

Effects of Alcohol on Human Aggression: An Integrative Research Review

Brad J. Bushman and Harris M. Cooper
University of Missouri—Columbia

This review used quantitative and qualitative techniques to integrate the alcohol and aggression literature. The primary purpose of the review was to determine if a causal relation exists between alcohol and aggression. The main meta-analysis included 30 experimental studies that used between-subjects designs, male confederates, and male subjects who were social drinkers. Studies using other designs or subject populations were integrated with meta-analytic procedures when possible and summarized descriptively when not. The results of the review indicate that alcohol does indeed cause aggression. However, alcohol effects were moderated by certain methodological parameters.

. . . O thou invisible spirit of wine,
if thou hast no name to be known by,
let us call thee devill! . . .
O God, that men should put an enemy
in their mouths to steal away their brains!
that we should, with joy, pleasance, revel
and applause, transform ourselves into beasts!
—William Shakespeare, *Othello* (II, iii)

For hundreds of years, it has been assumed that individuals behave more aggressively while under the influence of alcohol. Correlational evidence offers some support for conventional wisdom. For instance, numerous studies have found a strong relation between alcohol intoxication and homicide. In an early book, MacDonald (1961) reviewed 10 studies and found that the proportion of murderers who had been drinking before their crimes ranged from .19 to .83, with a median of .54. More recent studies have reported similar findings among both adult (Holcomb & Anderson, 1983) and juvenile (Sorrells, 1977) assailants. In addition, alcohol intoxication has been linked to other types of aggression, such as assault (Myers, 1982), wife abuse (Gayford, 1975), and rape (Johnson, Gibson, & Linden, 1978).

Correlational evidence, however, does not allow one to infer that alcohol is the *cause* of violent crime (e.g., Brain, 1986). Experimental data must be used to make such inferences. Thus, the present review includes only studies that used the experimental method to examine the relation between alcohol and aggression.

Prototypical Methods in Experimental Alcohol–Aggression Research

Although each experimental study of alcohol and aggression has unique components, many studies have common features.

We thank Bill Holcomb for his assistance with this project. In addition, we extend gratitude to Russ Geen and Ken Sher for their helpful comments on an earlier draft of this article and to Chris Cooper, Alicia Peabody, and Shelly Wade for providing coder reliability measurements. Last, but not least, we express sincere appreciation to those researchers who provided unpublished reports and previously unpublished data.

Correspondence concerning this article should be addressed to either Brad J. Bushman or Harris M. Cooper, Department of Psychology, University of Missouri, Columbia, Missouri 65211.

The typical laboratory procedures for administering beverages and measuring aggression are described in the following sections.

Beverage Administration

To administer alcoholic and nonalcoholic beverages, most researchers use either a placebo or a balanced placebo design. In the placebo design, all subjects are told that they will receive alcohol. However, only some subjects are actually given alcohol; the other subjects are given a placebo. Occasionally, an additional condition is added to the placebo design. Subjects in this group are informed that they will receive a nonalcoholic beverage and they are given a nonalcoholic beverage. This cell serves as a control condition because subjects receive neither alcohol nor the expectancy of alcohol. The influence of alcohol-related expectancies on aggression can be determined by comparing the placebo group with the control group.

In the balanced placebo design (Marlatt & Rohsenow, 1980), half of the subjects are told that they will receive an alcoholic beverage and half are told that they will receive a nonalcoholic beverage. Within each of these groups, half of the subjects are given alcohol and half are not. Thus, the balanced placebo design includes an “antiplacebo” condition. Subjects in this group are informed that they will receive a nonalcoholic beverage, but they are given an alcoholic beverage. The pure pharmacological effect of alcohol can be ascertained by comparing the antiplacebo group with the control group.

Measures of Aggression

For this review, we have adopted Baron’s (1977) definition of aggression: “Aggression is any form of behavior directed toward the goal of harming or injuring another living being who is motivated to avoid such treatment” (p. 7). Buss (1961) has proposed that aggressive acts can be classified by using combinations of three categories: *physical–verbal*, *active–passive*, and *direct–indirect*. The studies in the review used three types of aggression measures: *verbal–active–direct*, *verbal–active–indirect*, and *physical–active–direct*. Passive acts of aggression (e.g., sit-in demonstration, refusing to speak to another person) and

indirect acts of physical aggression (e.g., setting a booby trap for another person) have not been used in experimental studies of alcohol and aggression.

Verbal aggression is considered to be direct when the victim is actually present and indirect when the victim is absent. Generally, direct verbal aggression is measured by recording a subject's vocal comments to one or more individuals, and indirect verbal aggression is measured by means of a questionnaire.

Direct physical aggression is generally measured by means of a teacher-learner task (Buss, 1961), a reaction-time task (Taylor, 1967), or a variation of one of these two procedures. In the Buss (1961) procedure, a subject and a confederate are told that the study is concerned with the effects of punishment on learning. A rigged "lottery" is used to assign the real subject to the teacher role and the confederate to the learner role. When the learner makes an incorrect response on the task, the teacher is told to punish him by means of electric shock. By using different buttons, the teacher can vary both the intensity and the duration of shock given to the confederate.

In the Taylor (1967) procedure, the subject competes with an ostensible opponent on a reaction-time task. At the beginning of each trial the subject sets the level of shock he wants the opponent (confederate) to receive if his response is slower. At the end of each trial, the subject is informed of the level of shock the opponent set for him to receive on the trial. The loser then receives the indicated intensity of shock. In actuality, the experimenter determines who wins and loses and the feedback and shock levels given to the subject. The dependent variable is the intensity of shock the subject sets for the opponent.

Explanations of Intoxicated Aggression

Several theories have been proposed to explain the relation between alcohol and aggression. Graham (1980) has suggested that most of these theories can be placed into one of four categories depending on the role each assigns to alcohol: direct cause, indirect cause, indirect cause conditional on motive for drinking, or predisposition-situation. A brief description of each category follows.

Direct Cause

According to this explanation, alcohol causes aggression directly by anesthetizing the center of the brain that normally prevents aggressive responding. Disinhibition theorists contend that alcohol facilitates aggression "not by 'stepping on the gas' but rather by paralyzing the brakes" (Muehlberger, 1956, p. 40).

Indirect Cause

According to this explanation, alcohol facilitates aggression indirectly by causing certain cognitive, physiological, and emotional changes that increase the probability of aggression. Some of the cognitive changes that accompany alcohol consumption include impaired intellectual functioning, inaccurate assessment of risks, and reduced awareness. Alcohol has also been shown to change arousal levels and to increase emotional lability.

Motive for Drinking

According to this explanation, certain motives lead people to drink, and these motives interact with alcohol to facilitate aggression. Two drinking motives have been proposed: anxiety reduction and power concerns. Horton (1943) argued that people drink primarily to reduce anxiety, and that anxiety reduction is accompanied by a decrease in aggressive restraints. Horton also claimed that there is a positive relation between the strength of the anxiety drive motivating drinking and the strength of the aggressive response. McClelland and his colleagues instead contended that "men drink primarily to feel stronger" (McClelland, Davis, Kalin, & Wanner, 1972, p. 334), especially those men who are preoccupied with power concerns. McClelland further assumed that thoughts of personal power that accompany alcohol consumption may be manifested as aggression.

Predispositional-Situational Factors

According to this explanation, the relation between alcohol and aggression is a spurious one based on a relation between predispositional or situational factors and aggression. Some researchers (e.g., Boyatzis, 1975) have claimed that certain types of individuals are predisposed to behave aggressively and that drinking occasions provide an acceptable outlet for aggression. Others (e.g., Lang, Goeckner, Adesso, & Marlatt, 1975) have argued that the psychological effects of alcohol are more important determinants of aggression than the pharmacological effects of alcohol. People apparently expect alcohol to increase aggression. Those who behave aggressively while intoxicated can therefore "blame the bottle" for their actions. MacAndrew and Edgerton (1969) claimed that violence and other antisocial behaviors occur when alcohol is consumed because, in many societies, drinking situations are culturally agreed-on "time-out" periods. Other researchers have emphasized the role of situational variables on intoxicated aggression (e.g., Graham, La Rocque, Yetman, Ross, & Guistra, 1980). Drinking often occurs in crowded, noisy, smoky, and provocative environments. Past research has shown that these aversive stimuli can facilitate aggression (Berkowitz, 1983).

We should note, as Graham (1980) did, that these four categories are not mutually exclusive or, perhaps, even independent. For example, the direct-cause hypothesis seems to make the same assumptions as the anxiety-reduction hypothesis. Even though Graham's framework lacks conceptual clarity, we chose it because it is fairly comprehensive and unbiased.

Although we were not able to formally test the preceding explanations, we can make a few statements that are relevant to some of these theories. For example, if alcohol directly causes aggression, then antiplacebo subjects should behave more aggressively than control subjects. Alcohol-related expectancies have also been manipulated experimentally in some studies. If these expectancies are important determinants of aggression, then placebo subjects should behave more aggressively than control subjects.

Past Reviews

Although several reviews have described the relation between alcohol and violent crime (e.g., Pernanen, 1976), few attempts

have been made to review experimental studies of alcohol-mediated aggression. We were able to locate four reviews devoted to this topic (Hull & Bond, 1986; Pihl, 1983; Steele & Southwick, 1985; Taylor & Leonard, 1983¹).

