

# Alcohol and Aggression: Three Potential Mechanisms of the Drug Effect

R.O. Pihl and J. Peterson<sup>1</sup>

The alcohol-aggression relationship is multifactorial and interactive. Indeed, given the results from numerous expectancy studies completed by and alluded to by Lang (this volume), alcohol per se does not even need to be a necessary condition. Just the belief that one has consumed alcohol can sometimes suffice. Factors operating at the level of the person and the environment/culture each affect the response to the drug and in turn are altered by the resultant feedback. Thus, the two current approaches to explaining drug-related violence of either focusing on the characteristics of the person consuming the drug or what the drug is doing to the individual are both valid.

Unfortunately, the involvement of myriad factors has led to the large degree of variability within and between studies, which, as Lang pointed out, appears to be an endemic problem. In order to begin to grasp the sources of confusion, attention needs to be directed at delineating more specifically the role of the factors that comprise the interaction. Given a

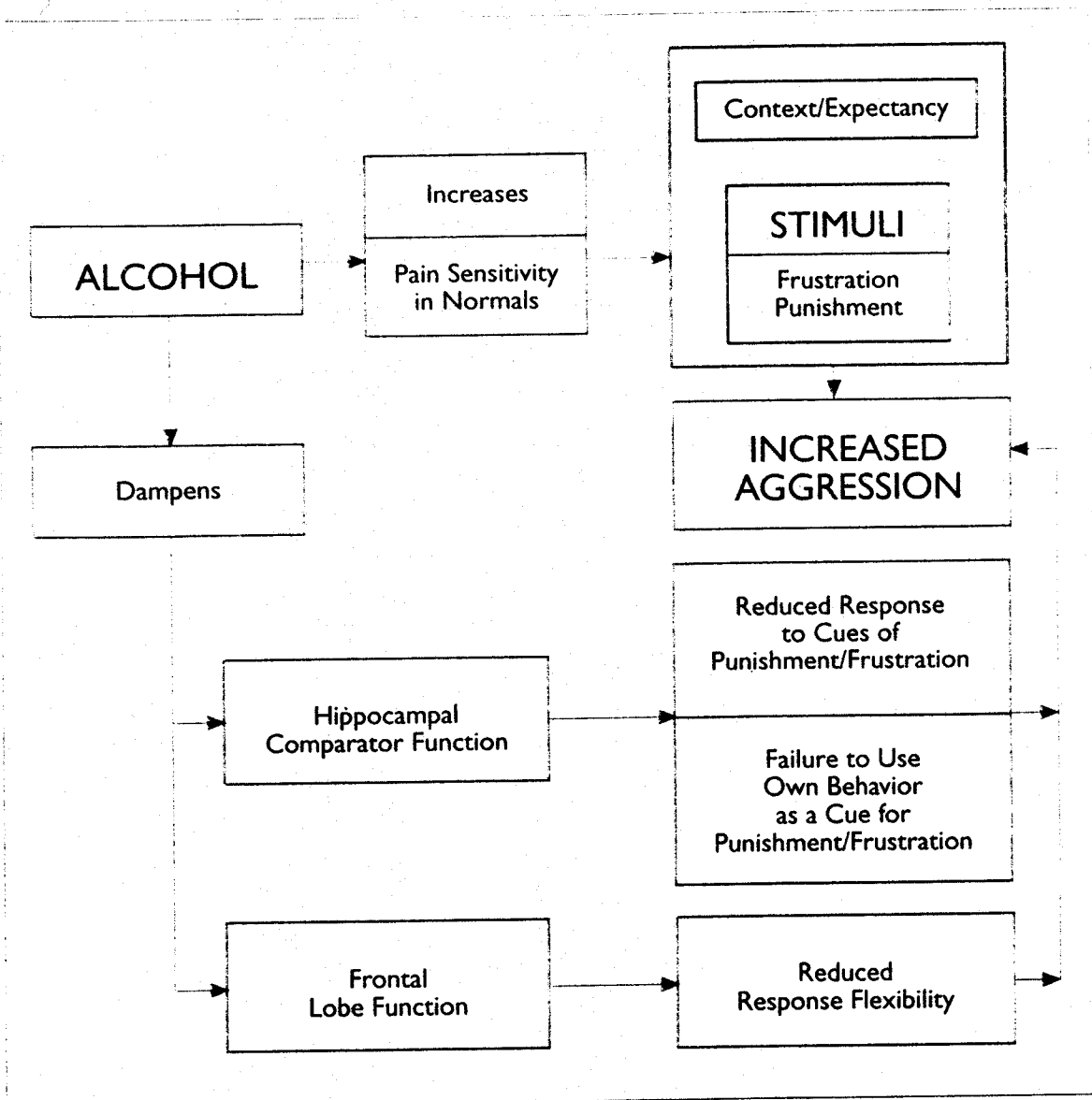
solid foundation, perhaps then pieces of the puzzle will begin to fit with greater predictability.

