REVIEW PAPER

Drugs and Aggression: Correlations, Crime and Human Manipulative Studies and Some Proposed Mechanisms

R.O. Pihl, Ph.D.[1], Jordan Peterson, Ph.D.[2]

[1]Departments of Psychology and Psychiatry, McGill University, Montreal, Quebec, Canada

[2]Department of Psychology, Harvard University, Cambridge, Massachusetts, USA

Violence and drugs seem to mix readily. In this paper, relevant correlational studies are briefly reviewed and methodological issues are discussed. With a focus on alcohol, facts pointing to some type of causal relationship are presented both from crime and laboratory manipulative studies. Dose and rate-dependent anxiolytic, psychomotor stimulant and alteration in inhibitory and problem-solving cognitive functions are each in turn seen as precipatory to aggression. Drugs other than alcohol which interact with these systems are also discussed.

Key Words: aggression, alcohol, anxiety, cognition

INTRODUCTION

Crime statistics are quite consistent in showing a strong correlation between violent crimes (murder, assault, rape, family violence) and the consumption of certain drugs (Reiss and Roth 1993). Alcohol in particular seems to mix readily with violent behavior. Table 1 is a summation of recently reviewed crime studies (Murdoch et al 1990). These investigations are typically beset with numerous and severe methodological problems. Thus, the implication of causal mechanisms between alcohol and the commission of a violent crime is often challenged. Retrospective reports, lack of physical measures, the time elapsed between the occurrence of the crime and the measurement of abuse, and the absence of control comparisons are common complications. Further, it is possible that intoxicated individuals may be simply more susceptible to arrest than sober criminals. Additionally, the large cultural variability in aggression when intoxicated that is reported by anthropologists is often quoted by those with a suspicious view of this data (see MacAndrew and Edgerton 1969 for a review) as is the high percentage of victims of

violence who also have been found to have been drinking. Thus, perhaps the relationship is simply situational and temporal describing where and when violence is most likely to occur. Two facts derived from crime studies suggest that this notion of a simple fortuitous relationship is wrong. First, the range for the means of those drinking, typically heavily, during the commission of a violent crime ranged from 24% to 85%, while an analysis of nonviolent crimes determined a much lower comparison figure of 12% to 38%. Second, the prototypic violent event sequence is drinking — provocation — violence, where the provocation is most frequently in the idiom of a verbal argument.

Explanations at a purely pharmacological level would be both incomplete and misleading. Cultural and subcultural sanctions, history of abuse, history of reinforcement for aggression, the degree and attribution of provocation and intent, and gender and age are all factors which contribute to aggression. Even where drugs are involved, these and other factors may be primarily responsible. Nadelman (1989) has pointed out how violent behavior is often required as part of the "rules" of the illegal drug business and is often necessary for the user to acquire money to purchase drugs. Lang (1993) and his coworkers have shown how psychological expectancy regarding certain drugs and the proclivity for violence

Table 1

A summary statement of 26 studies of alcohol's involvement in violent crimes

Studies	Countries	Cases	% Offenders Drinking	% Victims Drinking
26	11	9,304	61.53	45.16

may be a powerful determinant of the response. The litany of findings concerning alcohol expectancies and aggression is impressive. For example, it is well documented that people expect alcohol and certain drugs to increase aggressiveness (Kidder and Cohn 1979; Southwick et al 1981); that young children (aged five to 12 years) have adopted this expectancy (Lang et al 1992); that expectations vary with culture (Lang 1992), dose (Southwick et al 1981) and type of beverage (Lang et al 1983); that the more experienced the drinker, the more salient the expectancy (Cameron 1981), a fact true for both men and women (Rohsenow 1983); that a drinking woman is seen as more sexually available (George et al 1988) and more personally responsible if raped (Richardson and Campbell 1982) or abused by her husband (Richardson and Campbell 1980); and, that intoxicated victims of any crime are seen as more responsible for their predicament than nondrinkers (Pillmore 1985). It is worth noting that expectancies have been shown to predict alcohol-related aggression (Darmen and George 1988). For example, the specific expectancy that whiskey is more provocative than wine or beer concurs with experimental data from intoxicated adults (Takala et al 1957; Boyatzis 1974; Pihl et al 1983). The relationship between these processes and drug effects is interactive as social, psychological and pharmacological factors modify each other and, in turn, are altered by the resultant behavior. However, it is equally misleading and misinformative to ignore the effects of powerful drugs and to explain the relationship as purely cultural or psychological. Different drugs alter different basic physiological/motivational processes which directly and indirectly alter the likelihood of aggressive behavior. These fundamental processes which vary individually represent an evolutionary legacy, at times essential for survival, which, when rendered askew in terms of our present societal context, can result in an outcome of mayhem. All too often naive Rousseauians and their ilk ignore the fact that we all present with a preexisting biology that is at times, in some people, exceedingly fragile. Speculation on how particular drugs and their mode of action increase the likelihood of violent behavior can help explain some of the variance of the drug/violence relationship as well as increase to our knowledge of brain functioning and the phenomenon of aggression. As a consequence, this paper, after some comments about the definition and measurements of aggression, focuses in turn on three speculative drug effects which putatively increase the likelihood of aggression.