Two book chapters have summarized the alcohol and aggression literature. Of the 13 experimental studies reviewed by Pihl (1983), 7 were conducted in his own laboratory. From the data, Pihl concluded that both alcohol and expectancy facilitate aggressive behavior. In the Taylor and Leonard (1983) chapter, 10 of the 17 experimental studies reviewed were conducted by Taylor and his colleagues. The authors deduced that a strong positive relation exists between alcohol dose and physical aggression.

This article is not the first attempt to use quantitative techniques to synthesize the alcohol and aggression literature. Two previous meta-analytic studies have been published. In the earliest article, Steele and Southwick (1985) investigated the effects of alcohol on social behavior in general. Of the 35 articles used in the meta-analysis, 14 examined the relation between alcohol and aggression. Steele and Southwick concluded that alcohol can increase aggression by reducing inhibitions.

In the second meta-analytic study, Hull and Bond (1986) examined the effects of alcohol and expectancy on social and non-social behaviors. Only studies that used the balanced placebo design were included in the review. Five of the 34 articles examined the relation between alcohol and aggression. Hull and Bond concluded that alcohol, but not expectancy, increases aggression.

Present Review

The present review uses both quantitative and qualitative methods to integrate the alcohol and human aggression literature. The results of the review are divided into three sections. Meta-analytic procedures are used in the first two sections, whereas descriptive procedures are used in the third section. The main meta-analysis, reported in the first section, includes 30 studies that used between-subjects designs, male confederates, and male subjects who described themselves as social drinkers. The influence of moderator variables on the results of these studies is also investigated. In the second section, meta-analytic procedures are used to integrate studies that used other designs or subject populations. In the third section, descriptive summaries are given of studies that were too few in number to be integrated by using meta-analytic procedures.

Method

Literature Search Procedures

Formal and informal channels were used to search the literature. First, the reference sections of recent reviews on alcohol and aggression were combed (Hull & Bond, 1986; Pihl, 1983; Steele & Southwick, 1985; Taylor, 1983; Taylor & Leonard, 1983). Second, three on-line computer reference data bases were searched: *Psychological Abstracts* (1967–1988), *Dissertation Abstracts International* (1861–1988), and *DRUGINFO and Alcohol Use and Abuse* (1968–1988). The terms used to describe aggression (*attack, fight, dominant, aggression, violence, agnostic, hostility, and anger*) were the same descriptors used by the International Society for Research on Aggression in their journal, *Aggressive*

Behavior. The aggression keywords were paired with three alcohol terms: *alcohol, ethanol, and intoxicant*. Various forms of the keywords were also used (e.g., *aggress, aggression, aggressive*). Although the search was not restricted by language, it was restricted to studies that used *human* subjects. The computer retrieved all studies that used the paired keywords in the title, in the abstract, or as descriptors. The abstracts of the retrieved studies were then examined.

To obtain recently published studies not yet indexed by the on-line reference data bases, the contents of issues from seven journals published between January 1986 and June 1989 were inspected: *Aggressive Behavior, British Journal of Addiction, Journal of Abnormal Psychology, Journal of Personality and Social Psychology, Journal of Research in Personality, Journal of Studies on Alcohol, and Personality and Social Psychology Bulletin*.

More informally, unpublished, in-press, or recently published studies were requested from all authors who had published an experimental study on alcohol and human aggression in the 1980s. One in-press and two unpublished studies were received from the authors who responded.

Criteria for Relevance

Because the primary purpose of this review was to make a causal statement about the effects of alcohol on aggressive behavior, we used two exclusion criteria. First, correlational studies were excluded from the review. Second, studies that used aggressive state measures were excluded unless they also used behavioral measures of aggression. An aggressive state is a combination of thoughts, emotions, and behavior tendencies that are elicited by stimuli capable of evoking aggression. Although such a state should heighten the likelihood of aggression, it would not be classified by most psychologists as aggressive "behavior."

Coding Frame

The information listed in Appendix A was extracted from the report of each study.² These data have been divided into four categories: source characteristics, participant characteristics, experiment characteristics, and primary study results.

Intercoder Reliability

From the pool of relevant studies, 15 were randomly selected for coding by the primary coder and three undergraduate research assistants. With one exception, the reliability coefficients (Fleiss, 1971) ranged from 0.85 to 1.00 with a median of 1.00. One characteristic, nonaggressive response alternative available or unavailable, had low reliability because of a misinterpretation by one of the three undergraduate coders.

Meta-Analytic Procedures

Two meta-analytic procedures were used in the present review: (a) the estimation of average effect sizes and 95% confidence intervals, and (b) homogeneity analyses to determine whether the effect sizes were drawn from the same population.

One problem that arises in estimating average effect sizes is deciding what constitutes an independent hypothesis test. The present review used a shifting unit of analysis (Cooper, 1989). First, each statistical test was coded as if it were an independent event. For example, if a single

¹ Taylor published another chapter in 1983. However, because there is a great deal of overlap between the Taylor (1983) and the Taylor and Leonard (1983) chapters, only the latter, more extensive review was discussed.

² Further information about the coding frame and coding reliability can be obtained by writing Brad J. Bushman or Harris M. Cooper.

study contained two measures of aggression (e.g., shock intensity, shock duration) and two types of alcohol and placebo groups (e.g., beer, vodka), a total of four effect sizes would be coded (e.g., shock intensity-beer, shock intensity-vodka, shock duration-beer, shock duration-vodka). For the estimate of alcohol's overall effect on aggression, the four effect sizes would be averaged so that the study would contribute only one effect size. For an analysis in which the effects of alcohol are compared for different measures of aggression, the study would contribute two effect sizes (e.g., shock intensity, shock duration). Two effect sizes would also be contributed to an analysis that compared the effects of different types of alcohol (e.g., beer, vodka). Thus, the shifting unit approach retains as much data as possible without violating too greatly the independence assumption that underlies the validity of quantitative synthesis procedures.

We calculated estimates of average effect sizes by weighting each d index by the number of participants on which it was based. Average effect sizes were computed for each of the four comparison types (i.e., alcohol vs. control, alcohol vs. placebo, antiplacebo vs. control, placebo vs. control). We also calculated 95% confidence intervals for the average effect sizes.

Homogeneity analysis compares the amount of variance exhibited by a set of effect sizes with the amount of variance expected if only sampling error is operating. If the total variance is greater than the sampling error, the analyst proceeds to test whether study characteristics explain the "excess" variance. Hedges and Olkin (1985) described how the homogeneity analysis can be carried out by using the General Linear Model program of the Statistical Analysis System computer package (SAS, 1985). The WEIGHTED statement is used to minimize the weighted residual sums of squares. The sums of squares due to the tested influences are treated as chi-square statistics.

Results

Results From the Main Meta-Analysis

The main meta-analysis included 30 studies that used between-subjects designs, male confederates, and male subjects who described themselves as social drinkers (see Appendix B). Operationally, all subjects reported consuming less than 20 drinks per week.

Distribution of the data. Because extreme, unrepresentative values can seriously distort tests or estimates of parameters, some statisticians recommend discarding outlying observations before performing statistical analyses (e.g., V. Barnett & Lewis, 1978). Tukey's (1977) box plot procedure was used to identify extreme outliers in the distributions for the four comparison types. Tukey's procedure identified four extreme *positive* outliers in the data set, two for alcohol versus control comparisons and two for alcohol versus placebo comparisons. The four effect sizes were calculated from two studies reported by Zeichner and Pihl (1979, 1980) that were conducted for Zeichner's (1978) doctoral dissertation. The box plots for the four types of comparisons, with the outliers removed, are shown in Figure 1.

The normality assumption was tested by using the Shapiro-Wilk (1965) W statistic if there were 50 observations or fewer for the comparison or by using the Kolmogorov (1933) D statistic if there were more than 50 observations. The analyses showed no significant departures from normality: alcohol versus control, $W = 0.97$, $p = .54$; alcohol versus placebo, $D = 0.08$, $p > .15$; antiplacebo versus control, $W = 0.93$, $p = .43$; and placebo versus control, $W = 0.95$, $p = .47$.

Because many research reports did not include standard deviations for the dependent measures of aggression, we calcu-

lated several d indexes from statistical tests that used pooled variance terms. For those studies that did report standard deviations, the experimental and control group standard deviations were pooled to calculate effect sizes. Hartley's (1950) $F(\max)$ statistic was therefore used to test whether the control and experimental group variances were homogeneous. For the meta-analysis, 30 studies yielded 116 effect sizes. Standard deviations were available for 78 of the 116 effect sizes. Overall, 86% of the $F(\max)$ tests were nonsignificant at the .05 level and 92% were nonsignificant at the .01 level. Therefore, pooling the standard deviations was considered an acceptable procedure.

Differences between the four comparison types. The average effect sizes and confidence intervals for the alcohol versus control, alcohol versus placebo, antiplacebo versus control, and placebo versus control comparisons were as follows: 0.25 (0.11, 0.40), 0.61 (0.51, 0.70), 0.06 (-0.17, 0.29), and 0.10 (-0.10, 0.29), respectively. On examining the data, we noticed that the average effect size for the placebo versus control comparison was positive even though the alcohol versus placebo effect size was larger than the alcohol versus control effect size. To discover the cause for this anomaly, the studies were separated into two groups: those that contained alcohol, placebo, and control conditions and those that contained only two of these three conditions. We then analyzed the two groups of studies separately. The results of these two analyses as well as the overall analysis are displayed in Table 1.

