

# A Biosocial Model of the Alcohol-Aggression Relationship\*

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**ABSTRACT.** Four of alcohol's dose- and rate-dependent pharmacological properties may increase the likelihood of human aggression. As an anxiolytic, alcohol is capable of reducing the inhibitory effect of fear on manifestation of aggressive behavior. As a psychomotor stimulant, alcohol can potentiate aggressive behavior, once evoked, or lower the threshold for such evocation. Alcohol-related disruption of certain higher order cognitive functions may reduce the inhibitory control generally exercised by previously established knowledge and

decrease ability to plan in the face of threat or punishment. Finally, alcohol's ability to increase pain sensitivity may increase the likelihood of defensive aggression. Discussion of the nature and relevance of these pharmacological properties is structured according to a heuristic and synthetic schema, predicated upon consideration of an inhibitory neuropsychological structure—the individually and culturally determined general expectancy set. (*J. Stud. Alcohol*, Supplement No. 11: 128-139, 1993)

**T**HE FRENCH mathematician/philosopher of science Henri Poincare noted that “a pile of bricks is not a house.” Individual studies of a given phenomenon, lacking synthetic analysis, retain at best the potential of building blocks, particularly within the confines of modern society, glutted with poorly integrated information. Models give form to chaos, adding intelligibility to the apparently nonsensical, providing the structure and meaning of melody to what would otherwise remain a cacophony of individual notes. It might nonetheless appear premature to present a model synthesizing a set of phenomena whose mere existence remains subject to debate. Roizen (in press), for example, has challenged the viability and utility of the alcohol-aggression model per se after analysis of the vast cultural variability characterizing expression of alcohol-related aggression. However, the presence of tremendous variability does not necessarily invalidate a given supposition. It merely renders the presentation of that supposition more difficult.

Studies describing criminality in Western society consistently support the idea that alcohol intoxication is linked to aggressive behavior (Brain, 1986; Collins, 1981; Pernanen, 1976, 1981), both in terms of its commission and its reception. Murdoch et al. (1990) examined 9,304 criminal cases, reported in 26 studies, drawn from 11 countries. Sixty-two percent of violent offenders were drinking at the time of, or shortly before, commission of the crime. Forty-five percent of victims were intoxicated when victimized. Criminal acts that were actively aggressive were much more likely to be perpetrated by drunken individuals than were nonaggressive criminal acts. Those few studies that directly assessed physical measures of in-

ebriation have concluded that individuals involved in violence were often drinking heavily, to levels well above that defined by legal intoxication (Mayfield, 1976; Shupe, 1954).

Controlled laboratory studies also provide support for the hypothesis that alcohol intoxication predisposes individuals to expression of aggression. Bushman and Cooper (1990) summarized their meta-analysis of 30 experimental studies with the statement that “alcohol does indeed cause aggression. However, alcohol effects [are] moderated by certain methodological parameters” (p. 341). The data derivable from studies of criminal behavior, in combination with the results of controlled laboratory studies, constitute sufficient reason to posit the existence of a profound but complex alcohol-aggression relationship, and furthermore make it apparent that any model representing this relationship must be flexible enough to account for extant cultural, situational and experimentally induced variability.

The relationship between alcohol intoxication and violent behavior necessarily remains incomprehensible, in the absence of a clearly defined concept of aggression. Many theorists have contributed to the development of such a definition. Bushman and Cooper (1990) adapted Baron's (1977) description of aggression for application in the course of their review, defining an aggressive act as a “behavior directed toward the goal of injuring another living being, who is motivated to avoid such treatment” (p. 341). Buss (1961) determined that aggressive behavior could be classified as *physical/verbal*, *active/passive*, and/or as *direct/indirect*. Valzelli (1981) categorized aggression as *instrumental*, or as *hostile*. Instrumental aggressive acts are directed toward attainment of reward. Hostile aggression, by contrast, is devoted towards minimization of aversive conditions. These propositions are all predicated

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on the idea that aggressive acts are those intended to culminate in the production of an aversive state—in the aggressor or another—and it is on this broadened definition that the proposed model rests. But what constitutes an aversive state?

Various lines of convergent research, adroitly integrated by Gray (1982, 1987), suggest that aversive states fall into two categories: that of *pain*, physical and psychological, and that of *fear*. Physical and psychological pain emerge as a consequence of punishment. Punishing stimuli, which detrimentally affect the adaptive integrity of an organism, elicit three broad categories of response: fight or flight (Gray, 1987), manifestation of affiliation-seeking behaviors (such as crying or other distress behaviors) (Panksepp et al., 1985) and extinction of learned behavior (Gray, 1982). Punishment can take place at the physical level, and take the form of physical damage, or at the psychological level, and take the form of challenge to expectancy. Frustrations or disappointments (defined by Gray [1982] as absences of *expected* reward) might be considered psychological punishments, in that their manifestation depends on the internal psychic state (the expectancies) of the organism being frustrated or disappointed. Fear, by contrast, emerges as a consequence of threat. Threatening stimuli, which signal the potential for punishment, result in the inhibition of ongoing behavior—from the objective standpoint—and in the generation of fear, from the subjective level of analysis (Gray, 1982). Threatening stimuli are threatening either because they are novel, and are therefore of unspecified significance, or because they have consistently occurred in close association with punishment and serve as a punishment cue (Gray, 1982). The threat system serves to replace reliance on punishment during the course of socialization. In this manner, anxiety substitutes for pain (Pihl and Peterson, 1992).

Aggressive acts therefore appear (more specifically) as those intended to *punish* (to produce physical or psychological pain) or to *threaten*. This clarified definition can be further improved by an analysis of the conditions under which aggression is elicited. Defensive aggression is usually associated with subjection to punishment (Gray, 1982) and might be considered an unconditioned response, modifiable by learning. Instrumental aggression, by contrast, is associated with reward-seeking (including threat/punishment-avoidant) behavior (Valzelli, 1981). Its expression appears more purely dependent on learning—on learning the association between the application of punishment or threat, and the contingent receipt of a reward. It also appears reasonable and necessary to posit that aggressive acts may be classified as prosocial, or as antisocial, at least with regards to the human expression of behavior. Under certain circumstances, human cultures justify the application of punishment or threat to modify the expression of behavior that contravenes the moral code

embedded in the culturally determined expectancy set, described in some detail below. The application of aggression under such conditions might be considered prosocial. Antisocial aggressive acts, by contrast, involve the application of punishment or threat, and the simultaneous contravention of an established social more. Synthesis of this information leads to generation of a  $2 \times 2 \times 2$  definition of aggression, in which intentionally threatening or punishing actions are manifested in an instrumental or defensive manner, in a pro- or antisocial context.