The definition and measurement of aggression

Worthy of reiteration is the fact that aggression is a multifactorially produced response. No aggressive acts are alike in terms of their motivation, goal and social-cultural context. They are not treated as if they are alike in the course of interpersonal interaction. Aggressive conduct is frequently rewarded and even idealized, under certain circumstances, but punished and vilified under others. Researchers have characterized aggressive acts as physical/verbal, active/passive, direct/indirect (Buss 1961) and as instrumental or hostile/defensive (Valzelli 1981). Instrumental aggression is concerned with reward attainment while defensive aggression is designed to minimize aversive conditions. In this latter definition, aggressive acts culminate in the elimination of an aversive state in an aggressor and typically produce an aversive state in another. Bushman and Cooper (1990), in their recent review of experimental studies on alcohol and aggression, describe an aggressive act as "behaviour directed towards the goal of injuring another living being, who is motivated to avoid such treatment." We have noted elsewhere (Pihl et al 1993) that aversive states fall essentially into two categories: that of pain (psychological or physical) and that of fear. Pain emerges as a consequence of punishment, as a result of physical damage (in the case of sensory pain), social isolation (in the case of grief or loneliness) or challenge to expectancy (in the case of frustration or disappointment). Fear, in contrast, emerges as a consequence of threat (Grey 1982). Novelty, which may signal danger, and the likelihood of punishment can be threatening (Grey 1982). Aggressive acts, therefore, appear as those intended to punish or to threaten. The various forms of aggression may have the same goal — the production of an aversive state — but for completely different motivations. Expression of defensive and instrumental aggression, in the human case, takes place in a social context and has social consequences. In recognition of this fact, it also appears necessary to note that aggression may take prosocial and antisocial forms.

While the assessment of aggression in crime situations is obvious, less apparent is the motivation and role of a drugged condition. Experimental paradigms which afford controlled environments, both social and pharmacological, have employed a variety of paradigms, ranging from laboratory barroom social encounters (Boyatzis 1974) to actual barroom experiments (Murdoch and Pihl 1988a; 1988b), to contrived dyadic interactions (Taylor and Chermac 1993). The most used approach is the Buss Aggression Task, and specifically the Taylor Variation (Taylor 1983). In this task a subject
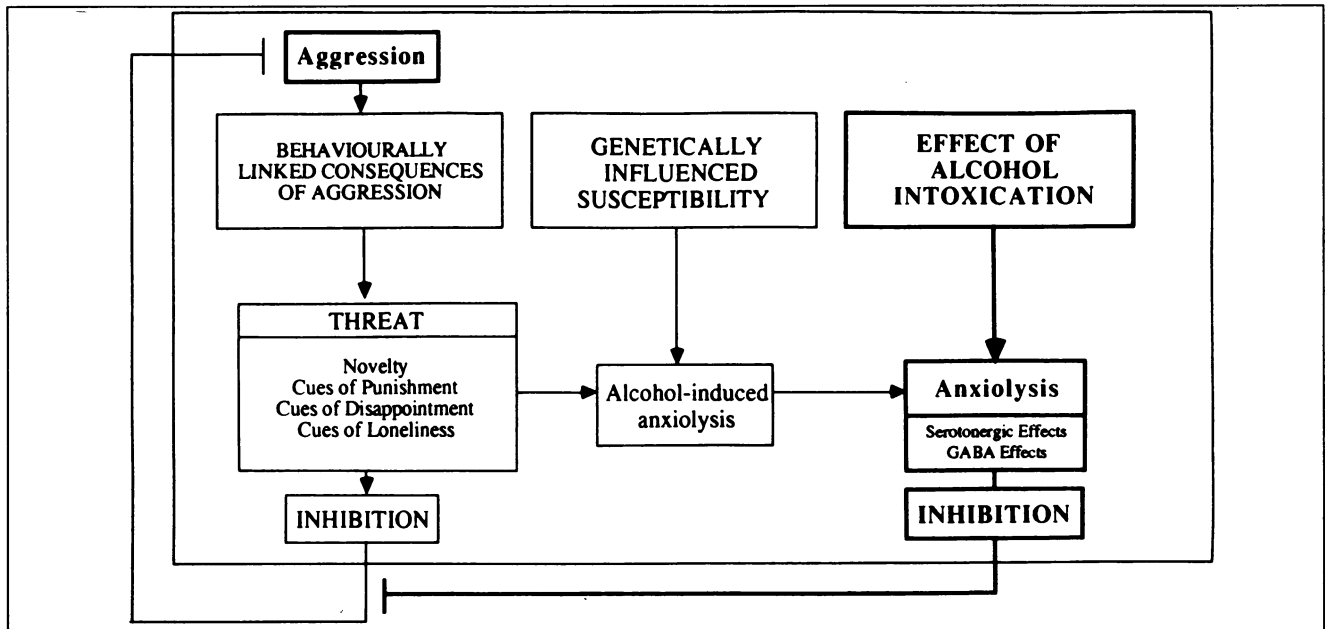
Fig. 1.   Alcohol's hypothesized effect on the threat system and the increased likelihood of aggression.

