

## Provocation, Acute Alcohol Intoxication, Cognitive Performance, and Aggression

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This study investigated the relationships between provocation, acute alcohol intoxication, impaired frontal-lobe function, and aggressive behavior. The authors ranked 114 men according to their performance on two neuropsychological tests associated with frontal-lobe function. Forty-eight men (24 with scores in the upper and 24 with scores in the lower performance quartiles) participated in the full study. Half completed an aggression task while intoxicated, the remainder while sober. Aggression was defined as shock intensity delivered to a sham opponent. Shock intensity significantly increased as a main effect of provocation, alcohol intoxication, and lower cognitive performance. Furthermore, provocation interacted significantly with test performance such that individuals in the lower cognitive performance quartile responded to increased provocation with heightened aggression.

Violence presents a serious and growing challenge to North American society. More than a million and a half individuals are victims of violent crime each year (U.S. Bureau of the Census, 1991); 20,000 are murdered (Prothrow-Stith, 1990). Aggression can be defined as any behavior directed toward the goal of harming another living being who is motivated to avoid such treatment (Baron & Richardson, 1994). Various antecedents to aggressive behavior have been implicated, including social, situational, and individual determinants. A major social determinant of aggression is provocation, in the form of verbal insult, physical attack, or other noxious stimuli (Hammock & Richardson, 1992).

One of the most important situational determinants of aggression is acute alcohol intoxication, which is associated with more than half of all homicides, assaults, rapes, and cases of family violence (Murdoch, Pihl, & Ross, 1990). Bushman and Cooper's (1990) meta-analytic review of over 30 controlled laboratory studies concluded that alcohol-intoxicated individuals are significantly more verbally and physically aggressive. However, every one who drinks does not become aggressive, and the study of characteristics of predispositions to aggressive responses becomes particularly relevant.

Many individual determinants including personality traits, attitudes, genetic factors, and sex differences have been implicated in aggressive behavior (Baron & Richardson, 1994; Wood, Wong, & Chachere, 1991). The occurrence of a violent act in

response to external factors is thought to require the interaction of various cognitive processes, including planning of behaviors and consciousness of the act and its consequences (Tancredi & Volkow, 1988). As the frontal cortices are thought to be involved in the highest level of goal-directed activity, including the organization and planning of behavior (Damasio, 1979; Luria, 1980), the present study focused on individual differences in performance on neuropsychological tests of frontal-lobe function.

Some neuropsychological evidence associates frontal-lobe deficits with poorer regulation of human social behavior. Individuals with frontal-lobe damage often exhibit a "disinhibition syndrome" characterized generally by impulsivity and socially inappropriate behavior (Hecaen & Albert, 1978; Miller, 1987). If damage occurs early enough in life, it can result in the development of pervasive abnormalities of affective and social behavior (Eslinger & Damasio, 1986) and the inability to accommodate social impulses into the total personality structure (Ackerly & Benton, 1948).

Furthermore, some research links various forms of antisocial behavior with poor performance on putative frontal-lobe function measures (Buikhuisen, 1987; Lueger & Gill, 1990; Moffitt, 1990). However, one review concluded that despite the evidence supporting a specific relationship between violent criminal behavior and frontal-lobe dysfunction, alternative explanations could not be ruled out (Kandel & Freed, 1989).

A theoretical explanation for how impaired frontal-lobe function might lead to impaired regulation of social behavior is that there is a disturbance of the synthesis of external and internal cues underlying the regulation of complex behavior (Luria, 1980). As a consequence, behavior is governed more by impulse, current focus of attention, or salient stimulus cues than by rules or plans (Luria, 1980). Although this condition would not necessarily lead to increased aggression under normal circumstances, it might in situations where the salient cue is provocation and where peripheral or less contingent cues that inhibit aggression, such as fear of violence-related consequences, are

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lessened. Thus, an individual with impaired frontal-lobe function would be expected to demonstrate increased aggression under provocative conditions. Furthermore, as the behavior of this individual is governed more by the stimulus cue, level of frontal-lobe function would interact with provocation.

