

Alcohol-Related Aggression during the College Years: Theories, Risk Factors and Policy Implications*

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ABSTRACT. *Objective:* The purpose of this article is to present an overview of the research literature on alcohol-related aggression with a focus on college students. *Method:* Data from both survey studies and experimental laboratory investigations conducted on college students are reviewed. Various methodological approaches to studying the alcohol-aggression relation, and their associated limitations, are then presented and discussed. *Results:* The literature indicates that alcohol consumption facilitates aggressive behavior and increases the risk of being the victim of a violent act, particularly in heavy drinkers. Results from these studies are then placed into a context by reviewing 12 influential theories of aggression and alcohol-related aggression. On the basis of these

theories and empirical data, a preliminary risk profile is presented to help identify which factors are likely to be important in predicting who will and who will not become aggressive after drinking alcohol. *Conclusions:* Although much research is still needed to elucidate the intricate causes of alcohol-related aggression, current prevention efforts might focus on modifying key risk factors such as poor cognitive functioning and inaccurate expectations about the effects of alcohol. Other prevention efforts directed specifically at college students might focus on helping them to identify risky situations that might facilitate the expression of intoxicated aggression. (*J. Stud. Alcohol*, Supplement No. 14: 129-139, 2002)

ENTRANCE INTO college marks a time of significant change in the life of a young adult. For many students, college is a place where drinking alcohol either begins or increases in frequency. Subsequent to this rise in drinking, a substantial number of college students develop alcohol-related problems. Fortunately, for the majority of students, this rise in “college drinking” diminishes after they graduate, at which time they go on to live more productive lives (Chen and Kandel, 1995; Gotham et al., 1997). However, what is problematic for these individuals are the consequences of their drinking during the college years.

Very little research has been conducted to characterize the prevalence and patterns of alcohol-related aggression in college students. Although not specifically intended to target college students, a large study on adolescent development found that of 391 young adults between 18 and 22 years of age (mostly college students), 30% of males and 25% of females reported having engaged in a fight while intoxicated (H. White, personal communication, 1999). In another large study that sampled college students from 140 U.S. colleges in 1993 and then resampled students from 130 of these schools in 1997, it was found that 19-24% of students reported being intoxicated while exhibiting verbal aggression, 9-10% reported being intoxicated while engaging in property damage and 4-6% reported being intoxi-

cated when apprehended by police (Wechsler et al., 1998). However, these numbers were found to be substantially higher in heavier drinkers (Wechsler et al., 1994, 1998). Furthermore, another report on this sample indicated that a large proportion of college students reported being victimized by intoxicated individuals. Specifically, 12% reported being pushed, hit or assaulted; 20% reported being the recipients of unwanted sexual advances; and 22% reported being involved in verbally aggressive interactions (Wechsler et al., 1995). Again, these percentages were found to be significantly higher in heavy drinkers. Interestingly, this suggests that one is more likely to be victimized by an intoxicated assailant if one is a heavy drinker. Finally, when considering schools with high levels of student drinking, 61% of college administrators indicated that physical assaults were a moderate or major problem at their school, 53% indicated a problem with damage to campus property and 86% indicated a problem with sexual assault (Wechsler et al., 1995).

Methodological Approaches and Limitations

The studies described above indicate that alcohol-related aggression is a serious problem both on and off our college campuses. However, methodological issues surrounding the correlational nature of these findings preclude the formulation of statements suggesting a causal relationship between alcohol use and aggressive behavior. At best, what can be determined from these investigations is the percentage of individuals apprehended for, or reporting, an alcohol-related

*Preparation of this article was supported, in part, by National Institute on Alcohol Abuse and Alcoholism grant ROI-AA-11691 and the University of Kentucky Research Challenge Trust Fund.

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incident. Another difficulty is one of base rates. That is, the above results do not indicate whether alcohol is associated with aggression at a higher rate than would be expected by chance alone. Furthermore, there is the problem of directionality. That is, does alcohol cause aggression or do aggressive individuals tend to drink more? Other limitations of some of the above studies include insufficient information regarding whether alcohol was present at the time of the transgression and a reliance on self-report methods that are troubled by response biases, problems in recollection and problems with making inferences about another person's state of intoxication. Despite these limitations, these studies portray a compelling relation between alcohol and aggression, particularly on college campuses. Nevertheless, it is clear that more survey studies utilizing innovative methodologies are needed to characterize the alcohol-aggression relation in college students. The literature pertaining to sexual assault among college students serves as a good example of worthy approaches for documenting the alcohol-aggression link with survey data (Abbey, this supplement).