### **Role of the General Expectancy Set: Elicitation, Construction and Modification of Aggression**

An expectancy state is a dynamic model of what is going to happen in the future as a consequence of behaviors manifested in the present (Luria, 1980). Expectancy states appear to arise as an emergent property of knowledge generated during novelty-induced exploratory activity (Pihl and Peterson, 1992). Novelty motivates exploratory behavior, designed to determine the actual significance of the novel occurrence, for reward or for punishment (Gray, 1982). Application of language (Luria, 1976, 1980) and imitation allows for the direct communication of such knowledge from individual to individual. This means that an expectancy state is a socioculturally determined construct. Mesquita and Frijda's (1992) recent review describes the culture-specific nature of such expectancy states, or cognitive schemas, and details evidence suggesting that the nature of these schemas modifies event appraisal, determines the behavioral repertoire in general and alters various processes involved in the regulation of emotion.

The general expectancy set (GES)—composed of all extant expectancies—appears to provide the context for interpretation of environmental stimuli. This set serves an exceptionally complicated general function: inhibition of anxiety and novelty-induced psychomotor activity as a consequence of learning (Sokolov, 1969). It subsumes expectancies about the specific behavioral effects of alcohol (which may include the belief that drinking increases levels of interpersonal aggression [Brown et al., 1980; Lang and Sibrel, 1989; Roizen, 1983]) and constitutes the historically and individually determined structure upon which alcohol exercises its pharmacological action (as outlined below). It is the consequence of this necessarily idiosyncratic and culture-specific expectancy-set/drug interaction, modeled schematically and presented in Figure 1, which determines the highly variable nature of the alcohol-aggression relationship.

The general expectancy set performs three functions relevant to consideration of the alcohol-aggression relationship. These three functions determine elicitation, construction and modification (facilitation and inhibition) of aggression. First, the GES provides the context within

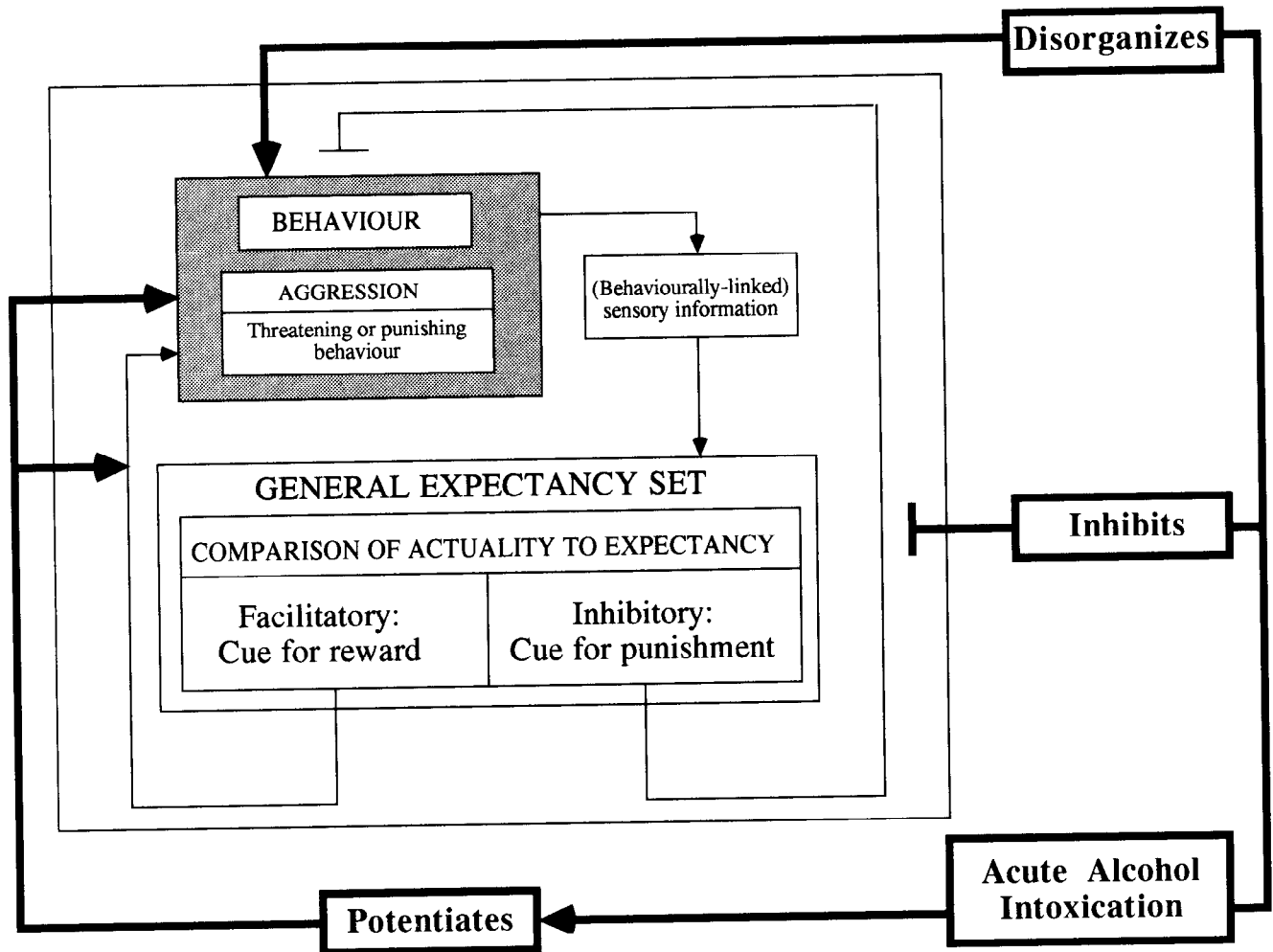


FIGURE 1. The effect of acute alcohol intoxication on aggression: Inhibition of fear, potentiation of reward (and of cues for reward) and disorganization of behavior

which "objective" stimuli are subjectively interpreted, and therefore determines what is novel by exclusion (all that is not represented is novel); what is threatening (all that has been experienced in contiguous association with punishment, and all that is novel, is threatening); a certain subset of what is punishing (determines what is disappointing or frustrating—what constitutes the absence of an expected reward); and a certain subset of what is rewarding (such as the absence of an expected threat or punishment). Second, the GES also constitutes the repertoire of culturally determined behaviors from which aggressive responses can be selected for application, in response to eliciting stimuli. Intended behaviors that engender aversive states may be very elaborate and complex in their construction. In the case of an animal, the construction of such behaviors is limited to the creative capacity (or the innate potential) of the animal. In the human case, the capacity for aggressive action is a consequence of the sum total of cultural endeavor, insofar as that has been internalized during the course of socialization. Finally, the

GES determines the conditions under which aggressive behavior becomes subject to fear-predicated (punishment-expected) inhibition or hope-induced (reward-expected) potentiation. Fear inhibits the expression of aggression when such expression has been linked with punishment in the past. Likewise, hope facilitates the expression of aggression when such expression has been linked with reward in the past. Cultural context determines, in large part, the circumstances under which aggressive responding will be associated with socially predicated reward (and therefore potentiated as prosocial) or punishment (and therefore limited as antisocial).