This brief paper is designed to focus explicitly on the question of how alcohol may directly affect psychological mechanisms that would increase the likelihood of aggressive behavior. Three specific mechanisms that we have recently explored in detail elsewhere (Pihl and Peterson 1993; Peterson and Pihl 1990) will be discussed and results of laboratory alcohol/aggression studies presented in their support. These mechanisms are an increase in pain sensitivity in normal individuals, a decrease in the use of cues regarding one's own behavior, and a decrease in frontal lobe functioning and a concomitant loss of alternative problem-solving strategies. Figure 1 presents a schematic of these three mechanisms and their putative effect in relationship to potentially increased aggression.

Laboratory studies of the alcohol-aggression relationship provide the advantages of control, precision, and the ability

---

<sup>1</sup>Departments of Psychology and Psychiatry, McGill University, 1205 Dr. Penfield, Montreal, Canada H3A 1B1



**FIGURE 1**

A theoretical model of the effect of alcohol on mechanisms that increase the likelihood of aggressive behavior.

to specify variables, circumstances usually absent in nonlaboratory situations. However, the disadvantages of such studies include the range of limitations referred to by Lang (this volume) and, in particular, the use of often arguable measures of aggression and the manipulation of variables conservatively labeled "artificial." The aggression measures utilized in the

studies described below were the intensity and duration of an electric shock that one subject administered to another presumed subject (actually a computer) in a reaction time competitive task. This procedure, labeled the Buss-Taylor Task, has had a number of modifications, the majority of which involve use of a provocative aversive stimulus being delivered to the subject by

the "partner" when a trial is lost. Aggression scores obtained with this procedure have been shown to be both reliable and valid (Bernstein et al. 1987) and are related to aggression rated by peer (Williams et al. 1967) and by self (Shembert et al. 1968) as well as to a history of antisocial behavior (Hartman 1969). The procedure is also the "method of choice" in alcohol and aggression studies, which have demonstrated an expectancy effect, an alcohol effect, variability relative to type of alcohol consumed, and dose, provocation, and attributional effects (for reviews, see Taylor 1983; Pihl 1983; Pihl and Ross 1987; Lang and Sibrel 1989; Bushman and Cooper 1990).

### **PAIN SENSITIVITY**

Pain, broadly defined to include frustration and the absence of expected rewards, is easily the most apparent eliciting stimulus for aggression. This literature is voluminous and consistent and is really only criticized because it does not account for the totality of aggressive behavior. Thus if alcohol was in some way to increase sensitivity to pain, a persuasive explanatory factor would be evident. Unfortunately, alcohol consumption linked with increased pain sensitivity seems counter-intuitive. "Feeling no pain" when intoxicated is part of the popular vernacular, an idea that seems to mix much better than alcohol and aggression. Indeed, alcohol has been used as an anaesthetic (Mullin and Luckhardt 1934; Wolff et al. 1942).

However, as mentioned previously, alcohol effects are not ubiquitous. Rather, they are related to dose, rate of adminis-

tration, time passed since consumption, subject characteristics, previous drinking history, and undoubtedly other factors. Thus a drug which has analgesic properties at high dosages may in some individuals have quite the reverse properties at lower dosages. Grey (1982) has reported in a series of studies with rats an increased sensitivity to pain while alcohol intoxicated. Specifically, these animals showed reduced flinch and jump thresholds to electric shocks. Gustafson (1986) has further noted increased subjective ratings by humans of sensitivity to electric shocks, when given alcohol over a placebo.