The interaction between frontal-lobe function and alcohol intoxication may be important for the following reason. Alcohol interferes with selected aspects of higher order cognition dependant on the intact structure of the prefrontal cortex (Peterson, Rothfleisch, Zelazo, & Pihl, 1990). Furthermore, acute alcohol intoxication interferes with the ability to integrate previously acquired knowledge in the formulation of behavioral strategies in a provocative situation (Lau & Pihl, in press), an ability thought to be influenced in part by the frontal cortex (Pihl, Peterson, & Lau, 1993). As alcohol interferes with cognitive abilities associated with the frontal lobes and impaired frontal-lobe function is thought to lead to increased aggression in provocative situations, individuals with reduced frontal function would be more vulnerable to the aggression-increasing effects of alcohol. This potential interaction may be important in understanding individual differences in alcohol-related aggression.

We designed the present study to investigate the main effects and interactions between provocation, acute alcohol intoxication, cognitive abilities associated with frontal lobe function, and aggression. We hypothesized that (a) all individuals would be more aggressive as provocation increased, (b) intoxicated individuals would be more aggressive than sober individuals, (c) individuals with lower cognitive performance would be more aggressive than those with higher cognitive performance, (d) provocation would interact with cognitive ability such that individuals with lower cognitive performance would respond to increased provocation with greater increases in aggression, and (e) intoxicated condition and cognitive ability would interact, such that alcohol would increase aggression more for lower cognitive performers.

## Method

### Participants

One hundred and fourteen volunteer nonalcoholic male social drinkers (a score of less than 5 on a short form of the Michigan Alcohol Screening Test; Pokorny, Miller, & Kaplan, 1972), aged 18–40, in good physical and mental health, were recruited through newspaper advertisements. Those receiving medical treatment that contraindicated alcohol consumption, who had sustained a serious injury to the head, or were familiar with psychological experimentation were excluded from participation. Women were excluded due to gender differences in physical aggression (Eagly & Steffen, 1986).

The remaining volunteers were assigned to one of two groups on the basis of their performance on two neuropsychological tests: total number of trials and incorrect guesses on the spatial conditional associative-learning task (Petrides, 1985) and the total number of errors on the self-ordered pointing task (Petrides & Milner, 1982). *Z*-score transformations were calculated for these three scores, which were added to determine a cumulative *z* score for each person. Participants whose cumulative *z* scores fell in the upper and lower performance quartiles (UQ and LQ, respectively) of the distribution were selected to complete the entire protocol.

### Apparatus

Aggression was elicited and assessed with the Taylor (1967) competitive reaction-time task. In this study, the task board consisted of eight buttons, numbered consecutively from one to eight. Red lights situated above each button indicated the shock level chosen by the opponent when lit. An Apple II computer was used to run the aggression task and to record data. Shocks were administered through the Mark I Behaviour Modifier (Farrall Instruments), connected to an electrode attached to the inner forearm, below the elbow of the nondominant hand. Each person monitored administrations of shocks to his fictitious opponent by viewing a direct current ammeter provided for that purpose. A pre-recorded videotape of the opponent receiving instructions regarding performance of the aggression task was played to each man on a Sony television connected to a Quasar VCR. This tape served to reinforce his belief in the existence of the opponent. The men's blood alcohol level (BAL) was determined using an Alco-sensor III (Thomas Ltd.). The men were also asked to rate themselves on a 7-point Likert-type "how drunk" scale. A score of 1 represented a rating of sobriety; 7 represented a rating of the most intoxicated the man had ever been.

The spatial conditional associative-learning task and self-ordered pointing task were used to separate participants into the two groups. In the former, each one of six randomly placed lamps was paired with one of six white cards. None of the men were informed of the pairings. The lamps were randomly lit, one at a time whereupon each man was to touch the cards one at a time until he touched the one that was paired with the lamp. Each person's task was to learn these associations so that when a given light was presented, the correct card would be chosen. Individuals with unilateral frontal-lobe damage have been shown to perform poorly on this task; the impairment on this task appears to be due to difficulties in learning to choose from a set the appropriate response to a given stimulus (Petrides, 1985).<sup>1</sup>

In the self-ordered pointing task, participants were presented 12 representational drawings of familiar objects arranged in a 3 × 4 matrix on each of 12 pages. The same designs appeared on each page; however, the positions of the drawings were different and randomly determined for each page. Participants were told to point to a different design on each page without choosing any given design more than once. This test theoretically measures organizational ability and sequencing of responses rather than the reproduction of sequences preorganized by the experimenter. Individuals with unilateral frontal-lobe damage are significantly impaired on this task; the deficits can be attributed either to poor monitoring of responses or poor organizational strategies, or both (Petrides & Milner, 1982).<sup>2</sup>

The Information, Block Design, and Vocabulary subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) were administered to provide estimates of full-scale IQ (Brooker & Cyr, 1986) and overall cognitive ability.

