EFFECTS OF ALCOHOL ON CARDIOVASCULAR REACTIVITY AND THE MEDIATION OF AGGRESSIVE BEHAVIOUR IN ADULT MEN AND WOMEN

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Abstract — Aims: Recent models have proposed several pharmacological means by which alcohol may produce heightened aggression, among them that alcohol may both hyper-arouse the reward system and diminish the threat detection system. The current study examined these hypotheses employing heart rate and blood pressure as physiological indices of arousal, examining whether arousal differed by alcohol group, and if this related to level of aggression. Methods: Participants were 32 males and 32 females, aged 18–30 years, screened for physical and psychological disorder, who competed on the Taylor aggression paradigm. The gender groups were further split into half sober, half intoxicated. Arousal was measured at baseline, post-beverage consumption, and post-aggression paradigm. Results: Participants in the sober condition initially demonstrated slight heart rate elevations and blood pressure decreases, but showed little arousal in response to the aggression paradigm, whereas sober participants demonstrated considerable arousal on both indices. Intoxicated participants were more aggressive than sober controls; men and women did not differ significantly. Regression analyses demonstrated that change in systolic blood pressure from post-beverage consumption to post-aggression paradigm acts as a mediating variable in the alcohol–aggression relationship. Conclusions: These results lend support to the stress-response dampening model of the alcohol–aggression relationship, and moreover suggest that the magnitude of intoxicated aggression is related to the magnitude of that dampening.

INTRODUCTION

One of the most important situational determinants of interpersonal aggression is alcohol intoxication. One study demonstrated that more than half of perpetrators and approximately 45% of victims of violent crime consumed alcohol prior to the offence in question (Murdoch et al., 1990). Furthermore, several meta-analyses of relevant experimental work have demonstrated unequivocally that intoxicated participants are more verbally and physically aggressive than sober controls (Bushman and Cooper, 1990; Bushman, 1993). Thus, although the manifestation of aggression is clearly multifactorial and complex (Raine, 1993; Pihl and Peterson, 1995), alcohol intoxication appears to be of considerable importance. However, the mechanisms through which alcohol elicits heightened aggression are not completely understood.

Recent models (Pihl and Peterson, 1995; Pihl and Hoaken, 2002) have suggested several distinct but not necessarily mutually exclusive pharmacological effects of alcohol that may increase the likelihood of aggressive behaviour. Included in these are the altering effects alcohol has on pain sensitivity (Gustafson, 1985; Stewart et al., 1992), and the altering effects alcohol has on certain cognitive abilities, specifically the so-called 'executive cognitive functions' (Hoaken et al., 1998; Giancola, 2000). The models also suggest that alcohol may both hyper-arouse the system that mediates reward (Conrod et al., 2001), as well as diminish the system that governs threat detection (Pihl et al., 1993).

It is widely agreed that, whatever the mechanism, low and moderate doses of alcohol clearly lead to stimulation in both animals and humans. In rats, for example, alcohol produces increased locomotion, rearing and exploration (Smoothy and Berry, 1984; Erickson and Kochhar, 1985); in humans subjective increases in power, expansiveness, euphoria and energy are reported (Pihl and Peterson, 1995; Conrod et al., 2001). Alcohol has also been shown to lead to increased heart rate in humans (Rush et al., 1989; Conrod et al., 1997). In certain populations, such as young males at high risk for alcoholism, this heart rate response is especially pronounced (Stewart et al., 1992), and sometimes manifests behaviourally as increased sensation-seeking (Finn et al., 1992), approach behaviour and dominance (Pihl and Peterson, 1995), as well as conduct disorder and antisocial personality (Pihl et al., 1990). Escalation of these sorts of behaviours following alcohol use leads to greater confrontation, greater provocation, and, as a result, greater likelihood of an aggressive or violent encounter.

Paradoxically, alcohol has also been associated with patterns of cardiovascular dampening. For example, although alcohol consumption alone has been shown to lead to increases in resting heart rate, it has, at the same time, occasioned decreases in resting blood pressure in both animals (Piano et al., 1991) and humans (Higgins et al., 1993; Rush et al., 1989).