The results showed that the average placebo versus control effect size was in fact negative for studies in which alcohol, placebo, and control conditions were yoked together, $d(+)$ = -0.10. The positive placebo versus control effect in the overall analysis may have been due to two large effect sizes from one study that included only placebo and control groups (Alioto, 1974). These two factors account for the apparently discrepant pattern of results.

A homogeneity analysis was conducted to determine if there were effect-size differences among the four comparison types. This analysis showed that the effect sizes from the four comparison types were heterogeneous, $\chi^2(3, N = 116) = 25.76$, $p < .0001$. Pairwise tests revealed that the average effect size for alcohol versus placebo comparisons was larger than the average effect sizes for the alcohol versus control, $\chi^2(1, n = 88) = 9.81$, $p = .0017$; antiplacebo versus control, $\chi^2(1, n = 68) = 13.66$, $p = .0002$; and placebo versus control, $\chi^2(1, n = 72) = 10.93$, $p = .0009$, comparisons.

We then performed homogeneity analyses to determine whether study results within each comparison type were similar. The analyses revealed that the antiplacebo versus control, $\chi^2(3, n = 12) = 15.08$, $p = .0018$, and the placebo versus control, $\chi^2(5, n = 16) = 15.85$, $p = .0073$, comparisons were heterogeneous, whereas the alcohol versus control, $\chi^2(9, n = 32) = 13.30$, $p = .15$, and alcohol versus placebo, $\chi^2(23, n = 56) = 24.38$, $p = .38$, comparisons were homogeneous. Because there were only four studies that contributed antiplacebo versus control effect sizes, and six studies that contributed placebo versus control effect sizes, analyses for the influence of moderator variables on these two comparison types were not performed. Even though the best explanation for study differences within the alcohol versus control and alcohol versus placebo comparisons was sampling error, we conducted a priori moderator tests.

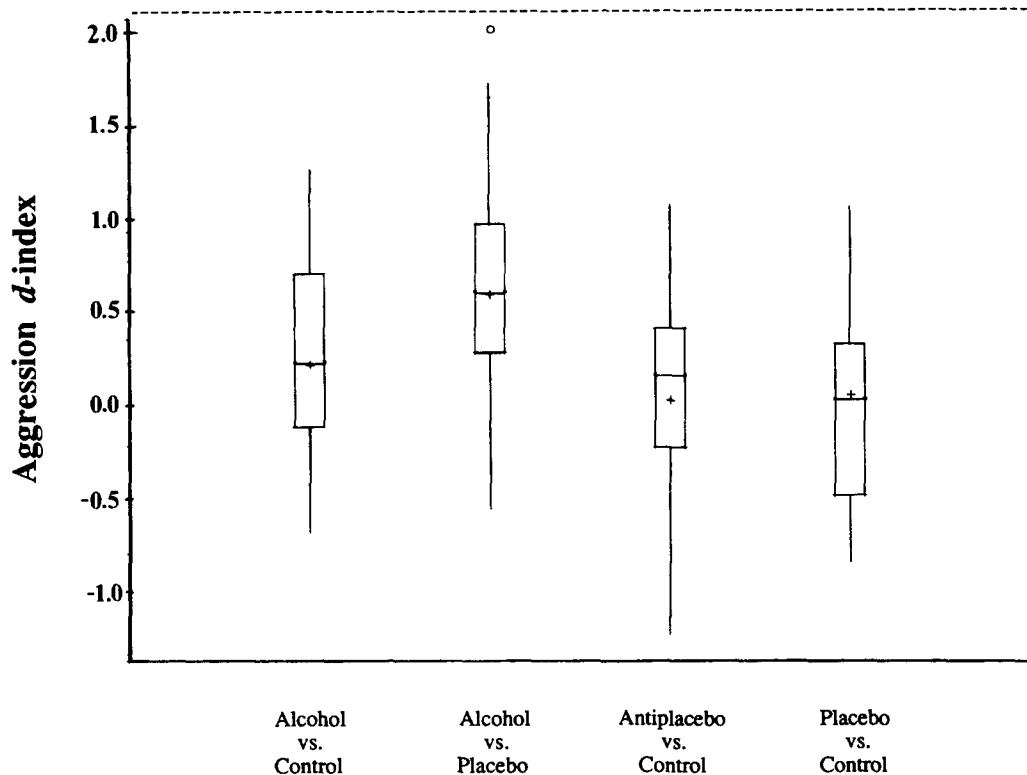


Figure 1. Box plots for the four types of comparisons (+ = sample mean, O = mild outlier)

Influence of moderator variables on alcohol effects. Homogeneity analyses revealed that five variables moderated alcohol versus control effect sizes (see Table 2). Effect sizes were smaller

Table 1
Effect Sizes and Confidence Intervals for the
Four Comparison Types

Comparison	Study		
	Yoked	Unyoked	Total
Alcohol versus control			
$d(+)$	0.11	0.38	0.25
n	14	18	32
95% C.I.	-0.10, 0.33	0.18, 0.58	0.11, 0.40
Alcohol versus placebo			
$d(+)$	0.26	0.69	0.61
n	14	42	56
95% C.I.	0.04, 0.48	0.58, 0.79	0.51, 0.70
Antiplacebo versus control			
$d(+)$			0.06
n			12
95% C.I.			-0.17, 0.29
Placebo versus control			
$d(+)$	-0.10	1.03	0.10
n	14	2	16
95% C.I.	-0.31, 0.12	0.56, 1.50	-0.10, 0.29

Note. Yoked studies contained alcohol, control, and placebo groups; unyoked studies contained only two of these three groups. $d(+)$ = average weighted effect size; n = number of observations; C.I. = confidence interval.

if subjects were distracted from focusing on internal cues during the alcohol ingestion period than if they were not distracted, $\chi^2(1, n = 32) = 7.93, p = .005$. Effect sizes were smaller for studies in which the experimenter was blind to alcohol and expectancy manipulations than for studies in which the experimenter was not blind to conditions, $\chi^2(1, n = 32) = 7.37, p = .007$. Effect sizes were larger when subjects were required to make an aggressive response than when they were given a non-aggressive response alternative, $\chi^2(1, n = 32) = 4.72, p = .03$. Effect sizes were larger when the highest button was associated with the stimulus level described as "definitely unpleasant" than when a middle button was associated with the same level of pain, $\chi^2(1, n = 22) = 5.83, p = .016$. Finally, effect sizes were larger when subjects fasted from food for 4 hr than when they fasted from food for 2 hr, $\chi^2(1, n = 26), p = .032$.

We used multiple regression analysis to determine the best linear combination of moderator variables for alcohol versus control comparisons. Initially, all significant moderators were included in the model. The influence of multicollinearity on the least squares estimates was tested by using variance inflation factors (see Neter, Wasserman, & Kutner, 1983). Partial sums of squares were analyzed to determine the contribution of each moderator variable if it was the last added to the model. The final model was obtained by sequentially eliminating moderator variables that made nonsignificant unique contributions.

The alcohol versus control regression model contained two moderator variables: experimenter blind or not blind to conditions, $\chi^2(1, n = 117) = 40.87, p < .0001$, and nonaggressive response alternative available or unavailable, $\chi^2(1, n = 117) =$

Table 2
*Significant Effects for Qualitative Moderators Variables
 Included in the Coding Frame*

Characteristic	<i>d</i> (+)	<i>n</i>	95% C.I.
Alcohol versus control			
Subject distracted or not distracted while consuming beverage			
Distracted	-0.03	17	-0.23, 0.16
Not distracted	0.61	15	0.39, 0.83
Experimenter blind or not blind to alcohol and expectancy manipulations			
Blind	-0.13	11	-0.38, 0.12
Not blind	0.46	21	0.28, 0.65
Nonaggressive response alternative available or unavailable			
Available	0.04	18	-0.16, 0.23
Unavailable	0.56	14	0.34, 0.79
Noxious stimulus intensity threshold			
Highest button	0.82	8	0.49, 1.14
Middle button	0.08	14	-0.14, 0.29
Hours fasted from food			
2 hr	-0.11	10	-0.38, 0.16
4 hr	0.38	16	0.17, 0.59
Alcohol versus placebo			
Subject distracted or not distracted while consuming beverage			
Distracted	0.47	31	0.35, 0.60
Not distracted	0.80	25	0.65, 0.95
Experimenter blind or not blind to alcohol and expectancy manipulations			
Blind	0.28	15	0.11, 0.49
Not blind	0.76	41	0.64, 0.87
Type of alcohol			
Vodka	0.70	39	0.59, 0.82
Other distilled beverages	0.75	6	0.41, 1.10
Absolute alcohol	0.28	9	0.06, 0.50
Beer	0.26	2	-0.22, 0.76
Confederate retaliation			
Possible	0.69	47	0.59, 0.79
Impossible	0.13	9	-0.12, 0.38

Note. *d*(+) = average weighted effect size; *n* = number of observations; C.I. = confidence interval.

26.73, $p < .0001$. These two variables explained 31% of the variability in alcohol versus control effects.