In addition to these studies, a large number of controlled experimental laboratory investigations have also demonstrated a strong relation between alcohol use and aggressive behavior. Experimental studies are advantageous over correlational studies in that their design structure allows for the formulation of causal inferences. Furthermore, for the purposes of this article, another important reason for paying attention to the results of experimental studies is that although many of these studies utilize samples of convenience, these samples tend to consist mainly of college students.

Most experimental studies that have investigated the alcohol-aggression relation in college students have measured aggression using the Taylor Aggression Paradigm (TAP; Taylor, 1967). Using the TAP, subjects compete against a fictitious opponent under the guise of a reaction time (RT) task. Prior to each RT trial, subjects select 1 of 10 shock intensities that they wish to administer to their opponent. An RT trial then follows. In the event that the subject wins the trial, his or her opponent ostensibly receives the selected shock. In the event that the subject loses the trial, the subject receives a shock ostensibly from his or her opponent. To manipulate the level of provocation, subjects receive both high and low intensity shocks. In actuality, no opponent exists. The TAP operationalizes aggressive behavior as the average shock intensity selected over trials. In some modifications of the TAP, shock duration is also used as a dependent variable. The TAP is a useful tool for assessing the effects of alcohol on aggressive behavior. This is accomplished by comparing shock intensity and duration selections between sober and intoxicated persons.

The TAP has been criticized for having a number of limitations, including adhering to demand characteristics, measuring constructs other than aggression and not incor-

porating a nonaggressive response option (Tedeschi and Quigley, 1996). However, a number of theorists have responded to these and other concerns (Anderson and Bushman, 1997; Berkowitz and Donnerstein, 1982; Giancola and Chermack, 1998). Furthermore, they have shown that the construct validity of the TAP is well established, in part, by studies demonstrating its convergent and discriminant validity. Convergent validity has been shown through positive associations between shock selections and self-report measures of physical assault, behavioral hostility and outwardly directed anger (Giancola and Zeichner, 1995a; Hammock and Richardson, 1992). Discriminant validity has been demonstrated through the lack of relations between shock selections and measures of guilt, suspicion, resentment, inwardly directed anger (Giancola and Zeichner, 1995c), helping and competition (Bernstein et al., 1987). Additional data supporting the construct validity of the TAP come from studies showing that adolescents with high teacher ratings of aggression are more aggressive on a modified version of the TAP than adolescents with low ratings (Shemberg et al., 1968; Williams et al., 1967). Violent offenders also respond more aggressively on the TAP compared with non-violent offenders (Hartmann, 1969; Wolfe and Baron, 1971). Although the criticisms marshaled against the TAP reveal some of its limitations, available data indicate that the TAP is a good index of aggression. Because most tools that assess complex social constructs such as aggression are less than perfect, it is recommended that a better manner in which to measure these variables is to employ multiple and diverse metrics.

In most investigations that have used the TAP to study alcohol-related aggression, subjects have typically been assigned to one of three beverage groups: alcohol, no alcohol or placebo. Placebo groups are used to control for the possibility that aggression is the result of the mere belief that one has consumed alcohol. Although some studies have shown full (Lang et al., 1975) or partial (Pihl et al., 1981) placebo effects on aggression, the majority of investigations have found that the mere belief that alcohol has been consumed does not significantly influence aggressive behavior in college students (e.g., Chermack and Taylor, 1995; Giancola and Zeichner, 1995b; Zeichner and Pihl, 1979, 1980). Moreover, three large meta-analytic reviews concur that believing that alcohol has been consumed plays a negligible role in affecting aggression (Bushman and Cooper, 1990; Hull and Bond, 1986; Steele and Southwick, 1985).

Parenthetically, a methodology termed the balanced placebo design was created to separate the pharmacological effects of alcohol from placebo effects. This design involves the use of the three groups described above as well as a group of subjects who receive alcohol but are told that they are consuming a nonalcoholic beverage (i.e., "antiplacebo" condition). Although this design is theoretically useful, it is not practically useful because of the near impossibility of

convincing antiplacebo subjects that they have not consumed alcohol, particularly at the higher doses needed to facilitate aggression (Martin and Sayette, 1993). To the author's knowledge, only two studies have used the balanced placebo design to examine the effects of alcohol on aggression as measured by the TAP (Lang et al., 1975; Pihl et al., 1981). Results were mixed in that only one study found an increase in aggression for the antiplacebo group (Pihl et al., 1981).