#### **Alcohol Effects: Inhibition of Threat, Potentiation of Psychomotor Activity, Disruption of Cognitive Function and Sensitization to Pain**

Alcohol has four relatively well-defined dose and time-related pharmacological effects, which have the potential to (independently and cumulatively) increase the expres-

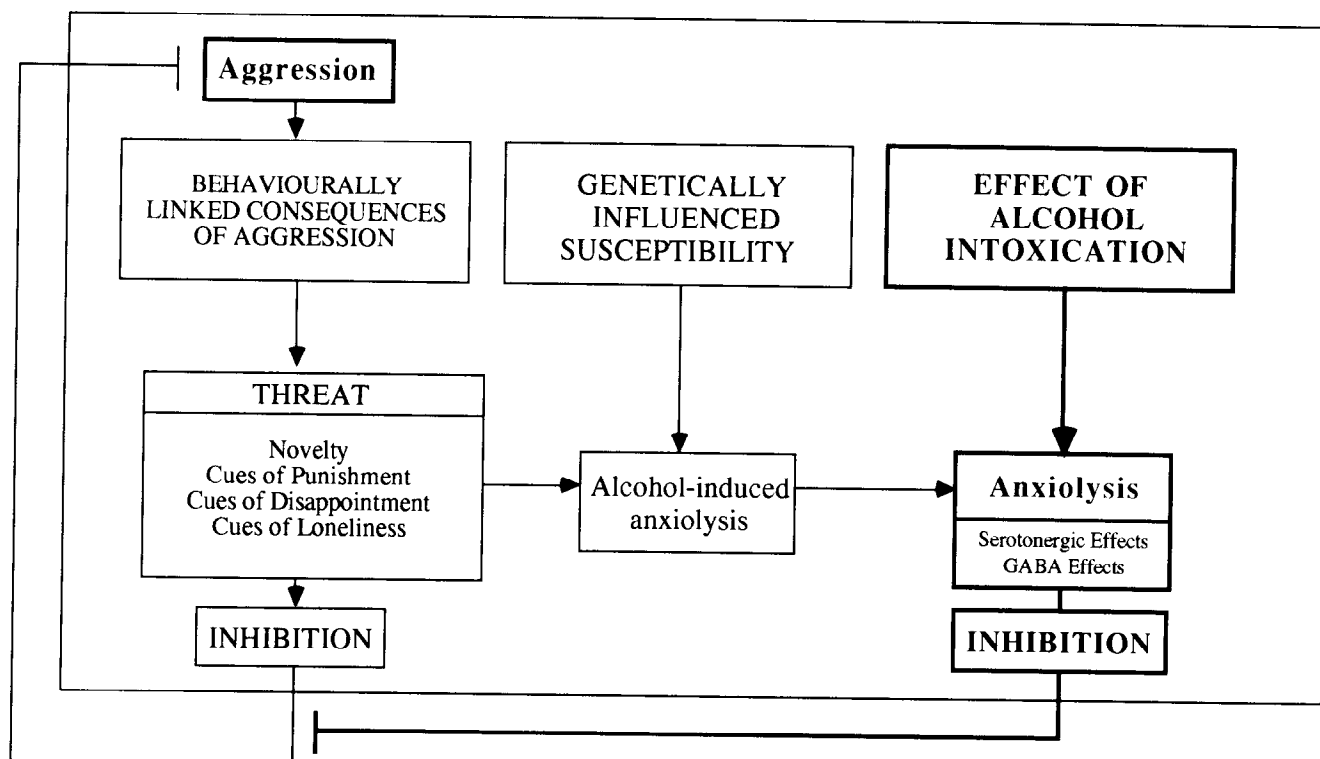


FIGURE 2. Alcohol elimination of threat sensitivity and its potential effect on aggression

sion of aggression. The first of these effects, which might be described as anxiolytic, reduces threat-related inhibition of behavior; the second, which might be described as stimulant, potentiates psychomotor activity; the third interferes with specific aspects of higher order cognitive function; and the fourth produces (population-specific) increases in pain sensitivity.

*Elimination of threat*

The anxiolytic effects of alcohol, which are comparable to those produced by benzodiazepines or barbiturates, appear manifested via pharmacological effects on the functioning of the anxiolytic-sensitive septal/hippocampal, novelty/threat detection system (Gray, 1982, 1987). It appears possible that anxiolysis emerges as a consequence of alcohol's effect on GABA, the brain's major inhibitory neurotransmitter. Alcohol enhances the ability of GABA to open the CL-ion channel, at the GABA-benzodiazepine-CL-receptor complex, and additionally directly potentiates CL-ion intake (Warneke, 1991; Zorumski and Isenberg, 1991). These two actions increase the firing rate of GABA neurons and enhance their inhibitory action. The hippocampus contains a number of GABAergic neurons, arranged in "recurrent inhibitory circuits" (Gray, 1987, p. 301), and alcohol may play its anxiolytic role at this level, more indirectly, through potentiation of serotonergic

activity (LeMarquand et al., 1992) or through a number of other potential mechanisms (Gray, 1987).

The septal/hippocampal system appears responsible for the inhibition of ongoing behavior as a consequence of exposure to threat or novelty (Gray, 1982, 1987). Alcohol, as an anxiolytic, reduces the inhibitory effect fear normally exercises on expression of dangerous behavior—behavior whose manifestation has been linked in the past to receipt of punishment or threat (Gray, 1982, 1987). This means that alcohol may facilitate the expression of aggression when such expression is under the inhibitory control of fear (and when the potential aggressor is sensitive to the anxiolytic properties of alcohol). The relationship between alcohol-induced anxiolysis and aggression is outlined schematically in Figure 2.