### Procedure

Respondents to newspaper advertisements, who met inclusion criteria, were asked not to consume drugs or alcohol for at least 24 hr prior to testing. All participants signed an informed consent form and provided demographic data including age, years of education, subjective

<sup>1</sup> Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic area 8 of the dorsolateral frontal cortex (Petrides, Alivisatos, Evans, & Meyer, 1993).

<sup>2</sup> Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic areas 46 and 9 of the mid-dorsolateral frontal cortex (Petrides et al., 1993).

report of alcoholic beverages consumed per week, and salary code. The latter was defined as annual personal income where each point represented an increment of \$5,000. The participants then completed the battery of neuropsychological and intelligence tests.

Men in the UQ or LQ competed in the Taylor aggression task, usually within 7 days of the first testing session. Half of the men in each quartile were randomly assigned to the alcohol condition, the remainder to the sober condition. In the alcohol condition, the men were administered 1 milliliter per kg of body weight of 95% alcohol USP units in three drinks of a 1:7 alcohol:orange juice solution. In the sober condition, the men were administered three drinks of juice of equivalent volume. In each condition, participants were told explicitly what they were drinking.

Drinks were consumed over a 20-min period. A 20-min waiting period followed to allow the men in the alcohol condition time to reach near-peak BALs. BALs were then taken and each person rated himself on the "how drunk" Likert scale.

Each man's pain threshold for electric shock was determined by delivering a series of shocks from 0–255 units (0–5.61 milliamperes [mA], which increased stepwise by 5 units (5 units = 0.11 mA) at a constant rate. Each man was to press a button in response to any shock he regarded as painful (a) to stop the administration of the shock and (b) to reduce the level of the next shock by one step. Therefore, the next shock was one step lower than the shock that induced pressing the button. Pressing the button on 3 consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the man's pain threshold.

The aggression task was then introduced as a competitive reaction-time task. Each man was instructed to select a shock level that he would deliver to his opponent after winning a reaction-time trial. Following the reaction-time task, the person would be informed of the opponent's shock choice. The one who lost would receive that shock. Shock levels 1–8 increased linearly from 15% to 100% of the person's given pain threshold. If he won, he administered the previously chosen shock to his opponent. The experimenter then left briefly, telling each man that he was about to verify the readiness of the opponent. On his return, the experimenter stated that instructions were about to be delivered to the opponent, and that this delivery could be viewed on the television monitor. In fact, what was actually presented was a prerecorded videotape of a fictitious opponent. Three practice trials were then conducted.

The task itself consisted of 26 consecutive trials including a block of 12 trials followed by a transition trial, a second block of trials, and a final trial. The opponent's shock choices ranged from 1–4 in the first block and from 5–8 in the second block of trials. The order of wins and losses as well as the opponent's shock choices were randomly assigned by the computer. The opponent's shocks were all of the same duration. All participants received three shocks at each level alternately winning one trial and losing two trials versus winning two trials and losing one trial. All participants lost the transition trial and won the final trial. In both cases the opponent's shock choice was a 5.

Following the aggression task, the men completed a short questionnaire to verify the success of the deception. They rated their own and their opponent's performance on the aggression task and described how effective they thought the task was at measuring their reaction time. All the men were debriefed and the necessity for deception was fully explained. No one was adversely affected by the deception, according to self-report. The experimenter rated each man's deception on a Likert-type scale from 1–6. A score of 1 represented a rating of "not deceived"; 6 represented a rating of "totally deceived." Intoxicated participants were retained in the laboratory until their BAL dropped below one third of 1%. Each person was paid \$5 an hour to compensate for lost time.