In our research with individuals from multigenerational alcoholic families, we have failed to demonstrate increased pain sensitivity when intoxicated and, in fact, have shown just the opposite in this narrowly defined population (Stewart et al., submitted). These latter results are consistent with the literature showing that alcoholics in general are more sensitive to pain stimulation than controls when sober and more sensitive to the pain-reducing effects of alcohol than others (Brown and Cutter 1977). In effect, alcohol seems to normalize a sober overreactivity to pain in this population. Interestingly, these at risk for alcoholism individuals appear to be less aggressive when intoxicated on the Buss-Taylor Task than controls who are not at risk (Pihl et al. 1990).

### **REDUCED CUES TO PUNISHMENT AND FRUSTRATION**

Disinhibitory theories of intoxicated aggression are perhaps the most promi-

ment (Graham 1980; Bushman and Cooper 1990). Often these theories are expressed in informational terms (Hull 1981; Pihl et al. 1981; Steele and Josephs 1988). There is even a commonality, albeit strained, with psychoanalytic theorizing regarding reduction in superego control. Grey (1982, 1987) has written two books that explore what he calls the behavioral inhibition system, which in response to threat halts ongoing activity and initiates sensory motor cognitive activity directed toward analysis and response to specific, threatening cues. This system is presumed to comprise a neurological circuit involving the septum and hippocampus and their interconnections with other limbic and cortical structures. Grey presents an array of experimental literature to demonstrate that anxiolytics, including alcohol, seem to operate differentially on the behavioral inhibition system. While these drugs may actually enhance response to punishment and frustration, they are seen as reducing associated cues related to fear and anxiety. This effect is probably explanatory for alcohol-affected stress-response dampening seen in sons of multigenerational male alcoholics (Pihl et al. 1990) and in the reduction of anxiety following drinking in anxiety-sensitive women (Stewart et al., in press).

One aspect of human socialization is the process of teaching children to regard aspects of their own behavior as a threat to their own well-being and to that of others. Individuals who do not learn this connection threaten the integrity of the social group. Thus, well-socialized indi-

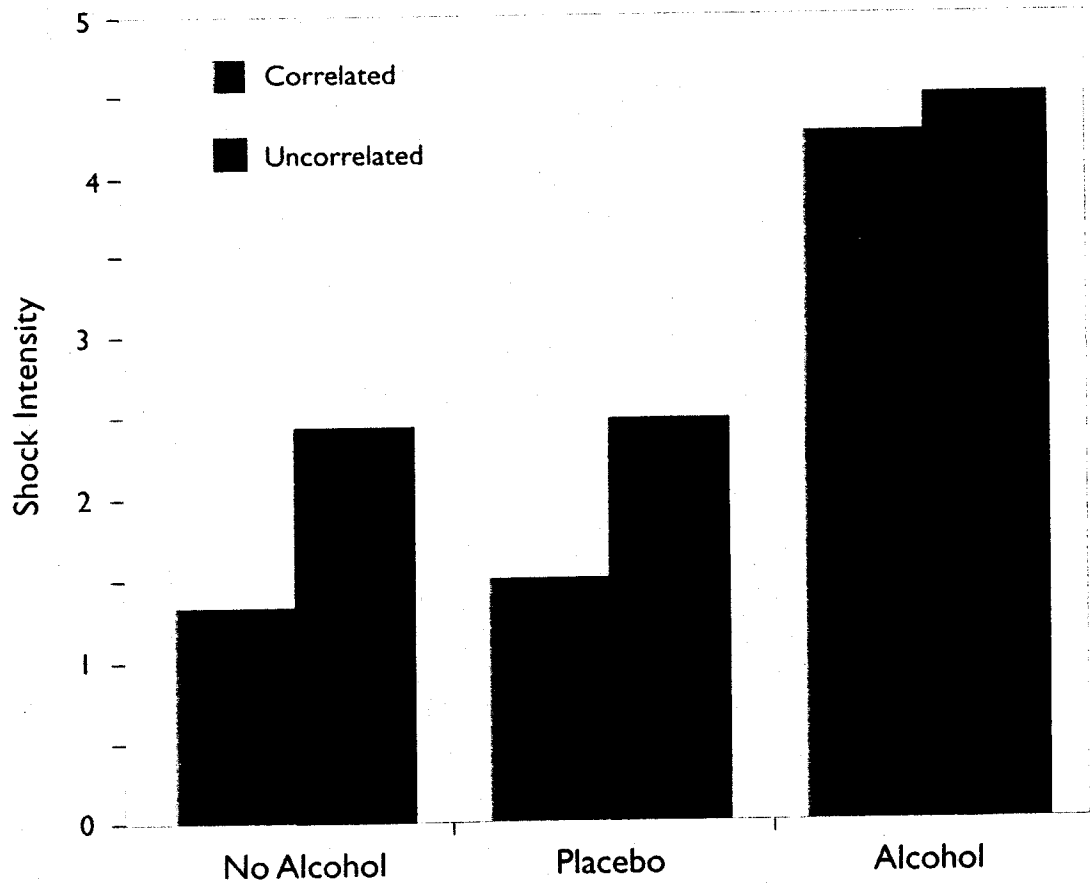
viduals engage in aggressive acts only in extremely limited situations. To engage in aggression outside of this narrow range of justifiable situations in itself should be a cue for punishment and frustration. As alcohol disinhibits all behaviors under the general inhibitory control of fear, of which cues of one's own aggression is primary, intoxicated individuals should participate in dangerous situations where this behavior would normally be inhibited by threat of danger.