competes with another subject, typically bogus. Following "wins", the subject administers a preselected aversive stimulus to his/her opponent and *vice versa*. The intensity and duration of the aversive stimulus administered by the subject are taken as indices of the subject's choice of level of aggression. Peer-rated high aggressors (Williams et al 1967) and individuals with antisocial histories (Hartman 1969) are typically more aggressive on this task. Furthermore, there is consistency among a large number of studies showing an alcohol effect (Bushman and Cooper 1990).

The complex nature of aggression means a decreased likelihood that manipulation of a pharmacological and/or biochemical function will have an easily comprehensible consequence. Nonetheless, there are at least three dose- and time-related pharmacological effects which can be shown to alter the likelihood of aggressive responding. These effects can operate independently or cumulatively. They include alterations to the threat system, potentiation of psychomotor activity and alterations of certain cognitive functions. In turn, each of these effects operates through a general expectancy set, a conceptualized model which will be elaborated later.

Drugs and the threat system

Aggression elicited by punishment (in the case of defensive aggression) or by cues of reward (in the case of instrumental aggression) remains under the tonic inhibitory control of threat under normal conditions. This means that, during the course of socialization in most cultures, normal individuals learn to associate the manifestation of aggression with the increased likelihood of receipt of punishment. The resultant

anxiety associated with the appreciation of danger plays an important role in protecting individuals from damage. This is true if the danger is associated with particular elements in the external environment, with novelty or with the expression of particular motivated behaviors. Thus, although variable for most situations, particularly among the properly socialized, cues to become aggressive should elicit anxiety (i.e., the threat of being punished).

Anxiety is soluble in alcohol and other drugs, so to speak. Anxiolytics reduce the protective control of anxiety (including threat contingent upon one's own aggression) and, thus, dose-appropriate intoxicated individuals are more likely in general to engage in dangerous activities. Gray (1982, 1987) has concluded that anxiety appears to protect a person from punishment primarily through inhibiting behavior whenever novel stimuli previously paired with punishment are present. In the extreme, anxiety is debilitating and is currently redressed with drugs such as the benzodiazepines, the barbiturates and alcohol. It is often argued that each of these drugs in turn can be negatively reinforcing because of the relief from distressing anxiety. But, in terms of aggression, it is the behavioral inhibiting nature of anxiety, the normal functioning of the threat system designed to preserve the integrity of the organism by avoiding those situations of potential risk, that is diminished. While the type of aggression is arguable, studies in rats (Blanchard et al 1993), clinical reports (see Woods et al 1987) and a few manipulative studies (Taylor 1990) link the benzodiazepines and the barbiturates to increased aggression, particularly at moderate dosages. These drugs, along with alcohol, effectively dampen the anxiolytic sensitive septo-hippocampal novelty threat detection system