Homogeneity analyses revealed that five variables moderated alcohol versus placebo effect sizes. Effect sizes were smaller if subjects were distracted from focusing on internal cues during the alcohol ingestion period than if they were not distracted, $\chi^2(1, n = 56) = 5.61, p = .018$. Effect sizes were smaller if the experimenter was blind to conditions than if the experimenter was not blind to conditions, $\chi^2(1, n = 56) = 13.05, p = .0003$. The type of alcohol also influenced the size of effects, $\chi^2(3, n = 56) = 7.76, p = .05$. Effect sizes were larger for studies that used vodka or other distilled beverages than for studies that used absolute alcohol or beer. Effect sizes were larger when the confederate could retaliate than when the confederate could not retaliate, $\chi^2(1, n = 56) = 6.00, p = .014$. Finally, effect sizes increased

by 0.06 as alcohol concentration increased by 1%, $\chi^2(1, n = 47) = 9.01, p = .0027$. The percentage of absolute alcohol in beverages ranged from 7% to 25%. The data for the four reliable qualitative moderator variables are listed in Table 2.

Regression analysis showed that two moderator variables significantly contributed to the prediction of alcohol versus placebo effects: experimenter blind or not blind to conditions, $\chi^2(1, n = 117) = 34.32, p < .0001$, and opponent retaliation possible or impossible, $\chi^2(1, n = 117) = 26.66, p < .0001$. The three other moderator variables were eliminated from the model because they were not reliable. A linear combination of the two reliable terms accounted for 30% of the variability in alcohol versus placebo effects.

The effects of several moderator variables were not tested because they were included in only one or two studies. These variables, as well as a statistical summary of their effects, are listed in Table 3.

Meta-Analysis of Studies That Used Other Designs or Subject Populations

Sex differences in alcohol-mediated aggression. Four alcohol and aggression experiments have treated sex of the subject as a factor. In the two studies conducted by Rohsenow and Bachorowski (1984), the confederate was the same sex as the subject. In a third experiment by Ratliff (1984), the confederate was male for all subjects. In the fourth study, Bond and Lader (1986), the sex of the confederate was not specified. Two additional studies have used female confederates. In one study, the subjects were male (Richardson, 1981); in the other study, the subjects were female (Walker, 1982).

Statistical analyses of the results from the six studies showed one significant sex of confederate effect for antiplacebo versus control comparisons, $\chi^2(1, n = 7) = 6.15, p = .013$, and one sex of confederate trend for alcohol versus placebo comparisons, $\chi^2(1, n = 8) = 3.51, p = .061$. For the antiplacebo versus control comparisons, the average effect size was $-0.30 (-0.70, 0.11)$ for men and $0.51 (0.02, 1.00)$ for women. For the alcohol versus placebo comparisons, the average effect size was $-0.04 (-0.44, 0.37)$ for men and $0.47 (0.12, 0.88)$ for women. Thus, from the results of these studies, we would conclude that, when intoxicated, both men and women behave more aggressively toward a female target than toward a male target.

Effects of heavy drinking habits on alcohol-mediated aggression. Although no alcohol and aggression study has treated drinking history as a factor, six experiments have used male heavy drinkers and male confederates. All six studies used the balanced placebo design. Three of the six studies were conducted by Rohsenow and Bachorowski (1984). The average number of drinks consumed by subjects in Experiments 1, 2, and 3 was 28.23, 25.65, and 27.65, respectively. Subjects in the study by White (1987) imbibed an average of 23.03 drinks per week. Lang et al. (1975) and R. K. Barnett (1979) used the Drinking Practices Questionnaire to classify subjects as heavy social drinkers.

We performed statistical analyses on the data from the six studies. The effect sizes and confidence intervals for the four comparisons were as follows: alcohol versus control, $-0.24 (-0.42, 0.12)$; alcohol versus placebo, $-0.06 (-0.29, 0.23)$; antiplace-

Table 3
Moderator Variables Not Included in the Main Meta-Analysis

Study	Variable	Interaction with alcohol
Bailey, Leonard, Cranston, & Taylor (1983)	Aggression task performed under self-aware or non-self-aware conditions	<i>ns</i> (alcohol vs. placebo)
Gustafson (1985)	Aggressive or nonaggressive film segment shown	<i>ns</i> (alcohol vs. placebo)
Gustafson (1986)	Subjects told or not told that the confederate planned to retaliate with high shock levels	No difference between alcohol and placebo subjects in the threat condition. In the standard condition, alcohol subjects were more aggressive than placebo subjects.
Gustafson (in press)	Positive versus negative mood induction	<i>ns</i> (alcohol vs. placebo)
Heermans (1980)	Misattribution of arousal: tranquilizer versus no drug, energizer versus no drug	<i>ns</i> (alcohol vs. control) <i>ns</i> (alcohol vs. control)
Jeavons & Taylor (1985)	Presence or absence of a low aggression norm	Within the no-norm condition, alcohol subjects were more aggressive ($d = 0.93$) than placebo subjects. In the norm condition, there was no difference between alcohol and placebo subjects.
Myerscough (1984)	Sleep deprivation versus no sleep deprivation	<i>ns</i> (alcohol vs. placebo)
Pihl & Zacchia (1986)	Positive versus negative mood induction	<i>ns</i> (alcohol vs. control) <i>ns</i> (alcohol vs. placebo) <i>ns</i> (placebo vs. control)
Schmutte & Taylor (1980)	High or low pain feedback from provocative confederate	Pain feedback enhanced aggression in subjects with high blood alcohol levels and reduced aggression in placebo subjects.
Taylor & Gammon (1976)	Pressure or no pressure to reduce aggression	<i>ns</i> (alcohol vs. control) <i>ns</i> (alcohol vs. placebo)
Taylor, Gammon, & Capasso (1976)	Subjects told or not told that the confederate planned to use low shock levels	No difference between alcohol and placebo subjects in the no-threat condition. In the standard condition, alcohol subjects were more aggressive than placebo subjects.
Taylor & Sears (1988)	No pressure, mild pressure, or strong pressure to increase aggression	No difference between alcohol and placebo subjects in the no-pressure situation ($d = 0.00$). Alcohol subjects were more aggressive than placebo subjects in the mild pressure ($d = 0.12$) and strong pressure ($d = 0.31$) situations.
White (1987)	Attractive versus unattractive confederate	<i>ns</i> (alcohol vs. control) <i>ns</i> (alcohol vs. placebo) <i>ns</i> (antiplacebo vs. control) <i>ns</i> (placebo vs. control)
Zeichner, Pihl, Niaura, & Zacchia (1982)	Subjects were forced to attend to, distracted from attending to, or received no instruction about the noise level set by the confederate.	In all conditions, alcohol subjects were more aggressive than placebo subjects. Regarding shock intensity, effects were highest for no-instruction ($d = 0.67$), followed by forced attention ($d = 0.24$) and distraction ($d = 0.08$). For shock duration, effects were highest for forced attention ($d = 1.15$), followed by no-instruction ($d = 0.65$), and distraction ($d = 0.07$).

Note. *ns* = statistically nonsignificant result.

cebo versus control, -0.20 ($-0.40, 0.13$); and placebo versus control, -0.33 ($-0.48, 0.05$). The confidence intervals for all four comparison types include the value zero. Thus, these results indicate that alcohol and expectancy manipulations have little effect on the amount of aggression displayed by male heavy drinkers.

Alcohol and aggression studies using within-subjects manipulations of alcohol dose. Four alcohol and aggression studies have used repeated measures designs with different doses of ethanol. All four studies included placebo conditions. Bennett, Buss, and Carpenter (1969) and Gustafson (1984) used three doses of alcohol: 0.79, 0.53, and 0.26 g/kg. Cherek, Steinberg, and Vines

(1984) used five doses of alcohol: 0.99, 0.79, 0.40, 0.20, and 0.10 g/kg. Cherek, Steinberg, and Manno (1985) used three doses of alcohol: 0.46, 0.23, and 0.12 g/kg.

We conducted a weighted regression analysis to examine the influence of alcohol dose on effect sizes for the four studies. The analysis showed that the dose effect was not significant, $\chi^2(1, N = 14) = 0.23, p = .63$.

Descriptive Summaries of Other Studies

More naturalistic studies of alcohol and aggression. Most experiments that have examined the relation between alcohol and

aggression have been conducted in a laboratory setting. However, a study by Boyatzis (1974) was conducted in a more natural setting: a comfortable room in a modern office building. Subjects were told that the research involved studying the leisure-time behavior of men. Subjects were assigned to a distilled spirits, beer, or nonalcoholic beverage group. Subjects were free to order drinks from the bar throughout the evening and were encouraged to play such games as darts, cards, and dice. The men were videotaped early in the session, in the middle of the session, and late in the session. An "aggressive-behavior" score was obtained for each subject during each filming period by summing across several behavior categories. The results showed that subjects in the alcohol conditions were more aggressive than subjects in the control condition. In addition, men who consumed distilled spirits were more aggressive than men who consumed beer, especially as the night wore on.

In experiments that use a reaction-time task, the real subject generally competes with either a confederate or an apparatus. In an experiment conducted by Leonard (1984), both partners in the dyad were real subjects. Subjects were both intoxicated, both sober, or one partner was intoxicated and the other was sober. The results showed that intoxicated dyads set higher shock levels than sober dyads. The intensity levels set by subjects in mixed dyads were in between, but not significantly different from, the levels set by intoxicated and sober dyads. The intoxicated members of mixed dyads, however, set more intense shocks than did the sober members of mixed dyads.