Most of the research on the cardiovascular effects of alcohol consumption has developed out of what was first referred to as the tension-reduction hypothesis of alcohol consumption (Conger, 1951, 1956), which is more contemporarily referred to as alcohol stress-response dampening (SRD; Levenson et al., 1980). What this theory argues, essentially, is that alcohol is consumed because it is negatively reinforcing; people drink because they have learned that alcohol ameliorates the adverse effects of various psychosocial stressors. Investigations of the SRD hypothesis demonstrated that, when faced with stressful or provocative stimuli and/or situations, individuals who had consumed alcohol show a muting of typical psychophysiological stress responses (reviewed in Sayette, 1993). For example, alcohol’s dampening effect on heart rate has been shown in response to non-social stimuli, such as an aversive shock (Eisenhofer et al., 1986; Levenson et al., 1987) and a loud noise (Lehrer and Taylor, 1974), as well as stressful social interactions (Wilson et al., 1980; Sher and Walitzer, 1986). Alcohol has also been shown to enact a SRD effect on both systolic (Zeichner et al., 1985; Niaura et al., 1988) and
diastolic (Eisenhofer et al., 1986; Wilson et al., 1989) blood pressure. It should be noted that, while many studies have shown significant alcohol-induced cardiovascular SRD, this pattern of results is not always consistently demonstrated (Sayette, 1993). Interpretation of this literature is made difficult due to the inconsistency of the methodologies involved. In order to account for the idiosyncratic pattern of response to the many different methodologies, one theory postulates that alcohol diminishes stress responses not directly, but by means of appraisal disruption; that is, the more complex the paradigm intended or expected to elicit a stress response, the more likely the manifestation of alcohol SRD (Sayette, 1993).

The cardiovascular SRD effect is relevant to the alcohol–aggression relationship inasmuch as threat is typically seen as inhibiting aggressive responses (Ito et al., 1996). Specifically, those who have reason to fear that their aggression will be punished are less likely to engage in aggressive acts. Threat without the inhibiting effect of alcohol has been associated with large and consistent increases in blood pressure and heart rate (Holmes and Will, 1985; Gerin et al., 1992; Sinha et al., 1992; Farrington, 1997). These physiological responses can be seen as ‘reminders’ of the socialization process; arousal means threat, and threat means punishment. Thus, fear should adaptively inhibit the types of behaviours that might initiate an aggressive interaction. However, alcohol dampening of the threat-detection system means that the threat-induced inhibition of aggressive responding is itself inhibited, and socially and interpersonally inappropriate behaviours are more likely to be manifested.

In summary, alcohol’s psychomotor stimulant effects may lead to increased sensation-seeking and approach behaviour — clearly behaviours which carry an element of danger. However, at the same time, alcohol’s dampening effects on stress reactivity may attenuate the magnitude of the inhibitory effect that anxiety or fear normally would exercise over the expression of potentially dangerous behaviours. As anxiety cues are reduced, individuals may be more likely to engage in behaviour that has been previously associated with punishment or threat. Both of these effects can be seen to theoretically predispose individuals to alcohol-elicted increases in aggressive behaviour. Although there is much indirect evidence to support these contentions, direct evidence remains scarce.

Furthermore, while there is an abundance of evidence supporting a positive relationship between alcohol and aggression in men, relatively few studies have been performed with women, most of them recently. Although it has generally been argued that men are more aggressive than women (Bettencourt and Miller, 1996), there are studies which do not support this contention of gender differences (Archer, 2000; Hoaken and Pihl, 2000; Giancola et al., 2002). Moreover there has been considerable controversy about the conditions under which the main effect of gender on aggression holds (Eagly and Steffen, 1986). Although there is a paucity of research comparing autonomic dampening responses to alcohol among women, there is some evidence that the genders respond similarly (Stewart and Pihl, 1994). Due to the disagreement regarding the aggression-eliciting effects of alcohol in women, and the paucity of data investigating biological mediating factors, this investigation tested and compared male and female participants.

The current study intended to examine the effects of alcohol on cardiovascular indices of the reward and threat system, namely heart rate and both systolic and diastolic blood pressure, relative to non-intoxicated controls. Furthermore, it intended to investigate whether the patterns of arousal manifested in response to alcohol change when there is an alcohol–provocation interaction; specifically, intoxicated and sober participants will compete on the Taylor (1967) aggression paradigm in order to assess the interactive effects of alcohol and this activity on cardiovascular indices of arousal. The study employed a $2 \times 3$ mixed-model with beverage condition (alcohol vs no alcohol) as a between-subjects factor, and phase (pre-drink baseline, post-drink baseline and post-provocation) as a within-subjects factor.