A series of more than 20 studies on the alcohol-aggression relation, conducted by Taylor and colleagues, using the TAP, documented robust and reliable findings. College students who received alcohol evidenced higher levels of aggression than those who received placebo or nonalcoholic beverages (e.g., Bailey and Taylor, 1991; Chermack and Taylor, 1995; Leonard, 1989; Taylor and Gammon, 1975; Taylor et al., 1976). Using a modified version of the TAP, Pihl and colleagues also found (in more than 10 studies) that college students who received alcohol administered higher shock intensity levels and longer shock durations compared with those who received a placebo or a nonalcoholic control beverage (e.g., Hoaken et al., 1998; Lau and Pihl, 1994; Pihl et al., 1990; Pihl and Zacchia, 1986; Zeichner and Pihl, 1979). Furthermore, using other modifications of the TAP, Giancola and colleagues replicated the above results (Giancola et al., in press; Giancola and Zeichner, 1995b,c, 1997; Zeichner et al., 1994, 1995). Clearly, the results of these studies support a strong relation between acute alcohol consumption and aggressive behavior in college students.

Many of the investigations reviewed above, and others, have been included in meta-analytic studies. As would be expected, the results of these studies support the contention that acute alcohol intoxication facilitates aggressive behavior. For example, Bushman and Cooper (1990) determined that the average effect sizes for alcohol versus placebo conditions and placebo versus nonalcohol conditions were 0.61 and 0.10, respectively. In a later study, Bushman (1993) reported similar mean effect sizes for these comparisons (i.e., 0.49 and 0.0028). In summary, these statistics clearly indicate that acute alcohol consumption significantly increases the expression of aggressive behavior in college students.

Theoretical Perspectives

General theories of aggression

Clearly, there is a need for effective prevention interventions aimed at attenuating alcohol-related aggression in college students. However, it has been argued that for prevention interventions to make a significant and lasting impact, they must spawn from theoretically based empirical research that elucidates the causal structure of the alcohol-

aggression relation (Chermack and Giancola, 1997). Given the important need for theory in guiding research, some important models of alcohol-related aggression are presented below. However, insofar that such specialized models are a subset of more general theories, it would be useful first to review some broad theories of aggression. Geen (1990) and Berkowitz (1993) provided two such prominent contemporary theories.

Geen's (1990) major premise is that the elicitation of aggression is dependent on the interaction of two general factors. The first involves "background variables" such as genetics, physiology, temperament, personality, social-cultural expectations and exposure to violence. According to Geen, deviations on these variables serve to predispose toward aggression. The second factor involves frustrating or provocative environmental stimuli that produce stress, arousal and anger. These stimuli can take many forms such as a verbal or physical attack, family conflict, hot temperatures and physical pain. Geen explained that the manner in which these provocative or frustrating stimuli are interpreted will moderate the amount of stress, arousal and anger that is experienced, which will then affect whether aggression is or is not expressed. Specifically, if an aversive situation is interpreted as justifiable or nonintentional, the result will be little arousal and anger, which will lead to little or no aggression. Conversely, if a situation is interpreted to be malicious or arbitrary, the result will be high levels of arousal and anger and thus a higher probability of an aggressive response. Finally, Geen added that even in a highly aroused or angered state, the expression of aggression can still be moderated by paying attention to alternative nonaggressive means of coping with the situation. However, if such attentional resources are lacking, the probability of an aggressive response will be heightened.

Berkowitz (1993) put forth a theory postulating that the desire to behave in an aggressive manner is the result of experiencing negative affect. Negative affect is defined as any unpleasant feeling that can be brought on by a number of factors such as frustration, insults, attacks, hot temperatures and noise. Berkowitz made the point that it is not the direct effect of such instigating factors that produces aggression (i.e., damaged self-image, being punched in the face), but instead the psychological damage (i.e., negative affect) that they produce. According to the theory, the experience of negative affect results in the activation of aggression- or fear-related cognitions, feelings and expressive-motor and physiological reactions that are associated with both basic fight and flight tendencies. Once the primary reactions to an aversive event have occurred, more differentiated feelings later arise as the result of higher order cognitive processing (e.g., making causal attributions, thinking about possible consequences of aggression, paying attention to social rules). According to Berkowitz, this higher order reasoning differentiates the original more ba-

sis experience, thus intensifying some of its aspects and suppressing others. Therefore, an initial basic response to aggress can be modified by "thinking" about alternative nonaggressive solutions to the situation.

Although these theories have not been covered in great detail, both attempt to explain aggressive behavior by implicating fairly broad constructs and processes (i.e., arousal, anger, negative affect, social cognition). Furthermore, both theories also suggest that variations in a number of key individual difference variables (e.g., biology, personality) and situational variables (e.g., provocation, temperature) are crucial for the expression of aggression. These models are important because they provide good overarching explanations of aggressive behavior and offer excellent conceptual frameworks from which to test more specific hypotheses about the causes of aggression.

Alcohol and aggression: Disinhibition and expectancy models

The disinhibition model is considered to be a very general explanation of the alcohol-aggression relation. It contends that alcohol has a direct effect on aggression by pharmacologically disinhibiting brain centers important in maintaining inhibitory control over behavior (Graham, 1980). This model has limited empirical support because not all persons become aggressive when they drink alcohol.

