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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 prestress 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; Lienert & 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

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(Steffen, Nathan, & Taylor, 1974), increase in skin conductance level (Jones, Parsons, & Rundell, 1976), and decreased cardiac contractility (Child, Kovick, Levisman, & Pearce, 1979; Knott & Beard, 1972). This complex pattern of physiological changes includes indications of both stimulant (i.e., increased HR, increased skin conductance level) and relaxant (i.e., decreased muscle tension, decreased contractility) properties. Because of this complexity it seems unrealistic to expect naive subjects to show agreement in their 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; McNamee, 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" (Conger, 1951, 1956) is a model for relating alcohol and stress that has generated much controversy (e.g., Cappell & Herman, 1972). The hypothesis proposes 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 individuals 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, & Marlatt, 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 authors found that subjects who believed they had consumed alcohol had smaller percentage increases in heart rate in response to the stressor, but did not differ from subjects who believed they had consumed tonic in self-reported anxiety or in behavioral response measures. There were no effects attributable to consumption of alcohol per se. Abrams and Wilson (1979) essentially repeated this procedure with female subjects interacting with a male confederate, adding a measure of skin conductance and more detailed behavioral observations. They concluded that believing alcohol was consumed resulted in "increased levels of physiological arousal" (p. 169).[1] Again, no effects of consumption of alcohol were found.

Because the studies conducted by Polivy et al. and by Wilson and Abrams suggested a potential role for cognitive factors in mediating the relationship between alcohol and stress, the four-cell design was adopted for the present investigation. This investigation is also characterized by the use of two different stressors (electric shock and self-disclosing speech); monitoring of a number of physiological responses selected from the cardiovascular, electrodermal, respiratory, and skeletal muscle systems; and adoption of a continuous self-report of anxiety. Finally, in keeping with concerns raised earlier in this introduction, the experimental design allows separation of effects of alcohol on prestress levels from its effects on the magnitude of response to stress.

Method

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 telephone screening that elicited information about typical drinking patterns to obtain a sample of moderate to heavy social drinkers. The criteria for inclusion were (a) alcohol consumed more than once a week on the average and (b) typical consumption of two or more drinks per occasion (subjects selected for this study consumed an average of 12.6 ounces (357.2 g) of absolute alcohol per week). Respondents were excluded if they were under 21 years of age or if they reported having been arrested for any offense involving alcohol.

Subjects were told during the initial phone contact that they might receive alcohol in the experiment and that they would either make a self-disclosing speech or receive an electric shock, which was described as "painful but not harmful." In addition they were asked to complete a questionnaire prior to their arrival at the laboratory, to abstain from eating or drinking alcohol for at least 4 hours before the experiment, and to refrain from driving themselves to the experiment (taxi service was provided as needed). Subjects were paid $7.50 each for participating.

Apparatus

Physiological. Data were obtained for a number of physiological variables using a system designed for on-line analysis consisting of a Grass Model 7 polygraph and a PDP 11/10 minicomputer. The system enabled detection and averaging of physiological data during the course of the stressor portion 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; the computer timed the interval between successive heart beats in msec (HR = 60,000/IBI in msec). (b) Respiration rate intercycle interval (ICI)—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 inspirations. (c) General somatic activity (ACT)—an electromagnetic sensor placed under the subject's chair detected movement in all planes. (d) Skin conductance level (SCL)—a constant voltage device was used to pass a small current through surface electrodes attached to the medial phalanges of the first and third fingers. (e) Pulse transmission times—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-PTT) and at the finger (F-PTT). 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) was obtained during the stressor portion of the experiment through the use of an "anxiety dial" modeled after one used by Blankstein, Pliner, and Constantinou (Note 1). Subjects manipulated the dial pointer in reference to a 10-point scale anchored by the legends "extremely calm" and "extremely tense." The dial was attached to a potentiometer that produced a proportional voltage. Using a simple calibration

[1] Data from this study were handled quite differently from those of the earlier study with male subjects, and interpretation of results is difficult. A separate analysis of changes in their measures of heart rate and skin conductance in response to the interaction was not reported. The multivariate treatment of the data that is reported includes measurement periods before and after the interaction. Our examination of their figures portraying mean heart rate and percentage of change in skin conductance suggests that subjects who believed they had consumed alcohol had faster heart rates and higher skin conductance levels prior to the interaction and that these prestressor differences were maintained throughout the experiment.

formula, the computer was able to monitor this voltage and thus continuously track the dial position.