Consistent with previous studies, it was hypothesized that intoxicated participants would demonstrate slightly increased resting heart rate and slightly decreased resting blood pressure in response to an intoxicating dose of alcohol (Rush et al., 1989; Bruce et al., 1999). In terms of response to provocation and aggression, it was hypothesized that the intoxicated participants would manifest greater aggression, but would demonstrate dampened heart rate and blood pressure reactivity to provocation relative to sober controls. Finally, although results of recent studies with similar procedures have demonstrated inconsistent results in terms of gender differences (e.g. Giancola and Zeichner, 1995; Hoaken and Pihl, 2000), men were expected to be more aggressive than women in their responses, regardless of intoxication.

**SUBJECTS AND METHODS**

**Subjects**

A total of 64 non-alcoholic social drinkers (32 men, 32 women) between the ages of 18 and 30 years, in good physical and mental health, were recruited through local newspaper and campus advertisements. The criteria for non-alcoholic status were a score of 5 or less on the Michigan Alcoholism Screening Test (MAST; Pokorny et al., 1972) and a brief clinical interview based on DSM-IV criteria regarding previous or present drug or alcohol-related problems. Participants who reported medical treatment that contraindicated alcohol consumption, any current or prior cardiovascular condition, a daily consumption of 25 or more cigarettes, a history of serious head injury, a diagnosed learning disability, pregnancy, or familiarity with psychological experimentation were excluded from participation. In order to control for fluctuations in aggressive behaviour and alcohol effects that may be associated with phase of the menstrual cycle, female participants were tested during the follicular phase of their menstrual cycle (days 5–10 of cycle), as estimated by self-reports of the length of their three previous menstrual periods (Sutker et al., 1987). All participants were requested to fast for 4 h prior to the testing session. Participants were compensated at a rate of $5 per hour.

**Measures and apparatus**

Aggression was elicited and assessed with a modified version of the Taylor (1967) competitive reaction-time task. In this study, the task board consisted of eight buttons, numbered consecutively from one to eight. Red lights situated above
each button lit up to indicate the shock level chosen by the opponent. An IBM-compatible personal computer was used to run the aggression task and to record data. Shocks were administered via the Mark I Behaviour Modifier (Farrall Instruments, Grand Islands, NB, Canada), connected to an electrode attached to the inner forearm, below the elbow of the non-dominant hand. Each participant monitored administrations of shocks to their fictitious opponent by viewing a DC ammeter provided for that purpose. A pre-recorded videotape of a same-sex sham opponent receiving instructions regarding performance on the aggression task was played to the participant to reinforce the participant’s belief in the existence of the opponent.

Dependent cardiovascular measures were heart rate (in beats per minute) and systolic and diastolic blood pressure (in mmHg); and measurements were recorded using a portable Sunbeam digital monitor (Model 7621). The Sunbeam monitor uses the oscillometric principle for determining brachial pressure. According to the manufacturer, it is accurate to within ±3 mmHg for blood pressure. This monitor received the highest rating for accuracy in a test of 15 commercially available models (Consumers Union, 1992). Each determination requires approximately 30 s. The blood pressure cuff was placed on the participant’s non-dominant arm, above the elbow. Blood pressure and heart rate values were recorded as the average of three seated readings taken at each of the three measurement periods. The first measurement period occurred soon after the participant arrived at the laboratory, prior to the consumption of the alcohol or orange juice, but subsequent to a seated rest period during which informed consent was obtained (pre-beverage baseline). The second measurement period occurred 40 min following the first measurement period, subsequent to the consumption of the beverage, and before commencing the aggression paradigm (post-beverage baseline). The third measurement was immediately following the aggression paradigm (post-provocation) and was considered a proxy for reactivity to the provocation.

Procedure

Upon arriving at the laboratory, the participant’s blood-alcohol concentration (BAC) was measured to ensure sobriety; this was conducted using an Alco-Sensor III breathalyser (Intoximeters Inc., St Louis, MO, USA). Participants were randomly assigned to the alcohol or no alcohol condition. In the alcohol condition, participants were administered 1 ml per kg of body weight of 95% alcohol USP units in three drinks of a 1:7 alcohol:orange juice solution. In the sober condition, the participants were administered three drinks of orange juice of equivalent volume to that in the alcohol condition, based on their body weight. In each condition, participants were told explicitly what they were drinking. Drinks were consumed at a constant rate over a 20-min period. A 20-min absorption period followed to allow the participants in the alcohol condition time to reach near peak BAC. BACs were then assessed with the Alco-Sensor III and recorded.

