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Greater Reinforcement From Alcohol for Those at Risk: Parental Risk, Personality Risk, and Sex

Robert W. Levenson, Oliver N. Oyama, and Patricia S. Meek
Indiana University

The effects of a high dose of alcohol (1 g ethanol/kg body weight) on physiological and self-report responses to two stressors (electric shock and self-disclosing speech) were compared with the effects of a placebo in three groups of nonalcoholic subjects considered to be at heightened risk for alcoholism by virtue of their (a) having an alcoholic parent (parental risk) or (b) matching a prealcoholic personality profile (personality risk), or (c) having an alcoholic parent *and* matching a prealcoholic personality profile. These high-risk groups were contrasted with a low-risk group that had neither risk factor. Male and female subjects were tested in each group with appropriate controls for drinking experience and, for female subjects, phase of menstrual cycle. Results indicated that a potentially reinforcing effect of alcohol (its capacity to attenuate physiological responses to stress) was more pronounced in high-risk subjects than in low-risk subjects. This relation was found for both parental risk and personality risk factors and in both male and female subjects.

The effects produced by a given dose of alcohol can vary greatly from drinker to drinker. This observation is borne out both by folk wisdom and by laboratory experimentation. Although such individual differences might at one time have been dismissed as random error, the trend in recent alcohol research has been to try to uncover the ways in which variation in the actions of alcohol is linked to variation in characteristics of the drinker. To consider both of these sources of variation at once merges two strands of past investigation that have for the most part proceeded independently. As for the actions of alcohol, some of the best studied have been the impact of alcohol on cognitive performance, on emotion and mood, on physiological responding, on inner states, and on psychomotor performance. As for factors of individual variation, some of the best studied have been past drinking history (and the tolerance that it bestows), sex, racial and ethnic heritage, the presence of parental alcoholism, and a number of different personality characteristics. In selecting from among the many possible combinations of factors of individual variation and different effects of alcohol, we were guided by a desire to obtain data that could link individual variation and variation in the effects of alcohol within a reinforcement model of the etiology of alcoholism.

We considered it desirable to select individual difference factors related to heightened risk for alcoholism and to select effects of alcohol that could be construed as being positive and reinforcing for the drinker. The hypothesis was simple: Individuals at heightened risk for alcoholism are provided by nature or by nurture with a propensity for receiving an extra increment of alcohol's reinforcing effects. Thus all other things being equal, this predisposition causes people at risk to experience drinking as being more rewarding than do others. Incrementally greater reward leads to engagement in incrementally greater amounts of drinking behavior and thus to a more rapid progression along the path toward alcohol dependence and, ultimately, to alcoholism. In common parlance, we hypothesized that people at risk for alcoholism would receive a greater "bang for their buck" when they drank and, further, that this "bang" would be a reinforcing one.

Stress-Response Dampening

Because this experiment attempts to extend our earlier research in this area (Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sher & Levenson, 1982), the logic underlying this research program should be briefly reviewed. Initially, we chose to study alcohol's capacity to reduce the magnitude of psychophysiological responding generated by a stressful situation. This capacity can be viewed as reinforcing, by virtue of its reduction of the disruption caused by a stressful event, and it fits comfortably with earlier influential tension-reduction models (Conger, 1951, 1956). Face validity is reasonable as well because drinkers regularly assert that one reason they drink is to help them deal with life's stresses. Our initial efforts were directed toward establishing the existence of this stress-response-dampening effect. We (Levenson et al., 1980) were able to demonstrate that a stress-response-dampening effect of alcohol (a) existed in male subjects at a dose of 1 g/kg, (b) was overwhelmingly pharmacologic as opposed to being a result of psychological expectancy, and (c) was most pronounced in cardiovascular measures of heart rate and pulse transmission time to the ear. These findings set the stage for our subsequent explorations of sources of individual variation, which led to the present study.

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Oliver N. Oyama is now at the Pain Therapy Center of Columbia and the University of South Carolina School of Medicine, Columbia. Patricia S. Meek is now at the University of California, San Francisco.

Correspondence concerning this article should be addressed to Robert W. Levenson, who is now at the Department of Psychology, University of California, Berkeley, California 94720.

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Risk by Virtue of Personality

We first studied variation in alcohol's stress-response-dampening effect that was related to a prealcoholic personality profile measured by two self-report inventories: the MacAndrew Alcoholism Scale (MAC; MacAndrew, 1965) and the Socialization subscale of the California Psychological Inventory (So; Gough, 1969). The MAC is an empirically derived test based on items from the Minnesota Multiphasic Personality Inventory. Its validity as a measure of risk for alcoholism rests on several studies showing its capacity to discriminate alcoholics from nonalcoholics and to predict subsequent alcoholism in nonalcoholic individuals (e.g., Hoffman, Loper, & Kammeier, 1974; MacAndrew, 1979; Saunders & Schuckit, 1981). The relation between the So and risk for alcoholism is based on more indirect evidence, consisting of the marked similarity between the traits the So purports to measure and a constellation of traits shown in prospective studies to characterize people who become alcoholics in later life (e.g., J. C. Jones, 1968; McCord & McCord, 1960; Robins, Bates, & O'Neal, 1962; Schuckit, Gunderson, Heckman, & Kolb, 1976).