Three studies we have completed present some support for this theory. Each of these studies used a modified version of the Buss-Taylor Aggression Task. Unlike the majority of studies using the paradigm in which subjects receive and give electric shocks, the first three studies described below involve subjects receiving aversive tones but delivering shocks. In the first experiment (Zeichner and Pihl 1979), 72 male social drinkers between the ages of 18 and 35 were divided into six groups. There were three drug conditions and two contingency conditions. The drug conditions were alcohol, placebo, and sober; individuals participated in the aggression task after receiving 1.32 mL/kg of 95-percent USP alcohol or placebo or nothing and were randomly assigned to one of two contingency conditions. In the first condition, the intensity of the aversive tones the subject received was correlated with the shocks they delivered; in the second condition, the tones they received were unrelated to their own behavior. Figure 2 illustrates significant differences in the no-alcohol and placebo conditions between individuals who received correlated versus

uncorrelated consequences. This result confirms the well-known conclusion in the aggression literature that the behavior we emit is related to the behavior we receive. Disconfirming this well-known fact, there was no difference in correlated and uncorrelated responding when intoxicated (average blood alcohol level 0.092), with subjects failing to modify their behavior as a result of the consequences. Because it appeared subjects were not processing information relevant to the conse-

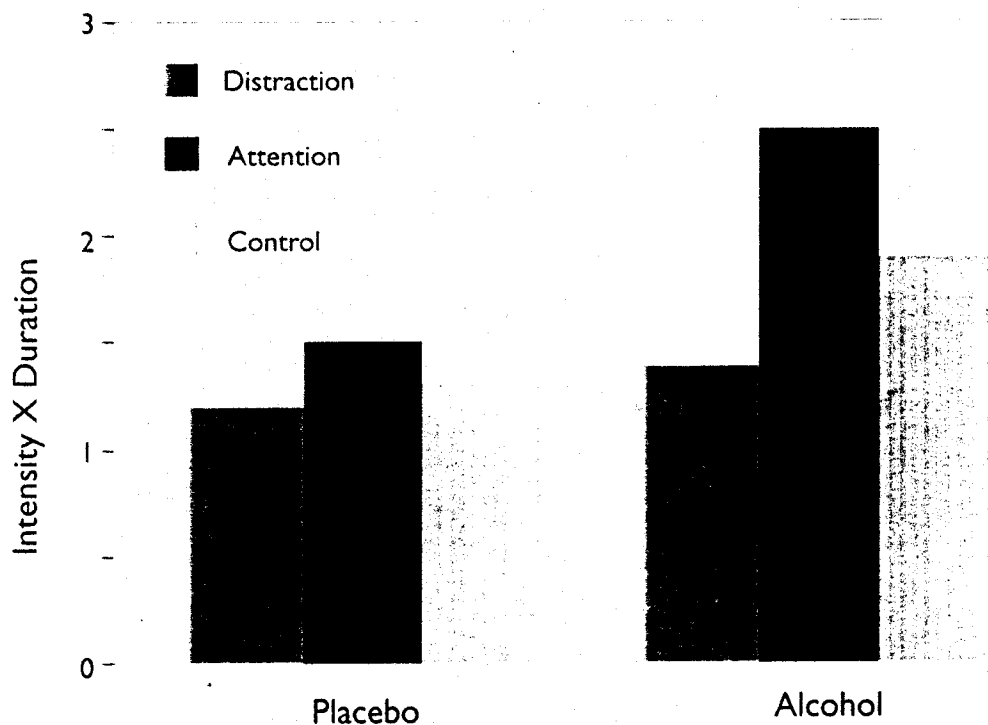
quences of their own behavior, a second study was designed as follows.