Fear logically retards the expression of motivated aggression when that expression would expose the aggressor to receipt of punishment or threat as a consequence of interpersonal retaliation, or of the revenge of society as such. The tendency for intoxicated individuals to become victimized can also be comprehended from this perspective. Fear produced in response to threat (to cues of imminent punishment) play an adaptive role—that of helping individuals avoid harm. If the anxiety (the behavioral inhibition) such cues evoke is pharmacologically reduced, then the chance for harm inevitably increases. Intoxicated individuals may therefore prove more likely to participate

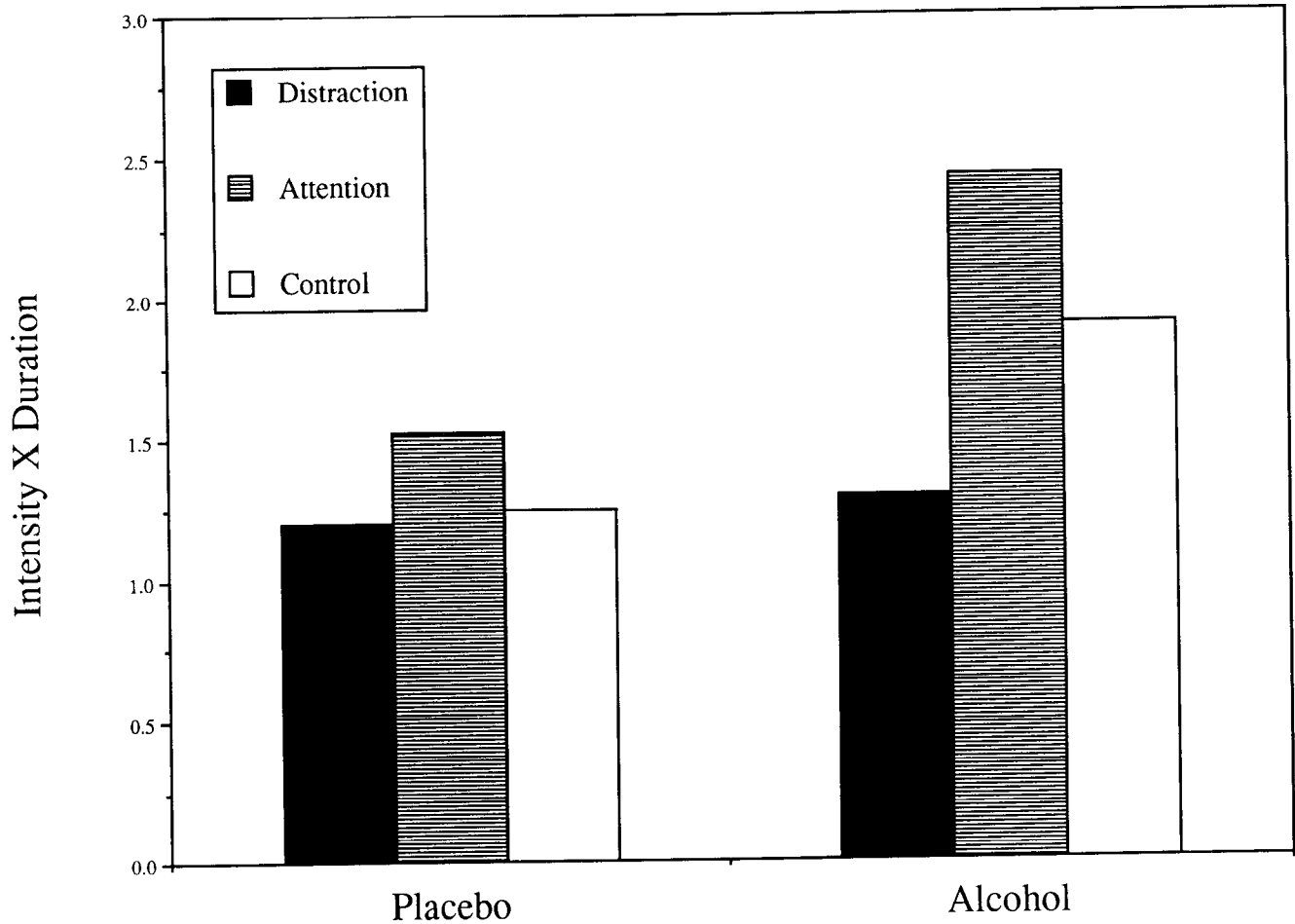


FIGURE 3. The interaction between alcohol intoxication and intensity by duration of shock administered during participation in a Buss-Taylor aggression task under normal (control), distraction and forced-attention conditions

in dangerous activities that would inspire fear in the sober state.

Various experimental studies have demonstrated alcohol-induced threat-inhibition-reduction potentiation of aggression (Cherek et al., 1985; Kelly et al., 1988; Leonard, 1989; Shuntich and Taylor, 1972; Taylor and Gammon, 1975; Taylor et al., 1976). Three studies from the McGill Alcohol Research laboratory are detailed. Zeichner and Pihl (1979) administered placebo, no alcohol or 1.32 ml of 95% alcohol/kg of body weight to three groups of young males. Subjects assigned to each of these three dose groups were required to participate in one of two modified Buss-Taylor paradigms: one contingent, in which they were subjected to aversive tones whose intensity was correlated with the shocks they delivered; and one non-contingent, in which they received aversive tones whose intensity was not correlated with their behavior. Subjects who consumed placebo or no alcohol modified their behavior appropriately and reduced the intensity of the shock they delivered. Alcohol-intoxicated subjects, by contrast, failed to modify their aggression in response to

the threat (the punishment cue) integrally part of the experimental task. Analysis of this experiment led Zeichner and Pihl to suggest that alcohol interfered with an individual's ability to "process information pertinent to the consequences of their own behavior" (p. 159). In keeping with this hypothesis, Zeichner et al. (1982) administered the same dose of alcohol or placebo to two groups of young males prior to their participating in another modified Buss-Taylor task, and then asked them either to record the level of shock they administered and received, thereby forcing them to attend to their behavior or to complete a series of mathematical problems while administering/receiving shocks, and to thereby become distracted. Forced attention actually increased alcohol's potentiation of aggression whereas distraction reduced it. Results are graphically presented in Figure 3. The acutely intoxicated participants were not more aggressive because they were unaware, verbally or cognitively, of what they were doing and of the consequences of their behavior, but because that knowledge no longer served an inhibitory function, perhaps because it no longer produced fear.