In the majority of alcohol and aggression studies, the subject and confederate are strangers. In an experiment conducted by Smith, Parker, and Nobel (1975), subjects were male-female couples who were either spouses or close friends. Each couple participated in a high dose (1.18 g/kg), a low dose (0.73 g/kg), and a placebo condition. During the study, subjects were instructed to talk about anything that was important to them at the time. Conversations were tape recorded and later scored for aggressive content. The results showed no effect for alcohol dose on direct verbal aggression.

Different types of alcohol. Two studies, not included in the main meta-analysis, examined the influence of different types of alcohol on aggression. In the earliest study (Takala, Pihkanen, & Markkanen, 1957), experimental subjects participated in two sessions, one that used a 1.00 g/kg dose of brandy and one that used a 1.00 g/kg dose of beer. Control subjects were selected from a different population. In the study, small groups of subjects were asked to discuss several topics (e.g., the right of police to use arms). Conversations were tape recorded and later scored by means of the Bales Recording System. The results showed that verbal aggression was highest in the brandy condition, second highest in the beer condition, and lowest in the control condition.

Subjects who participated in the Taylor and Gammon (1975) experiment were randomly assigned to receive either a high (0.99 g/kg) or a low (0.33 g/kg) dose of vodka or bourbon. Vodka and bourbon placebo groups were not included in the design. After consuming beverages, subjects competed with an ostensible opponent who increased shock levels across trials on a reaction-time task. The results showed that subjects who received the high dose of alcohol were more aggressive than subjects who received the low dose of alcohol. A three-way interac-

tion showed that under high levels of provocation, subjects who received a high dose of vodka were more aggressive than subjects who received a high dose of bourbon. This pattern was reversed for the low dose groups.

Alcohol and other drugs. Alioto (1974) investigated the effects of marijuana- and alcohol-related expectancies on aggressive behavior. In both the marijuana and alcohol groups, half of the subjects expected to receive an active drug and half expected to receive a placebo. In reality, all subjects were given an inert substance. The results showed that subjects who expected marijuana were least aggressive, whereas those who expected alcohol were most aggressive.

In another study, Taylor, Vardaris, Rawtich, Gammon, Cranston, and Lubetkin (1976) tested the effects of delta-9-tetrahydrocannabinol (THC), the active ingredient in marijuana, and alcohol on aggression. Subjects received a high dose of THC (0.3 mg/kg), a low dose of THC (0.1 g/kg), a high dose of alcohol (0.99 g/kg), a low dose of alcohol (0.33 g/kg), or a placebo. After consuming beverages, subjects competed with a confederate on a reaction-time task. The results showed that subjects who received alcohol set more intense shocks than subjects who received THC, but only in the high-dose conditions. It should be noted, however, that these data are difficult to interpret because the placebo shock levels were not reported.

An experiment conducted by Bailey (1987) examined the combined effects of alcohol and propranolol on aggression. Propranolol is a beta-adrenergic antagonist that is thought to decrease somatic symptoms of arousal. Propranolol has been found to entirely abolish heart-rate response to unpredictable shock given during a reaction-time task (Langer et al., 1985). It has been used to treat anxiety symptoms (Granville-Grossman & Turner, 1966), alcohol withdrawal symptoms (Carlsson & Johansson, 1971), and episodes of rage and violent behavior in patients with brain disorders (Williams, Mehl, Yudofsky, Adams, & Roseman, 1982).

Subjects in the study were given 60 mg propranolol plus 0.99 g/kg alcohol, 0.99 g/kg alcohol, or a placebo drink. After consuming beverages, subjects competed with a confederate on a reaction-time task. The results showed that subjects in the alcohol condition were significantly more aggressive than subjects in the propranolol-alcohol or placebo conditions. The latter two conditions did not differ from each other.

Discussion

The evidence from the main meta-analysis indicates that alcohol does indeed cause aggressive behavior. The average effect sizes for both alcohol vs. control, $d(+) = 0.25$, and alcohol vs. placebo, $d(+) = 0.61$, comparisons were significantly greater than zero. These alcohol effect sizes are similar in magnitude to the effect sizes reported for other independent variables in aggression research (e.g., Eagly & Steffen, 1986; Hearold, 1986). Alcohol also appears to influence aggressive behavior as much or more than it influences other social and nonsocial behaviors (Hull & Bond, 1986; Steele & Southwick, 1985). Thus, alcohol and aggression effects are by no means trivial.

Differences Between the Four Comparison Types

Recall that of the four comparison types, only the alcohol versus placebo and alcohol versus control comparison effects

were greater than zero, and that the effect size for the former comparison was significantly larger than the effect size for the latter. Siegel (e.g., Siegel & MacRae, 1984) has proposed a theory of drug tolerance that can account for the difference between the two reliable comparisons. The theory, which is based on Pavlovian conditioning, suggests that the administration of a drug constitutes a classical conditioning trial. The drug's active ingredient serves as the unconditional stimulus (UCS) that produces an unconditional pharmacological response (UCR). Stimuli in the environment that accompany drug administration (e.g., taste and smell of alcohol) constitute the conditional stimulus (CS). Because most drugs produce disturbances in homeostatic systems, the conditional response (CR) may be *opposite* in direction to the UCR. The compensatory CR is an attempt to reestablish a state of equilibrium by reducing the effects of the drug. A placebo beverage (which smells and tastes like alcohol) would therefore elicit a compensatory CR, whereas a control beverage would not. Thus, we would expect a larger positive effect for the alcohol versus placebo comparison than for the alcohol versus control comparison. In addition, the effect for the placebo versus control comparison should be negative. This is the exact pattern of results found for studies in which alcohol, placebo, and control groups were yoked together.³

Although this reasoning can account for the differences between three of the comparisons, it does not explain the null antiplacebo versus control effect. According to Siegel (e.g., Siegel & MacRae, 1984), subjects in the antiplacebo group should behave more aggressively than subjects in the control group because they receive the UCS without the inhibiting effects of the CS. Although Siegel's theory is not entirely consistent with the data, we offer it as a stimulus for future theoretical research.

To account for the results from all four comparisons we must invoke another explanation. Taylor and Leonard (1983) have argued that subjects in the antiplacebo group

may realize that the experimenter attempted to deceive them concerning the contents of the beverage. Thus, it is extremely likely that they would be suspicious concerning other facets of the experiment and would tend to monitor their behavior. This increased self-awareness would be expected to *reduce* aggressive responding. (p. 94, emphasis added)

The same logic could explain the behavior of subjects in the placebo group. These subjects, who expect to receive an alcoholic beverage, may become suspicious when they do not experience the intoxicating effects of alcohol.

One possible test of Taylor and Leonard's (1983) explanation would be to examine the effects of alcohol content on responses to manipulation check questions (see Appendix A, Experiment Characteristic 16). If beverage manipulations were effective, answers to such questions should *not* be influenced by alcohol content. For antiplacebo versus control comparisons, 3 (75%) of the 4 studies reported a main effect for alcohol content. For alcohol versus placebo comparisons, 13 of the 24 studies provided manipulation check data. Of these 13 studies, 10 (77%) reported a main effect for alcohol content. Therefore, these data indicate that subjects in the antiplacebo and placebo groups generally see through the beverage deception.

It is the opinion of the authors that the alcohol versus control comparison provides the best estimate of the effects of alcohol

on aggression. In real life, the psychological and pharmacological effects of alcohol occur together. Furthermore, this estimate is not affected by the methodological problems that have been noted. We would therefore encourage alcohol and aggression researchers to include control groups in their study designs.

Relevance of the Results to the Four Explanations of Intoxicated Aggression

Recall that Graham (1980) has divided theories of intoxicated aggression into four categories. Although we could not formally test each explanation, the results from this review are relevant to some of the theories.

The direct-cause explanation states that alcohol increases aggression by anesthetizing the region of the brain that controls aggressive inhibitions. The antiplacebo versus control comparison should provide the best test of this hypothesis because it yields the pharmacological effects of alcohol uncontaminated by expectancies. The average effect size for antiplacebo versus control comparisons was 0.06, which was not significantly different from zero. Thus, there appears to be little support for the hypothesis that alcohol directly affects aggression.

The predispositional-situational explanation assumes that intoxicated aggression is the result of factors concomitant with alcohol consumption. One such factor is alcohol-related expectancies. The placebo versus control comparison should provide the best test of this hypothesis because it yields the psychological effects of alcohol uncontaminated by pharmacological effects. The average effect size for antiplacebo versus control comparisons was 0.10, which was not significantly different from zero. Thus, the pure psychological effects of alcohol do not seem to increase aggressive behavior.

It appears that neither the pure pharmacological effects of alcohol nor the pure psychological effects of alcohol are important determinants of aggression. It is possible that both effects must occur together for alcohol to cause aggression. These statements must be qualified, however, because of the methodological problems with the antiplacebo and placebo manipulations that were noted in the previous section.