This study (Zeichner et al. 1982), the results of which are depicted in figure 3, attempted to require subjects, whether intoxicated or not, to attend to the consequences of their own behavior. In this study there were two drug conditions: subjects received either 1.32 mL/kg of 95-percent USP alcohol or placebo. In the first of the three behavioral conditions, the intention was to have subjects actually pay less



**FIGURE 2**

Shock intensity delivered by males who consumed no alcohol, placebo, or alcohol, when shock reception was correlated or uncorrelated with shock administered.

**FIGURE 3**

Shock intensity x duration delivered by males who consumed a placebo or alcohol, during forced distraction, forced attention, and while attending normally.

attention to the consequences of their behavior by having them complete a mathematical problem concurrent with participation in the competition task. This was labeled the distraction condition. In the second experimental condition, subjects were required to focus specifically on the consequences of their own behavior as well as the behavior of their competitor. They were required to write down the level of the shock that they administered and the level of the tone they received. The third control condition involved the correlated alcohol condition of the previous study. Figure 3 illustrates that distraction resulted in a significant reduction in alcohol-related

aggression, and that individuals forced to attend to the consequences of their own behavior when intoxicated were the most aggressive. From these results one could conclude that awareness of behavior and its consequences may not be as important as presumed. Rather, the affective component of this knowledge appears no longer to be accompanied by fear. The results suggest information is being processed, at least verbally, but no longer inhibits behavior.

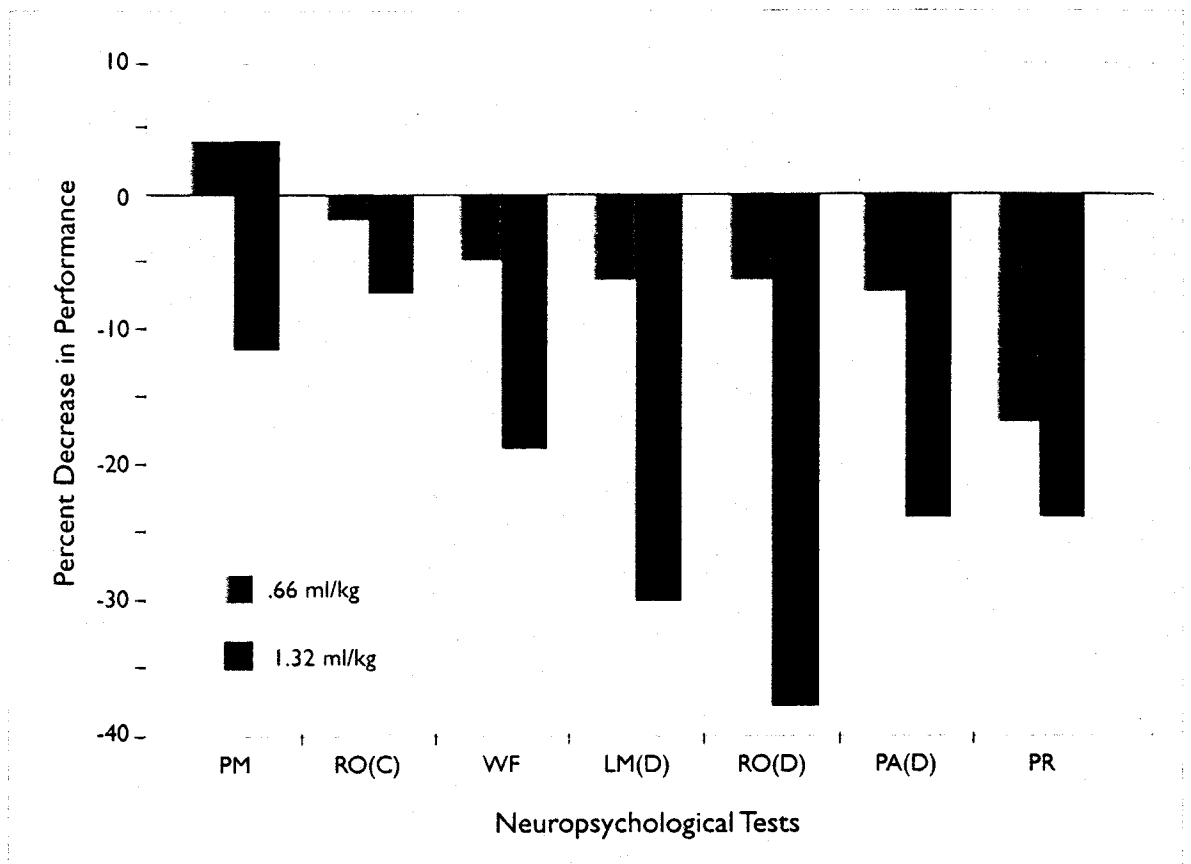
A third study (Zeichner and Pihl 1980) also supports the position that threat is affected by alcohol. This study employed three drug groups: alcohol 1.32 mL/kg, placebo, and control. Subjects