Finally, Zeichner and Pihl (1980) tested three groups of young males administered alcohol (as above), placebo or no beverage who shocked a (fictitious) opponent in response to that opponent's delivery of tones varying in aversiveness under two conditions of intent—neutral and malicious. In the former condition, participants were told they would receive tones whose aversiveness was fixed, a priori, according to a schedule established by the experimenter. In the latter condition, subjects were told that tone aversiveness could be chosen, voluntarily, by their opponent. Tone aversiveness in both conditions was actually predetermined in both cases. Intoxicated subjects were more aggressive, in general, than nonintoxicated individuals. In addition, the intoxicated participants were immune to inhibition of aggression induced by consideration of intent, although the placebo and no-alcohol subjects reduced their shock administration in the neutral condition. Intoxicated subjects reacted to increased intensity of stimuli received with increased level of shock administered, regardless of intent. Placebo and no-alcohol subjects, by contrast, reacted to intensity in the malicious condition only. These results can be comprehended by reference to cultural factors—to cultural construction of the general expectancy set. In Western society, application of threat or punishment is supposed to remain restricted by consideration of intent. The expression of aggression in the absence of consideration of intent or ability is generally followed by receipt of punishment or frustration. Consideration of intent, and consequent modification of behavior, is fundamental to well-socialized behavior. Acutely intoxicated individuals in the Zeichner and Pihl (1980) study did not modify their aggressive behavior according to the socially defined moral code, and responded straightforwardly to intensity of punishment received. The inhibition normally produced by the threat inherent in breaking a social rule appears to have been dissolved in alcohol, so to speak.

#### *Potentiation of psychomotor activity*

The psychomotor effects of alcohol, which are simplistically comparable to those produced by cocaine or amphetamine, appear manifested via pharmacological effects on the functioning of the stimulant-sensitive dopaminergic psychomotor exploratory system (Pihl and Peterson, 1992). Alcohol appears to manifest its psychomotor stimulant properties in part by activating (increasing the firing rate) neurons in various areas of the mesocorticolimbic dopaminergic system (Gessa et al., 1985; Harris et al., 1992). This stimulant effect appears rewarding and unconditioned (Wise and Bozarth, 1987). The precise mechanism whereby this effect occurs remains unspecified, and appears complex (Samson et al., 1992). Alcohol may increase dopaminergic firing directly, as its perfusion into ventral tegmental area slice preparation medium has dem-

onstrated (Brodie et al., 1990). It appears to increase DA or DA metabolite levels in the ventral striatum as well, and potentiates DA release from the nucleus accumbens, compared to striatum (reviewed in Samson et al., 1992). Some researchers have hypothesized that alcohol-related DA neuron activation may occur indirectly, in a very complex manner, as a consequence of its potentiation of GABAergic inhibition, by inhibiting the effect of neurons that inhibit DA firing in the ventral tegmental area (Harris et al., 1992). Still others have identified a potential (and equally complex) role for serotonin (Wise and Bozarth, 1987). Ethanol administration appears, in general, to serve as a 5-HT agonist, to facilitate serotonergic neurotransmission. 5-HT and 5-HT<sub>3</sub> agonists are capable of stimulating the release of striatal dopamine (Bladina et al., 1988). In addition, 5-HT<sub>3</sub> antagonists block ethanol-induced increases in dopamine release in the nucleus accumbens (Carboni et al., 1989; Yoshimoto et al., 1992), perhaps by restricting firing of 5-HT neurons that synapse on dopaminergic neurons, or through the action of 5-HT<sub>3</sub> receptors located on dopaminergic neurons. It is of interest in this regard to note that Pihl et al. (1992) demonstrated that individuals administered a 5HT-precursor (L-tryptophan)-depleted diet, crossed with alcohol, were more aggressive while participating in a modified Buss-Taylor task than those given tryptophan augmentation and no alcohol.

Whatever the mechanism, and under certain restricted conditions of dose and timing, alcohol is clearly capable of serving as a psychomotor stimulant. Susceptibility to this effect appears population specific. Young men at heightened familial risk for alcoholism, for example, appear particularly sensitive to alcohol's ability to increase heart rate (Stewart et al., 1992). Heart-rate acceleration has been linked by Fowles (1980) to stimulation of Gray's (1982) behavioral activation system (BAS), whose functions are in turn associated with dopaminergic activity, and with reward. Psychomotor stimulants nonspecifically potentiate motor activity (Wise, 1988). The psychomotor stimulant effects of alcohol may therefore potentiate the expression of aggression under any conditions where (sober) aggression would normally be elicited, and perhaps where the potential for aggression has been released during intoxication by alcohol-induced inhibition of fear, under those conditions where such intoxication produces psychomotor stimulant effects. Bailey and Taylor's (1991) study supports this general conclusion. These authors tested three groups of young males: high, moderate and low aggressive (by self-report), administered either 0.98 or 0.16 ml/kg of 95% ethanol (i.e., vodka) prior to participation in a competitive reaction-time task. Initial ratings of aggressiveness appear as a measure of a priori probability of aggressive motor behavior. As might be predicted from this perspective, intoxicated high and moderate aggressives increased shock levels administered more rap-