The objective measures of aggression were the intensity of shock each man selected for each provocation level (1–8) for trials following a loss (receipt of a shock) and a win (receipt of information regarding the opponent's choice of shock level). The first measure reflects an individ-

ual's response to physical provocation, whereas the second measure reflects an individual's response only to the opponent's intentions. The first shock choice was made before the first reaction-time test but after three practice trials with the opponent, and so it was not included in the analysis.

## Results

### Participant Measures

The two tests were completed by 114 men. Fifty-six men with LQ and UQ cumulative test  $z$  scores for the entire distribution were selected for further participation in the study; 48 of them (24 UQ and 24 LQ) completed the entire protocol. (Two men from the LQ were not deceived and were excluded from the analysis, and 6 others—2 from the LQ and 4 from the UQ—did not return to complete the testing.) Mean demographics by quartile were as follows: for age, LQ  $M = 23.6$ ,  $SD = 5.3$ , UQ  $M = 24.4$ ,  $SD = 5.1$ ; for years of education, LQ  $M = 12.7$ ,  $SD = 2.2$ , UQ  $M = 14.9$ ,  $SD = 1.8$ ; for salary code, LQ  $M = 1.9$ ,  $SD = 1.2$ , UQ  $M = 2.4$ ,  $SD = 1.4$ ; for beverages per week, LQ  $M = 6.6$ ,  $SD = 7.3$ , UQ  $M = 7.8$ ,  $SD = 7.1$ ; and for IQ, LQ  $M = 97$ ,  $SD = 7$ , UQ  $M = 111$ ,  $SD = 11$ . Separate two-tailed  $t$  tests revealed that quartile groups differed in mean years of education and IQ,  $t(45) = 3.85$ ,  $p < .0001$ , and  $t(46) = 5.24$ ,  $p < .0001$ , respectively.

### Neuropsychological Test Measures

Separate two-tailed  $t$  tests revealed significant differences ( $ps < .0001$ ) between each quartile's mean scores for the conditioned associative-learning task trials (CAT: LQ  $M = 175$ ,  $SD = 12$ ; UQ  $M = 51$ ,  $SD = 17$ ;  $t[46] = -29.1$ ) and total number of conditioned associative-learning task errors (CAE: LQ  $M = 159$ ,  $SD = 54$ ; UQ  $M = 20$ ,  $SD = 11$ ;  $t[46] = -12.2$ ), and for self-ordered pointing errors (SOPE: LQ  $M = 5.1$ ,  $SD = 2.4$ ; UQ  $M = 1.5$ ,  $SD = 1$ ;  $t[46] = -6.8$ ). Correlations between CAT and CAE on the conditional-associative learning task and SOPE on the self-ordered pointing task were as follows: CAT and CAE,  $r = .83$ ,  $p < .0001$ ; CAT and SOPE,  $r = .45$ ,  $p < .0001$ ; CAE and SOPE,  $r = .39$ ,  $p < .0001$ ; coefficient  $\alpha = .69$ .

### Alcohol Measures

Participants were tested on the aggression paradigm sober (BAL  $M = 0\%$ ,  $SD = 0.00$ ) or intoxicated (BAL  $M = < \text{one tenth of } 1\%$ ,  $SD = 0.02$ ; "how drunk" scale  $M = 3.8$ ,  $SD = 1.1$ ). Separate two-tailed  $t$  tests revealed that the mean BAL for the intoxicated subjects of the LQ ( $M = \text{one tenth of } 1\%$ ,  $SD = 0.01$ ) and UQ ( $M = \text{less than one tenth of } 1\%$ ,  $SD = 0.03$ ) and mean "how drunk" ratings of the LQ ( $M = 3.8$ ,  $SD = 1.4$ ) and UQ ( $M = 3.8$ ,  $SD = 0.8$ ) did not differ significantly.

### Deception Measure

Typically, the men did not question the existence of the opponent when completing the short questionnaire to verify the success of the deception. All but two were classified as being deceived. A 2 (quartile)  $\times$  2 (drug) analysis of variance (ANOVA) conducted on the 6-point deception scale revealed that the

mean deception rating of the LQ ( $M = 4.1$ ,  $SD = 1.3$ ) and UQ ( $M = 4.3$ ,  $SD = 1.0$ ) did not differ significantly, nor were there any significant interactions.

