Alcohol and Stress Response Dampening: Pharmacological Effects, Expectancy, and Tension Reduction

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Alcohol consumption and alcohol expectation were separately evaluated in terms of effects on psychophysiological levels prior to stress and reduction of the magnitude of response to stress. Ninety-six male, experienced drinkers were assigned to eight conditions in a between-subjects design in which beverage consumed (alcohol or tonic), beverage expected (alcohol or tonic), and stressor (self-disclosing speech or threat of shock) were manipulated. Dosage for subjects receiving alcohol was 1 g ethanol/kg body weight. Results indicated strong effects of alcohol consumption on pre-stress levels, consisting of accelerated heart rate (HR), lower HR variability, higher skin conductance, longer pulse transmission time (PTT), higher "cheerfulness" and lower "anxiety" (ANX). This pattern of effects is related to previous unsuccessful attempts to specify a simple relationship between alcohol consumption and "tension." In addition, alcohol consumption significantly reduced the magnitude of the HR, PTT, and ANX responses of subjects to the stressors. No effects attributable to alcohol expectation were found. These results are integrated with the existing literature concerned with pharmacological and cognitive effects of alcohol as they pertain to stress, psychophysiological responses to stress, and "tension reduction."

The relationship between alcohol and stress has long been a concern in the alcohol research literature. It is ironic that after extensive study, little consensus exists as to the nature of this relationship. Similarly, it is not uncommon for social drinkers to assert that alcohol has a beneficial action vis-à-vis stress without being able to precisely formulate the nature of this action.

Effects of Alcohol on Response to Stress

A number of studies have shown that alcohol reduces the magnitude of the physiological response to stressful stimuli. This reduction in response magnitude has been demonstrated for the electrodermal response to loud tones (Carpenter, 1957; Greenberg & Carpenter, 1957), the electrodermal response to verbal stimuli (Coopersmith, 1964; Liebert & Traxel, 1959), and the cardiac response to loud tones (Lehrer & Taylor, 1974). It should be noted that these studies used simple laboratory stressors such as tones and high affect words. Studies that used "real-life" stressors and included measures of affective responses will be discussed later when the role of cognitive mediators of the effects of alcohol is addressed.

Effects of Alcohol on Resting Levels

Physiological effects of alcohol (during the ascending limb of absorption) have been shown to include heart rate (HR) acceleration (Dengerink & Fagan, 1978; Naitoh, 1972), reduction of forehead muscle tension...
in skin conductance level (Jones, Parsons, & Steffen, 1974), increase in cardiac contractility to expect naive subjects to show agreeable tension, decreased contractility) properties. Because of this complexity it seems unrealistic to expect naïve subjects to show agreement in the subjective labeling of this state based on induced physiological changes. Indicative of this are reports of alcohol's increasing self-reported anxiety (Dengerink & Fagan, 1978; McNamara, Mello, & Mendelson, 1968; Mendelson, LaDou, & Solomon, 1964; Steffen, Nathan, & Taylor, 1974) and contradictory reports of alcohol's decreasing self-reported anxiety (Polivy, Schueneman, & Carlson, 1976; Warren & Raynes, 1972; Williams, 1966). On the basis of these results, attempts to view effects of alcohol on resting psychological and physiological levels in simple, or unidimensional, terms seem ill advised.

Tension Reduction

The "tension reduction hypothesis" (Cronger, 1951, 1956) is a model for relating alcohol and stress that has generated much controversy (e.g., Cappell & Herman, 1972). The hypothesis presumes a drive reduction model, which requires that the organism be in some high drive state (e.g., "tension") and emit the response of consuming alcohol. The response is then reinforced by virtue of its ability to reduce the drive state. We find application of this model to typical experimental paradigms in human research to be problematic. Few experiments on alcohol and stress, for example, have first induced a verified state of high tension, then had subjects consume alcohol, and then measured changes in the state of tension. More typical procedures are to have subjects consume alcohol in an uncontrolled state of tension and then measure changes in affective and physiological levels or to have subjects consume alcohol and then introduce a stressful stimulus to determine whether normal responses to stress are altered. Additionally, there are special problems incurred when "tension" is operationalized in terms of one or two physiological measures; in this case there does not exist any simple physiological index of "tension" that holds across individual and situations.