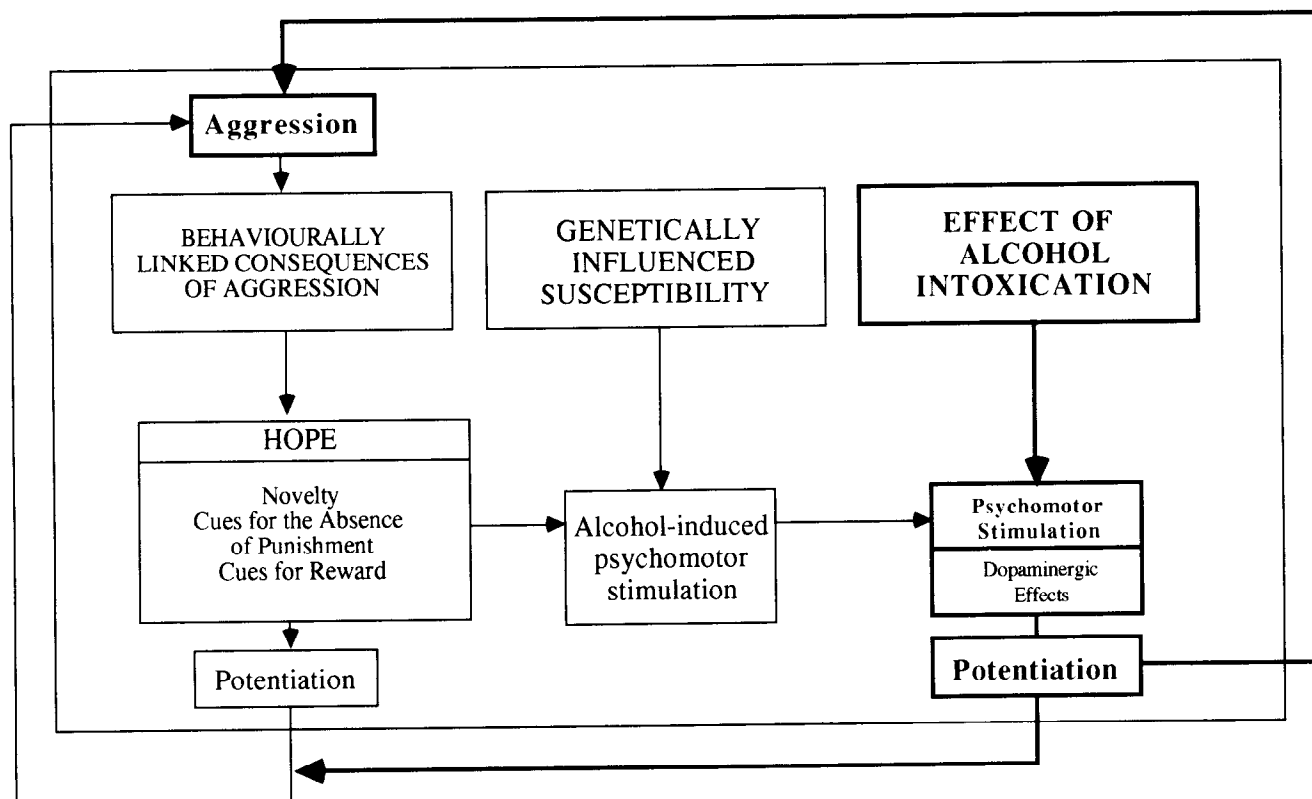


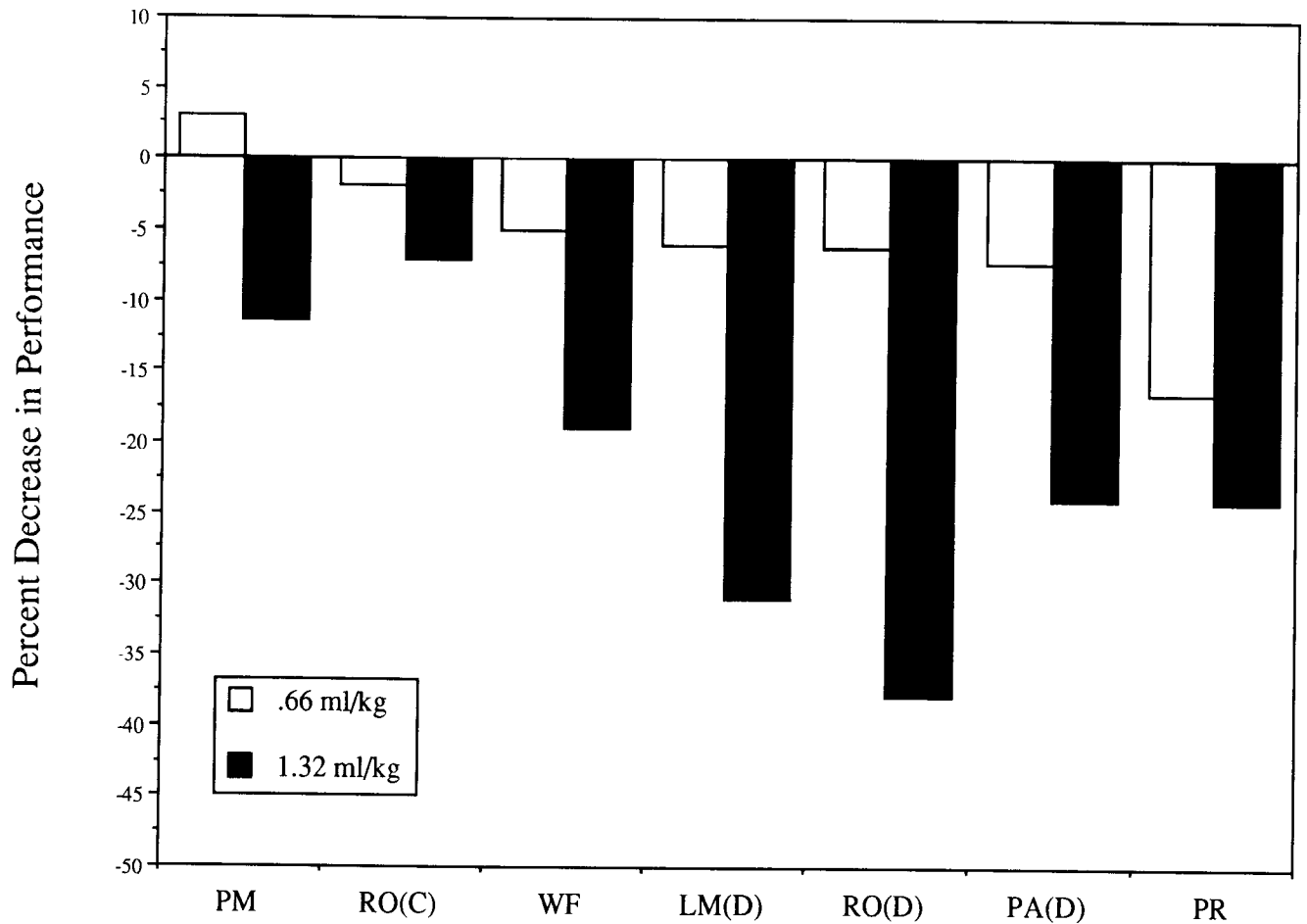
FIGURE 4. Alcohol psychomotor stimulation and its potential effects on aggression

idly, as a function of provocation, than intoxicated low aggressives. It is also of interest to note, in passing, that psychomotor stimulants lower the threshold for stimulus-potentiation of dopaminergic activity for activation of reward (Wise, 1988). This means that alcohol may additionally potentiate the expression of aggression under those conditions where such expression is rewarding (and perhaps under conditions where such expression heightens the expectation of reward). The complex relationship between the psychomotor stimulant properties of alcohol and aggression is presented schematically in Figure 4.