In direct opposition is the expectancy model, which stipulates that it is not the pharmacological properties of alcohol that facilitate aggression, but rather the mere belief that one has consumed alcohol (MacAndrew and Edgerton, 1969). This model rests on the assumption that people have a priori beliefs that alcohol will lead to aggression. As noted above, previous studies have demonstrated negligible differences in aggression between subjects receiving a placebo beverage versus those who knowingly drank a nonalcoholic beverage. These data are typically used to argue against the position that alcohol expectancies affect aggressive behavior. However, this is an erroneous argument because placebo manipulations do not take into account individual differences in beliefs that alcohol will increase aggression. That is, it may be that placebo manipulations are indeed effective in increasing aggression but only in persons who believe that alcohol will increase aggression. The few published studies that take into account individual differences in alcohol expectancies for aggression have shown modest to good support that expectancies interact with alcohol to increase aggression (Chermack and Taylor, 1995; Dermen and George, 1989; Leonard and Senchak, 1993).

Another model, a more refined version of the disinhibition explanation, is the indirect cause model (Graham, 1980). This model suggests that alcohol detrimentally affects certain psychological and/or physiological processes that then

lead to the expression of aggressive behavior. Some of the most prominent contemporary theories of alcohol-related aggression are variants of the indirect cause model. Specifically, most of them are cognitive models that suggest that alcohol disrupts a specific type of cognitive function that then increases the probability of aggression. Due to their influential nature in the current research literature on alcohol-related aggression, seven of these models are reviewed below.

Cognitive models

Pernanen (1976) hypothesized that alcohol consumption increases the probability of an aggressive reaction by reducing the number of available psychological coping mechanisms that rely on conceptual/abstract reasoning. According to this model, alcohol creates a "narrowing of the perceptual field" (p. 415), which reduces the ability to detect both internal and external cues that may provide crucial information about another person's intentions in a precarious situation. Consequently, a reduction in these cues will result in a random or an arbitrary interpretation of the other person's intentions. Accordingly, when intoxicated, it is this tendency to interpret incoming information as random or arbitrary (especially if the incoming information is aggressive in nature) that will increase the probability of a violent response.

Taylor and Leonard (1983) postulated that aggressive behavior is determined by the relative balance of a combination of both instigative (e.g., threats, insults) and inhibitory (e.g., anxiety, norms of reciprocity) cues present in hostile interpersonal situations. Instigative cues increase the probability of an aggressive encounter, whereas inhibitory cues decrease that probability. These theorists reasoned that the cognitive disruption produced by alcohol reduces the number of information sources (i.e., cues) that one can attend to in any given situation. Therefore, aggressive behavior is most likely to occur in a context where instigative cues are paramount as opposed to a situation dominated by inhibitory cues.

Steele and Josephs (1990) proposed an attention allocation model in which alcohol interferes with information processing in such a manner as to disrupt the ability to allocate attention to multiple aspects of a situation effectively. Accordingly, alcohol creates a "myopic" or narrowing effect on attention, which results in attention being allocated only to the most salient aspects of a particular situation and not to other less salient cues. Alcohol will therefore decrease the ability to extricate important meaning from less salient, possibly inhibitory, cues. It is thus maintained that in a conflict or a provocative situation, alcohol's myopic effect on attention may facilitate aggression by forcing attention to the most salient (i.e., provocative) aspects of that situation and not to other less salient (i.e., inhibitory) cues.

As can be seen quite clearly, Taylor and Leonard's (1983) and Steele and Josephs' (1990) models are very similar (i.e., both maintain that alcohol impairs the ability to attend to inhibitory cues). The main difference between the two models is that Steele and Josephs explicitly posited the hypothetical mechanism of inhibition conflict as a determinant of when alcohol will, and will not, facilitate aggression. Inhibition conflict refers to the magnitude of conflict between two opposing response tendencies (Steele and Southwick, 1985). According to Steele and colleagues (Steele and Josephs, 1990; Steele and Southwick, 1985), a considerable degree of inhibition conflict must be present if alcohol is to facilitate aggression. Steele and Josephs' model predicts that an intoxicated person is likely to attack another individual in the presence of both inhibitory and instigatory cues (high conflict) because attention will be focused on the most salient cues (i.e., provocative/instigatory). However, in the absence of any inhibitory cues (low conflict), the model predicts that the effects of alcohol will be irrelevant. That is, without inhibitory cues, an attacker will be just as likely to emit an aggressive response in either an intoxicated or a sober state due to the lack of any internal or external proscriptions against aggression. Similarly, if no provocative cues are present, a person should not react aggressively whether intoxicated or sober. Parenthetically, the mechanism of inhibition conflict is nonetheless implicit in Taylor and Leonard's model.

