically increase. This means that drunk people may continue to participate in dangerous situations, when their behavior should be inhibited by the threat of danger.

This general theory can help integrate different aspects of the literature regarding alcohol and aggression that seem surprising on first consideration. Zeichner and Pihl (1979) examined the behavior of three groups of young men administered placebo, no-alcohol, or 1.32 mL of 95% USP alcohol/kg of body weight. Subjects in these three dose groups were further subdivided into two modified Buss-Taylor paradigms: one in which they received aversive tones, whose intensity was correlated with the shocks they delivered, and one in which they received aversive tones, whose intensity was unrelated to their own behavior. Subjects who had received placebo or no-alcohol modified their behavior according to the logical contingency: They reduced the intensity of the shock they delivered in the correlated condition. Intoxicated subjects failed to modify their behavior in a similar manner (Figure 14.5).

Originally, Zeichner and Pihl (1979) hypothesized that alcohol reduced an individual's ability to "process information pertinent to the consequences of their own behaviour" (p. 159). In accordance with this hypothesis, Zeichner, Pihl, Niaura, and Zacchia (1982) tested two groups of young men, who received 1.32 mL of 95% USP alcohol/kg of body weight

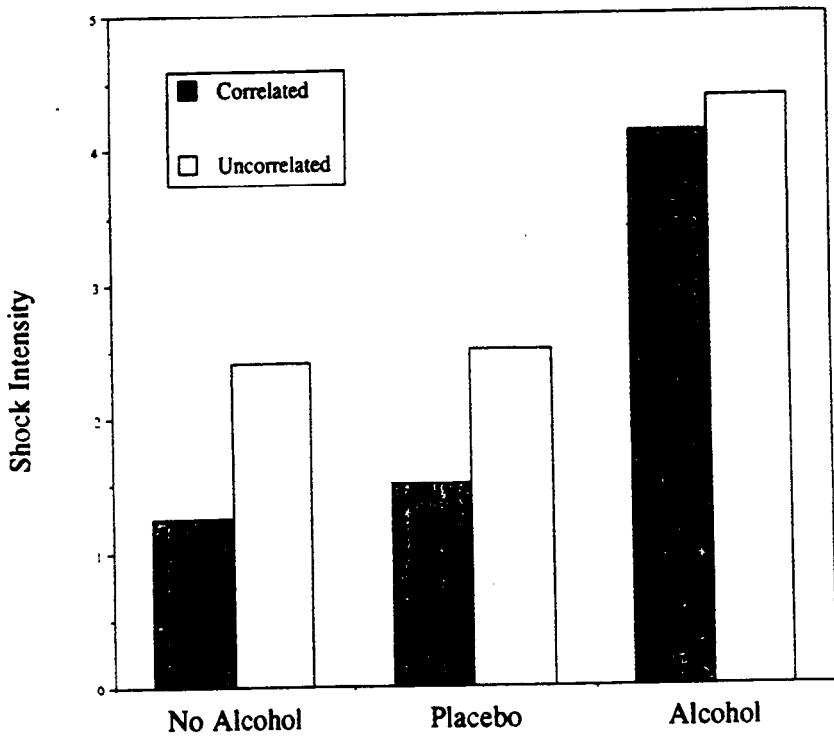


Figure 14.5. Shock Intensity Delivered by Males Who Consumed No Alcohol, Placebo, or Alcohol, When Shock Reception Was Correlated or Uncorrelated With Shock Administered

or placebo, requiring them either to complete mathematical problems or to write down the level of shock they were administering and receiving, while participating in a modified Buss-Taylor task. Distraction actually reduced the effect of alcohol on aggression, while forced attention increased it (Figure 14.6). The intoxicated subjects were aggressive not because they were unaware of their behavior and its consequences, but because that knowledge was no longer accompanied by fear. The information was still being processed, at least by the verbal system, but it no longer had an inhibitory effect on behavior.

Furthermore Zeichner and Pihl (1980) tested three groups of young men, who received placebo, no-alcohol, or 1.32 mL of 95% USP alcohol/kg of body weight, who shocked a (sham) partner, in response to that