Cognitive Mediators: Expectation Effects

Owing to its long history of use by the general public, alcohol has become associated with a set of beliefs and expectations concerning its effects. Since these expectations may be independent of the actual pharmacological effects of alcohol and may be evoked merely by the belief that alcohol is being consumed, it has been important to control for subjects' expectations in alcohol research. Marlatt, Demming, and Reid (1973) introduced an appropriate four-cell design for separating the effects of consuming alcohol from the effects of believing alcohol has been consumed (i.e., expectation effects). A number of subsequent studies applied this design to the examination of behavior associated with alcohol, with the result that behaviors such as increased aggression (Lang, Goeckner, Adesso, & Marlett, 1975) and increased sexual arousal in males (Wilson & Lawson, 1976) were found to be associated with the belief that alcohol was being consumed, and not with the consumption of alcohol per se.

Application of this design to the alcohol-stress relationship has also been undertaken. Polivy et al. (1976) found that expectation of alcohol resulted in higher levels of self-reported anxiety in anticipation of stress. In contrast, alcohol consumption resulted in lower levels of anxiety. Wilson and Abrams (1977) examined the effects of alcohol and expectation on the responses of males to interacting with a female confederate. The typical drinking pattern and physiological response measures to stress were used to determine whether normal reactions to stress are altered. Additionally, there are special problems incurred when "tension" is operationalized in terms of one or two physiological measures; in this case there does not exist any simple physiological index of "tension" that holds across individual and situations.

Subjects

Ninety-six male students were chosen from a group of volunteers who responded to an advertisement in the campus newspaper of Indiana University offering payment for participation in an experiment involving alcohol and stress. Subjects were chosen on the basis of a proportional voltage. The system employed was an electronic sphygmomanometer, and blood alcohol concentration (BAC) was assessed using a Smith and Wesson Model 900 Breathalyzer.

Apparatus

Physiological. Data were obtained for a number of physiological variables using a system designed for analysis of physiological interactions. A two-dimensional coordinate system and a PDP 11/03 minicomputer. The system enabled detection and averaging of physiological data during the course of the experiment as well as printing and storage of these data for subsequent analysis. Using this system, the following data were obtained: (a) Heart rate interbeat interval (IBI)—the electrocardiogram was detected using miniature surface electrodes placed on opposite sides of the chest; (b) Respiration rate (interval (IC)—a thermistor clipped to the inner surface of the nostril responding to the temperature difference between inhaled and exhaled air provided the respiratory signal; the computer timed the interval between successive breaths in msec (HR = 60.0001IBI in msec), (c) Photoplethysmographic devices attached to the pinna of the ear and the middle finger were used to determine the interval between the R-wave of the electrocardiogram and the arrival of the pulse wave at the ear (E-FTP) and at the finger (F-FTP). Changes in these transmission times reflect changes in cardiac contractility and/or blood pressure (Newlin & Levenson, 1979). In addition to these on-line measures, manual determination of systolic blood pressure was accomplished using an electronic sphygmomanometer, and blood alcohol concentration (BAC) was assessed using a Smith and Wesson Model 900 Breathalyzer.

Nonphysiological. A continuous self-report of anxiety (ANX) prior to the administration of the experiment through the use of an "anxiety dial" modeled after one used by Blankstein, Fliner, and Constantine but modified so that instead of a dial pointer in reference to a 10-point scale anchored by the legends "extremely calm" and "extremely tense," the dial was utilized to assign a proportional voltage. Using a simple calibration from driving themselves to the experiment (taxi service was arranged), and that these prestressor differences were maintained throughout the experiment.