Methodological Parameters That Moderated Alcohol Versus Control and Alcohol Versus Placebo Effects

Some of the characteristics extracted from the studies moderated alcohol effects. For alcohol versus control comparisons, effect sizes were smaller when the experimenter was blind to conditions than when the experimenter was not blind to conditions, and were larger when an aggressive response was required than when a nonaggressive alternative was available. For alcohol versus placebo comparisons, effects were smaller for blind studies than for nonblind studies and larger for studies in which the confederate was free to retaliate against the subject than for studies in which confederate retaliation was not possible.

³ Similar findings were obtained for balanced placebo studies. The average effect sizes and confidence intervals were as follows: alcohol versus control, $d(+) = 0.14 (-0.09, 0.36)$; alcohol versus placebo, $d(+) = 0.31 (0.09, 0.54)$; antiplacebo versus control, $d(+) = 0.06 (-0.17, 0.29)$; and placebo versus control, $d(+) = -0.13 (-0.35, 0.10)$.

Table 4
*Percentage of Studies Reporting Information for
 Characteristics Included in the Coding Frame*

Type of characteristic	Meta-analysis		All studies	
	n	%	n	%
Participant characteristics				
Average age of subjects	7	23	18	37
Drinking history	16	53	28	57
Average number of drinks per week	4	13	15	31
Experimental characteristics				
Hours fasted from drugs and alcohol	15	50	24	49
Time of day alcohol consumed	3	10	9	18
Blood alcohol level before task	16	53	29	58
Blood alcohol level after task	15	50	29	58
Surveillance during alcohol consumption	8	27	19	39
Subject distracted or not distracted while consuming beverage	17	57	31	63
Experimenter blind or not blind to alcohol and expectancy manipulations	11	37	21	43
Beverage manipulation successful or unsuccessful	14	47	25	51
Noxious stimulus intensity threshold	18 ^a	60	22 ^b	49
Subject told the confederate is intoxicated or is sober	14	30	24	49

Note. $N = 30$ for the main meta-analysis. $N = 49$ for all studies.

^a Based on 29 studies that used physical measures of aggression.

^b Based on 41 studies that used physical measures of aggression. Not included in the table are characteristics reported by more than 70% of the studies.

Reporting Study Characteristics

It should be noted that many of the research reports failed to include information about variables that could potentially moderate alcohol effects. Table 4 contains the percentage of studies reporting information for the characteristics included in the coding frame. Those characteristics that were reported more than 70% of the time have been omitted from the table.

Results From Secondary Meta-Analyses

We also used quantitative procedures to examine studies that used designs and subject populations different from those in the main meta-analysis. Null effects were obtained for studies that used within-subjects manipulations of alcohol dose. However, the relation between alcohol dose and level of aggression was in the predicted direction (i.e., positive). Nonsignificant findings were also obtained for studies that used male heavy drinkers. It is possible that drinking habits moderate alcohol effects. For example, Goldberg (1943) found that heavy drinkers were more tolerant of alcohol effects than either moderate drinkers or abstainers, presumably because they had habituated to the pharmacological properties of the drug. On the other hand, the sex of the target did influence alcohol-related aggression. When intoxicated, both men and women behaved more aggressively toward a female target than toward a male target. Aggression against a woman is generally considered antisocial behavior. Consuming alcohol may either reduce normal inhibitions or

provide subjects with an "excuse" for engaging in behavior that would ordinarily be regarded as unacceptable.

Conclusions

In conclusion, the results of the review indicate that alcohol does indeed facilitate aggressive behavior. The effects of alcohol on aggression were similar to the effects of other independent variables on aggression. In addition, alcohol appears to influence aggressive behavior as much or more than it influences other social and nonsocial behaviors.

We would like to offer three suggestions to guide future alcohol and aggression research. First, if a balanced placebo design is not used, a control condition should be added to a placebo design. The alcohol versus control comparison may provide the best estimate of the effects of alcohol on aggression. Those who use antiplacebo and placebo conditions should take special steps to ensure that beverage manipulations are successful. Second, attention must be paid to variables that could potentially moderate alcohol effects. A description of these variables should also be included in the research report. Third, researchers should continue to test and develop theories of intoxicated aggression. For, as Kant once remarked, research without theory is blind.

References

- Alioto, J. T. (1974). The effects of expectancy of receiving either marijuana or alcohol on subsequent aggression in provoked high and low users of these drugs. *Dissertation Abstracts International*, 35, 4637B. (University Microfilms No. ADG74-27722, 0000)
- Bailey, D. S. (1987). The combined effects of alcohol and propranolol on human aggression. *Dissertation Abstracts International*, 48, B. (University Microfilms No. ADG87-19575, 8712)
- Bailey, D. S., Leonard, K. E., Cranston, J. W., & Taylor, S. P. (1983). Effects of alcohol and self-awareness on human physical aggression. *Personality and Social Psychology Bulletin*, 9, 289-295.
- Barnett, R. K. (1979). The effects of alcohol, expectancy, provocation, and permission to aggress upon aggressive behavior. *Dissertation Abstracts International*, 40, 4993B. (University Microfilms No. ADG80-09229, 0000)
- Barnett, V., & Lewis, T. (1978). *Outliers in statistical data*. New York: Wiley.
- Baron, R. A. (1977). *Human aggression*. New York: Plenum Press.
- Bennett, R. M., Buss, A. H., & Carpenter, J. A. (1969). Alcohol and human aggression. *Quarterly Journal of Studies on Alcohol*, 30, 870-877.
- Berkowitz, L. (1983). Aversively stimulated aggression: Some parallels and differences in research with animals and humans. *American Psychologist*, 38, 1135-1144.
- Bond, A., & Lader, M. (1986). The relationship between induced behavioral aggression and mood after the consumption of two doses of alcohol. *British Journal of Addiction*, 81, 65-75.
- Boyatzis, R. E. (1974). The effect of alcohol consumption on the aggressive behavior of men. *Quarterly Journal of Studies on Alcohol*, 35, 959-972.
- Boyatzis, R. E. (1975). The predisposition toward alcohol-related interpersonal aggression in men. *Quarterly Journal of Studies on Alcohol*, 35, 959-972.
- Brain, P. F. (1986). Multidisciplinary examinations of the "causes" of crime: The case of the link between alcohol and violence. *Alcohol and Alcoholism*, 21, 237-240.