Each participant’s pain threshold for electric shock was determined by delivering a series of shocks from 0 to 255 units (0–5.63 ma) which increased stepwise by 5 units at a constant rate. Each participant was instructed to press a button in response to any shock s/he regarded as painful: (1) to stop the administration of the shock; (2) to reduce the level of the next shock by one step. The next shock therefore was one step lower than the shock that induced pressing the button. Pressing the button upon three consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the participant’s pain threshold.

The aggression task was then introduced as a competitive reaction-time task. Each participant was instructed to select a shock level that s/he would deliver to his/her opponent after winning a reaction-time trial. Following the reaction time task, the participant would be informed of the opponent’s shock choice. If the participant ‘lost’ that trial, s/he received that shock. Shock levels 1–8 increased from 28 units for level 1 to 100% of the participant’s given pain threshold for level 8, with intermediate shock levels being equal to 28 units plus 23, 31, 39, 76, 84 and 92% of the difference between the participant’s given pain threshold and the initial 28 unit level. The nature of the increases of the shock intensity was decided upon in order to define clearly those shocks thought to be minimally provoking (level 1–4) and those thought to be maximally provoking (levels 5–8). If the participant had ‘won’ the reaction time trial, s/he would then administer the previously chosen shock to his/her opponent.

Following the instructions, the experimenter then left the room briefly, telling each participant that s/he was about to verify the readiness of the opponent. Upon his return, the experimenter stated that instructions were about to be delivered to the opponent, and that this delivery could be viewed on the TV monitor as a review of the instructions. As noted earlier, the participant was actually watching a pre-recorded videotape of a fictitious opponent receiving instructions.

The task itself consisted of 26 consecutive trials, including a block of 12 trials followed by a transition trial, a second block of 12 trials, and a final trial. The opponent’s shock choices ranged from 1 to 4 in the first block and 5 to 8 in the second block of trials. The computer randomly assigned the order of wins and losses as well as the opponent’s shock choices. The opponent’s shocks were all of either 1- or 2-s duration. All participants received three shocks at each level, alternately winning one trial and losing two trials versus winning two trials and losing one trial. If the participant was to receive two shocks at a certain level, they would receive one each of 1- and 2-s duration. In both the transition trial and the final trial the opponent’s shock choice was a 5. The objective measure of aggression was the mean shock selected for both the high and low provocation conditions. This measure reflects the magnitude of an individual’s aggressive response to both low and high provocation.

Following the task and assessment of heart rate and blood pressure, participants were given a questionnaire to assess their perception of the opponent, as well as their subjective rating of their aggression level to probe the completeness of the deception. The participants were then debriefed. Debriefing included an explanation of the deception, its necessity, and a request to refrain from revealing the deception to others. Participants were compensated for their time, and if in the sober condition, were allowed to leave. Participants in the alcohol condition were retained in the laboratory until their BACs dropped to 0.02% (20 mg/dl).
RESULTS

Subject measures

A total of 64 participants were deemed admissible to participate in the study and were tested on the Taylor paradigm. Participants were divided into four groups: Group 1 consisted of males who consumed alcohol \((n = 16)\); Group 2 consisted of sober males \((n = 16)\); Group 3 consisted of females who consumed alcohol \((n = 16)\); and Group 4 consisted of sober females \((n = 16)\). Analysis of variance (ANOVA) conducted on demographic and associated variables indicated no differences between the alcohol and no-alcohol groups in terms of age, years of education, consumption of alcoholic beverages per week, or cigarettes per day. An ANOVA was also conducted on BACs (Table 1). There were no significant differences between post-beverage and post-provocation measures. There was also no gender difference, either at post-beverage or post-provocation.

Aggression measure

A \(2 \times 2 \times 2\) mixed-design ANOVA was conducted on mean shock selections, with provocation as a repeated measure and gender and alcohol condition as between-participant factors (Fig. 1). There was no significant three-way interaction, nor were any of the three-two-way interactions significant. There was a significant main effect of provocation \([F(1,58) = 62.24, P < 0.001]\). There was also a significant main effect of alcohol condition \([F(1,58) = 6.71, P < 0.05]\). There was no main effect of gender \([F(1,58) = 2.34, P = 0.13]\). However, analysis of simple main effects allowed better understanding of the relationship between variables.