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Alcohol and Stress

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Testing of experimental hypotheses was generally accomplished using planned comparisons by t-test. As most comparisons involved testing means from interactions including both-between-groups and within-subject factors, a pooled error term was calculated following the procedure presented by Kirk (1968). To avoid problems associated with determination of the exact number of degrees of freedom associated with this pooled term, a most conservative procedure was adopted in which the number of degrees of freedom for the comparisons was smaller than the two associated with the error terms contributing to the pooled error.

Group Differences Prior to Drking

There were no differences across the eight experimental groups in predrinking mood, blood pressure, or BAC. Of this, we were able to utilize the postdrinking data directly without any correction for predrinking levels.

Effects on Prestress Levels

Analysis of postdrinking mood scale data revealed higher self-reported "cheerfulness" for subjects who had consumed alcohol compared to those who had consumed tonic, (F(1, 48) = 12.48, p < .001). There were no effects on the other mood subscale scores or on blood pressure. The average BAC for subjects who consumed alcohol was .09%

Another indication of the effects of alcohol consumption was obtained from analysis of the physiological and ANX data from pre-stress Periods 1–14 of the stressor phase of the experiment. From the 46-period ANOVAS, significant main effects for beverage consumption were found for IBI, IBV variability, SCL, F-PTT, and ANX. When planned comparisons were performed on Beverage Consumed x Period means of stressor Periods 1–14 for these variables, it was found that subjects who had consumed alcohol had higher HR (i.e., shorter IBIs), lower IBV variability, higher SCL, longer F-PTT, and lower ANX than subjects who consumed tonic. In Table 1 the relevant means and F and t values are presented.

As regards expectancy effects, we were not able to find differences between sub-

In all conditions, a squirt of lime juice was added to the beverage, which was then divided into three glass containers. The total amount of liquid consumed was in the same proportion to body weight (e.g., approximately 35 oz. (1039 ml) of liquid for a 145 lb. (66 kg) subject).

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Effects on Magnitude of Response to Stressors

To provide an overall impression of the effects of the stressors, we have plotted the response profiles for IBI, ACT, SCL, E-PTT, and ANX for subjects in the shock (Figure 1) and speech (Figure 2) conditions. Examination of these figures reveals strong responses in all variables to both the initiation of the countdown sequence and to the stressor in both conditions. The response consisted of faster HR (decreased IBI), increased ACT, increased SCL, decreased E-PTT (and decreased F-PTT), and increased ANX—all of which are indicative of a more aroused psychophysiological state. Comparison of the two figures will also reveal that the response profiles for the two stressors are more alike than dissimilar. The major differences occurred in ACT (which remained relatively elevated throughout the 3-min. speech for subjects in that condition) and in ANX (subjects in the speech condition tended not to adjust the “anxiety dial” during their speeches).

Prior to analyzing the effects of alcohol and expectancy on responses to the stressors, we had to decide whether it would be necessary to analyze the data from subjects in the shock and speech conditions separately. To do this we examined the Stressor × Beverage Consumed × Period and the Stressor × Beverage Expected × Period interactions from the 46-period ANOVA for differences in the effects of beverage consumed or beverage expected between the shock and speech stressors. As none of these interactions were significant, subjects from the shock and speech conditions were combined to analyze the effects of alcohol and expectancy on responses to the stressors. Actual consumption of alcohol was found to reduce the psychophysiological response to stress. Examination of the Beverage Consumed × Period interactions from the difference score ANOVA revealed significant interactions for IBI, ACT, and ANX. To articulate the nature of these effects, we isolated the periods of peak response to the stressors, which occurred near the start of the countdown and shortly after the shock or at the end of the speech. We then compared the magnitude of the responses in these periods and found that subjects who consumed alcohol had a smaller HR increase (i.e., smaller IBI decrease) and a smaller E-PTT decrease to the start of the countdown than subjects who consumed tonic (Table 2). Further, in response to the stressor, subjects who consumed alcohol had smaller HR increases, smaller E-PTT decreases, and smaller ANX increases than subjects who consumed tonic (Table 2).