Pihl et al. (1993) posited a biosocial model of intoxicated aggression in which cognitive functioning is but a single aspect of a multidimensional mechanism. According to these theorists, acute alcohol consumption disrupts the functioning of the prefrontal cortex and its subcortical connections, especially the hippocampus, which, according to Pihl et al., "is involved in the recognition of threat" (p. 134). Thus, by disrupting these neural regions and circuits, alcohol eliminates signals of punishment through its anxiolytic effects (i.e., reduces fear reactions), resulting in decreased inhibitory control over behavior. Pihl et al. also posited that aggressive responses are enhanced through alcohol's psychomotor stimulant properties and an increased sensitivity to cues of physical pain.

Hull (1981) proposed a general model of the effects of alcohol on self-awareness in which it is suggested that alcohol intoxication engenders aggressive behavior through a reduction in self-awareness. According to his model, alcohol disrupts self-awareness by interfering with the higher order cognitive encoding of self-relevant information necessary to attain a self-aware state. Such interference then purportedly disrupts the ability to evaluate self-relevant social and environmental information that putatively provides feedback concerning appropriate forms of behavior. Without access to this information, Hull posited the heightened probability of aggressive behavior.

Sayette (1993) advanced an appraisal disruption model of alcohol's effects on stress. Ito et al. (1996) invoked this

model to account for the alcohol-aggression relation. According to Sayette, if alcohol is consumed before the onset of anxiety-eliciting cues, it will disrupt the cognitive appraisal of those cues, thus resulting in anxiolysis. In such a case, as noted by Ito et al., alcohol may facilitate aggression indirectly by reducing fear and inhibition. This model shares a commonality with that of Pihl et al. (1993) in that both make the point that alcohol disrupts, in essence, the same cognitive ability (i.e., recognition of threat [Pihl et al.] and information appraisal [Sayette]), which then facilitates aggression through an attenuation of fear and inhibition.

Giancola (2000a) advanced the idea that all of the cognitive abilities implicated in the above models are components of a more general construct termed executive functioning. Executive functioning is defined as a higher order cognitive construct involved in the planning, initiation and regulation of goal-directed behavior (Luria, 1973, 1980; Milner, 1995). The cognitive abilities subsumed within this construct include attentional control, previewing, information appraisal, strategic goal planning, abstract reasoning, temporal response sequencing, self- and social monitoring, abstract reasoning, cognitive flexibility, hypothesis generation and the ability to organize and adaptively utilize information contained in working memory (Kimberg and Farah, 1993; Stuss and Benson, 1984). Giancola argued that, compared with models that invoke only one cognitive ability, a more general model that incorporates a cluster of conceptually and empirically related abilities would more accurately reflect the richness and complexity of the cognitive mechanisms influencing the alcohol-aggression relation. Based on data showing that low executive functioning is related to increased aggression and that acute alcohol consumption disrupts executive functioning, Giancola put forth a new model. This model postulates that (1) executive functioning mediates the alcohol-aggression relation in that acute alcohol intoxication disrupts executive functioning, which then heightens the probability of aggression and (2) executive functioning moderates the alcohol-aggression relation in that acute alcohol consumption is more likely to facilitate aggressive behavior in persons with medium to low, rather than high, executive functioning.

Beginning to Sketch a "Risk Profile" for the Alcohol-Aggression Relation

Although research shows that acute alcohol consumption is related to the expression of aggressive behavior, there is a wide range of individual differences among these data. In other words, not all people become aggressive when they drink. Therefore, it can be argued that alcohol does not directly cause aggression solely through its pharmacological actions (Bushman and Cooper, 1990). Rather, accumulating evidence indicates that intoxicated aggression is

the product of individual difference and contextual variables interacting with alcohol pharmacodynamics (Chermack and Giancola, 1997). Currently, very little is known about the manner in which these latter variables, and their interactions, serve as underlying mechanisms of intoxicated aggression. Therefore, a useful task for investigators would be to identify which traits characterize individuals who typically exhibit intoxicated aggression and which situational conditions are most likely to facilitate such behavior. Below is a brief examination of some individual difference and contextual variables that may serve as "risk factors" for alcohol-related aggression.

Individual difference variables

Dispositional aggressivity. Dispositional aggressivity, typified by the tendency to be aggressive across a range of situations, has been shown to be strongly related to self-reported husband-to-wife violence (Leonard and Senchak, 1993) and violent behavior in male college students (Dermen and George, 1989). Interestingly, dispositionally aggressive individuals, such as those with antisocial personality disorder or conduct disorder, are also characterized by low executive functioning (Malloy et al., 1990; Moffitt and Henry, 1989). Only one study has assessed the combined effects of acute alcohol consumption and dispositional aggressivity on aggression as measured by the TAP in college students (Bailey and Taylor, 1991). Acute alcohol consumption increased aggression in men with high levels of dispositional aggressivity but not in those with low or moderate levels.