- Buss, A. H. (1961). *The psychology of aggression*. New York: Wiley.
- Carlsson, C., & Johansson, T. (1971). The psychological effects of propranolol in the abstinence phase of chronic alcoholics. *British Journal of Psychiatry*, *119*, 605–606.
- Cherek, D. R., Steinberg, J. L., & Manno, B. R. (1985). Effects of alcohol on human aggressive behavior. *Journal of Studies on Alcohol*, *46*, 321–328.
- Cherek, D. R., Steinberg, J. L., & Vines, R. V. (1984). Low doses of alcohol affect human aggressive responses. *Biological Psychiatry*, *19*, 263–267.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Cooper, H. M. (1989). *Integrating research: A guide for literature reviews* (2nd ed.). Beverly Hills, CA: Sage.
- Eagly, A. H., & Steffen, V. J. (1986). Gender and aggressive behavior: A meta-analytic review of the social psychological literature. *Psychological Bulletin*, *100*, 309–330.
- Fleiss, J. L. (1971). Measuring nominal scale agreement among many raters. *Psychological Bulletin*, *76*, 378–382.
- Friedman, H. (1968). Magnitude of an experimental effect and a table for its rapid estimation. *Psychological Bulletin*, *70*, 245–251.
- Gayford, J. J. (1975). Wife battering: A preliminary survey of 100 cases. *British Medical Journal*, *1*, 194–197.
- Goldberg, L. (1943). Quantitative studies on alcohol tolerance in man. *Acta Physiologica Scandinavica*, *5*(Suppl. 16), 7–128.
- Graham, K. (1980). Theories of intoxicated aggression. *Canadian Journal of the Behavioral Sciences*, *12*, 141–158.
- Graham, K., La Rocque, L., Yetman, R., Ross, T. J., & Guistra, E. (1980). Aggression and barroom environments. *Journal of Studies on Alcohol*, *41*, 277–292.
- Granville-Grossman, K. L., & Turner, P. (1966). The effect of propranolol on anxiety. *Lancet*, *1*, 788–790.
- Gustafson, R. (1984). Alcohol, frustration, and direct physical aggression: A methodological point of view. *Psychological Reports*, *55*, 959–966.
- Gustafson, R. (1985). Alcohol-related aggression: A further study of the importance of frustration. *Psychological Reports*, *57*, 683–697.
- Gustafson, R. (1986). Threat as a determinant of alcohol-related aggression. *Psychological Reports*, *58*, 287–297.
- Gustafson, R. (in press). Human physical aggression as a function of alcohol, frustration, and subjective mood. *International Journal of the Addictions*.
- Hartley, H. O. (1950). The maximum *F*-ratio as a short-cut test for heterogeneity of variance. *Biometrika*, *37*, 308–312.
- Hearold, S. (1986). A synthesis of 1043 effects of television on social behavior. In G. Comstock (Ed.), *Public communication and behavior* (Vol. 1, pp. 65–133). New York: Academic Press.
- Hedges, L. V., & Olkin, I. (1985). *Statistical methods for meta-analysis*. New York: Academic Press.
- Heermans, H. W., Jr. (1980). The effect of alcohol, arousal, and aggressive cues on human physical aggression in males. *Dissertation Abstracts International*, *41*, 3630B. (University Microfilms No. ADG81-05011, 0000)
- Holcomb, W. R., & Anderson, W. P. (1983). Alcohol and drug abuse in accused murderers. *Psychological Reports*, *52*, 159–164.
- Horton, D. (1943). The function of alcohol in primitive societies: A cross-cultural study. *Quarterly Journal of Studies on Alcohol*, *4*, 199–320.
- Hull, J. G., & Bond, C. F., Jr. (1986). Social and behavioral consequences of alcohol consumption and expectancy: A meta-analysis. *Psychological Bulletin*, *99*, 347–360.
- Jevons, C. M., & Taylor, S. P. (1985). The control of alcohol-related aggression: Redirecting the inebriate's attention to socially approved conduct. *Aggressive Behavior*, *11*, 93–101.
- Johnson, S. D., Gibson, L., & Linden, R. (1978). Alcohol and rape in Winnepeg, 1966–1975. *Journal of Studies on Alcohol*, *39*, 1887–1894.
- Kolmogorov, A. N. (1933). Sulla determinazione empirica di una legge di distribuzione [Concerning the empirical determination of a distribution law]. *Giornale dell' Istituto Italiano degli Attuari*, *4*, 83–91.
- Lang, A. R., Goeckner, D. J., Adesso, V. J., & Marlatt, G. A. (1975). Effects of alcohol on aggression in male social drinkers. *Journal of Abnormal Psychology*, *84*, 508–518.
- Langer, A. W., McCubbin, J. A., Stoney, C. M., Hutcheson, J. S., Charlton, J. D., & Obrist, P. A. (1985). Cardiopulmonary adjustments during exercise and an aversive reaction time task: Effects of beta-adrenergic blockade. *Psychophysiology*, *22*, 59–68.
- Leonard, K. E. (1984). Alcohol consumption and escalatory aggression in intoxicated and sober dyads. *Journal of Studies on Alcohol*, *45*, 75–80.
- MacAndrew, C., & Edgerton, R. B. (1969). *Drunken comportment: A social explanation*. Chicago: Aldine.
- MacDonald, J. M. (1961). *The murderer and his victim*. Springfield, IL: Charles C Thomas.
- Marlatt, G. A., & Rohsenow, D. J. (1980). Cognitive processes in alcohol use: Expectancy and the balanced placebo design. In N. K. Mellow (Ed.), *Advances in substance abuse: Behavioral and biological research* (pp. 159–199). Greenwich, CT: JAI Press.
- McClelland, D. C., Davis, W. N., Kalin, R., & Wanner, E. (1972). *The drinking man*. New York: Free Press.
- Muehlberger, C. W. (1956). Medicolegal aspects of alcohol intoxication. *Michigan State Bar Journal*, *35*, 38–42.
- Myers, T. (1982). Alcohol and violent crime re-examined: Self-reports from two subgroups of Scottish male prisoners. *British Journal of Addiction*, *77*, 399–413.
- Myerscough, R. P. (1984). The combined effects of alcohol and sleep deprivation on human aggression. *Dissertation Abstracts International*, *46*, 694B. (University Microfilms No. ADG85-08389, 8508)
- Neter, J., Wasserman, W., & Kutner, M. H. (1983). *Applied linear regression models*. Homewood, IL: Irwin.
- Pernanen, K. (1976). Alcohol and crimes of violence. In B. Kissin & H. Bergleiter (Eds.), *Biology of alcoholism* (Vol. 4, pp. 351–443). New York: Plenum Press.
- Pihl, R. O. (1983). Alcohol and aggression: A psychological perspective. In E. Gotthel, K. A. Druley, T. E. Skoloda, & H. M. Waxman (Eds.), *Alcohol, drug abuse and aggression* (pp. 292–313). Springfield, IL: Charles C Thomas.
- Pihl, R. O., & Zaccchia, C. (1986). Alcohol and aggression: A test of the affect-arousal hypothesis. *Aggressive Behavior*, *12*, 367–375.
- Ratliff, K. G. (1984). Enhancing control or losing it: Sex differences in alcohol's effect on social responses to provocation. *Dissertation Abstracts International*, *45*, 3957B. (University Microfilms No. ADG85-04773, 8506)
- Richardson, D. (1981). The effect of alcohol on male aggression toward female targets. *Motivation and Emotion*, *5*, 333–344.
- Rohsenow, D. J., & Bachorowski, J. (1984). Effects of alcohol and expectancies on verbal aggression in men and women. *Journal of Abnormal Psychology*, *93*, 418–432.
- SAS Institute (1985). *SAS user's guide: Statistics* (Version 5 ed.). Cary, NC: Author.
- Schmutte, G. T., & Taylor, S. P. (1980). Physical aggression as a function of alcohol and pain feedback. *Journal of Social Psychology*, *110*, 235–244.
- Shapiro, S. S., & Wilk, M. B. (1965). An analysis of variance test for normality. *Biometrika*, *52*, 591–611.
- Siegel, S., & MacRae, J. (1984). Environmental specificity of tolerance. *Trends in Neurosciences*, *7*, 140–143.

- Smith, R. C., Parker, E. S., & Nobel, E. P. (1975). Alcohol and affect in dyadic social interaction. *Psychosomatic Medicine*, 37, 25-40.
- Sorrells, J. M. (1977). Kids who kill. *Crime and Delinquency*, 23, 312-320.
- Steele, C. M., & Southwick, L. (1985). Alcohol and social behavior I: The psychology of drunken excess. *Journal of Personality and Social Psychology*, 48, 18-34.
- Takala, M., Pihkanen, T. A., & Markkanen, T. (1957). *The effects of distilled and brewed beverages: A physiological, neurological, and psychological study* (Publication No. 4). Helsinki, Finland: The Finnish Foundation for Alcohol Studies.
- Taylor, S. P. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality*, 35, 297-310.
- Taylor, S. P. (1983). Alcohol and human physical aggression. In E. Gottheil, K. A. Druley, T. E. Skoloda, & H. M. Waxman (Eds.), *Alcohol, drug abuse and aggression* (pp. 280-291). Springfield, IL: Charles C Thomas.
- Taylor, S. P., & Gammon, C. B. (1975). Effects of type and dose of alcohol on human physical aggression. *Journal of Personality and Social Psychology*, 32, 169-175.
- Taylor, S. P., & Gammon, C. B. (1976). Aggressive behavior of intoxicated subjects: The effect of third-party intervention. *Journal of Studies on Alcohol*, 37, 917-930.
- Taylor, S. P., Gammon, C. B., & Capasso, D. R. (1976). Aggression as a function of the interaction of alcohol and threat. *Journal of Personality and Social Psychology*, 34, 938-941.
- Taylor, S. P., & Leonard, K. E. (1983). Alcohol and human physical aggression. In R. G. Geen & E. I. Donnerstein (Eds.), *Aggression: Theoretical and empirical reviews* (Vol. 2, Issues in Research). New York: Academic Press.
- Taylor, S. P., & Sears, J. D. (1988). The effects of alcohol and persuasive social pressure on human physical aggression. *Aggressive Behavior*, 14, 237-243.
- Taylor, S. P., Vardaris, R. M., Rawtich, A. B., Gammon, C. B., Cranston, J. W., & Lubetkin, A. I. (1976). The effects of alcohol and delta-9-tetrahydrocannabinol on human physical aggression. *Aggressive Behavior*, 2, 153-161.
- Tukey, J. W. (1977). *Exploratory data analysis*. Reading, MA: Addison-Wesley.
- Walker, A. L. (1982). Effects of alcohol on aggression in female social drinkers: A balanced placebo study. *Master Abstracts International*, 21, 4285. (University Microfilms No. ADG13-20298, 0000)
- White, R. K. (1987). Expectancy and pharmacological effects of alcohol on punitiveness with attractive and unattractive children. *Dissertation Abstracts International*, 48, 893B. (University Microfilms No. ADG87-10766, 8709).
- Williams, D. T., Mehl, R., Yudofsky, S., Adams, D., & Roseman, B. (1982). The effect of propranolol on uncontrolled rage outbursts in children and adolescents with organic brain dysfunctions. *Journal of the American Academy of Child Psychiatry*, 21, 129-135.
- Zeichner, A. (1978). Alcohol and aggression: The role of behavior contingencies and instigator intent. *Dissertation Abstracts International*, 39, 4605B. (University Microfilms No. ADG05-30172, 0000)
- Zeichner, A., & Pihl, R. O. (1979). Effects of alcohol and behavior contingencies on human aggression. *Journal of Abnormal Psychology*, 88, 153-160.
- Zeichner, A., & Pihl, R. O. (1980). Effects of alcohol and instigator intent on human aggression. *Journal of Studies on Alcohol*, 43, 714-724.
- Zeichner, A., Pihl, R. O., Niaura, R., & Zacchia, C. (1982). Attentional processes in alcohol-mediated aggression. *Journal of Studies on Alcohol*, 43, 714-724.