In the shock condition a brief, unpleasant shock was administered through electrodes attached to an adjustable wristband. The shock was produced by a constant current device, with shock onset and duration controlled by the computer.

Finally, a light-emitting diode (LED) digital display was used as a countdown device during the stressor portion of the experiment. The display, which was situated on a table in front of the subject, consisted of ¾-inch (1.9 cm) red digits and was controlled by the computer.

Procedure

Several days prior to participating in the laboratory session, subjects came to the Psychological Clinic to complete a questionnaire package that contained measures of anxiety in response to specific situations (Endler & Okada, 1975), assertiveness and social skills (Levenson & Gottman, 1978), quantity and frequency of drinking, and two measures related to alcohol abuse (MacAndrew, 1965; Seltzer, Vinokur, & Van Rooijen, 1975). On the day of the scheduled experimental session, subjects underwent the following procedure:

Predrinking phase (15 min.). On arriving at the laboratory, subjects were met by an experimental assistant who recorded weight, height, oral temperature, systolic and diastolic blood pressure, and BAC. Each subject then completed a self-report mood inventory (Nowlis, 1965) and signed an informed consent statement.

Administration of beverages (1 hr, 45 min.). The experimenter removed the appropriate tonic and/or vodka bottles from a refrigerator and gave them to the assistant, who was blind to their contents. Subjects, who were randomly assigned to one of four conditions of expectancy (expect alcohol or expect tonic) and consumption (consume alcohol or consume tonic), watched the assistant measure the beverages into a graduated cylinder. The contents of the bottles corresponded to the four conditions to which subjects were assigned as follows: (a) Expect alcohol–consume alcohol. Subjects observed the assistant measure Popov's 80-proof vodka (1 g ethanol/kg body weight) from a vodka bottle and add four times the amount of Sunrise tonic from a tonic bottle (we had previously determined that subjects could not reliably detect the presence of vodka at this dilution). (b) Expect alcohol–consume tonic. The procedure was identical to that of Condition a except the vodka bottle actually contained decarbonated tonic. (c) Expect tonic–consume alcohol. Subjects observed the assistant measure an appropriate amount of liquid from a tonic bottle. The bottle actually contained a mixture of 1 part vodka to 4 parts tonic. (d) Expect tonic–consume tonic. The procedure was identical to that of Condition c except the tonic bottle contained only tonic.

In all conditions, a squirt of lime juice was added to the beverage, which was then divided into three glasses. Regardless of condition, 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, 7 oz. (208 ml) of which were 80-proof vodka if the subject actually received alcohol.

Each subject was seated in a private room and told he must consume the beverages within 45 min. The assistant entered the room every 15 min. to bring in the next glass and every 8 min. to remind the subject of the time remaining. Assistants were told not to engage in conversation with the subject, to maximize the extent to which they remained blind to the subject's true condition. Subjects were given magazines and newspapers to read while drinking the beverages and during the 40-min. absorption period that followed the drinking session.

During the absorption period, subjects were again given the mood inventory to complete and measures of temperature and blood pressure were taken. Just prior to entering the experimental chamber, a final Breathalyzer sample was obtained for each subject, but BAC was not yet computed.

Stressor phase (35 min.). Subjects were escorted to the experimental chamber and seated in a comfortable chair while the experimenter attached the electrodes, explaining the purpose of each. While the electrodes were being attached, the assistant entered the room to give bogus feedback about the final BAC reading, based on the bottles used to mix the drinks. The assistant, who was blind to the actual BAC reading, stated either: "His BAC is .11, so he's drunk," if part of the beverage had been poured from a vodka bottle, or "His BAC is .00, so he's sober," if the drinks had been poured only from tonic bottles. Thus, the assistant was blind to the contents of the drinks at all times when interacting with the subject and only later learned the subject's true BAC. The experimenter who attached the electrodes was aware of the subject's true condition during the 10-min. interaction period required for electrode placement but refrained from discussing the content of the drinks with the subject. After all the electrodes had been attached, the subject was read one of the following sets of instructions:

> *(Shock condition).* After about 10 or 15 minutes, you will see the number 360 appear on the display in front of you and the numbers will begin to count down. At that time, pick up the clipboard beside your chair and read the instructions, which will inform you when the shock will be delivered. (The instructions read, "The number on the display in front of you is counting by seconds from 360 to 0. You will receive a shock when the number reaches 0.")