Using these two scales as indexes of risk for alcoholism, we found, in two experiments (one using just the MAC, the other using the MAC and So in combination), high-risk male subjects to receive more of the stress-response-dampening benefits from alcohol consumption than did their low-risk counterparts (Sher & Levenson, 1982). This was the first strong support for our hypothesis that individual differences in the effects of alcohol are organized around personality variables related to risk for subsequent alcoholism.

Risk by Virtue of Parental Alcoholism

Although the case for a relation between having a certain set of personality traits and being at heightened risk for alcoholism is admittedly somewhat tenuous, the risk associated with having an alcoholic parent is better established. This heightened risk (often estimated as being 3 to 5 times that of people who do not have an alcoholic parent) is thought to be mediated by genetic factors, learning, and imitation, with different authors arguing for a different mix among these factors (e.g., Cadoret, 1976; Cotton, 1979; El-Guebaly & Offord, 1976; Frances, Timm, & Bucky, 1980; Goodwin, 1976, 1979; Harburg, Davis, & Caplan, 1982; McKenna & Pickens, 1981; Smart & Fejer, 1972).

In the case of the MAC and So personality risk measures, we were not aware of any previous work that linked these factors to individual differences in the effects of alcohol. For the risk associated with parental alcoholism, however, a number of such studies do exist. Having alcoholic parents has been linked with individual differences in the effects of alcohol on resting levels of muscle tension (Schuckit, Engstrom, Alpert, & Duby, 1981), on facial flushing (Schuckit & Duby, 1982), and in rates of alcohol metabolism (Schuckit & Rayses, 1979).

Findings of heightened risk for alcoholism among children of alcoholic parents, combined with findings of differential effects of alcohol associated with having an alcoholic parent, suggested that parental alcoholism would be an important factor to study in relation to psychophysiological responses to stress. Nonetheless, it seemed unwise to abandon the personality risk measures that had proved useful in previous studies. Thus the present study was designed to study subjects with alcoholic parents, subjects with the prealcoholic personality, subjects with both risk factors, and control subjects having neither risk factor. Subject recruitment for such a study represented a major undertaking; nonetheless, we decided to include one additional factor in the design.

Sex

In all of our previous experiments, we used only male subjects. The omission of female subjects has been a glaring problem in much of the experimental literature on alcohol. When female subjects have been included, however, a number of interesting sex differences have emerged (Abrams & Wilson, 1979; Sutker, Allain, Brantley, & Randall, 1982; Wilson & Abrams, 1977). Thus for the present study we decided to include both male and female subjects because this would enable us to determine whether previous findings concerning alcohol's stress-response-dampening effects in men would replicate with female subjects. Furthermore, this would provide an opportunity to determine whether relations that personality and parental risk factors had with individual differences in the effects of alcohol were consistent across sexes.

Method

Subjects

Telephone screening. We used newspaper advertisements to recruit subjects between the ages of 21 and 35 years to participate in "alcohol research." Respondents were screened by telephone to establish that they (a) met our criteria for moderate social drinking (i.e., consumed at least two drinks on at least two occasions per week, or at least four drinks on at least one occasion per week), (b) had never been institutionalized for alcoholism, (c) had never been arrested for an alcohol-related offense (including driving under the influence of alcohol or public intoxication), (d) had not participated in one of our earlier alcohol studies, (e) had no major health problems, and (f) were not taking any medication that contraindicated the use of alcohol.

Questionnaire battery. Subjects who passed the initial telephone screening were asked to come to the psychology department at Indiana University to complete a battery of questionnaires that included (a) a measure of quantity and frequency of drinking; (b) three versions of the Michigan Alcoholism Screening Test (MAST; Selzer, 1971), all to be completed by the subject (one concerning the subject, one concerning the subject's biological father, and one concerning the subject's biological mother); (c) the MAC; and (d) the So. Subjects whose own MAST score was greater than 7 were excluded from participation in the experiment. In all, 1,213 subjects completed the questionnaire package for a payment of $3.50.

Risk-group designations. There were four risk groups: (a) low risk (33 men, 31 women), (b) personality risk (36 men, 31 women), (c) parental risk (30 men, 32 women), and (d) personality and parental risk (26 men, 23 women). Criteria for inclusion in the four risk groups were based on scores on the MAC, So, and parental MASTs (see Table 1). A total of 243 subjects participated in the laboratory experiment, and each was paid $10.

To keep the design balanced, we established cutoffs before the first subject was tested. For the MAC and So, the median scores obtained in our previous research using these questionnaires (Sher & Levenson, 1982) were relaxed by 1 scale point. This 1-point adjustment made it more feasible to fill the risk group cells for both male and female sub-

Table 1
Criteria for Risk Groups

Group	Measure				
	MAC		So		MAST
Low risk (33 men, 31 women)	≤19	and	≥37	and	Both parents ≤3
Personality risk (36 men, 31 women)	≥21	and	≤35	and	Both parents ≤8
Parental risk (30 men, 32 women)	≤30	or	≥36	and	At least one parent ≥9
Personality and parental risk (26 men, 23 women)	≥21	and	≤35	and	At least one parent ≥9

Note. MAC = MacAndrew Alcoholism Scale. So = Socialization subscale of the California Psychological Inventory. MAST = Michigan Alcoholism Screening Test.

jects (female subjects meeting the criteria were more difficult to find). To spread the risk groups somewhat, the low-risk cutoffs for the MAC and the So were set 1 point away from the high-risk cutoffs. The cutoff scores for parental alcoholism on the MAST were based on the existing literature. Although Selzer (1971) originally proposed a MAST cutoff score of 5 for identifying alcoholics, higher cutoffs have often been used (e.g., Moore, 1972). Because we were using parental MAST scores based on the child's rating, conservatively strict cutoffs were adopted (9 for parental alcoholism and 3 for no parental alcoholism) in hopes of minimizing false positives.