postulated by Gray (1982). Anxiolytics have been shown to operate directly on gamma-aminobutyric acid (GABA), the brain's major inhibitory neurotransmitter. Alcohol has been shown to affect the CL-ion channel at the GABA benzodiazepine receptor (Warnecke 1991; Zorumski and Eisenberg 1991). It may also play a role at this level by affecting serotonergic activity (LeMarquand et al 1994). The threat system is illustrated schematically in Figure 1 which suggests that if the normal inhibitory response of threat itself is inhibited, then aggression is more likely. In a sense, the consequences of one's aggressive actions, which were previously inhibited because of the possibility of punishment, are themselves inhibited and, thus, the brakes are removed or at least the power to stop is markedly decreased. The finding that victims of violence are also often intoxicated and the sociological evidence which shows that intoxicated victims are often culpable in their own demise/predicament suggests such a disregard for potential danger. In fact, the review of crime studies by Murdoch et al (1990) demonstrates that individuals who actively precipitate their own violent demise are much more likely to be intoxicated than other victims of crime. These authors further suggest that the probability of violence is greatest when both members of a dyad are intoxicated, less so when only one is intoxicated and least when both individuals are sober.

Figure 2 presents the results of two studies which, taken together, illustrate the diminished functioning of the threat system. In the study illustrated in Part A of Figure 2 there were 72 young men randomly assigned to three drug conditions: no alcohol, placebo and alcohol. In the alcohol condition, subjects received an intoxicating dose of 1.32 ml/kg of 95% proof alcohol which was to be consumed in 20 minutes. After an additional 20 minute waiting period with subjects still on the rising limb of the blood alcohol curve, each group was split randomly into two additional conditions. In one situation, subjects participated in a Buss-Taylor Paradigm where, for every loss in the competitive game, they received an aversive tone equivalent to the intensity of a shock they delivered to their opponent. In this "correlated" condition the placebo and no alcohol groups behaved as expected and significantly reduced the intensity of the shocks they delivered to match the level of aversiveness they received. The same, however, could not be said for subjects who were intoxicated. These subjects (m BAL 0.089) behaved much like the other three noncorrelated groups, where the aversive stimulus they received was unrelated to the aversive stimulus they delivered. However, both groups actually received the same levels of aversive stimulation. The authors of this study (Zeichner and Pihl 1979) concluded that alcohol was most likely interfering with the ability of an individual to process information concerning the effect of the subject's own behavior. Variations on this theme have been offered as explanations by a number of writers (Taylor 1983; Steele and Josephs 1990) to explain the alcohol-aggression relationship.