Effects of alcohol within gender factor. There was a simple main effect of alcohol in men in both low provocation \([F(1,28) = 4.58, P < 0.05]\) and high provocation conditions \([F(1,28) = 4.65, P < 0.05]\), with intoxicated men demonstrating greater aggression than sober men. Conversely, there was no simple main effect of alcohol in women, at either the low \([F(1,28) = 1.59, P = 0.22]\) or high provocation conditions \([F(1,28) = 1.07, P = 0.31]\).

Effects of gender within alcohol factor. In the sober condition, there was no significant simple main effect of gender in either the low or high provocation condition; that is, men’s and women’s aggressive responses were no different when sober. In the alcohol condition, there was also no significant main effects of gender at low provocation; however, at high provocation there was a trend, demonstrating intoxicated men to be more aggressive than intoxicated women \([F(1,28) = 3.10, P < 0.10]\).

Physiological measurements

Baseline measures. A multivariate ANOVA was conducted on the four groups in terms of the heart rate, systolic blood pressure and diastolic blood pressure measurements at baseline shown in Table 2. As expected, there was a significant effect of gender on both systolic \([F(1,60) = 55.58, P < 0.001]\) and diastolic \([F(1,60) = 5.07, P < 0.05]\) blood pressure (men as a group have greater blood pressures due to greater body mass). There was no significant difference between any of the groups in terms of heart rate. All subsequent analyses were conducted on mean change from baseline; due to the significant gender differences, graphical representations of the data will be split by gender, but all analyses will continue to include gender as a factor.

Heart rate changes due to alcohol and provocation. As can be observed in Fig. 2, differences were observed when comparing heart rate measured post-beverage with heart rate measured at pre-beverage baseline, and when comparing heart rate measured post-provocation with heart rate measured post-beverage. (It should be noted that pre-drink baseline change scores of zero have been included in all figures to improve interpretation.) In order to test this, we conducted a \(2 \times 2 \times 2\) mixed design ANOVA, with heart rate measurement (post-beverage and post-provocation) as a repeated measure, and gender and alcohol conditions as between-participants factors. The three-way interaction was not significant. The interaction between gender and measurement time was not significant, nor was the interaction between gender and alcohol. However, there was a significant interaction between alcohol and measurement time \([F(1,60) = 5.87, P < 0.05]\). There was no significant effect of gender in terms of either change from pre-beverage baseline to post-beverage baseline or post-beverage baseline to post-provocation. There was a significant effect of alcohol from pre-beverage baseline to post-beverage baseline \([F(1,62) = 6.38, P < 0.05]\) with the alcohol groups demonstrating significant increases in heart rate relative to the no alcohol groups. There was also a significant effect of alcohol from post-beverage baseline to post-provocation.
Table 2. Pre-drinking baseline measures of systolic blood pressure, diastolic blood pressure and heart rate as a function of gender and beverage condition

<table>
<thead>
<tr>
<th>Gender and test condition</th>
<th>Systolic blood pressure (mmHg)</th>
<th>Diastolic blood pressure (mmHg)</th>
<th>Heart rate (beats per minute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men/no alcohol</td>
<td>119.75 (7.41)</td>
<td>69.31 (8.69)</td>
<td>67.81 (10.86)</td>
</tr>
<tr>
<td>Men/alcohol</td>
<td>123.37 (9.66)</td>
<td>68.37 (7.57)</td>
<td>69.12 (8.32)</td>
</tr>
<tr>
<td>Women/no alcohol</td>
<td>102.68 (9.97)</td>
<td>63.12 (7.65)</td>
<td>71.25 (12.30)</td>
</tr>
<tr>
<td>Women/alcohol</td>
<td>105.68 (9.98)</td>
<td>65.50 (8.21)</td>
<td>69.93 (9.15)</td>
</tr>
</tbody>
</table>

Values are means ± SD for 16 subjects per group.

\[ F(1,62) = 6.65, P < 0.05 \] with non-intoxicated groups demonstrating much greater heart rate increases to provocation than intoxicated groups.