> *(Disclosure condition).* After about 10 or 15 minutes, you will see the number 360 appear on the display in front of you and the numbers will begin to count down. At that time, pick up the clipboard beside your chair. Read the speech topic and replace the clipboard. (The topic was "What I like and dislike about my body and physical appearance.") When the number on the display reaches 0, look into the camera, state your name, and begin your speech. Please stop talking when the number 9999 appears on the display (this occurred 3 min. after the start of the speech.) Try to be as open and honest as possible. Graduate students in clinical psychology will rate the videotape of your speech for openness, defensiveness, and other psychological variables.

Before leaving the room, the experimenter instructed subjects on the use of the anxiety dial and reminded them to continuously monitor their anxiety levels and adjust the dial accordingly throughout the experiment.

The entire session lasted 23 min., consisting of 7 min. of prestressor recording, 6 min. of countdown followed by the stressor, and 10 min. of additional recordings. In the speech condition these latter 10 min. included the 3-min. speech.

Postexperimental phase (10 min.). At the end of the session, the experimenter entered the room, removed the electrodes, and escorted the subject to another room for final recording of BAC and temperature and administration of a questionnaire designed to assess the effectiveness of the experimental manipulations. Subjects were then debriefed and driven home.

Results

Data were collected in this experiment before and after drinking (mood, blood pressure, and BAC), during the 23-min. stressor sequence, and during the postexperimental debriefing. Preliminary analysis of variance (ANOVA) was performed on all data as follows. The mood, blood pressure, BAC, and postexperimental questionnaire scores were analyzed in a series of 2 × 2 × 2 (Stressor × Beverage Consumed × Beverage Expected) ANOVAS. The physiological data (IBI, IBI variability, ICI, ACT, SCL, E-PTT, F-PTT) and continuous self-report of anxiety (ANX) obtained during the stressor phase were analyzed by dividing the 23 min. of data into 46 periods of 30 sec duration. These data were then analyzed with two series of ANOVAS. The first series were 2 × 2 × 2 × 46 (Stressor × Beverage Consumed × Beverage Expected × Period) ANOVAS with a repeated measure on the last factor. In the second series of ANOVAS, data from Periods 1–14 were averaged (these corresponded to the 7 min. preceding the start of the countdown) and difference scores were computed for the remaining Periods 15–46 by subtracting the average prestress value from each period value. These difference scores were submitted to a series of 2 × 2 × 2 × 32 (Stressor × Beverage Consumed × Beverage Expected × Period) ANOVAS with a repeated measure on the last factor.

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 used in these comparisons was the smaller of the two associated with the error terms contributing to the pooled error.

Group Differences Prior to Drinking

There were no differences across the eight experimental groups in predrinking mood, blood pressure, or BAC. Because 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, $F(1, 88) = 12.48$, $p < .001$. 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 prestress Periods 1–14 of the stressor phase of the experiment. From the 46-period ANOVA, significant main effects for beverage consumed were found for IBI, IBI variability, SCL, F-PTT, and ANX. When planned comparisons were performed on Beverage Consumed × Period means of prestress Periods 1–14 for these variables, it was found that subjects who had consumed alcohol had faster HR (i.e., shorter IBIs), lower IBI 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-

Table 1
Effects on Prestress Levels (Periods 1–14)

Measure	Consume		Beverage consumed		Expect		Beverage expected
	Alcohol	Tonic	F	$t(88)$[a]	Alcohol	Tonic	F
IBI (msec)	764	805	4.84*	1.83*	797	772	<1
IBI σ (msec)	51	67	9.78**	2.11*	55	63	2.01
ICI (msec)	3417	3578	1.34	—	3431	3561	<1
ACT	2.8	2.8	<1	—	2.8	2.8	1.48
SCL (mho)	19.3	14.4	5.04*	−1.92*	17.6	16.2	<1
F-PTT (msec)	235	222	8.72**	−2.45**	223	234	2.04
E-PTT (msec)	198	191	3.69	—	195	194	<1
ANX	2.0	2.8	7.17**	2.31*	2.4	2.5	<1

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.
[a] t test comparing average of prestress Trials 1–14 from Beverage Consumed × Period interaction.
* $p < .05$. ** $p < .01$.

jects expecting alcohol and those expecting tonic in any of the postdrinking mood variables or blood pressure. Similarly, in the 46-period ANOVA there were no significant main effects for beverage expected for any of the physiological variables or for ANX (Table 1).