Control for phase of menstrual cycle. One complication engendered by the inclusion of female subjects is the possibility that the effects of alcohol vary as a function of phase of the menstrual cycle (B. M. Jones & Jones, 1976a, 1976b). To obtain some control over this source of variation, all female subjects in the present study were scheduled for the experiment in a 5-day window between 5 and 9 days after the end[1] of their last menstrual period. We selected this window to avoid the menstrual period itself as well as the premenstrual period, which is accompanied by large changes in hormone levels. If a woman missed a scheduled appointment and could not be rescheduled in the current 5-day window, she was rescheduled for that window in the following month.

Verification of parental alcoholism. Participants in the experiment were asked for permission to contact each of their biological parents to ask if they would be willing to complete the MAST. Permission was granted to contact a total of 256 parents from 152 different subjects. We mailed each parent a separate package consisting of a cover letter, a MAST, and a stamped return envelope. Total confidentiality was promised to both children and parents; parents were not provided with details of the experiment in which their child had participated, and parents' responses were not made available to the children. We received completed MASTs from 171 parents (67% of those contacted) of 114 experimental subjects (75% of those giving permission), with respondents fairly evenly distributed across the experimental conditions.

Apparatus

Physiological. Acquisition and online analysis of physiological functions were accomplished by using a system consisting of a Grass Model 7 polygraph and a DEC PDP 11/10 minicomputer. The resolution of this system is 1 ms for measures of time and 1 mV for measures of amplitude.

Six physiological measures were obtained. (a) Heart rate: Beckman miniature electrodes with Redux paste were placed in a bipolar configuration on opposite sides of the subject's chest. Heart rate was expressed as the cardiac period or interbeat interval (IBI) between successive R waves on the electrocardiogram (EKG). (b) Pulse transmission time to the finger (FPTT): Pulse transmission time was determined by measuring the time interval between the R wave of the EKG and the arrival of pulse pressure wave at the middle finger of the nondominant hand. A Grass photoplethysmograph detected the finger pulse. (c) Pulse transmission time to the ear (EPTT): The time interval between the R wave of the EKG and the arrival of the pulse pressure wave at the ear was measured by using a Hewlett Packard photoplethysmograph attached to the pinna of the subject's ear to detect the ear pulse. (d) Skin conductance level (SCL): A constant voltage device passed a small voltage between Beckman regular silver/silver-chloride electrodes attached to the palmar surface of the middle phalanges of the first and third fingers of the nondominant hand. The electrolyte was sodium choloride in Unibase, which allows for long-term recording without hydration-related problems. (e) General somatic activity (ACT): An electromechanical transducer attached to the platform under each subject's chair generated an electrical signal proportional to the amount of subject movement in any direction. This signal was monitored by the computer, thus providing an index of global somatic muscle activity.

The preceding five measures had all been obtained in our earlier studies and their biological meaning was described in Sher and Levenson (1982). We added a sixth measure to the present study to obtain additional information about peripheral vascular effects of alcohol. (f) Finger pulse amplitude (FPA): The trough-to-peak amplitude of the finger pulse wave was computed. This measure reflects the amount of blood flow in the finger and the underlying processes of vascular dilation and constriction. Sympathetic nervous system activation (primarily alpha-adrenergic) results in constriction of this vasculature.

Continuous rating of anxiety. In addition to the six physiological measures, a continuous self-report of perceived anxiety (ANX) was obtained by using a rating dial. This device consisted of a rotatable pointer dial whose pointer travelled over a 180-degree scale. This scale consisted of 10 divisions anchored by *extremely calm* (0°) and *extremely tense* (180°). Subjects were instructed to keep their dominant hand on the dial and to adjust it so that it always reflected their current level of tension. Subjects were free to change the dial setting as often as they wished throughout the experiment. The rating dial was attached to a potentiometer in a voltage-dividing circuit that allowed the computer to determine precisely the dial position.

Procedure

The procedures were modeled after those used in our earlier studies (Levenson et al., 1980; Sher & Levenson, 1982). Upon arriving at the laboratory, subjects signed an informed consent form and confirmed that they had complied with our instructions not to consume alcohol for 24 hr prior to the experiment and not to eat for 4 hr prior to the experiment. A blood alcohol reading was obtained using a Smith and

[1] With hindsight we realized that use of the beginning of the menstrual period would have been more accurate.

Wesson Model 900 Breathalyzer to verify that subjects had not consumed alcohol.

Beverage procedures. Height and weight were determined, and subjects were asked to gargle with Chloraseptic to attenuate taste acuity. Subjects were randomly assigned to one of two conditions of dose (1 g ethanol/kg body weight or 0 g ethanol/kg body weight) with all subjects in both conditions told that they would be consuming alcohol. The assistant poured a quantity of unsweetened grapefruit juice (equal to 3 times the weight-appropriate dose of vodka) into a graduated cylinder. A Popov's vodka bottle was removed from the refrigerator, and the amount of liquid appropriate to a 1 g/kg dose was poured into the graduated cylinder. In the 1 g/kg condition, the bottle contained Popov's 80-proof vodka. In the 0 g/kg condition, the bottle contained decarbonated tonic. Pilot research had indicated that subjects could not reliably detect the taste of vodka in this 1:3 ratio with grapefruit juice. The resulting drink was then divided equally into three glasses, and two ice cubes were added to each glass. In the 0 g/kg dose conditions, the rims of the glasses had previously been rubbed with vodka to enhance the impression that vodka was actually being consumed.