were randomly assigned to one of two intent conditions. In an intent condition called neutral intent, subjects were told that the aversive stimuli they were receiving from their competitor was actually fixed according to a predetermined schedule developed by the experimenter. In the malicious condition subjects were told that the aversive stimuli they were receiving was chosen by their competitor. Actual aversive stimulation was identical in both groups. The results were quite similar to those presented in figure 2 (the contingency study). That is, control or placebo subjects' aggressive behavior was restricted by considerations of intent, with malicious intent evoking significantly more aggression than neutral intent. Again, as with the consequences study, intoxication obviated this relationship. The notion of intent is basic to our conceptualizations of justice; to aggress against another without consideration of intent suggests a breakdown in socially defined norms, which in turn we would argue are threat based. Inhibition of aggression involved in the threat of breaking the social rule now seems to have been eliminated by intoxication.

### **Decreased Frontal Lobe Functioning**

Figure 4 presents the results of a recent study we completed (Peterson et al. 1990) in which a battery of neuropsychological tests was administered to individuals who had been randomly assigned to one of three doses of alcohol (placebo, 0.66 mL/kg and 1.32 mL/kg of 95-percent USP alcohol) within a balanced placebo design. In this procedure half the subjects were

told that they were receiving alcohol while the other half were told they were receiving placebo. Thus, drug and expectancy are putatively crossed. Alcohol expectancy effects were found for only 2 of the 20 tests—digit span and the Young-Pihl Memory Test. Furthermore, under the dosages studied, alcohol seemed to have little effect on intellectual functioning as measured by standard IQ tests. What alcohol did seem to affect was performance on tasks associated with delayed memory and cognitive ability often associated with the functioning of the prefrontal cortex. Significantly and specifically affected, notably by the higher intoxicating dose, were such tasks as assessment, planning and foresight, organization of behavior, abstract conceptualization, memory transfer of information, and tasks involving complex motor behavior. We concluded from these results that alcohol did not seem to affect previously learned knowledge but rather the ability to deal with the threatening or novel. It has been suggested (Luria 1980; Peterson and Pihl 1990) that the prefrontal cortex is critical in the formulation of verbal and motor strategies aimed at dealing specifically with issues of threat or novelty. In a very recent study (Lau et al. 1992), frontal lobe functioning was crossed with alcohol, and provocation and aggressive behavior were assessed. In this study 114 male social drinkers were administered two tests developed at the Montreal Neurological Institute, and putative of frontal lobe functioning, the spatial conditional associative learning task (Petrides 1985), and the self-ordered pointing task



**FIGURE 4**

The percent decrease in performance for subjects given two dosages of alcohol from the norm of sober controls on a battery of neuropsychological tests. PM=Porteous Maze; RO(C)=Ray Osterreith copy; WF=Word Fluency; LM(D)=Logical Memory Delayed; RO(D) = Ray Osterreith delayed; PA(D)=Paired Associates Difficult; PR=Pursuit Rotor.