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, E-PTT, 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 after the start 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 expecting 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.

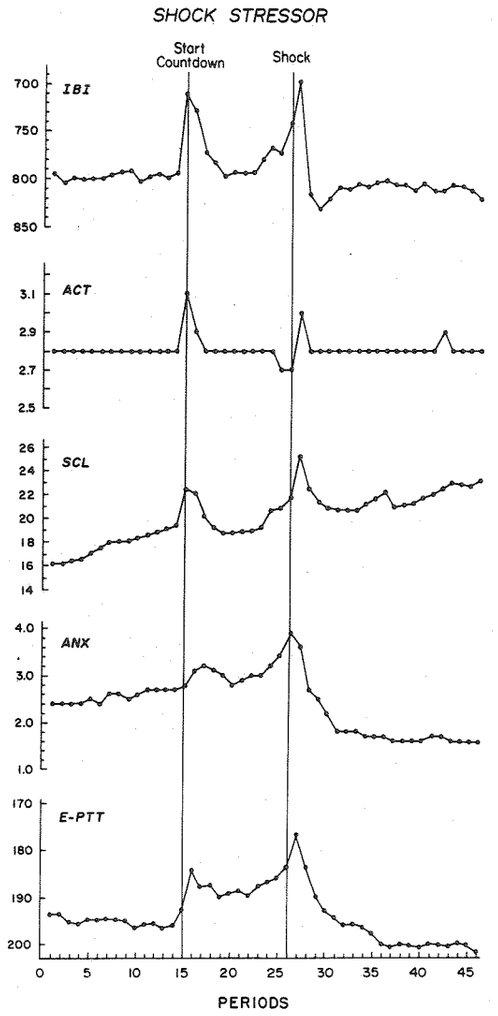

Figure 1. Profile of responses to shock stressor. (All variables are scaled so that the upward direction indicates higher levels of arousal; $N = 48$. IBI = heart rate interbeat interval; ACT = general somatic activity; SCL = skin conductance level; ANX = self-reported anxiety; E-PTT = ear pulse transmission time.)

Verification of Experimental Manipulations

We attempted to assess the success of our manipulations by having subjects complete a postexperimental questionnaire that asked them to estimate how drunk they were after drinking, during the stressor portion of the experiment, and at the present time. They were further asked to estimate the number of ounces of "hard liquor" they had

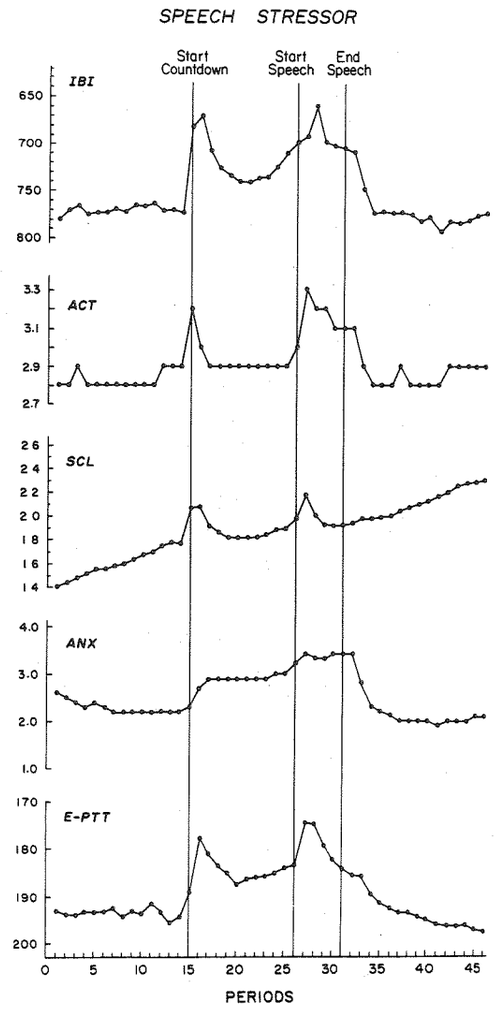

Figure 2. Profile of responses to speech stressor. (All variables are scaled so that the upward direction indicates higher levels of arousal; $N = 48$. IBI = heart rate interbeat interval; ACT = general somatic activity; SCL = skin conductance level; ANX = self-reported anxiety; E-PTT = ear pulse transmission time.)