**Systolic blood pressure.** As can be observed in Fig. 3, little differences were observed when comparing systolic blood pressure measured at pre-beverage baseline to that measured at post-beverage baseline; however, there was noticeable change in systolic blood pressure from post-beverage baseline to post-provocation. In order to test this, we conducted a \( 2 \times 2 \times 2 \) mixed design ANOVA, with systolic blood pressure measurement (post-beverage baseline and post-provocation) as a repeated measure, and gender and alcohol conditions as between-participants factors. The three-way interaction was not significant. The interaction between gender and measurement time was not significant, nor was the interaction between gender and alcohol. The interaction between alcohol and measurement time just failed to reach significance \( [F(1,60) = 3.89, P = 0.053] \). There was, however, an overall main effect of alcohol \( [F(1,60) = 21.26, P < 0.001] \).

There was no significant effect of gender in terms of either change from pre-beverage baseline to post-beverage baseline or post-beverage baseline to post-provocation. There was no significant effect of alcohol from pre-beverage baseline to post-beverage baseline; however, there was a significant effect of alcohol from post-beverage baseline to post-provocation, with the no-alcohol groups demonstrating \( [F(1,62) = 19.07, P < 0.001] \) significantly greater systolic blood pressure elevations than the alcohol groups during this period (Fig. 3).

**Diastolic blood pressure.** As can be observed in Fig. 4, little differences were observed when comparing diastolic blood pressure measured at pre-drink baseline to that measured post-beverage baseline; again, however, there was noticeable change in diastolic blood pressure from post-beverage baseline to post-provocation. In order to test this, a \( 2 \times 2 \times 2 \) mixed design ANOVA was conducted, with diastolic blood pressure measurement (post-beverage baseline and post-provocation) as a repeated measure, and gender and alcohol conditions as between-participants factors. The three-way interaction was not significant. The interaction between gender and measurement time was not significant, nor was the interaction between gender and alcohol. The interaction between alcohol and measurement time just failed to reach significance \( [F(1,60) = 2.39, P = 0.128] \). There was, however, an overall main effect of alcohol \( [F(1,60) = 6.04, P = 0.018] \).
mixed design ANOVA was conducted, with diastolic blood pressure measurement (post-beverage baseline and post-provocation) as a repeated measure, and gender and alcohol conditions as between-participants factors. The three-way interaction was not significant. The interaction between alcohol and measurement time was not significant, nor was the interaction between gender and alcohol. The interaction between gender and measurement time approached significance \( [F(1,60) = 3.57, P = 0.063] \). There was an overall main effect of alcohol \( [F(1,60) = 13.34, P < 0.005] \) and measurement time \( [F(1,60) = 14.15, P < 0.001] \).

There was a trend for gender in terms of change from pre-beverage baseline to post-beverage baseline \( [F(1,62) = 3.65, P = 0.061] \), with women demonstrating greater diastolic blood pressure decreases in response to alcohol than men. There was no gender effect in terms of post-beverage baseline to post-provocation measurement of diastolic blood pressure. There was no significant effect of alcohol from pre-beverage baseline to post-beverage baseline; however, there was a significant effect of alcohol from post-beverage baseline to post-provocation, with the no-alcohol groups demonstrating \( [F(1,62) = 6.02, P < 0.05] \) significantly greater diastolic blood pressure elevations than the alcohol groups during this period.

**Relationship between physiological response and aggression**

Regression analyses were conducted to determine the extent to which alcohol and situational factors interacted to affect the physiological indices, and the extent to which this physiological change accounted for variance in aggression manifested. Because there were no significant gender effects, groups were collapsed, and regression coefficients are therefore based on all participants. Two stepwise regressions were conducted, the first on mean shock selected in the low provocation condition; the second on mean shock selected in the high provocation condition. Six predictive variables were included in the stepwise regression. They were: heart rate change from pre-drink baseline to post-beverage baseline; heart rate change from post-beverage baseline to post-provocation; systolic blood pressure change for those same two periods; and diastolic blood pressure change for those same two periods. The first stepwise regression (low provocation) failed to find any one factor which accounted for a greater percentage of the variance than any other. However, the second stepwise regression, for mean shock selected at high provocation, entered one factor, change in systolic blood pressure from post-beverage to post-provocation \( [F(1,60) = 5.91, P = 0.018, R^2 = 0.090] \). The standardized \( \beta \) coefficient = –0.299, indicating that the lesser the blood pressure reactivity, the greater the aggression manifested in the high provocation condition.

**Analysis of mediation**

The above analyses suggest that physiological change, specifically changes in systolic blood pressure from post-beverage to post-provocation, is related to level of aggression manifested. The next step was to examine statistically the possibility that this physiological change might mediate the relationship between alcohol and aggression at high provocation. In order to appropriately test this hypothesis, a series of regression analyses were conducted in accordance with the guidelines detailed by Baron and Kenny (1986).