Subjects were taken to a separate room outfitted with a comfortable chair and were provided with magazines to read. Subjects were given the first glass of beverage with instructions to finish it within 15 min. The second and third glasses were brought in at 15-min intervals. At the end of this 45-min drinking period, a second breathalyzer reading was taken, and subjects were moved to another room to have the recording electrodes attached.

Stressor procedures. To continue with the deception, while the electrodes were being attached, the assistant delivered bogus blood alcohol concentration (BAC) feedback by stating, "His (her) BAC is .075, he (she) should be feeling pretty drunk about now." The assistant then returned to the original room and actually computed the BAC obtained on the last reading.

The experimenter instructed the subject on the use of the rating dial (ANX) and explained that the experiment would involve giving a speech on a topic that would be revealed later and receiving an electric shock. Subjects were told that each of these events would be signaled by a 6-min countdown. At this point a third breathalyzer reading was obtained.

The structure of the experiment was as follows. The first 7 min of the session constituted a prestressor baseline period, during which resting levels of the physiological measures and of ANX were assessed. Then the number 360 appeared on a digital display device, cuing the subject to pick up a clipboard at the side of the chair. On the clipboard were instructions to prepare a 3-min speech on the topic "What I like and dislike about my body and physical appearance." The instructions indicated that the display device would be counting down by seconds, and when it reached 0 the speech was to be delivered. Thus this "countdown phase" of the experiment lasted for 6 min. When the display reached 0 the subject began the speech, continuing until the display signaled the end of 3 min. A 5-min postspeech baseline ensued. Then the 360 appeared again, beginning a 6-min countdown to the electric shock. A single shock was administered to the wrist, and then, a final 5-min postshock baseline ended the session.

Within risk group, sex, and dose conditions, half of the subjects received the speech stressor first and the shock stressor second, as just described. The other half received the shock stressor first and the speech stressor second, with the instructions altered appropriately.

At the end of the 32-min stressor sequence, physiological recording devices were removed, and a fourth breathalyzer reading was obtained. Subjects were then prepared for a separate 15-min procedure in which cortical-evoked potentials were measured. These cortical data are not presented in this article. Following this final procedure, a debriefing questionnaire was administered, and a fifth and final breathalyzer test was given. The subject was then completely debriefed, and a taxi was provided to take him or her home.

Data Analysis

The data analysis was designed to parallel that used in our earlier studies. The analysis had two main components: (a) prestressor levels of all physiological measures and ANX and (b) physiological and ANX reactivity to the shock and speech stressors. Within each of these components, we examined four kinds of effects: (a) *alcohol effects*—those attributable to the consumption of alcohol; (b) *risk-group effects*—differences in the effects of alcohol related to risk status; (c) *sex effects*—differences in the effects of alcohol related to sex; and (d) *Risk × Sex effects*—differences in the effects of alcohol related to the interaction of risk grouping and sex.

Prestressor levels. For the analyses of prestressor levels, 2 × 2 × 2 × 2 (Parental Risk × Personality Risk × Sex × Dose) analyses of variance (ANOVAS) were performed for the average of the first 7 min of physiological and ANX recording (this constituted the 7-min period prior to the start of the countdown to the first stressor).

Reactivity. For the analyses of reactivity to the stressors, we computed difference scores by subtracting the prestressor average from each of the 102 10-s measurement periods that constituted the countdowns, stressors, and interstressor interval. Rather than carrying out a repeated-measures ANOVA with 102 measurement periods, we reduced the data by calculating the average physiological levels for six 20-s periods of maximal interest: (a) the first 20 s of the countdown prior to the shock, (b) the last 20 s of the countdown prior to the shock, (c) the first 20 s after the shock, (d) the first 20 s of the countdown prior to the speech, (e) the last 20 s of the countdown prior to the speech, and (f) the first 20 s of the speech. Selection of these reactivity periods was based on findings from our previous studies using these stressors (Levenson et al., 1980; Sher & Levenson, 1982); their locations are indicated in Figure 1 for several of the physiological variables. The resulting ANOVAS were 2 × 2 × 2 × 2 × 2 × 3 (Parental Risk × Personality Risk × Sex × Dose × Stressor Type × Stressor Period) with stressor type and stressor period treated as within-subjects factors.

When significant F ratios were obtained for within-subjects factors having more than two levels (e.g., stressor period), a Geisser-Greenhouse conservative F test was performed. If this conservative test indicated that the F ratio might not be significant, the Huynh-Feldt epsilon was computed and the degrees of freedom reduced accordingly. Unless otherwise noted, all reported F ratios remained significant at the .05 level with these reduced degrees of freedom.

Planned comparisons. Differences between the 1 g/kg and 0 g/kg doses of alcohol were of primary interest in this experiment. This reflected the two primary research questions: (a) What does alcohol do? and (b) Are there differences in what alcohol does that are associated with risk factors for alcoholism or sex? Thus within significant main effects or interactions of interest, the analytic strategy was always to compare means from comparable subjects at the two doses.