### Pain Threshold Measures

A 2 (quartile)  $\times$  2 (drug) ANOVA conducted on pain threshold revealed a significant main effect of quartile,  $F(1, 44) = 9.00$ ,  $p < .01$ . Individuals in the UQ had lower pain thresholds for electric shock than those in the LQ,  $M = 99$ ,  $SD = 77$ , and  $M = 171$ ,  $SD = 88$ , respectively.

### Shock Intensity Measures

A 2 (quartile)  $\times$  2 (drug)  $\times$  2 (response condition: loss-win)  $\times$  8 (provocation) mixed design ANOVA, with response condition and provocation as repeated measures, conducted on shock intensity was done to compare the men's responses following wins and losses. This analysis revealed a significant main effect of response condition,  $F(1, 44) = 26.52$ ,  $p < .0001$ , with participants choosing higher mean shock intensities following a loss and receipt of a shock ( $M = 4.1$ ) than after winning a trial and administering a shock ( $M = 3.5$ ). In addition, there was a significant interaction between response condition and provocation,  $F(7, 308) = 3.27$ ,  $p < .01$ . As a result, the data for the two response conditions were analyzed separately.

A 2 (quartile)  $\times$  2 (drug)  $\times$  8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a loss, using Huyn-Feldt conservative degrees of freedom, revealed significant main effects for quartile ( $M_s = 3.5$  and  $4.7$  for UQ and LQ, respectively;  $F[1, 44] = 7.29$ ,  $p < .01$ ); drug ( $M_s = 3.7$  and  $4.5$  for UQ and LQ, respectively;  $F[1, 44] = 4.04$ ,  $p < .05$ ); and provocation ( $F[7, 308] = 21.20$ ,  $p < .0001$ ). The effect for provocation was decomposed into a linear and pooled-nonlinear trend. The results indicated a significant linear trend,  $F(1, 308) = 136$ ,  $p < .0001$ , but no significant nonlinear trend,  $F(6, 308) = 2.07$ .

Furthermore, there was a significant interaction between provocation and quartile,  $F(7, 308) = 2.67$ ,  $p < .05$ . The interaction between quartile and the linear trend for provocation was significant,  $F(1, 308) = 11.6$ ,  $p < .001$ , indicating that the slopes of the two quartiles differed. Thus, individuals of the LQ showed a greater increase in mean shock intensity as provocation increased (Figure 1).<sup>3</sup> Analysis of the pooled nonlinear portion of the interaction did not reveal any additional differential pattern of responding,  $F(6, 308) = 1.25$ .

A 2 (quartile)  $\times$  2 (drug)  $\times$  8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a win, revealed a significant main effect for provocation,  $F(7, 308) = 4.73$ ,  $p < .0001$ . The effect for provocation was decomposed into a linear and pooled-nonlinear trend. The results showed a significant linear trend,  $F(1, 308) = 30.8$ ,  $p < .0001$ , but no significant nonlinear trend,  $F(6, 308) = 0.40$ . The main effects for drug ( $M_s = 3.2$  and  $4.0$  for UQ and LQ, respectively),  $F(1, 44) = 3.29$ ,  $p = .0764$ , and quartile ( $M_s = 3.1$  and  $4.0$  for UQ and LQ respectively),  $F(1, 44) = 4.03$ ,  $p = .051$ , were marginally significant.