As can been seen in Fig. 5, the relationship between alcohol and aggression (represented by a standardized \( \beta \) coefficient) is considerably diminished when the third variable, change in systolic blood pressure from post-beverage to post-provocation, is controlled for. Baron and Kenny (1986) noted that any degree of reduction in the relationship between the criterion and the predictor is evidence of partial mediation;

![Change in Systolic Blood Pressure (Post-Beverage to Post-Provocation) (Mediator)](image)

![Alcohol Condition (Independent Variable)](image)

![Aggression at High Provocation (Dependent Variable)](image)

*Fig. 5. Regression analyses depicting the mediating role of change in systolic blood pressure from post-beverage to post-provocation on the alcohol–aggression relationship.*

Coefficients are standardized \( \beta \)s in a series of multiple regression analyses. Standardized \( \beta \)s in parentheses represent the relation between two variables after controlling for the influence of the third.
However, objective rules to what amount of reduction is theoretically noteworthy are not readily available. Two recent studies in the alcohol literature (Kushner et al., 2001; MacPherson et al., 2001) recommended two objective criteria: first, that the relation between the predictor and the criterion loses statistical significance when the mediator is controlled for, and secondly, that the magnitude of the relationship between the predictor and the criterion is diminished by at least 30% when the mediator is controlled for.

Our mediation analyses met both objective criteria. The relationship between alcohol and aggression was statistically significant when systolic blood pressure change was not controlled for ($\beta = 0.267, P > 0.05$), but was not statistically significant when blood pressure change was controlled for ($\beta = 0.158, P = 0.266$). A calculation of change in standardized $\beta$ coefficients demonstrated that controlling for systolic blood pressure change post-beverage to post-provocation produced a 40.8% decrease in the magnitude of the relationship between alcohol and aggression. Consequently, there appears to be some statistical evidence that individually specific physiological change in response to alcohol partially mediates the alcohol–aggression relationship.

**DISCUSSION**

This study is consequential inasmuch as it replicates previous studies regarding the alcohol–aggression relationship, but, moreover, because it provides direct support for a SRD model of that relationship. Moreover, the study contributes to a growing literature that suggests gender differences in alcohol-related aggression are not as great as once thought (Bettencourt and Miller, 1996). These results will be discussed in turn.

The results of this study, first of all, replicate literally dozens of others that demonstrate that, in between-group studies, an alcohol manipulation will make intoxicated participants act in a more aggressive fashion than their sober controls (Bushman, 1993). This is no longer at issue. However, what is still largely not agreed upon are the mediating variables that help explain this relationship, and a clear consensus on differential gender effects.

The demonstration that alcohol appears to attenuate cardiovascular responses to threat in men and women is consistent with existing findings (Sayette, 1993). However, that alcohol’s dampening of cardiovascular response to threat can be so directly related to the likelihood of aggressive response to provocation appears a novel finding. This result sheds light on the issue of why some people react aggressively when alcohol-intoxicated and others do not. The results of this study suggest that it may not simply be alcohol administration, but instead the individually specific susceptibility to alcohol’s SRD effects (Sher and Walitzer, 1986), that accounts for aggression. That is, at least in the high provocation condition, regression analysis appears to suggest that, although intoxicated, some participants still manifested a substantial stress response to the provocation, and act relatively non-aggressively, whereas those who manifest more pronounced alcohol-induced SRD are more likely to respond aggressively. Analyses consistent with those prescribed by Baron and Kenny (1986) suggest that systolic blood pressure change from post-beverage to post-provocation acts as a mediating variable for the alcohol–aggression relationship.

The fact that heart rate responses to alcohol and high provocation do not predict aggression, whereas blood pressure results do, might be considered an inconsistent finding, as both are generally considered sympathetic nervous system responses (Mezzacappa et al., 1996). However, dissociation of these two measures has been demonstrated in response to different types of stress and/or provocation, such as neuropsychological (Higgins et al., 1993) and intelligence test performance (Zeichner et al., 1985).