Alcohol expectancies. Alcohol expectancies are defined as beliefs about the effects of alcohol on behavior (Leigh, 1987). As noted above, some research suggests that intoxicated aggression results, in part, from the belief that alcohol increases aggression. It is well known that people vary in their belief that alcohol increases arousal, power, assertiveness, verbal aggression and physical aggression (Brown et al., 1980; Rohsenow and Bachorowski, 1984). Significantly, self-report studies indicate that the association between alcohol consumption and aggression is stronger among individuals who *expect* alcohol to increase aggression (Dermen and George, 1989; Leonard and Senchak, 1993). One published study, using the TAP, attempted to determine whether individual differences in alcohol-aggression expectancies would affect aggression under the influence of alcohol in male college students (Chermack and Taylor, 1995). Results indicated that under conditions of high provocation, intoxicated subjects with high expectancies about the effects of alcohol on aggression were more aggressive than were those with low expectancies.

Drinking history. Quantity of past alcohol consumption is positively related to self-reported aggression in male (Dermen and George, 1989) and female (West et al., 1990) social drinkers. Theory suggests that increased alcohol con-

sumption and aggressive behavior are both components of an overarching construct of "deviant behavior" (Jessor and Jessor, 1977; Pernanen, 1991). However, the underlying mechanisms, or causal dynamics, of that construct are not known. One laboratory study found that acute alcohol consumption increased aggression on the TAP in male college students, but only in those with *low*, rather than moderate or high, levels of past-year drinking (LaPlace et al., 1994). The authors hypothesized that alcohol's detrimental effects on cognition were greater in those with a low tolerance for alcohol compared with those with a higher tolerance.

Executive functioning. Low executive functioning capacity has been found to be related to increased aggression in young boys and young adult males; fighting in normal pre-adolescent boys; and increased disruptive, delinquent and physically aggressive behavior in adolescent females (Giancola and Zeichner, 1994; Giancola et al., 1996, 1998; Seguin et al., 1995). It has been hypothesized that low executive functioning facilitates the expression of aggression by impeding the cognitive regulation of behavior and interfering with the ability to generate alternative, nonaggressive responses in provocative situations (Giancola, 1995, 2000a).

Only one study has assessed the relation between executive functioning, acute alcohol consumption and aggression (Lau et al., 1995). Normal male college students were administered two neuropsychological tests of executive functioning and were then separated into "high" and "low" functioning groups. They were administered either an alcohol or a nonalcohol beverage and then tested on the TAP. Alcohol and low executive functioning had independent effects on aggression; however, an interaction between executive functioning and alcohol consumption was not observed. An interaction was predicted because theory suggests that alcohol increases aggression to a greater extent in individuals with medium to low, compared with high, executive functioning (Giancola, 2000a). Conclusions from this study are limited because only two executive functioning tests were used, and statistical power was too low to detect a significant Executive Functioning \times Alcohol interaction.

Hostile attributional biases. Research has shown that aggressive children are more likely than their nonaggressive counterparts to erroneously attribute hostile intent to another child's provocative actions, even if those actions are, from an objective standpoint, ambiguous in intent (Dodge, 1980; Dodge and Frame, 1982). Furthermore, hostile attributional biases have been shown to be positively related to undersocialized aggressive conduct disorder, reactive aggression and number of violent crimes committed in a sample of highly aggressive juvenile offenders (Dodge et al., 1990). These data suggest that erroneous hostile attributional biases may be, in part, responsible for increased aggression in children. Others have found that adults are also vulnerable to making erroneous hostile attributions in

ambiguous interactions (Epps and Kendall, 1995). Therefore, with respect to intoxicated aggression, it is possible that alcohol may disrupt information processing to the extent that an individual may distort and/or misinterpret ambiguous interpersonal information or cues, thus resulting in the attribution of a hostile bias, which may then lead to an increased probability of emitting an aggressive response.

Biochemistry. Both animal and human research have demonstrated a positive relation between testosterone levels and physical aggression (Volavka, 1995). Berman et al. (1993) found that healthy male college students with high levels of testosterone, measured in saliva, were more aggressive on the TAP than those with low levels. Moreover, heightened aggression has also been related to low levels of the brain neurotransmitter serotonin (Berman et al., 1997). Interestingly, a study using the TAP demonstrated increased aggression in healthy college males who received a tryptophan-depleted dietary mixture (Pihl et al., 1995). Tryptophan is the biochemical precursor for serotonin; its dietary depletion leads to lowered brain serotonin levels. Theorists have argued that serotonin is involved, in part, in the inhibition of behavior (Spoont, 1992; Volavka, 1995). As such, it may be that the aggression enhancing effects of alcohol are more likely to occur in individuals with higher baseline levels of testosterone and lower levels of serotonin.