Table 2
Effects of Beverage Consumed on Magnitude of Response During Peak Stress Periods

Measure: Change in	Beverage Consumed × Period F	Countdown			Stressor		
		Alcohol	Tonic	$t(88)$	Alcohol	Tonic	$t(88)$
IBI (msec)	3.34**	−75.6	−101.5	2.19*	−101.0	−141.3	3.42**
E-PTT (msec)	3.74**	−11.6	−15.6	1.91*	−16.0	−22.0	2.87**
ANX	3.98**	.38	.60	.81	.87	1.39	1.93**

Note. IBI = heartrate interbeat interval; E-PTT = ear pulse transmission time; ANX = self-reported anxiety.
* $p < .05$. ** $p < .01$.

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 ounces of alcohol consumed (see footnote to Table 3) to allow comparison with results from other experiments. Taken together, this pattern of results indicates that subjects' perceptions concerning their state of drunkenness were a function of both the actual beverage content and the expectation established by the experimenter. Thus, it seems warranted to conclude that at the relatively high dosage used, our manipulations had a significant impact on subjects' perceptions but were not able to completely override the effects of the actual beverage content.

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 rating the experiment as being more deceptive than subjects in the other conditions ($p < .01$ by Scheffé'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

Table 3
Subjects' Estimates of Drunkenness and Amount of Liquor Consumed

Measure	Consume			Expect		
	Alcohol	Tonic	$F(1, 88)$	Alcohol	Tonic	$F(1, 88)$
Drunkenness after drinking (1–10 scale)	5.7	2.4	88.4**	4.9	3.2	23.8**
Drunkenness while anticipating stressor (1–10 scale)	5.2	1.9	82.2**	4.3	2.8	14.5**
Drunkenness at present time (1–10 scale)	4.2	1.7	39.2**	3.4	2.5	4.4*
No. of ounces of hard liquor consumed[a]	5.2	2.0	44.8**	5.1	2.2	36.9**

[a] Individual cell means: consume alcohol–expect alcohol, 6.54; consume alcohol–expect tonic, 3.88; consume tonic–expect alcohol, 3.58; consume tonic–expect tonic, .46.
* $p < .05$. ** $p < .01$.

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 stressors 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 layperson's 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 sober 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 consumed. 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. 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-

anisms responsible for this effect. Several mechanisms may be hypothesized, including a direct pharmacologic effect on physiological responsiveness, with attenuated responsivity leading (by some mediating mechanism such as proprioceptive feedback) to the inference of lower levels of anxiety. An alternative explanation may be that the effect is an indirect result of alcohol's influencing a cognitive process, such as increasing distractibility (making the person attend less fully to the stressor) or altering evaluation of the level of threat (making the person feel the stress is less severe). Another question worthy of investigation is the possible existence of a relationship between individual differences in the magnitude of the SRD effect and the potential for development of problems of alcohol addiction or abuse. An experiment using a "high risk" methodology is presently being carried out in our laboratory to investigate this relationship.

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 of higher dosage, however, was increased likelihood that subjects would not believe our deception manipulations. We found this to be particularly true in the consume alcohol–expect tonic condition, in which subjects experienced a number of signs of intoxication and subsequently reported finding the experiment more "deceptive." Among studies of alcohol and stress using the .5 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 mouthwash, and alcohol smeared on the glasses 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 the amount of alcohol consumed to be 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 associated 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 the 1 g/kg dosage indicates that expectation 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

1. Blankstein, K. R., Pliner, P., & Constantinou, K. *Heart rate increases in an "incubation of threat" situation: Fear or coping?* Paper presented at the meeting of the Psychophysiological Research Society, September 1978.

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