As was the case in our analysis of prestress levels, we found no evidence that expectancy alcohol had any effect on the magnitude of response to stress. The Beverage Expected × Period interactions were not significant for any of our dependent variables.

**Table 1**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Consume Alcohol</th>
<th>Consume Tonic</th>
<th>Beverage consumed P</th>
<th>t(88)*</th>
<th>Expect Alcohol</th>
<th>Expect Tonic</th>
<th>Beverage expected P</th>
</tr>
</thead>
<tbody>
<tr>
<td>IBI (msec)</td>
<td>764</td>
<td>805</td>
<td>4.84*</td>
<td>1.85*</td>
<td>797</td>
<td>777</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>ICI (msec)</td>
<td>3417</td>
<td>3578</td>
<td>1.34</td>
<td></td>
<td>3431</td>
<td>3561</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>ACT</td>
<td>2.8</td>
<td>2.8</td>
<td>&lt;.01</td>
<td></td>
<td>2.8</td>
<td>2.8</td>
<td>1.48</td>
</tr>
<tr>
<td>SCL (mho)</td>
<td>19.3</td>
<td>14.4</td>
<td>5.04*</td>
<td>-.92*</td>
<td>17.6</td>
<td>16.2</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>F-PTT (msec)</td>
<td>235</td>
<td>222</td>
<td>8.72**</td>
<td>-2.45**</td>
<td>223</td>
<td>234</td>
<td>2.04</td>
</tr>
<tr>
<td>E-PTT (msec)</td>
<td>192</td>
<td>191</td>
<td>3.69</td>
<td></td>
<td>194</td>
<td>196</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>ANX</td>
<td>2.0</td>
<td>2.8</td>
<td>7.17**</td>
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Note. IBI = Heart rate interbeat interval; ICI = respiration rate intercycle interval; ACT = general somatic activity; SCL = skin conductance level; F-PTT = finger pulse transmission time; E-PTT = ear pulse transmission time; ANX = self-reported anxiety.

* t test comparing average of prestress Trials 1–14 from Beverage Consumed × Period interaction.

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Note. IBI = Heart rate interbeat interval; ICI = respiration rate intercycle interval; ACT = general somatic activity; SCL = skin conductance level; F-PTT = finger pulse transmission time; E-PTT = ear pulse transmission time; ANX = self-reported anxiety.

* t test comparing average of prestress Trials 1–14 from Beverage Consumed × Period interaction.
consumed and whether they thought the experiment was deceptive in any way. In Table 3 the results of the analysis of these data are presented. Subjects who were told they were drinking alcohol rated themselves as being more drunk and having consumed more ounces of liquor than subjects who were told they were drinking tonic. Similarly, subjects who consumed alcohol rated themselves as being more drunk and having consumed more ounces of liquor than did subjects who consumed tonic. Although the ANOVA on these data revealed no significant interactions of Beverage Consumed × Beverage Expected, the cell means for this interaction are presented for subjects' estimates of drunkenness and the expectation established by the beverage. Taken together, these data are presented.