Part B of Figure 2 presents the results of a study with subjects in a similar paradigm to Part A, although there were two groups, placebo and alcohol, in three conditions. The study (Zeichner et al 1982) which was designed to test alterations in the information processing model basically forced individuals in the alcohol situation to attend to their own behavior and the behavior of their opponent. In this forced attention condition, subjects recorded the level of
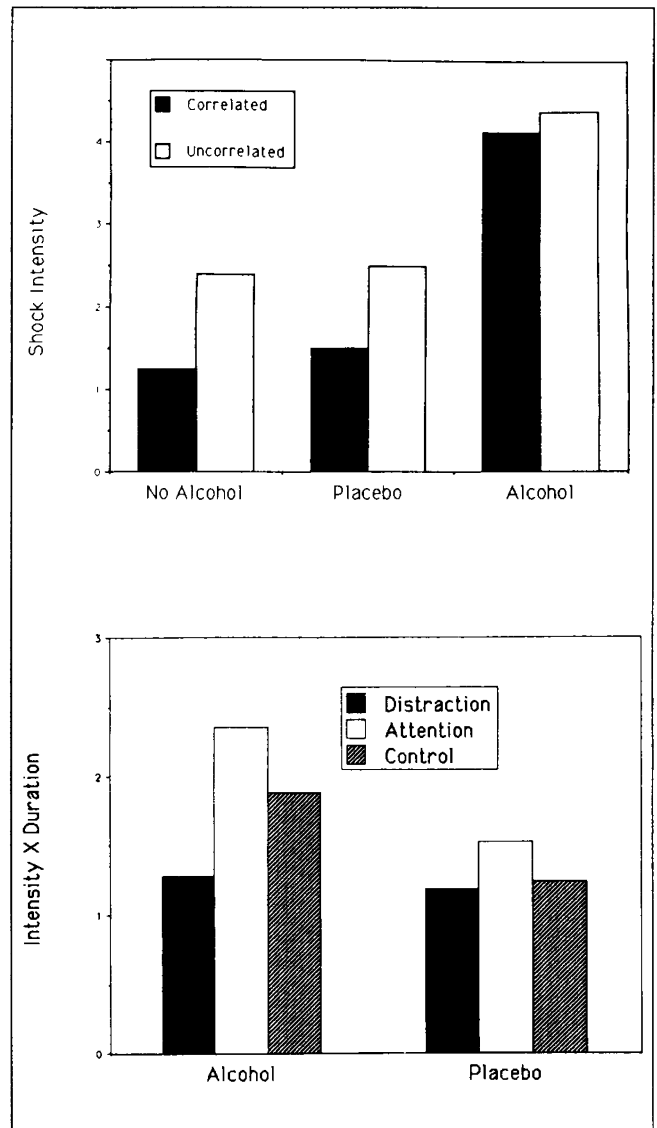
Fig. 2.   A. Delivered shock intensity by males in alcohol, placebo, and no alcohol groups under conditions where aversive stimuli received was correlated or uncorrelated with shocks given.
B. Shock intensity X duration response of subjects who had consumed either placebo or alcohol under conditions of distraction, forced attention or normal attention.
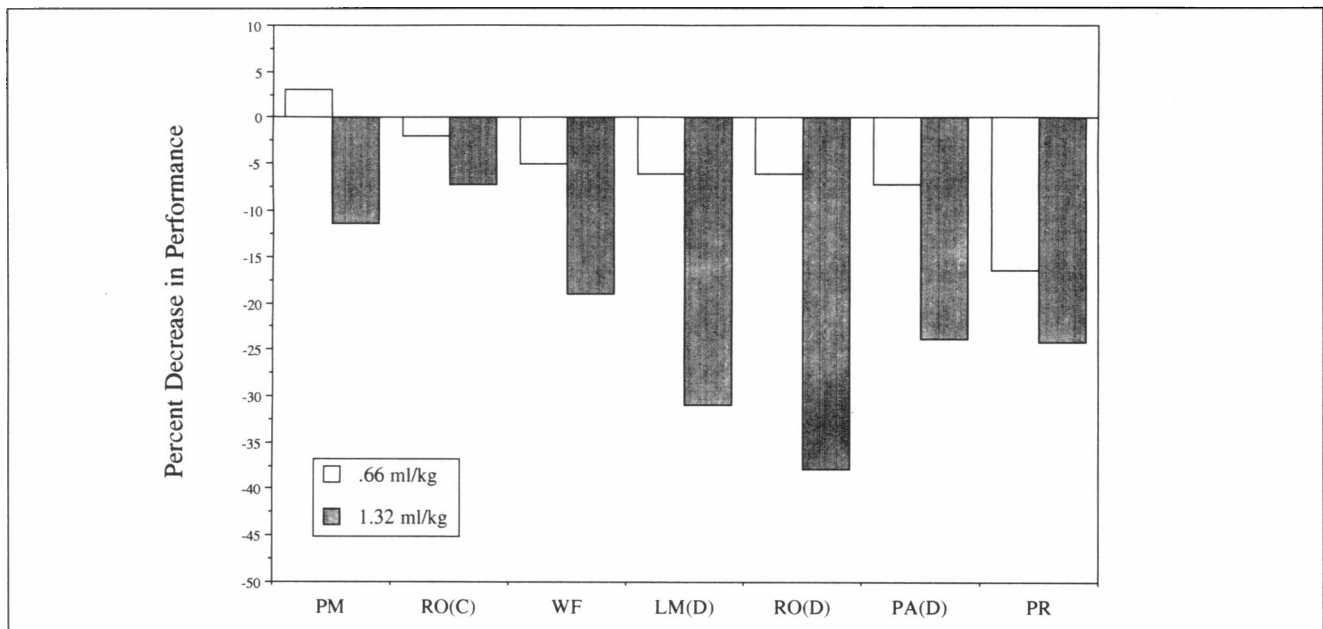
Fig. 3.   Percent decrease in performance from placebo response under two dosages of alcohol on seven neuropsychological
tests. (PM= Porteus Mazes, RO (C) = Rey Ostereith Copy, WF = Word fluency, LM (D) = Logical Memory delayed,
RO (D) = Rey Ostereith delayed, PA (D) Paired Associates Difficult, PR = Pursuit Rotor.