When significant effects and interactions involved more than two means, planned comparisons were carried out using t tests. The pooled error terms for these comparisons were calculated using established procedures for "split-plot" designs (Kirk, 1968); the number of degrees of freedom used was the smaller of two associated with the two pooled error terms. Because a number of these comparisons were performed, a Bonferroni adjustment was made to reduce the risk of Type I errors. To be considered significant at the .05 level (one-tailed), a t value had to surpass the critical value for the .005 level (one-tailed) of significance.

Results

Manipulation Checks

Ratings of intoxication and of the number of ounces of alcohol consumed were obtained from subjects at the end of the experiment. Compared with subjects who consumed the 0 g/kg

Figure 1. Heart rate interbeat interval (IBI), ear pulse transmission time (EPIT), and finger pulse amplitude (FPA) responses to stressors with periods of maximal reactivity indicated. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates higher levels of arousal. A = start of countdown to shock; B = end of countdown to shock; C = shock; D = start of countdown to speech; E = end of countdown to speech; and F = speech. Arrows point to the beginning of each 20-s reactivity period.)

dose, those who consumed the 1 g/kg dose rated themselves as being more intoxicated following drinking and as having consumed more ounces of alcohol. The means for these variables are presented in Table 2. For intoxication ratings, these findings were consistent across all factors of sex and risk. For ratings of ounces consumed, there was a significant higher order interaction of Parental Risk × Personality Risk × Sex × Dose, $F(1, 222) = 5.53$. Examination of the means of this interaction revealed that within all but two risk–sex groupings, subjects who received the 1 g/kg dose thought they consumed more ounces of alcohol than those who received the 0 g/kg dose. Within groupings of low-risk women and parental-risk men, subjects consuming the two doses did not differ in the number of ounces thought consumed.

Verification of Parental Alcoholism

Agreement between MASTs completed by subjects and MASTs completed by the parents who responded to our mailing was quite high, especially for fathers. The correlations between MAST scores completed by subjects and by their parents were significant both for fathers, $r(75) = .85$, and for mothers, $r(92) = .52$. A comparison between these two correlations using Fisher's z transformation revealed that the correlation with fathers' MASTs was significantly higher than the correlation with mothers' MASTs ($z = 4.34$).

Table 2
Subjects' Ratings of Intoxication and Consumption

Measure	Dose		$F(1, 222)$
	Alcohol (1 g/kg)	No alcohol	
Drunkenness after drinking[a]	6.74	3.01	238.20*
Estimated ounces of alcohol consumed	4.51	2.18	103.81*

[a] Rated on a scale ranging from 1 to 10.
* $p < .001$.

Effects of Alcohol on Prestressor Levels

Alcohol had a marked effect on prestressor levels of most physiological variables and ANX. Compared with subjects who consumed no alcohol, subjects who consumed alcohol had shorter IBI (i.e., faster heart rate), greater ACT, longer FPTT, larger FPA, and lower ANX (Table 3).

Effects of Alcohol on Reactivity to the Stressors

Alcohol had significant effects on reactivity to stress in all four cardiovascular variables (IBI, EPTT, FPA, and FPTT). Descriptions of these effects follow; means and t values for the major comparisons are presented in Table 4. Alcohol had no significant overall effects on reactivity for the noncardiovascular physiological measures (ACT and SCL) or for ANX.

Reduced IBI response to stress. Alcohol consumption reduced the magnitude of the IBI response to stress. This was indicated by a significant main effect for dose, $F(1, 227) = 14.61$. Both the Dose × Stressor Period, $F(2, 454) = 21.03$, and the Dose × Stressor Type × Stressor Period, $F(2, 452) = 6.96$, interactions were also significant. Examination of these interactions

Table 3
Alcohol Effects on Prestressor Levels of Physiological Measures and Anxiety

Measure	Dose		
	Alcohol (1 g/kg)	No alcohol	$F(1, 227)$
IBI (ms)	736.34	783.86	9.57*
ACT	0.40	0.32	6.33*
SCL (μmho)	8.22	6.80	3.54
FPT (ms)	240.38	230.24	12.22**
FPA	88.00	48.86	18.45**
EPT (ms)	175.19	171.02	3.42
ANX	3.54	3.97	4.34*

Note. IBI = heart rate interbeat interval. ACT = general somatic activity. SCL = skin conductance level. FPTT = finger pulse transmission time. FPA = finger pulse amplitude. EPTT = ear pulse transmission time. ANX = self-reported anxiety.
* $p < .05$. ** $p < .001$.

revealed that alcohol produced significant stress-response dampening of IBI only during the stressors and that this occurred for both the speech stressor, $t(227) = -3.80$, and the shock stressor, $t(227) = -5.89$.

Reduced EPTT response to stress. Alcohol consumption reduced the magnitude of the EPTT response to stress. This was indicated by a significant main effect for dose, $F(1, 225) = 4.55$. Examination of the significant Dose × Stressor Period interaction, $F(2, 450) = 23.37$, revealed that alcohol produced significant stress-response dampening of EPTT only during the stressors.

Increased FPTT and FPA response to stress. Alcohol consumption increased the magnitude of FPTT and FPA responses to stress. For FPTT, the main effect for dose was not significant, $F(1, 225) < 1$, but the interaction of Dose × Stressor Period was significant, $F(2, 450) = 11.31$. Examination of means within this interaction revealed that alcohol increased FPTT response only at the start of the countdown. For FPA, the main effect for dose was significant, $F(1, 225) = 5.76$, as was the interaction of Dose × Stressor Period, $F(2, 450) = 6.84$. Examination of means within this interaction revealed that alcohol increased the FPA response at the end of the countdown and during the stressor.