(Petrides and Milner 1982). Subjects were categorized by their performance on these tests; those in the upper and lower quartiles were selected for participation on the aggression task. Half of these subjects engaged in the task while sober and the other half after consuming 1 mL/kg of 95-percent USP alcohol. The aggression task itself was presented in two phases. For the first 13 trials, subjects received shocks in the lower half of their previously determined shock threshold, and in the second 13 trials, subjects received shocks in the upper half of their threshold. These con-

ditions were viewed respectively as low provocation and high provocation. All shocks were randomly assigned by the computer and were of the same duration. Each subject won and lost exactly half of the trials during both provocation conditions. The results of this study were a significant drug effect, group effect, provocation effect, and provocation by group interaction. Conclusions of seeming importance include that individuals who score low on two putative tests of frontal lobe functioning are more aggressive when sober than those with intact

functioning when provoked. This perhaps suggests a fundamental impairment in integrating inhibitory responses. In addition, the effect of an intoxicating dose of alcohol is to render those individuals who reflect intact functioning when sober as aggressive as their lower functioning comparison group. These results support the notion of an alcohol effect on frontal lobe functioning and a resultant increased likelihood of aggressive responding when sufficient provocation is present.

### SUMMARY

Increased pain sensitivity, reduced response to cues of punishment, and reduced response flexibility are alcohol-related effects that can increase the likelihood of aggressive responding. While individual factors as well as situational factors are crucial considerations in the aggression equation, the effects of alcohol should not be overlooked. For example, a particularly perplexing finding is the high percentage of victims of violence who also have been found to be intoxicated. In a recent review of 26 crime studies from 11 countries, 45 percent of victims were found to have been drinking (Murdoch et al. 1990). The three effects described above are just as applicable in explaining both this fact as well as the fact that 62 percent of the violent offenders were drinking, typically heavily.

Finally, it should be noted that there are clearly other drug effects, some of which are individualized (e.g., pathological intoxication and/or hypoglycemia), which undoubtedly also affect the alcohol/aggression relationship. Nevertheless,

to paraphrase Swift, "drinking is not just a mere pause from thinking but a respite from feeling too."

### ACKNOWLEDGMENTS

The research described herein was supported by the Medical Research Council of Canada, by the Douglas Hospital/McGill University Alcohol Research Group, and by the Brewers Foundation of North America.

### REFERENCES

- Bernstein, S.; Richardson, D.; and Hammock, G. Convergent and discriminant validity of the Taylor and Buss measures of physical aggression. *Aggressive Behav* 13(1):15-24, 1987.
- Brown, R.A., and Cutter, H.S. Alcohol, customary drinking behavior and pain. *J Abnorm Psychol* 86(2):179-188, 1977.
- Bushman, B.J., and Cooper, H.M. Effects of alcohol on human aggression: An integrative research review. *Psychol Bull* 107(3):341-354, 1990.
- Graham, K. Theories of intoxicated aggression. *Can J Behav Sci* 12:141-158, 1980.
- Grey, J.A. *The Neuropsychology of Anxiety: An Enquiry into the Function of the Septal-Hippocampal System*. Oxford: Oxford University Press, 1982.
- Grey, J.A. *The Psychology of Fear and Stress*. Cambridge: Cambridge University Press, 1987.
- Gustafson, R. Threat as a determinant of alcohol-related aggression. *Psychol Rep* 58(2):287-297, 1986.
- Hartman, D.P. Influence of symbolically modelled instrumental aggression and pain cues on aggressive behavior. *J Person Soc Psychol* 11(2):280-288, 1969.
- Hull, J.G. A self-awareness model of the causes and effects of alcohol consumption. *J Abnorm Psychol* 90(6):586-600, 1981.