shock they delivered and the level of the aversive stimulus
they received. The distracted group was a control group who
were required to complete mathematical problems while
administering and receiving aversive stimuli. The control
condition was the same as the uncorrelated condition in the
previous study. The results clearly indicate that the individu-
als who became most aggressive were those in the forced
attention group. Consequently, it is not a failure to process
information relative to one's own behavior that leads to
increased aggression when intoxicated. Rather, it would
appear that it is the affective meaningfulness of the informa-
tion which is altered. It is as if the subjects knew what was
happening but did not care. Another example of relevant facts
not mattering when intoxicated can be derived from the
results of another study (Zeichner and Pihl 1980) where the
intent of the competitive partner was manipulated. Under one
condition, the subject was told that his partner was freely
choosing the level of aversive stimulus that was being admin-
istered, called "malicious intent," whereas in another condi-
tion, labelled "neutral intent," the partner was simply
presenting an aversive stimulus according to a list provided
by the experimenter. Again, under placebo and no alcohol,
subjects significantly reduced their level of aggression when
they believed that their competitor had no control over his
behavior, a fact not true of those who were intoxicated. This
notion of intent which is integral to concepts of justice that
pervade our society also seems to be diluted by alcohol. It is
as if social norms were dismissed, which is easily accom-
plished when normal negative injunctions lack affective

impact. Without the inhibitory effect of threat, the inhibition
for breaking social rules evaporates.

Drugs and the potentiation of the psychomotor activation system

Stimulation of the psychomotor activation system appears
rewarding in an unconditioned sense (Wise and Bozarth
1987). Animals will directly infuse chemicals which activate
this system and which serve as powerful reinforcers for
behavior (Fibiger and Phillips 1988). These facts are used to
explain the high abuse potential of stimulant drugs such as
cocaine and amphetamine. Other drugs which affect this
system directly are nicotine, caffeine, cannabis and phency-
clidine (Wise 1988). Some drugs, like opiates and alcohol,
affect the system indirectly. There is evidence that drugs
which activate this system enhance other rewards and can
produce sensitization in the form of reversed tolerance and
cross sensitization where one drug increases the rewarding
properties of another (Wise 1988). The primary biochemical
effect of these drugs appears to be dopaminergic. Cocaine,
for example, prolongs the effect of released dopamine by
blocking reuptake. Amphetamines react in a similar way but
also release dopamine as well as other neurotransmitters
(Koob and Bloom 1988). Alcohol's effect on the dopaminer-
gic system appears to be related to dose. Low and moderate
doses stimulate dopamine release, particularly at the nucleus
accumbens. However, at high sedative doses lower levels of
dopamine result (Gessa et al 1985). In the rat, for example,

increased locomotion, exploration and rearing are noted with dopamine release while in humans, subjective sensations of involvement in useful activity and feelings of euphoria and expansiveness including increased power and energy are often reported (Gawin 1991). Whether in a rat or in a human, these behaviors and sensations increase the likelihood of confronting a provocative situation. Reiss and Roth (1993), in a recent report, conclude that low dosages of these drugs produce behaviors such as increased competitiveness. At high dosages, these drugs produce disorganized behavior and violent outbursts in some young male individuals have an extensive family history of alcoholism and who appear particularly susceptible to alcohol's ability to increase heart rate which has been taken as an indication of stimulation of this psychomotor activation system (Fowles 1980; Gray 1982). These individuals have shown trait characteristics of high sensation-seeking (Finn et al 1992) and often reflect histories of conduct disorder and antisocial personality (Pihl et al 1990). These behaviors have been linked to reward dominance and increased approach behavior where hesitation and withdrawal might be more appropriate. Thus, there is a potential for aggression to occur. One study using the Taylor Paradigm (Bailey and Taylor 1991) tested individuals grouped by self-report of degree of sober aggression. The high and moderately aggressive individuals demonstrated the most alcohol-related aggression. Drugs which stimulate the psychomotor system serve as potentiators of reward and, thus, aggression might readily be potentiated both in fact or in expectation.