Reduced rated stressfulness of shock. On the debriefing questionnaire administered at the end of the experiment, subjects were asked to rate the stressfulness of the speech stressor as well as the stressfulness and painfulness of the shock stressor using scales ranging from 0 to 10. Alcohol reduced the rated stressfulness of the shock stressor (alcohol = 5.67, no alcohol = 6.43) $F(1, 224) = 5.80$. Alcohol's reduction of the rated painfulness of shock stressor approached significance (alcohol = 4.94, no alcohol = 5.57) $F(1, 225) = 3.64$, $p = .054$.

Blood Alcohol Level: Risk Group and Sex Differences

The four blood alcohol measures taken after drinking (see Procedures section) were analyzed in a 2 × 2 × 2 × 2 × 4 (Parental Risk × Personality Risk × Sex × Dose × Time of Measurement) ANOVA. As would be expected, the main effect for dose, $F(1, 226) = 1302.81$, was highly significant. Of greater interest for the present study, however, was evidence that BAC levels reached after drinking differed as a function of personality risk.

Differences in BAC levels related to personality risk were reflected in a significant Personality Risk × Dose × Time of Measurement interaction, $F(3, 652) = 3.20$. Planned comparisons revealed that personality risk subjects obtained lower BAC levels at the end of drinking than did subjects without this risk factor, $t(226) = 4.54$. As can be seen in Figure 2, this difference between risk groups subsequently decreased, becoming nonsignificant at the final three measurement points. Only the personality risk factor showed a relation with BAC; all main effects and interactions involving the parental risk and sex factors were nonsignificant.

Effects of Alcohol on Prestressor Levels: Risk Group and Sex Differences

In general, little evidence was found that the effects of alcohol on prestressor levels were related to risk or sex. Where significant main effects for dose were found for prestressor levels, the higher order interactions with factors of risk and sex were not significant. The only exception was for IBI, for which a significant interaction of Parental Risk × Personality Risk × Dose was found, $F(1, 227) = 5.39$. Examining the means of this interaction revealed that alcohol produced significant increases in prestressor heart rate only in the personality risk group (alcohol = 709.31 ms, no alcohol = 793.39 ms), $t(227) = 2.84$. For EPTT, the dose main effect was not significant, but the interaction of Parental Risk × Sex × Dose was significant, $F(1, 225) = 5.01$. Examination of the means of this interaction revealed that alcohol lengthened prestressor EPTT levels only for

Table 4
Alcohol Effects on Cardiovascular Reactivity to Stressors

Measure and stressor period	Dose		
	Alcohol (1 g/kg)	No alcohol	$t(227)$
IBI (ms)			
Begin countdown	−39.76	−43.30	−0.51
End countdown	−60.61	−77.61	−2.47
Stressor	−99.49	−144.95	−6.61*
EPT (ms)			
Begin countdown	−2.44	−0.85	1.54
End countdown	−7.64	−9.34	−1.63
Stressor	−11.76	−16.07	−4.15*
FPA			
Begin countdown	−1.70	1.22	0.71
End countdown	−18.79	−6.27	3.04*
Stressor	−19.66	−6.76	3.13*
FPTT (ms)			
Begin countdown	−2.92	1.03	2.73*
End countdown	−8.16	−7.48	0.47
Stressor	−12.19	−14.18	−1.38

Note. IBI = heart rate interbeat interval. EPTT = ear pulse transmission time. FPA = finger pulse amplitude. FPTT = finger pulse transmission time.
* $p < .005$.

Figure 2. Breathalyzer readings in relation to personality risk. (The second reading was obtained after drinking; the third reading was obtained before the start of the prestressor period; the fourth reading was obtained after the stressor experiment; and the fifth reading was obtained at the end of the experimental session).

female subjects with no parental history of alcoholism (alcohol = 177.33 ms, no alcohol = 164.01 ms), $t(225) = -3.13$).

Effects of Alcohol on Reactivity to the Stressors: Risk Group and Sex Differences

There was considerable evidence that individual differences in the effects of alcohol on IBI, EPTT, ACT, and ANX reactivity to stress were related to risk factors for alcoholism. All effects of alcohol found to be unique to one or more risk groups are summarized in Table 5; more detailed descriptions follow.

IBI: Greater stress-response dampening associated with personality risk. As indicated earlier, the overall effect of alcohol was to diminish the magnitude of the IBI response to stress. This stress-response-dampening effect was found to be more pronounced in personality risk subjects than in those without this risk factor. This finding was reflected in a significant interaction of Personality Risk × Sex × Dose × Stressor Period, $F(2, 454) = 4.01$. For men, only subjects with the personality risk factor showed the stress-response-dampening effect of alcohol during the stressor period, $t(227) = -3.60$. For women, only subjects with the personality risk factor showed the stress-response-dampening effect at the end of the countdown, $t(227) = -3.02$. During the stressors, both high-risk women, $t(227) = -2.75$, and low-risk women, $t(227) = -4.38$, showed the stress-response-dampening effect.