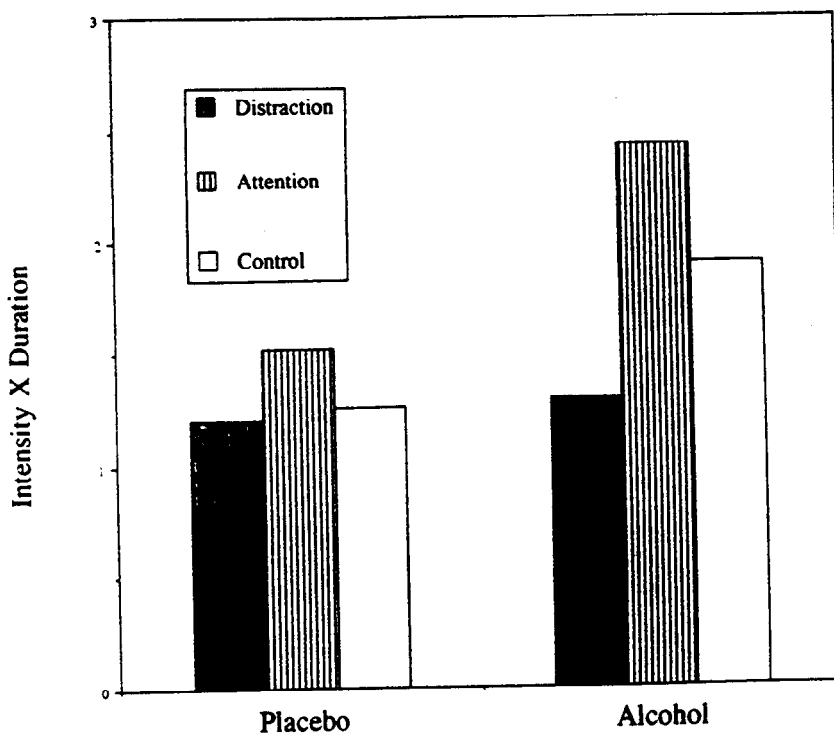


Figure 14.6. Shock Intensity \times Duration Delivered by Males Who Consumed a Placebo or Alcohol, During Forced Distraction, Forced Attention, and Normal Attention (Control)

partner's administration of tones of varying aversiveness, in one of two intent conditions (Figure 14.7). In the first (neutral) condition, subjects were told that the aversiveness of the tone they were to receive from the person they were to shock was fixed according to a predetermined schedule. In the second (malicious) condition, subjects were told that they would receive whatever tone their partner freely chose to deliver. Tones in both cases were actually delivered according to an identical predetermined schedule. The intoxicated subjects were more aggressive overall than subjects in the other two groups. They chose more intense shocks, in general. In addition, they delivered the more intense shocks for a longer duration, whereas the placebo and no-alcohol subjects reduced the duration of the more intense shocks. More important, the intoxicated subjects

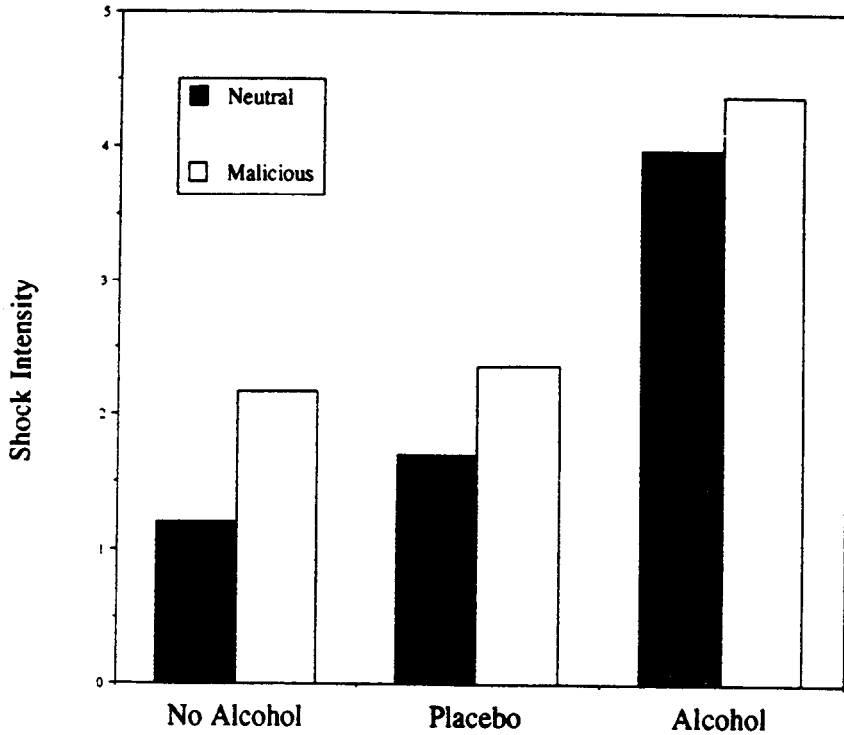


Figure 14.7. Shock Intensity Delivered by Males Who Consumed No Alcohol, Placebo, or Alcohol, Toward an Opponent of Neutral or Malicious Intent

were immune to the effects of intent on shock duration, although the placebo and no-alcohol subjects reduced their shocks in the neutral condition. Correlational analysis demonstrated that the intoxicated subjects reacted to increasing intensity of stimuli with increasing level of shock, regardless of intent, whereas the placebo and no-alcohol subjects reacted to intensity in the malicious condition only.