Some researchers have argued that the effect of alcohol on dopamine is secondary, acting possibly through the potentiation of GABA-related inhibition (Harris et al 1992) or as a 5-HT agonist because the latter has been shown to be capable of stimulating the release of dopamine (Bladina et al 1988). Although it appears that acute alcohol administration increases central 5-HT function (Lemarquand et al 1994), Virkkunen and Linnoila (1993) have suggested that this effect is transitory and that an alcohol induced 5-HT rebound may drive previously low 5-HT levels even lower. This rebound may potentiate aggression among susceptible individuals given exposure to appropriate repetitive or punishing stimuli. Reduced levels of the central 5-HT metabolite CSF 5-HIAA have been found in many impulsive and aggressive populations such as aggressive children (Kruesi et al 1990), children particularly cruel towards animals (Kruesi 1989), adults with histories of aggressive behavior (Brown et al 1982), criminal recidivists who commit violent crimes (Virrkunen et al 1987) and impulsive alcohol abusing individuals with a family history of male alcoholism (Linnoila et al 1989). The 5-HT depleted individual can be described as irritable, reward- and punishment-driven and poorly inhibited. Thus, this individual should be more likely to be aggressive when cues for such behavior are present.
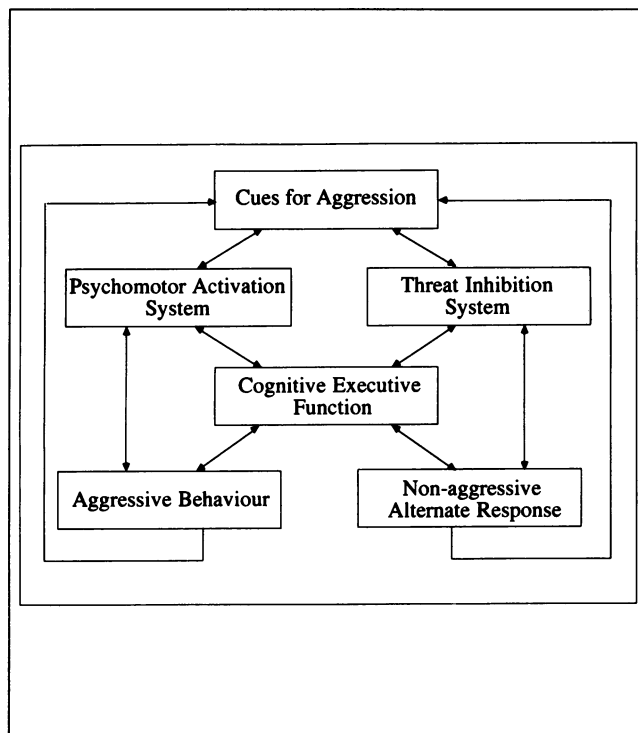

Fig. 4. Schematic of the interrelationship of three systems and centrality of the executive function in the determination of aggressive and nonaggressive responding.

Alteration in inhibitory and problem-solving cognitive functions

Jonathan Swift once remarked, "what is drinking but a mere pause from thinking." Indeed, it is evident that alcohol alters higher order functions which, when called upon, could diffuse the provocative situation and disarm aggressive intent. Figure 3 presents the results of a study (Peterson et al 1990) illustrating how relevant cognitive functions are affected by alcohol in a dose-dependent fashion. This large scale, balanced placebo designed study involved six groups of subjects with three dose groups of active placebo, 0.66 ml per kg or 1.32 ml per kg of 95% alcohol, crossed with the expectancy condition of being told that they were receiving alcohol or the condition that they were receiving a very low dose of alcohol. Expectancy effects were fundamentally absent but there were profound decrements in cognitive functioning, particularly at the higher dosage on tests that measured abstraction, delayed memory, planning and problem-solving, and motor skills that demanded self-monitoring. This particular pattern of deficit has often been linked theoretically to prefrontal cortical functioning. This particular brain area has been implicated in the application of previously established knowledge and in the gathering of

information in the face of novelty and threat. It is often referred to as serving an executive function. (Luria 1980; Peterson and Pihl 1990).

Recently Lau, Pihl and Peterson (1993) tested the relationship between the level of this type of cognitive functioning crossed with alcohol and performance on the Taylor Aggression Task. One hundred and fourteen nonalcohol male social drinkers were rank-ordered according to their performance on two neuropsychological tests (Self-ordered Pointing, Petrides and Milner 1982, and the Conditioned Association Test, Petrides 1985). Those subjects who performed in the upper and lower quartiles on a cumulative score were tested sober. Two other groups were tested while intoxicated with a dose of 1.00 ml/kg of 95% alcohol. The Aggression Task was presented in two parts: in the first part, the subject received shocks well below his subjectively determined pain threshold, termed "low provocation"; and, in the second part, shocks were near pain threshold, labelled "high provocation." All shocks, of course, were delivered according to an *a priori* random schedule. In the results there was an alcohol effect in which intoxicated subjects delivered significantly higher shocks to their opponents, a provocation effect in which individuals who received higher shock levels delivered higher shock levels, a cognitive effect in which individuals who scored in the lower quartile on the neuropsychological tests delivered higher levels of shock and a provocation by cognitive function interaction. In this interaction, the individuals in the lower cognitive performance quartile reacted most severely to higher levels of provocation. Alcohol also affected the high cognitive functioning group but only in the high provocation condition where they performed very aggressively, exactly like the low performing cognitive subjects. It would appear, therefore, that these types of cognitive functions play a profound role in controlling aggressive responses, most likely by providing alternatives and analyses of problem situations. Alcohol's effect, it would appear, functionally disrupts these processes. Figure 4 schematically represents the interrelationship of the cognitive system to the threat and psychomotor systems. The authors would argue that alcohol operates at each of these points, most likely differentially based on subject (perhaps genetic) characteristics, which, if executive function is diminished, further exacerbate the situation.