Subjects' Estimates of Drunkenness and Amount of Liquor Consumed

Table 3

Table 2

<table>
<thead>
<tr>
<th>Measure:</th>
<th>Beverage Consumed × Countdown Drink</th>
<th>Stressor</th>
<th>IBI (msec)</th>
<th>E-PTT (msec)</th>
<th>ANX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in Period</td>
<td>Alcohol</td>
<td>Tonic</td>
<td>r(88)</td>
<td>Alcohol</td>
<td>Tonic</td>
</tr>
<tr>
<td>IB (msec)</td>
<td>3.34**</td>
<td>-75.6</td>
<td>.01</td>
<td>-101.5</td>
<td>2.19*</td>
</tr>
<tr>
<td>E-PTT (msec)</td>
<td>3.74**</td>
<td>-11.6</td>
<td>.01</td>
<td>-15.6</td>
<td>1.91*</td>
</tr>
<tr>
<td>ANX</td>
<td>3.98**</td>
<td>.38</td>
<td>.60</td>
<td>.81</td>
<td>.87</td>
</tr>
</tbody>
</table>

Note. IBI = heart rate interbeat interval; E-PTT = ear pulse transmission time; ANX = self-reported anxiety.

*p < .05; **p < .01.

Our analysis of subjects' ratings of the deceptiveness of the experiment revealed a significant interaction of Beverage Consumed × Beverage Expected (F(1, 88) = 6.37, p = .013). With subjects who consumed alcohol and expected tonic, alcohol was perceived as more deceptive than subjects in the other conditions (p < .01 by Scheffe's method).

Discussion

Alcohol Effects on Prestress Levels

This experiment yielded data on the effects of alcohol on prestress levels of a number of physiological measures. Our finding that alcohol consumption produced increased HR and increased SCL is consistent with other published research. Similarly, our finding of prolonged F-PTT in response to alcohol provides support for the view that alcohol reduces myocardial performance, although we are unable to say with certainty whether cardiac contractility is the specific function affected. Our failure to find an effect of alcohol on our measure of skeletal muscle activity (ACT) is not consistent with Steffen et al.'s (1974) finding of decreased forehead muscle tension; however, differences between these measures and the use of hospitalized alcoholics by Steffen et al. could easily account for this discrepancy.

As we indicated earlier, the mixed pattern of stimulant effects (i.e., faster HR, higher SCL) and relaxant effects (i.e., prolonged F-PTT) produced by alcohol does not readily fit a simple label such as "tense," "relaxed," or "aroused." Yet subjects who consumed alcohol in our study did report feeling more "cheerful" and less "anxious." Unfortunately, we did not solicit information that would enable us to ascertain the basis of these self-reports, and thus we have no way of determining whether perceived physiological changes were important. Considering the number of contradictory findings on the effects of alcohol on self-report of anxiety that now exist in the literature, it would seem important in subsequent studies to devise ways of more thoroughly examining mood changes. Our review of the literature and our results indicate that the complex pattern of physiological changes produced by alcohol consumption is not likely to provide a simple key to understanding the mediational link between alcohol and mood. Researchers who attempt to infer mood from one or two physiological variables may reach unwarranted conclusions that could be avoided if a broader pattern of physiological changes were considered.

Finally, cursory examination of Figures 1 and 2 will reveal additional complexity in the measurement of self-reported anxiety beyond the selection of the appropriate assessment technique. Our results reveal that anxiety levels change throughout the course of the experiment, thus, the practice of assessing anxiety once or twice in an experiment (e.g., Abrams & Wilson, 1979; Polivy et al. 1976; Wilson & Abrams, 1977) can result in disparate findings if measurement periods are not comparable. In this regard, our data suggest the following periods as being worthy of differentiation when assessing anxiety and other mood variables: (a) before drinking, (b) after drinking and prior to explanation of stress manipulation, (c) during anticipation of stress, (d) following stress onset, and (e) following termination of stress.

Effects of Alcohol on Magnitude of Response to Stress

Our results clearly indicate that alcohol consumption is associated with reduction in the magnitude of response to stress. Specifically, we found attenuation of the magnitude of response to two kinds of stresses in both physiological (HR, E-PTT) and psychological (ANX) measures. This effect, which we will call "stress response dampening" (SRD), may be viewed as substantiating the in-persuasion claim that alcohol has a positive value when consumed in the context of a stressful situation. Thus, we have documented a nonplacebo effect associated with alcohol consumption, which could be applied to a better understanding of why people drink alcohol in stressful situations.