- Lang, A.R., and Sibrel, P.A. Psychological perspectives on alcohol consumption and interpersonal aggression: The potential role of individual differences in alcohol-related criminal violence. *Criminal Justice Behav* 16(3):299-324, 1989.
- Lau, M.; Pihl, R.O.; and Peterson, J.B. The role of cognitive factors and alcohol in aggression. *Can Psychol* 33(2a):429, 1992.
- Luria, A.R. *Higher Cortical Function in Man*. 2d ed. New York: Basic Books, Inc., 1980.
- Mullin, F., and Luckhardt, A.B. The effect of alcohol on cutaneous tactile and pain sensitivity. *Am J Physiol* 109(1):77-78, 1934.
- Murdoch, D.; Pihl, R.O.; and Ross, D. Alcohol and crimes of violence: Present issues. *Int J Addict* 25(9):1065-1081, 1990.
- Peterson, J.B., and Pihl, R.O. Information processing, neuro-psychological function, and the inherited predisposition to alcoholism. *Neuropsychol Rev* 1(4):343-369, 1990.
- Peterson, J.B.; Rothfleisch, J.; Zelazo, P.D.; and Pihl, R.O. Acute alcohol intoxication and cognitive functioning. *J Stud Alcohol* 51(2):114-122, 1990.
- Petrides, M. Deficits on conditional associative-learning tasks after frontal and temporal-lobe lesions in man. *Neuropsychologia* 23(5):601-614, 1985.
- Petrides, M., and Milner, B. Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia* 20(3):249-262, 1982.
- Pihl, R.O. Alcohol and aggression: A psychological perspective. In: Gottheil, E.; Druley, K.A.; Skodola, T.E.; and Waxman, M.H., eds. *Alcohol, Drug Abuse, and Aggression*. Springfield: C.C. Thomas, 1983. pp. 292-313.
- Pihl, R.O., and Peterson, J.B. Alcohol/drug use and aggressive behavior. In: Hodgins, S., and Shah, S., eds. *Mental Disorder and Crime*. New York: Sage, 1993. pp. 263-283.
- Pihl, R.O.; Peterson, J.B.; and Finn, P.R. Inherited predisposition to alcoholism: Characteristics of sons of male alcoholics. *J Abnorm Psychol* 99(3):291-301, 1990.
- Pihl, R.O., and Ross, D. Research on alcohol-related aggression: A review, and implications for understanding aggression. *Drugs Society* 1(4):105-126, 1987.
- Pihl, R.O.; Sita, A.; and Gagnier, J. Alcohol and aggression: A comparison of two groups of men with different patterns of sensitivity to alcohol (abstract). *Can Psychol* 31(2a):380, 1990.
- Pihl, R.O.; Zeichner, A.; Niaura, R.; Nagy, K.; and Zacchia, C. Attribution and alcohol-mediated aggression. *J Abnorm Psychol* 90(5):468-475, 1981.
- Shembert, K.M.; Leventhal, D.B.; and Allman, L. Aggression machine performance and rated aggression. *J Exp Res Person* 3(1):117-119, 1968.
- Steele, C.M., and Josephs, R.A. Drinking your troubles away. II: An attention-allocation model of alcohol's effect on psychological stress. *J Abnorm Psychol* 97(2):196-205, 1988.
- Stewart, S.; Achille, M.; Dubois-Nguyen, I.; and Pihl, R.O. The effects of alcohol administration on attention to threat in anxiety sensitive women. Submitted for publication.
- Stewart, S.H.; Finn, P.; and Pihl, R.O. A dose response study of the effects of alcohol on the perceptions of pain and discomfort due to electric shock in men at high risk for alcoholism. Submitted for publication.
- Taylor, S.P. Alcohol and human physical aggression. In: Gottheil, E.; Druley, K.A.; Skoloda, T.E.; and Waxman, H.M., eds. *Alcohol, Drug Abuse and Aggression*. Springfield: CC Thomas, 1983. pp. 280-291.
- Williams, J.F.; Meyerson, L.J.; Eron, L.D.; and Semler, I.J. Aggression and aggressive responses elicited in an experimental situation. *Child Dev* 38(1):181-190, 1967.

## Mechanisms of Drug Effect

Wolff, H.G.; Hardy, J.D.; and Goodell, H. Studies on pain: Measurement of the effect of ethyl alcohol on the pain threshold and on the "alarm reaction." *J Pharmacol* 75(1):38-49, 1942.

Zeichner, A., and Pihl, R.O. Effects of alcohol and behavior contingencies on human aggression. *J Abnorm Psychol* 88(2):153-160, 1979.

Zeichner, A., and Pihl, R.O. The effects of alcohol and instigator intent on human aggression. *J Stud Alcohol* 41(3):265-276, 1980.

Zeichner, A.; Pihl, R.O.; Niaura, R.; and Zacchia, C. Attentional factors in alcohol-mediated aggression. *J Stud Alcohol* 43(7):714-724, 1982.