Currently, very little is known about the acute effects of alcohol on testosterone and serotonin in the human brain. Animal research suggests that low doses of alcohol tend to enhance blood testosterone levels whereas high doses tend to have a suppressing effect (K. Miczek, personal communication, 2000). Animal research also suggests that acute alcohol consumption initially increases, but then decreases, brain serotonin levels (reviewed in LeMarquand et al., 1994). Although far less work has been conducted on humans than on animals, current research suggests that acute alcohol consumption depletes blood tryptophan levels, thus suggesting depletions in brain serotonin (reviewed in Badawy, 1998).

Gender. There exist only a small number of published studies on alcohol-related aggression in women. In a survey investigation, White et al. (1993) reported that adolescent males engaged in more alcohol-related aggression (e.g., fights, hurting someone, forced sex, vandalism, setting fires) than their female counterparts. In contrast, a laboratory study found that low doses of alcohol increased verbal aggression on an adjective checklist in females but not in males (Rohsenow and Bachorowski, 1984). In a study using a modified version of the TAP, Bond and Lader (1986) found that alcohol equally increased aggression (i.e., tone blasts) for men and women when they were exposed to low levels of provocation (i.e., low intensity tone blasts). However, when highly provoked, only men showed increased aggression with alcohol (Bond and Lader, 1986). In a study using a point subtraction task, Dougherty et al. (1996) showed

that alcohol increased aggression for women. In another study using men and women, Dougherty et al. (1999) reported that alcohol equally increased aggression for both genders. In contrast, however, Gustafson (1991) found that alcohol and provocation had no effects on aggressive responding (i.e., shock administration) in women.

Giancola and Zeichner (1995b) reported that alcohol increased aggression in the form of shock intensity and shock duration for men; however, it only slightly increased shock duration for women. Furthermore, high provocation (i.e., receiving high intensity shocks) increased aggression for men and women, regardless of whether they received alcohol. Hoaken and Pihl (2000) found that alcohol increased shock intensity and duration for men but not for women. Although alcohol did not affect aggression for women, higher levels of provocation increased their aggressive responding to the same degree as intoxicated men.

Contextual variables

Blood alcohol concentration limb effects. Studies have generally shown that a blood alcohol concentration (BAC) of .08% is typically sufficient to facilitate aggression (Gustafson, 1985; Pihl and Zacchia, 1986). Given this, the assumption has generally been that as long as one is at a BAC of .08% or higher, there is a greater likelihood for aggression. However, Giancola and Zeichner (1997) showed that this assumption is indeed correct, but only for the ascending limb of the BAC curve (when alcohol levels in the bloodstream are rising). That is, in a study measuring two different groups of male college students on the TAP, those tested on the ascending limb of the BAC curve (.08% BAC) were significantly more aggressive than those tested on the descending limb of the curve (.08% BAC). Those tested on the descending limb were no more aggressive than were sober control subjects. The authors explained this finding by noting that greater executive functioning deficits are found on the ascending limb compared with the descending limb of the BAC curve.

Alcohol type and dose. Intoxicated aggression varies depending on the type of alcoholic beverage that is consumed. Specifically, distilled beverages such as vodka and bourbon elicit significantly more aggression on the TAP compared with brewed beverages such as beer (Pihl et al., 1984). Further, the dose of alcohol administered also affects aggression. Research has shown that the relation between alcohol dose and aggressive behavior follows an inverted U-shaped curve. That is, at low doses that produce BACs around .03-.04%, alcohol produces rather small increases, if any, in physical aggression (reviewed in Pihl, 1983). Greater levels of aggression are typically seen at BAC levels of .08% or higher (Giancola and Zeichner, 1995b, 1997; Pihl, 1983). Of course, studies that produce excessively high BACs cannot be ethically conducted. However, based on

animal data and anecdotal reports, it is roughly estimated that, for most persons, BACs above .20-.30% will induce a biological and psychological state where aggression, and most other organized and complex behaviors, will not be possible.

Social pressure. It has been demonstrated that social pressure also helps to moderate the alcohol-aggression relation. In a study by Taylor and Sears (1988), confederates were asked to encourage sober and intoxicated male college students to behave more aggressively toward their opponent on the TAP. Results demonstrated that only intoxicated subjects were influenced by the confederates' suggestions to behave aggressively.