In Western society, aggressive behavior is restricted by considerations of intent. Acts of threat, punishment, or frustration directed toward another without consideration of that other's intent or ability are generally followed by punishment or frustration. The consideration of intent is a fundamental attribute of more sophisticated notions of justice. Intoxicated individuals in the Zeichner and Pihl (1980) study responded to the intensity of the punishing stimuli, and did not modify their behavior according

to socially defined behavioral norms. The inhibitory effect of the threat inherent in breaking a social rule appears to have been essentially eliminated by alcohol intoxication.

Hull (1981) has hypothesized that alcohol interferes with encoding processes fundamental to self-control, which are separate from attentional focus. He believed that alcohol produces a temporary chemical lesion attenuating or eliminating "the encoding of information in terms of its relevance for self" (p. 589). This line of reasoning is in accordance with the general theoretical outline set forth in this chapter. Hull also states, however, that alcohol might make subjects more dependent on the superficial aspects of the situation (p. 527). This notion might be clarified by noting that alcohol intoxication does not attenuate response to punishment or frustration (Gray, 1982, 1987), classes of stimuli that could not be called superficial, but reduces the inhibitory effect of threat.

The old psychoanalytic notion that alcohol reduces superego restrictions is interesting in light of the present theory, as well. The classical superego represented the internalization of societal standards. It is within a given social context that the initiation of or participation in threatening, punishing, or frustrating behaviors are defined as justifiable or not justifiable, or are defined as a threat to self or not. If alcohol appears to reduce the inhibitory effect of threat, while leaving social and verbal knowledge intact, it could be argued, within the psychoanalytic context, that alcohol therefore performs the function of freeing the ego from the restraints of the superego. This theory holds true, however, only when the drunken ego is being punished or frustrated. There is little evidence for a general release of "innate" aggressive tendencies (Bushman & Cooper, 1990).

Decrease in Response Flexibility

The notion that alcohol has a general disinhibitory effect is further weakened by consideration of the fact that there are many forms of cognition that remain intact during intoxication, at doses that are capable of heightening aggressive response. Peterson, Rothfleisch, Zelazo, and Pihl (1990) administered a battery of neuropsychological and motor tests to six groups of young males, administered three doses of alcohol (placebo, 0.66 mL/kg 95% USP ethanol, and 1.32 mL/kg 95% USP alcohol) under two expectancy conditions: told alcohol, and told no-alcohol. The results of the test battery indicated that alcohol had particularly negative effects on delayed memory (a function of the hippocampal system [Milner, 1964]), on some tests of cognitive ability associated with the prefrontal cortex, and on motor skills that demanded self-monitoring. By contrast, alcohol had virtually no effect on standard IQ tests such as the WAIS-R

Vocabulary and Information subtests, or on functions such as simple reaction time. Alcohol does not reduce previously learned knowledge about the nature of the world; what it reduces is the ability to deal with the threatening and novel.

The fact that alcohol impairs functions associated with the prefrontal cortex further complicates the alcohol-aggression relationship. The prefrontal cortex plays a critical role in the formulation of verbal and motor strategies that are designed to gather information in the face of novelty and/or threat (Luria, 1980; Peterson & Pihl, in press). In addition, the prefrontal cortex is also involved in the organization and sequencing of memories based on the acquisition of new information (Luria, 1980). The notion that alcohol affects the operation of the threat system implies that an intoxicated individual is unlikely to respond appropriately to signals of danger. Any additional prefrontal trouble is likely to render that intoxicated individual further incapable of acting in accordance with those signals, even when they remain sufficiently powerful to inhibit his or her ongoing behavior. An intoxicated, but ambulatory, individual appears therefore characterized by inappropriate fearlessness, and by impaired ability to plan and organize behavior. The combination of these two characteristics, serious enough in itself, is likely to be heightened in severity by the addition of reduced motor coordination, a potentially fatal lack in any number of potentially dangerous and strategically complicated situations.

Summary

Increased pain sensitivity, reduced response to cues of punishment and cues of frustrative nonreward, and reduced response flexibility can all apparently occur as a result of drinking. The presence of such factors independently or collectively is likely to increase the probability of aggressive behavior. In addition, the failure to use one's own behavior as a cue for punishment or frustration may characterize intoxicated victims, as well as aggressors.