#### *Disruption of higher order cognitive function*

The anxiolytic properties of alcohol appear to reduce behavioral application of information regarding threat, and this reduction appears to increase the probability of aggression in those situations where aggression would normally be inhibited by fear. The psychomotor stimulant properties of alcohol appear to facilitate the expression of aggression, once elicited, and/or to lower the stimulation threshold determining when such behaviors emerge. Peterson et al. (1990) have demonstrated, in addition, that alcohol is pharmacologically capable of interfering with certain aspects of cognitive function at blood alcohol concentrations commonly attained by voluntary drinkers. These authors administered a battery of neuropsychologi-

cal and motor tests to six groups of young males. Each group consisted of individuals administered one of three doses of alcohol (placebo, 0.66 ml/kg 95% USP ethanol, and 1.32 ml/kg 95% USP alcohol) under one of two expectancy conditions: (1) told alcohol and (2) told no alcohol. The results of the test battery indicated that alcohol had particularly detrimental effects on various tests of abstract, higher order cognitive ability, on delayed memory and on motor skills that demanded self-monitoring. These results are presented graphically in Figure 5. By contrast, alcohol had virtually no effect on standard IQ tests such as the WAIS-R Vocabulary and Information subtests (Wechsler, 1981), or on functions such as simple reaction time (Peterson et al., 1990). Alcohol appears particularly destructive to cognition in the case of those intellectual abilities that underly planning (theoretically dependent on the intact function of the prefrontal cortex), and the transformation of short-term information into long-term memory (dependent on the intact function of the hippocampal complex, which is involved in recognition of threat). The prefrontal cortex plays a vital role in the formulation of verbal and motor strategies, designed to gather information in the face of novelty and/or threat, or to otherwise deal with that threat (Luria, 1980; Peterson and Pihl, 1990), and in the application of previously established knowledge (which constitutes the GES) to regulation of behavior. The fact that alcohol interferes with the opera-



### Neuropsychological Tests

FIGURE 5. Dose-related effects of alcohol intoxication on cognitive performance: Tests associated with frontal function: PM (Porteus Maze), RO(C) (Rey-Osterreith, copy), WF (Thurstone Word Fluency); tests associated with hippocampal function: LM(D) (Logical Memory, Delayed), RO(D) (Rey-Osterreith, delay), PA(D) (Paired Associated, Delay); tests associated with motor function: PR (Pursuit Rotor) (Peterson et al., 1990)

tion of the threat/novelty registration system means that an acutely intoxicated individual will not respond properly to signals of imminent punishment. Alcohol-engendered prefrontal trouble is likely, in addition, to render the drunken individual incapable of acting in accordance with such signals, even when they still produce anxiety, in the intoxicated state.

Lau et al. (1992) examined the potential role of cognitive abilities associated with the function of the frontal lobe in mediating sober and alcohol-intoxicated aggressive responses to provocation. In the course of this study, 114 male social drinkers were administered two cognitive tests (developed at the Montreal Neurological Institute) potentially associated with the intact functioning of the frontal cortex: the spatial conditioned-association task (Petrides, 1985) and the self-ordered pointing task (Petrides and

Milner, 1982). Subjects were first categorized by quartile in accordance with their performance on these tests. Those who fell within the upper and lower quartile divisions were consequently selected for participation in a modified Buss-Taylor competitive-aggression task. Half of these subjects were tested sober; the other half while under the influence of 1.00 ml/kg of 95% USP alcohol. The aggression task itself was presented in two phases. During the first 13 trials subjects received shocks at an intensity below half their subjectively determined pain threshold. These trials constituted low provocation. In the last 13 trials, subjects received shocks at an intensity that exceeded half their subjectively determined pain threshold. These trials constituted high provocation. All subjects received shocks randomly, computer-delivered, at an intensity and duration determined a priori. Analysis of the results of