EPTT: Greater stress-response dampening associated with parental risk. As already indicated, the overall effect of alcohol was to diminish the magnitude of the EPTT response to stress. This effect proved to be more pronounced in subjects with the parental alcoholism risk factor than in subjects without this risk factor. This finding was reflected in significant interactions of Parental Risk × Dose, $F(1, 225) = 4.22$, Parental Risk × Dose × Stressor Period, $F(2, 450) = 3.12$ (epsilon adjusted $p = .051$), and Parental Risk × Dose × Stressor Type × Stressor Period, $F(2, 440) = 4.34$. Examination of the means of these

Table 5
Effects of Alcohol on Reactivity to Stressors Unique to Risk Groups

Risk grouping and effects	Dose: Alcohol (1 g/kg)	Dose: No alcohol	$t(227)$
High risk: Personality			
Decreases IBI response to stressors in males (ms)	−108.27	−158.38	−3.60*
Decreases IBI response to end of countdown in females (ms)	−33.10	−74.65	−3.02*
Increases ANX response to end of countdown	1.93	0.85	−2.68*
High risk: Parental alcoholism			
Decreases EPTT response to stressors (ms)	−8.81	−16.25	−5.07*
Decreases ACT response to shock stressor in males	0.32	0.87	4.74*

Note. IBI = heart rate interbeat interval. ANX = self-reported anxiety. EPTT = ear pulse transmission time. ACT = general somatic activity.
* $p < .005$.

interactions revealed that alcohol diminished the EPTT response only during the stressor period for subjects with a history of parental alcoholism, $t(225) = -5.07$, and that this effect occurred for both the speech stressor, $t(225) = -3.07$, and the shock stressor, $t(225) = -4.27$.

ACT: Greater stress-response dampening associated with parental risk. Unlike IBI and EPTT, alcohol had no overall effect on ACT reactivity. However, significant interactions of Parental Risk × Sex × Dose × Stressor Type, $F(1, 227) = 5.93$, and of Parental Risk × Sex × Dose × Stressor Type × Stressor Period, $F(2, 452) = 3.20$, were found. Examination of the means of these interactions revealed that alcohol reduced the magnitude of the ACT response to the shock stressor only for male subjects with a history of parental alcoholism, $t(227) = 2.62$, and that this effect was found only during the stressor period itself, $t(227) = 4.74$.

ANX: Greater stress-response enhancement associated with personality risk. As was the case with ACT, alcohol had no overall effect on ANX reactivity. However, a significant interaction of Parental Risk × Personality Risk × Dose × Stress Period was found, $F(2, 454) = 4.08$. Examination of the means of this interaction revealed that the only significant difference associated with alcohol consumption was for subjects with the personality risk factor at the end of the countdown, where the 1 g/kg dose was associated with a greater ANX response than was the 0 g/kg dose, $t(227) = -2.68$.

Effects of Alcohol on Reactivity to the Stressors: Parental Alcoholism as Reported by Parents

An additional internal analysis was carried out using data only from subjects whose parents had completed the MASTs. Because of the reduced sample size, a full factorial ANOVA strategy could not be used. Instead, within each dose condition, scores on MASTs completed by parents were correlated with subjects' physiological changes during the stressors and during the end of the countdown (the two stressor periods where relations with risk factors had been found in the analyses based on parental MASTs completed by the subjects). We used these correlations to examine the extent to which increasing levels of parental alcoholism (as indicated by the parent's own MASTs) were associated with the magnitude of physiological responses to the stressor in subjects who received alcohol and in those who did not.

In the alcohol condition, increasing levels of paternal alcoholism ($N = 37$) were associated with (a) smaller IBI response to the shock ($r = .35$), (b) smaller EPTT response to the shock ($r = .40$), (c) smaller EPTT response to the end of the countdown to the speech ($r = .37$), (d) smaller EPTT response to the speech ($r = .49$), (e) larger FPA response to the end of the countdown to the shock ($r = -.40$), and (f) larger FPA response to the shock ($r = -.46$). Maternal alcoholism in the alcohol condition ($N = 50$) was not associated with any differences in physiological response.

In the no alcohol condition, neither paternal alcoholism ($N = 40$) nor maternal alcoholism ($N = 44$) was associated with any differences in physiological response.

Effects of Alcohol on Prestressor Levels and on Reactivity to the Stressors: Additional Sex Differences

Beyond those interactions between sex and risk factors already reported, there were no other indications that alcohol differentially effected physiological variables or ANX in male and female subjects.

Discussion

Manipulation Checks

Subjects' ratings of intoxication and of the number of ounces of alcohol consumed were comparable with those obtained in a previous experiment in which all subjects were also told that they were consuming alcohol (Sher & Levenson, 1982). As in that previous study, the ploys used to increase the believability of the alcohol manipulation in the 0 g/kg condition met with some success; subjects in the present study reported experiencing a nontrivial level of intoxication (4.51 on a 10-point scale) and reported having consumed more than 2 oz of alcohol, but the deception was far from perfect. Lack of a balanced placebo design in this study, combined with the inability to completely deceive subjects, make it impossible to separate the pharmacologic effects of alcohol from those associated with psychological expectancies. In an earlier study that used the full balanced placebo design (Levenson et al., 1980), we concluded that pharmacological effects of alcohol were prepotent over the psychological effects at the 1 g/kg dose. The discussion that follows reflects our continuing belief in this conclusion.[2]

Verification of Parental Alcoholism

Because designation of risk was based on subjects' reports of their parents' drinking problems, a comparison between these reports and those obtained directly from their parents was needed. Correlations obtained between MASTs obtained from parents and from their children were significant and quite high. This level of agreement, combined with the conservative cutoffs adopted for the high- and low-risk groupings, lends confidence to the classification of risk status used in this experiment.