Previously, it was noted that theories of intoxicated aggression were neither mutually exclusive nor collectively exhaustive. Clearly, the theory presented in this chapter suffers from many of the same limitations. Other plausible, relatively well-documented but undiscussed interpretations of a pharmacological alcohol effect exist. Some individuals appear susceptible to states of pathological intoxication, or to hypoglycemia induced by alcohol consumption. These issues have been discussed in a previous review (Pihl & Ross, 1987). The expectancies an individual brings to the bottle should also be given due consideration. Each individual has power-

ful expectations regarding the effects of psychoactive substances, which are dependent on individual learning histories and cultural processes, and which may be used all too readily to justify intoxicated thought and action. The interested reader is referred to Lang and Sibrel's (1989) recent review of this literature. The personality of the individual who drinks is also relevant, as preexisting abnormalities in sensitivity to punishment, frustration, and their respective cues may well be compounded by alcohol-induced intoxication.

Other Drugs and Aggression

This chapter has focused on the alcohol-aggression relationship because there is a preponderance of evidence demonstrating its importance. Furthermore it would be reckless to discuss a "drug-aggression" relationship, with any degree of generality. Specificity of drug and dose action on aggression should be assumed automatically. There is not enough evidence available regarding most drugs of abuse and human aggression to draw even speculative conclusions. Unfortunately research in this area, difficult enough for legitimate, practical reasons, is made more complicated by bureaucratic restriction (Taylor, 1990).

Aggression is heightened or reduced by a wide variety of drugs, according to clinical reports. Cocaine, particularly in crack form (Honer, Gewertz, & Turey, 1987), THC (Nakas, 1973), phencyclidine (Fauman & Fauman, 1982), benzodiazepine (DiMascio, Shades, & Harmatz, 1969), and many other psychotropic medications, alone and in combination, have been noted to produce alterations in aggressive behavior. Unfortunately clinical reports are fraught with methodological problems (Pihl & Speirs, 1976). They are limited most severely by their inability to control for the nondrug state or trait characteristics of the patients being observed. "Chicken and egg" questions predominate, and increased violence, observed in a drug-abusing population, might as readily be attributed to unspecified premorbid characteristics of that population as to drug pharmacology per se. This is of course the reason that controlled investigations are necessary.

Laboratory studies utilizing the Buss-Taylor or similar tasks have been completed, using a wide variety of drugs. For each particular drug the literature is sparse, but some interesting findings have emerged. The ingestion of nicotine (Cherek, 1981) and caffeine or caffeinated coffee (Cherek, Steinberg, & Brauchi, 1983, 1984) decreases aggressive responding, below that obtained with placebo. In the case of nicotine, this result has been duplicated in studies of a variety of nonhuman species (Driscoll & Baettig, 1981). Although caffeine has stimulant properties, subjects typically report decreased irritability and improved mood post-ingestion (Goldstein, Kaizer,

& Whitby, 1969). THC seems to affect aggression in a similar manner, even under conditions of extreme provocation (Taylor, 1990; Taylor et al., 1976). The effects of barbiturates and benzodiazepines appear to be dose-dependent (like those of alcohol). At very low doses these drugs may decrease aggression (Cherek, 1990), while at moderate or high doses and over time they sometimes increase aggression (Taylor, 1990). Cocaine has also been demonstrated to heighten aggression (Taylor, 1990). The same thing is true of an amino acid mixture deficient in tryptophan (Pihl et al., 1991). The modes of action of most of these drugs remain unknown, and replication and extension of these basic findings is clearly necessary.

Final Comments

A final important factor clearly affecting the drug-aggression relationship is that of comorbidity. Clearly, the soup becomes a stew as co-occurring psychiatric problems are considered in addition to drug abuse and aggression. Many psychiatric problems accompany substance abuse. Antisocial and borderline personality disorders appear common among opioid addicts (Malow, West, Williams, & Sutter, 1989), for example. Antisocial personalities are characterized in part by their violent tendencies, and they are even more likely to abuse alcohol than opioids (Pihl, Peterson, & Finn, 1990). Conduct-disordered children also often become substance-abusing, antisocial adults (Pihl & Peterson, 1991). The theoretical model set forth in this chapter is not predicated on the assumption that alcohol produces aggression, only that it releases aggression, when it is provoked by frustration or by punishment, and when its expression is inhibited by fear. Abnormal levels of sober trait or state aggression (and levels of sober state or trait fear), like those characteristic of antisocial individuals, must necessarily affect the manner in which alcohol-intoxication alters behavior. Consideration of the interaction between preexistent personality characteristics and alcohol-induced aggression is, however, outside the scope of this chapter.

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