It may well be that, as seen with the other mechanisms, altered serotoninergic functioning could be involved in this deleterious process. The frontal area of the brain receives rich projections from the serotoninergic system and the conclusions of a number of reviews (Spoont 1992; LeMarquand et al 1994; Pihl and Peterson 1993) is that reductions in serotonin appear to produce disregulation of primary motivated behavior and decreased response to cues that normally control manifestation of such behavior. Thus, the 5-HT depleted individual is irritable, reward- and punishment-driven and poorly inhibited by threat. He or she is, therefore, probably more susceptible to alcohol's anxiolytic and psychomotor

stimulus effects. Spoont (1992) has speculated that what is upset is the integration of affect-cognition and behavior. The serotoninergic system's function in the frontal area might be compared to the conductor of an orchestra responsible for controlling the organization of the orchestra's constituent parts composed of potentially fractious but individualistic soloists who, for the purposes of music, must be melded into a harmonious unit. This role of maestro is not unlike that required to regulate central nervous system activity where sensitivity to the incoming sensory stimuli must be continuously modified, decreasing control and extending duration of psychomotor response to primary reinforcers and modulating sensitivity to cues such as punishment defined as threat that regulate driven behavior. Although speculative, drugs which could maintain 5-HT stability in the intoxicated individual might prove pharmacotherapeutic.

The general expectancy set

The general expectancy set represents an internalized paradigm, a statement of the context within which stimuli, in this case drugs, act. The notion has a basis in the theories of Luria (1980) and Sokolov (1969) as well as in the more elaborate hippocampal-comparator theories (Gray 1982; Taylor and Discenna 1986). This speculative structure is anchored in experience and learning. In most animals, it is the consequence of exploration. In humans, in addition to experience, it contains culture through language that the individual has incorporated. Each person has notions about what will happen in the future as a result of present behaviors. There are two primary functions for the system. First, it provides a context so that stimuli can be minimally categorized into those that are potentially threatening, punishing and rewarding. The survival of the organism depends on this fundamental categorization. Second, it determines when behavior, in this case aggressive behavior, becomes subject to threat inhibition or hope-induced potentiation. The three proposed mechanisms for drug effect on aggression act through this dynamic by diminishing the inhibition for aggressive behavior, by potentiating hope regarding reward outcome and by rendering a general disorganization to the system.

CONCLUSION

Laboratory manipulative studies confirm the phenomenon of increased aggression associated with certain drug consumption seen in crime statistics and offer clues as to some potential mechanisms. These mechanisms are viewed as causal only in the sense of how they can effect the likelihood of a behavior. The relative contribution of the myriad of complex interactive variables responsible for the final outcome no doubt have individualized vulnerability valences. Thus, while someday a grand general equation may be

possible, individual prediction will most likely remain at a relatively anemic problematic level.

Of the three proposed mechanisms, the pharmacological alteration in the threat system so that the inhibitions toward aggression are diminished, is the easiest to argue substantively. The animal literature has numerous examples of this phenomenon (Blanchard et al 1993) and the increased vulnerability of victims is readily incorporated. Seemingly askew is injunctive affect. Ask a drunk what will happen if he hits someone and the answer "there will be a fight" is likely, yet a punch nonetheless is delivered. The second proposed potentiator, psychomotor activation, is designed to explain the role of drugs that stimulate reward mechanisms and, at times and in certain populations, increase aggression. While these drugs have been linked to aggression, evidence for straightforward potentiation is relatively lacking and can be confused with drug-induced delusional states and withdrawal agitation, conditions which also increase aggressiveness and are not uncommon with this drug class.

Intuitively, diminished cognitive functioning seems apparent in the disorganized, inept and provocative behaviors of the intoxicated individual. Evidence supports this intuition and the link to increased aggressiveness. This type of induced deficit can independently raise the probability of violence but can also readily exacerbate the dimunition of threat inhibition or activation of the psychomotor system.

The three proposed mechanisms and the notion of an expectancy set are speculative but hopefully will serve both an organizational and heuristic purpose. The ideas are neither independent nor exhaustive but do provide explanatory value to some facts. The ultimate utility of these ideas, however, other than to those who must control intoxicated people, will require time and technologies more fanciful than the proposed ideas.

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