Based on our results, we would expect the SRD effect to be observable under certain experimental conditions. First, the effect should only be expected when a bona fide stressor is being used. For this reason, a potential stressor should be tested with comparable subjects to verify its capacity to produce reliable responses in the dependent measures. Second, we would expect the effect when higher (e.g., 1 g/kg) dosages of alcohol are used. This contention is based on the failure of other investigators to find effects of alcohol consumption on the response to stress at lower (.5 g/kg) dosages of alcohol. Third, we would anticipate the SRD effect to be most pronounced in two periods following absorption: during anticipation of stress and following stress onset.

The present experiment provides a needed demonstration of the SRD effect of alcohol consumption. An important remaining question is determination of the underlying mech-
Dosage, Deception, and Expectancy Effects

Despite our failure to find any effects attributable to the expectation of consuming alcohol, we do not view our results as a refutation of previously documented expectancy effects associated with alcohol. Rather, our data can be seen as illustrating a number of pharmacological effects of alcohol consumption. The study of expectation effects and alcohol is a relatively new research area. Although a number of different expectation effects have been demonstrated, there has been no basic research that has studied these effects under varying parameters of dosage, procedure, and subject demographics. In this expectation literature, the findings from research concerned with alcohol expectancy and stress can be summarized as indicating that alcohol expectancy is associated with higher levels of anticipatory anxiety (Polivy et al., 1976), smaller HR increase in response to stressful interactions (Wilson & Abrams, 1977), and “increased levels of physiological arousal” (Abrams & Wilson, 1979). An important characteristic of all of these studies is the use of a dosage of 5 g ethanol/kg body weight, as compared to the higher 1 g/kg dosage used in the present study.

Our selection of a relatively high dosage of alcohol enhanced the likelihood of our detecting reliable pharmacological effects. A related disadvantage, however, was increased likelihood that subjects would not believe our deception manipulations. We found this to be particularly true in the consumne alcohol—expect tonic condition, in which subjects expected to consume a number of signs of intoxication and subsequently reported finding the experiment “deceptive.” Among studies of alcohol and stress using the 1 g/kg dosage, complete deception of subjects was reported in both papers by Wilson and Abrams. Polivy et al. reported less complete deception, but they did not use the elaborate deception procedures (e.g., bogus BAC feedback, strong mountithal, and alcohol smeared on the glass in the placebo condition) used by Wilson and Abrams. In the only study in the expectation literature that used the 1 g/kg dosage, Lang et al. (1975) reported deception results quite similar to ours. Despite procedural differences between the two experiments, both found subjects’ perceptions of intoxication and altered antecedents influenced by what they were told they were drinking and by the actual beverage content. However, the possibility remains that more complete deceptions may be attainable by using more elaborate manipulations, and such procedures should probably be adopted in future research at the 1 g/kg dosage.

Our choice of a high dosage was undoubtedly a contributing factor to our failure to find reliable expectancy effects. At higher dosages, pharmacological effects may become relatively prepotent over expectancy effects, the latter being more discernible in the ambiguous state of intoxication and more potent with lower dosage. If this relationship does exist, it suggests that the four-cell consumption—expectation design may be more sensitive to alcohol effects at high dosages and more sensitive to expectancy effects at low dosages. Of course, Lang et al.’s (1975) finding of an expectation effect for a behavioral measure (increased aggression toward a confederate) at a 0.5 g/kg dosage indicates that expectancy effects can still be found at higher dosages. Nonetheless, a study of alcohol and response to stress that manipulated consumption, expectation, and dosage would help clarify these issues and would be a valuable addition to the literature.

Reference Note


References


MacAndrew, C. The differentiation of male alcoholic outpatients from nonalcoholic psychiatric outpatients by means of the MMPI. Quarterly Journal of Studies on Alcohol, 1965, 26, 238–246.