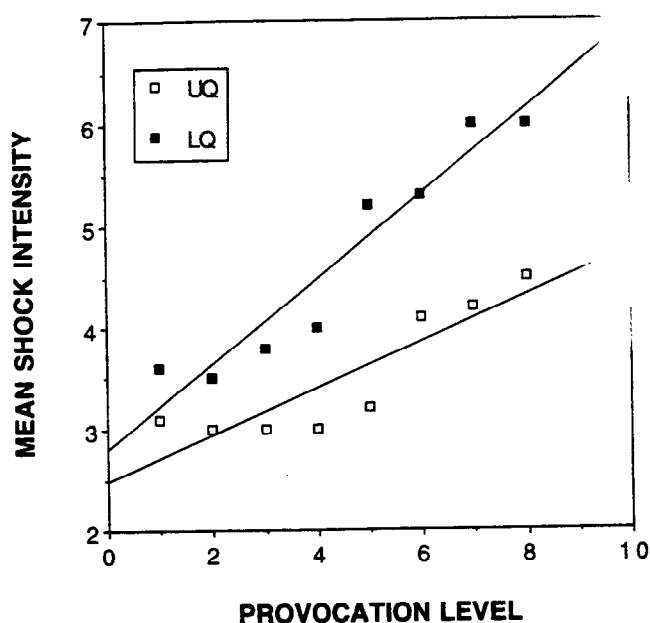


Figure 1. Mean shock intensity chosen after a loss, by provocation level, for cognitive performances in the upper and lower quartile (UQ and LQ, respectively) groups. See text for details.

### Discussion

The results of this study support the previously reviewed work that has shown that provocation heightens aggression during a competitive task and that alcohol intoxication increases aggression. In addition, it is significant that the results also show that individuals grouped according to performance on two neuropsychological tests of cognitive abilities associated with frontal-lobe function differ in degree of aggressive response. Specifically, individuals in the lower performance quartile become even more aggressive when provoked.

The notion that reduced frontal-lobe function may partially disinhibit aggressive behavior is one explanation for these results. If decreased frontal-lobe function impairs an individual's ability to use socially relevant information (Dimond, 1980),

<sup>3</sup> Separate correlations of IQ, years of education, and pain threshold on individual mean shock intensity revealed that IQ was significantly correlated with mean shock intensity ( $r = -.32$ ,  $p < .05$ ). However, neither years of education nor pain threshold were significantly correlated with shock intensity. In addition, IQ was not significantly correlated with scores on the two neuropsychological tests. To control for the possible confounding effect of IQ, this variable was included as a covariate in the above ANOVA. However, there was a significant interaction between quartile and IQ,  $F(1, 40) = 6.18$ ,  $p < .05$ , which violates the assumption of homogeneity of slopes on which the analysis of covariance rests. Therefore, IQ was included as a continuous independent variable in a fully saturated model as a main effect and in interaction with the categorical independent variables. These analyses revealed that the main effect of quartile was unaffected,  $F(1, 40) = 5.32$ ,  $p < .05$ . Furthermore, the main effect of IQ was not significant. Comparable analyses conducted on years of education and pain threshold did not substantially affect the results.

specifically through deficits in the internalization of inhibitory influences, these individuals should respond more aggressively when presented with provocation or punishment. This idea is consistent with observations that monkeys with frontal cortical ablations are more labile in social interactions and substantially more aggressive (Dimond, 1980). Furthermore, reduced performance on tasks reflecting abilities associated with frontal-lobe functioning has been shown to predict fighting in young boys (Seguin, Harden, Pihl, & Tremblay, 1993).

Contrary to expectations, there was no significant interaction between quartile and alcohol intoxication. The lack of an interaction may be due to a ceiling effect on shock intensity choice for LQ individuals. However, the mean shock intensity of approximately 5 chosen by both LQ groups is well below the maximum potential intensity of 8. Alternatively, the absence of an interaction may have been due to low statistical power.

The possibility that the results of this study were due to group differences in IQ is unlikely. Controlling for IQ in the analysis did not substantially affect the results. Thus, group differences in neuropsychological test scores predicted aggression beyond any prediction provided by IQ. Furthermore, IQ and neuropsychological test scores were not significantly correlated. A more likely explanation is that some IQ deficit is to be expected with a lifelong frontal deficit. Although the two neuropsychological tests used in this study are not thought to be particularly sensitive to variations in IQ (Petrides, 1985; Petrides & Milner, 1982), these tests were validated on individuals who had sustained frontal-lobe damage later in life, when IQ is not necessarily affected by such insult (Black, 1976).

It might be argued that this study measured degree of aggression in conditions where only an aggressive response was permitted. However, the presence of the lowest level shock button was clearly explained, and its use was not discouraged. In addition, there is research using paradigms that include a nonaggressive response option where alcohol has been shown to specifically affect aggressive responding (Cherek, Steinberg, & Manno, 1985).

In conclusion, the present study demonstrates that determinants such as provocation, drug effects, and the preexistent cognitive abilities of an individual increase the likelihood of aggression. More importantly, preexisting cognitive abilities and provocation have been shown to interact to predict aggressive behavior.

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