In terms of gender differences, while the intoxicated women in this study did appear to demonstrate slight increases in aggression in response to alcohol, these increases were not significant at either the low- or high-provocation condition. In contrast, alcohol significantly increased aggression in the men at both low and high provocation. The aggression of the intoxicated men was not significantly greater than intoxicated women at low provocation, but this gender difference approached significance at high provocation. These results, while not entirely consistent with previous studies, do correspond with recent studies that suggest that alcohol intoxication does not elicit as pronounced increases in aggression in women as it does in men (Hoaken and Phl, 2000; Giancola et al., 2002). The present results are also consistent with recent results showing that, if provoked sufficiently, women’s aggression will not always be less than that of men (Eagly and Steffen, 1986; Giancola et al., 2002). The fact that women experienced the same pattern of cardiovascular reactivity as men in both the alcohol and sober conditions but did not exhibit as pronounced a pattern of aggressive responding, may be related to socialization practices (Bandura, 1973).

A limitation of this study was that cardiovascular responses were not measured during the aggression task. It is possible that measures taken following the aggression paradigm are not representative of cardiovascular reactivity during the task, but, instead, represent recovery from stress. Future research should endeavour to measure arousal during the actual paradigm. It would also have been beneficial to assess individual differences in anxiety and hostility levels, factors known to influence significantly cardiovascular responding to interpersonal stress (Jorgensen et al., 1996). It might also prove beneficial to ask participants about their history of exposure to stressful events. Lastly, the extent to which alcohol dampened cardiovascular reactivity directly, or via interference in some appraisal capacity (e.g., Sayette, 1993), was not investigated. This also merits further investigation.

The present study might also be of interest in the context of contemporary efforts to understand the complex genetic antecedents and neurobiological concomitants of both alcoholism and aggression. Genetic research has long demonstrated a relationship between risk for alcoholism and likelihood of aggression (Goldman, 1993, 1996). For example, a subset of alcoholics identified to have altered serotonergic function have also been shown to frequently manifest impulsive and aggressive behaviours (Linnola et al., 1983); these behaviours were also demonstrably manifested by the adopted-out children of these individuals (Bohman, 1978; Bohman et al., 1987). This link is important to the present finding inasmuch as the serotonergic system has long been
thought to be responsible for the inhibition of behaviour in the presence of threat or cues for punishment (Soubrie, 1986). While most contemporary researchers would hold this contention to be simplistic, animal research continues to implicate different serotonin sub-receptor systems in differential responses to environmental stress (e.g. Neumaier et al., 2002). (See Pihl and Lemaquand, 1998, for a review of the complex relationship between the serotonergic system, alcohol and aggression.)

While a comprehensive discussion of neurobiology and/or genetics is beyond the scope of this paper, further directions for future research are apparent. For example, genetic research might attempt to link genotypes for aberrant serotonergic function to differential patterns of alcohol-related SRD. In addition, while tryptophan depletion studies have been conducted to help understand the relationship between function of the serotonergic system, alcohol and aggression (Pihl et al., 1995), no effort has been made to concurrently investigate psychophysiological arousal. Researchers might also profitably apply differential patterns of cardiovascular dampening and concomitant aggressivity to existing efforts at typology. For example, while Cloninger’s (1987) effort to distinguish Type I from Type II alcoholics has been criticized on the basis of poor reliability and validity (Glenn and Nixon, 1996), it is possible that differential psychophysiological SRD might prove a factor that reliably differentiates individuals theoretically thought to be more (Type II) versus less (Type I) likely to manifest aggression.

The current study presents findings that suggest the SRD effects of alcohol have different implications on physical aggression for men and women. While the cardiovascular responses between the sexes were equivalent, their pattern of aggression following provocation was not. The results may best be understood by considering the different socialization histories of men and women and the resulting experience they receive with different forms of aggression. Many approaches to the etiology of sex differences tend to consider such differences to be a product of learning, not heredity. In fact, it should be noted that most researchers do not propose that inherited biologically based sex differences account for all male–female differences in aggression. Instead, they generally propose that biological differences establish different back-grounds in the two sexes, against which environmental and situational forces operate.

In conclusion, irrespective of the gender differences, this study offers direct support for the arousal-dampening/ anxiolytic models of the alcohol–aggression relationship. Intoxicated participants, as a whole, responded more aggressi- vely than did their sober peers. They also demonstrated patterns of physiological arousal indicative of physiological arousal in response to alcohol alone, and patterns indicative of SRD when intoxicated and faced with a provocative situation. Perhaps most notably, this study appears to suggest that individually specific physiological changes in response to alcohol may at least partially mediate the highly complex relationship between alcohol and aggression.

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