Provocation. Provocation is a necessary ingredient in an interpersonal interaction if aggressive behavior is to occur. In their review of the literature on alcohol and violent crime, Murdoch et al. (1990) reported that verbal altercations tend to precede violent interactions. In her study of 307 assaultive criminals, Mayfield (1976) reported that "in 50% of the cases the victim attacked or made a move which was interpreted by the subject as an impending attack immediately prior to the assault" (p. 289). In the context of a provocative situation, research has shown greater aggression on the TAP in intoxicated, compared with sober, male college students (Taylor et al., 1979).

Clearly, multiple factors contribute to the expression of alcohol-related aggression. It should be made clear, however, that the risk factors described here are not an exhaustive list. Other traits that are potentially important in moderating the alcohol-aggression relation include age, perspective-taking, self-awareness, negative affect, temperament, affect regulation, emotionality, sensation seeking, anxiety, irritability, hostility, frustration tolerance, impulsivity, psychopathology, early physical abuse, perceived self-esteem and tolerance and sensitivity to alcohol. Unfortunately, there is no single profile that will predict intoxicated aggression in all persons. However, studying these and other variables is important because it will provide researchers with a better understanding of the mechanisms that underlie the alcohol-aggression relation.

Policy Implications

This article makes the point that acute alcohol consumption per se does not directly cause aggression. Instead, it argues that alcohol interacts with a host of individual difference and contextual variables to facilitate aggression. In other words, although alcohol does have some involuntary biological effects that predispose toward aggression (impairing brain functioning), there are also a number of psychological factors that contribute to alcohol-related aggression. Other than attempting to institute radical and clearly untenable preventative initiatives (e.g., alcohol prohibition, lacing alcoholic beverages with serotonin-enhanc-

ing and testosterone-reducing additives), changes in social policy will probably have little effect on the biological causes of alcohol-related aggression. However, one area in which both scientists and policy-makers can direct their attention is the development of psychological harm reduction strategies, strategies aimed at reducing the possibility of alcohol-related aggressive behavior.

This article identifies a number of psychological risk factors for intoxicated aggression. What appears to be needed are intervention programs aimed at modifying key risk factors so that alcohol consumption will be less likely to engender aggression. However, to be most effective, these interventions must be implemented in the proper context. For example, such programs could begin by educating people about the effects of alcohol on behavior. Specifically, it can be clarified that alcohol, in and of itself, does not cause aggression; it merely "drowns" the inhibitions that typically keep us from behaving aggressively or inappropriately. The message must be clearly sent that alcohol will not facilitate any behaviors for which there is no psychological predisposition. Given the early ages at which adolescents begin to consume alcohol in the United States, it would be prudent to begin such interventions at the junior high-school level and continue throughout the college years. This message could be conveyed through classroom teachings. Furthermore, fraternities, sororities, dormitories, athletic programs and other establishments and institutions could also be required to convey these messages to their members and residents. In addition, cognitive restructuring techniques could be similarly implemented to alter preexisting expectations that alcohol causes aggressive behavior (Darkes and Goldman, 1993).

With regard to executive functioning, interventions could be modeled after neuropsychological rehabilitation efforts aimed at strengthening cognitive functioning (Giancola, 2000b). Moreover, interventions could also be aimed at teaching social interaction and interpretation skills so that persons with aggressive or hostile dispositions and attributional biases can remain nonargumentative and non-violent when drinking. Such interventions would probably be implemented most successfully in specialized mental health clinics for disruptive, delinquent and violent children, adolescents and adults. Finally, interventions can also be used to educate about contextual influences on intoxicated aggression, particularly those that can be prevented or avoided (e.g., alcohol type, social pressure, provocation). Again, these messages can be conveyed in classrooms from junior high on to college as well as other college settings such as dormitories, fraternities and athletic programs.

The risk factors for intoxicated aggression that are listed in this article are clearly not specific to college students. However, when it comes to focusing on this special population, other well-known variables come into play that are as important, if not more important, than those listed above.

These would include contextual factors such as “keg” parties, sporting events, fraternity life and coed dormitories. It would also be important to consider other dispositional traits that could increase the probability of exposure to such “high-risk” contexts such as problem behaviors prior to college and preexisting attitudes that promote disinhibited behavior, violence and excessive drinking. Although many universities and colleges already have educational programs in operation, at various levels, to inform students about the dangers of alcohol, negative, and sometimes disastrous, outcomes are nonetheless still too high. Clearly, a problem cannot be effectively prevented or treated if the cause is not known. If effective policy aimed at reducing intoxicated aggression in college students is to be implemented, more research will be needed to understand how alcohol interacts with basic dispositional traits, environmental variables, problem behaviors and attitudes that are present before one arrives at college and contextual variables that are typically specific to college life.

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