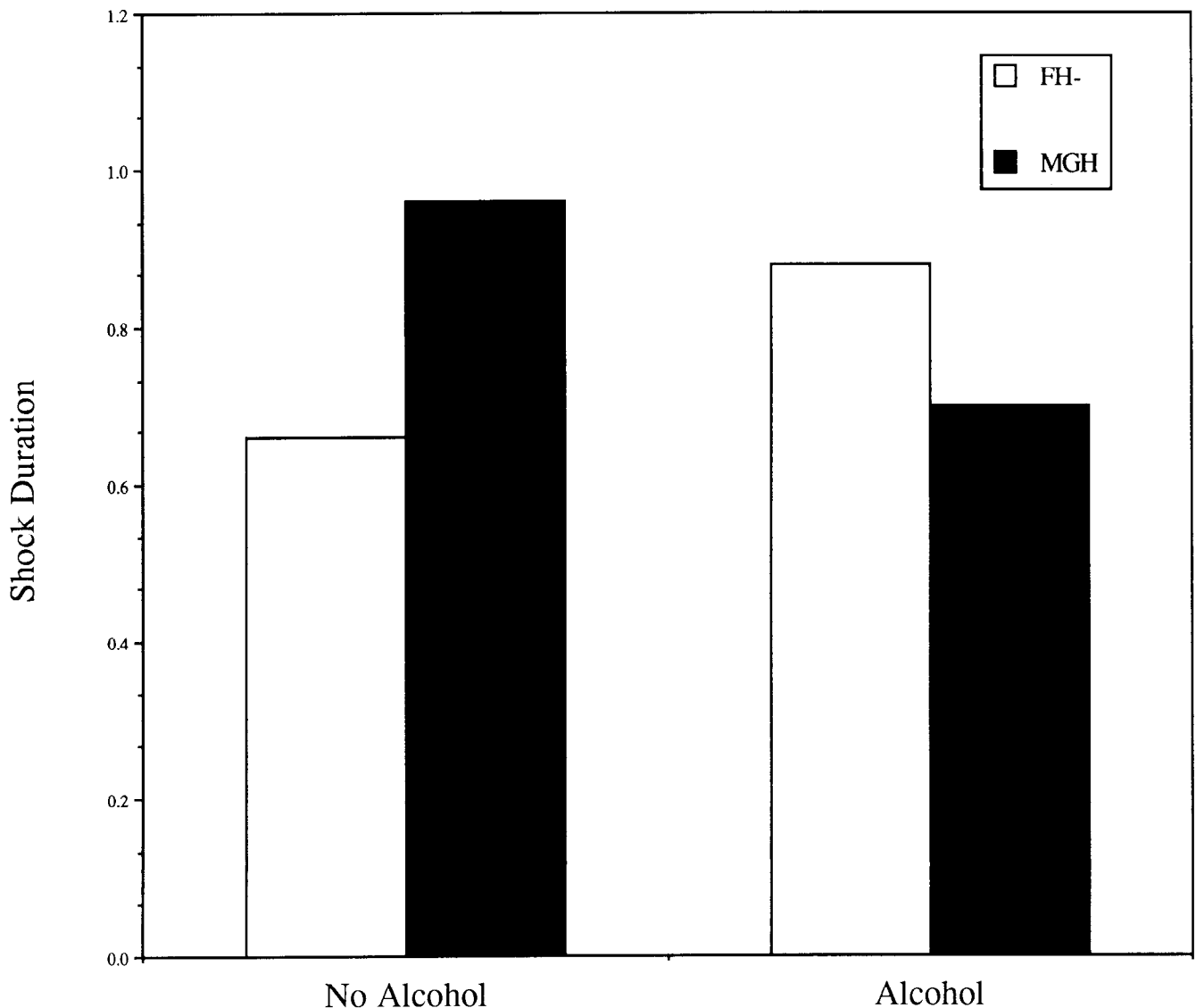


FIGURE 6. The interaction between shock duration administered during participation in a Buss-Taylor aggression task and alcohol intoxication, by family history of alcoholism (FH- = no family history of alcoholism for two generations; MGH = family history of male alcoholism, i.e., alcoholic father, paternal grandfather and paternal uncle, minimum)

this study demonstrated that aggression (measured as intensity of shock delivered by each subject) was affected by alcohol, in that alcohol intoxicated individuals delivered higher levels of shock overall; by cognitive function, in that individuals in the lower cognitive performance quartile delivered higher levels of shock, overall; by provocation, in that receipt of high levels of shock resulted in delivery of higher levels of shock; and by a provocation by cognitive function interaction, in that individuals in the lower cognitive performance quartile reacted to increased provocation with dramatically heightened increase in shock level delivered. This study suggests that individuals characterized by reduced cognitive ability, associated with the function of the prefrontal cortex, are less able or willing to inhibit their aggression when punished, in general,

and that this lessened inhibitory control is further reduced when punishment levels increase. These findings support the notion that specific aspects of higher order cognitive function are critical to the control of evoked aggression.

#### *Pain sensitivity*

Pain elicits defensive aggressions as an unconditioned response, as described previously. It may seem absurd, prima facie, to suggest that alcohol-intoxicated individuals might be more sensitive to pain, since alcohol use is popularly associated with pain reduction, and since alcohol has been used since time immemorial as an anaesthetic (Mullin and Luckhardt, 1934; Wolff et al., 1942). However, the fact that alcohol is sedating or hypnotic at high

doses does not necessarily mean that it is analgesic at lower, potentially stimulating doses. There is tremendous individual variability in sober and alcohol-intoxicated pain sensitivity. Gustafson (1985), for example, has demonstrated that alcohol-intoxicated subjects (as opposed to those given placebo) heighten their rating of pain sensitivity to electric shocks received while participating in a modified Buss-Taylor task. Gray (1982) reviewed studies suggesting that alcohol-intoxicated rats were characterized by reduced flinch and jump-thresholds to electric shock. Stewart et al. (in press) recently completed a study demonstrating population-specific sensitivity to alcohol-induced analgesia. These authors demonstrated that men at substantially heightened familial risk for the development of alcoholism were more sensitive to pain induced by electric shock while sober, as compared to low-risk controls, but that this difference in sensitivity disappeared under the influence of intoxicating doses of alcohol. Alcoholics also appear less sensitive to pain while intoxicated (as reviewed by Pihl et al., 1990). Pihl and associates (1990) have also demonstrated that males at heightened familial risk for alcoholism appear more aggressive when sober (when they are more pain sensitive) and less aggressive when intoxicated—at least while participating in the Buss-Taylor task. The results of this study (Pihl et al., 1990) are presented in Figure 6.

### Conclusion

Alcohol's specific pharmacological properties are becoming more clearly delineated. Consideration of these properties, within a context defined by appreciation of the general expectancy set, allows for deeper understanding of the alcohol-aggression relationship. There is little support for the idea that alcohol *causes* aggression. A drunken individual is not able to think up or act out complex patterns of aggressive behavior, which he or she could not conceive of or manifest while sober. Alcohol's effect on aggression appears more specific and more complex. Alcohol appears pharmacologically capable of reducing the inhibitory control of threat. This makes individuals more likely to exhibit behaviors that would normally be inhibited by fear. This heightens the probability that intoxicated persons will perpetrate, or become victims of, aggressive or otherwise dangerous behavior. Alcohol may also act as a psychomotor stimulant. Psychomotor stimulants amplify ongoing behavior, so to speak, and reduce the threshold for the elicitation of behavior, in general. This means that an alcohol-intoxicated individual, sensitive to the psychomotor stimulant properties of alcohol, needs less reason to be aggressive, and is likely to be more aggressive once provided with reason. Alcohol is also capable of increasing pain sensitivity, and therefore may lower threshold for elicitation of pain or punishment-elicited (defensive) aggression. Finally, alcohol interferes with selected aspects

of higher order cognition, particularly with those cognitive functions dependent on the intact structure of the prefrontal cortex. This cortical structure is critically involved in the regulation of ongoing behavior, as a consequence of previously learned information, and in planning in general.

There is wide individual variation in sensitivity to each of these alcohol effects. The most dangerous drunken individual should be one who is (1) capable of effectively manifesting a wide range of previously rehearsed (culturally or individually constructed) aggressive behaviors when sober, but generally inhibited in such expression by fear; (2) sensitive to the anxiolytic properties of alcohol; (3) sensitive to the psychomotor stimulant properties of alcohol; (4) more pain-reactive when drunk; and (5) impaired in the ability to plan and regulate behavior, when drunk. More specific assessment of this sensitivity should lead to enhanced comprehension of the alcohol-aggression relationship. Specification of such variability is well within the realm of possibility, at present levels of experimental sophistication.

The model presented in this article suffers from a number of limitations. It is speculative and may well be too inclusive. In general, there is a dearth of experiments that test the relationship between alcohol's known pharmacological properties and the expression of aggression. There is little direct support, for example, for the proposition that ethanol-induced psychomotor stimulation can potentiate aggression. However, research into the nature of alcohol's physiological (and psychological) effects is advancing rapidly, particularly as a consequence of sophisticated laboratory work conducted upon animals. Speculation has its dangers; however, it would be inappropriate to ignore the implications of modern animal research in the endeavor to further our comprehension of aggression.

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