Appendix A

Characteristics Extracted From Each Research Report

Source Characteristics

1. Year of publication
2. Publication outlet (i.e., master's thesis, doctoral dissertation, journal article, unpublished paper)

Participant Characteristics

1. Population sampled (i.e., college student, community, military)
2. Minimum and average age of subjects
3. Sex of subjects
4. Drinking history (i.e., social, heavy). Drinking history was based on self-report data provided by subjects.
5. Average number of drinks consumed per week
6. Motivation for participation (i.e., money, extra credit, course requirement)

Experiment Characteristics

1. Between- or within-subjects design
2. Type of comparison (i.e., alcohol vs. control, alcohol vs. placebo, antiplacebo vs. control, placebo vs. control)
3. Hours fasted from drugs and alcohol
4. Hours fasted from food
5. Time of day alcohol consumed (i.e., morning, 8:00 a.m.–1:00 p.m.; afternoon, 1:00–6:00 p.m.; evening, 6:00–11:00 p.m.)
6. Dose of absolute alcohol administered in g/kg units
7. Percent absolute alcohol in beverage
8. Type of alcohol (i.e., absolute alcohol, vodka, other distilled spirits, wine, or beer)
9. Time allowed for alcohol ingestion
10. Time allowed for alcohol absorption after ingestion
11. Blood alcohol level ascending or descending during aggression task
12. Laboratory study or contrived bar
13. Surveillance during alcohol consumption (i.e., subject alone, experimenter or assistant present, more than one person present)
14. Subject distracted or not distracted while consuming beverage
15. Experimenter blind or not blind to alcohol and expectancy manipulations
16. Beverage manipulation successful or unsuccessful. To check the effectiveness of beverage manipulations, researchers have asked subjects what type of beverage they received, what percentage of their drink was alcohol, how many ounces of alcohol they consumed, or how intoxicated they felt. For a study that used a placebo design, the beverage manipulation was considered successful if there was no effect for alcohol

content on the manipulation check questions. If a control condition was added, or if a balanced placebo design was used, a significant effect for expectancy was also required for the beverage manipulation to be considered successful.

17. Surveillance during aggression task (i.e., subject alone, experimenter or assistant present, more than one person present)

18. Level of provocation. Seventy-five college students (31 men and 44 women) were given brief descriptions of each of the experimental procedures and were asked to indicate on an 11-point scale how angry or provoked they would feel if they were in the study. The scale was anchored at one end by *not at all provoked* (0) and at the other end by *extremely provoked* (10). Ratings were then averaged and used as provocation values for the various experimental procedures. The ratings were, of course, sex appropriate (e.g., the average male ratings were used for studies with male subjects).

19. Main effect for provocation statistically significant or nonsignificant

20. Type of aggression (i.e., direct verbal, indirect verbal, shock or noise intensity, shock or noise duration, number of shocks or noises, money loss)

21. Nonaggressive response alternative available or unavailable

22. Number of dependent measure levels (i.e., 2, 3–5, more than 5)

23. Noxious stimulus intensity threshold. In some experiments the highest button (e.g., Button 10) was associated with the stimulus described as “definitely unpleasant,” whereas in other experiments a middle button (e.g., Button 5) was associated with the same stimulus level.

24. Number of task trials

25. Number of aggression opportunities

26. Sex of confederate

27. Confederate retaliation possible or not possible

28. Subject told that the confederate was intoxicated or sober

Primary Study Results

1. Direction of effect (i.e., positive, negative, null effect)
2. Magnitude of effect (i.e., *d* index). In this review the *d* index was used to determine the magnitude of an effect (Cohen, 1988). To compute the *d* index, the nontreatment group mean was subtracted from the treatment group mean and the result was divided by the pooled standard deviation. When means and standard deviations were not reported, but *t* tests or *F* tests with one degree of freedom in the numerator were given, *d* indices were calculated by using Friedman's (1968) formula. If *F* tests with multiple degrees of freedom in the numerator were reported, means and standard deviations were requested from the authors.

Appendix B

Studies Contributing Effects Sizes in the Main Meta-Analysis

- Alioto, J. T. (1974). The effects of expectancy of receiving either marijuana or alcohol on subsequent aggression in provoked high and low users of these drugs. *Dissertation Abstracts International*, 35, 4637B. (University Microfilms No. ADG74-27722, 0000)
- Bailey, D. S. (1987). The combined effects of alcohol and propranolol on human aggression. *Dissertation Abstracts International*, 48, B. (University Microfilms No. ADG87-19575, 8712)

- Bailey, D. S., Leonard, K. E., Cranston, J. W., & Taylor, S. P. (1983). Effects of alcohol and self-awareness on human physical aggression. *Personality and Social Psychology Bulletin*, 9, 289–295.
- Bond, A., & Lader, M. (1986). The relationship between induced behavioral aggression and mood after the consumption of two doses of alcohol. *British Journal of Addiction*, 81, 65–75.

- George, W. H., & Marlatt, G. A. (1986). The effects of alcohol and anger on interest in violence, erotica, and deviance. *Journal of Abnormal Psychology, 95*, 150-158.
- Gustafson, R. (1985a). Alcohol-related aggression: A further study of the importance of frustration. *Psychological Reports, 57*, 683-697.
- Gustafson, R. (1985b). Frustration as an important determinant of alcohol-related aggression. *Psychological Reports, 57*, 3-14.
- Gustafson, R. (1986a). Alcohol, frustration, and aggression: An experiment using the balanced placebo design. *Psychological Reports, 59*, 207-218.
- Gustafson, R. (1986b). Threat as a determinant of alcohol-related aggression. *Psychological Reports, 58*, 287-297.
- Gustafson, R. (1987). Alcohol and human physical aggression: An experiment using a "backward" balanced placebo design. *Journal of Social Behavior and Personality, 2*, 135-144.
- Gustafson, R. (1988a). Beer and male physical aggression in males. *Drug and Alcohol Dependence, 21*, 237-242.
- Gustafson, R. (1988b). *Wine and male physical aggression*. Manuscript submitted for publication.
- Gustafson, R. (in press). Human physical aggression as a function of alcohol, frustration, and subjective mood. *International Journal of the Addictions*.
- Heermans, H. W., Jr. (1980). The effect of alcohol, arousal, and aggressive cues on human physical aggression in males. *Dissertation Abstracts International, 41*, 3630B. (University Microfilms No. ADG81-05011, 0000)
- Jeavons, C. M., & Taylor, S. P. (1985). The control of alcohol-related aggression: Redirecting the inebriate's attention to socially approved conduct. *Aggressive Behavior, 11*, 93-101.
- Myerscough, R. P. (1984). The combined effects of alcohol and sleep deprivation on human aggression. *Dissertation Abstracts International, 46*, 694B. (University Microfilms No. ADG85-08389, 8508)
- Pihl, R. O., Smith, M., & Farrell, B. (1984). Alcohol and aggression in men: A comparison of brewed and distilled beverages. *Journal of Studies on Alcohol, 45*, 278-282.
- Pihl, R. O., & Zacchia, C. (1986). Alcohol and aggression: A test of the affect-arousal hypothesis. *Aggressive Behavior, 12*, 367-375.
- Pihl, R. O., Zacchia, C., & Zeichner, A. (1982). Predicting levels of aggression after alcohol intake in men social drinkers: A preliminary investigation. *Journal of Studies on Alcohol, 43*, 599-602.
- Pihl, R. O., Zeichner, A., Niaura, R., Nagy, K., & Zacchia, C. (1981). Attribution and alcohol-mediated aggression. *Journal of Abnormal Psychology, 90*, 468-475.
- Ratliff, K. G. (1984). Enhancing control or losing it: Sex differences in alcohol's effect on social responses to provocation. *Dissertation Abstracts International, 45*, 3957B. (University Microfilms No. ADG85-04773, 8506).
- Schmutte, G. T., Leonard, K. E., & Taylor, S. P. (1979). Alcohol and expectations of attack. *Psychological Reports, 45*, 163-167.
- Schmutte, G. T., & Taylor, S. P. (1980). Physical aggression as a function of alcohol and pain feedback. *Journal of Social Psychology, 110*, 235-244.
- Shuntich, R. J., & Taylor, S. P. (1972). The effects of alcohol on human physical aggression. *Journal of Experimental Research in Personality, 6*, 34-38.
- Taylor, S. P., & Gammon, C. B. (1976). Aggressive behavior of intoxicated subjects: The effect of third-party intervention. *Journal of Studies on Alcohol, 37*, 917-930.
- Taylor, S. P., Gammon, C. B., & Capasso, D. R. (1976). Aggression as a function of the interaction of alcohol and threat. *Journal of Personality and Social Psychology, 34*, 938-941.
- Taylor, S. P., Schmutte, G. T., & Leonard, K. E. (1977). Physical aggression as a function of alcohol and frustration. *Bulletin of the Psychonomic Society, 9*, 217-218.
- Taylor, S. P., Schmutte, G. T., Leonard, K. E., & Cranston, J. W. (1979). The effects of alcohol and extreme provocation on the use of a highly noxious electric shock. *Motivation and Emotion, 3*, 73-81.
- Taylor, S. P., & Sears, J. D. (1988). The effects of alcohol and persuasive social pressure on human physical aggression. *Aggressive Behavior, 14*, 237-243.
- Zeichner, A., Pihl, R. O., Niaura, R., & Zacchia, C. (1982). Attentional processes in alcohol-mediated aggression. *Journal of Studies on Alcohol, 43*, 714-724.

Received December 9, 1988

Revision received June 30, 1989

Accepted August 7, 1989 ■