Interestingly, when children completed MASTs for their parents, there was much higher agreement for fathers than for mothers (this higher agreement was obtained regardless of whether the child was male or female). Possible explanations for this finding include (a) statistical properties of the sample (e.g., different amounts of variability in the distribution of fathers' and mothers' MASTs) (b) more public drinking behavior of men in the age cohort represented by these parents, and (c) differences in accuracy of reporting by men and women.

[2] Use of the balanced placebo design by itself does not guarantee separation of the pharmacological and psychological effects of alcohol because subjects still have to believe what they are told they are drinking. With high doses and experienced drinkers, complete believability remains an elusive goal in the two deception cells of the balanced placebo design.

Effects of Alcohol

Prestressor levels. One advantage to maintaining similar procedures across studies is the opportunity that is provided to determine which findings replicate and which are less reliable. Alcohol's effects on prestressor levels have been strikingly consistent across this study and our two previous studies (Levenson et al., 1980; Sher & Levenson, 1982, Experiment 2). In all three studies, compared with the 0 g/kg condition, the overall effect of the 1 g/kg dose of alcohol was to (a) increase heart rate, (b) lengthen FPTT, and (c) not change EPTT. Effects that were significant in two studies and that were in the same direction and approached significance in the third study were that alcohol (a) increased SCL (significantly in our two earlier studies and nonsignificantly, $p = .058$, in the present study) and (b) reduced ANX (significantly in the present study and the earliest study and nonsignificantly in the other study).

There were two findings unique to the present study, one involving a new measure and the other involving an old one. The first of these was that FPAs were larger in the 1 g/kg condition than in the 0 g/kg condition. This measure had not been obtained in the previous studies. It was added to obtain a better understanding of alcohol's effects on the reactivity of the peripheral vasculature. The finding that alcohol increased blood flow in the finger is completely consistent with alcohol's known effect as a peripheral vasodilator, which is responsible for the flushing response associated with alcohol consumption. The second finding involved somatic activity, which was measured in all three studies. Only in the present study did alcohol have an effect on this measure, with greater somatic activity in the 1 g/kg dose than in the 0 g/kg dose.

The robustness of these prestressor effects is striking. Not only have most of them replicated across multiple experiments but they have proved to be quite robust to differences in psychological expectancy (Levenson et al., 1980), personality factors thought to predispose subjects to alcoholism (Sher & Levenson, 1982; and the present study), the presence of parental alcoholism (the present study), sex (the present study), and to various combinations of risk factors and sex (the present study). The only two exceptions to this characterization of findings occurred in the present study (i.e., alcohol increasing prestressor heart rate only in personality risk subjects and alcohol lengthening prestressor EPTT only in female subjects without a history of parental alcoholism). Taken together these findings support the position that the kinds of prestressor effects of alcohol studied in these experiments are largely pharmacological and that the modulating role played by psychological and individual difference factors is minimal.

Reactivity to stressors. As had been the case in each of our previous studies, alcohol significantly reduced the magnitude of the heart rate and EPTT responses to stress. We have previously referred to these effects as the "stress-response-dampening" effects of alcohol. In addition to consistency across studies in which physiological variables alcohol did effect, there has also been consistency in which physiological variables alcohol did not effect. In all of our studies, alcohol has had no significant overall effect on SCL or ACT responses to stress. This has also been the case for FPTT, with the exception of the start of the countdown in the present study.

Turning to the self-report realm, in the present study alcohol did not alter the ANX response to stress. This finding is consistent with our most recently published study (Sher & Levenson, 1982, Experiment 2) using these methods. Thus only in our first study (Levenson et al., 1980) did alcohol significantly reduce the ANX response to stress. However, on the present study's debriefing questionnaire completed at the end of the experiment, subjects who consumed alcohol rated the shock as being less stressful than did those who had not consumed alcohol.

Considering just the findings for IBI, EPTT, ANX, and the stressfulness ratings, the picture is overwhelmingly one of alcohol reducing cardiovascular responses to stress and reducing some aspects of the subjective distress produced by the stressors. However, this consistent picture is complicated by one deviation: the results for the newly added measure of FPA.

In a footnote to a recent article (Sher & Levenson, 1982, footnote 8), we noted our growing realization that the stress-response-dampening effect of alcohol might not exist in the periphery. Of particular concern at that time was our consistent finding that alcohol did not diminish the skin conductance response to stress, and some evidence that it might actually increase skin conductance responding, albeit nonsignificantly. The measure of FPA was added to the present study to obtain additional data on another aspect of peripheral responding, in this case related to the blood vessels rather than to the sweat glands.

In the present study, peripheral vasoconstriction (i.e., decreased FPA) was the modal response to the stressors, a finding in keeping with other stress research using this measure and the general understanding of the action of the sympathetic nervous system in response to stress (peripheral vasoconstriction is primarily mediated by the alpha-adrenergic branch of the sympathetic nervous system). However, in contrast to the effects of alcohol on the IBI and EPTT components of the cardiovascular system's response to stress (both of which were reduced in magnitude by alcohol), the magnitude of the FPA response to stress was increased by alcohol. Although we have not used this measure before, there is no reason to believe that this finding will not replicate as reliably as have the cardiovascular stress-response-dampening effects for IBI and EPTT.

Thus use of the term *stress-response dampening* to describe alcohol's effects on psychophysiological responses to stress needs to be restricted so that it refers only to certain aspects of the cardiovascular system's response to stress. On the basis of what is known about underlying mechanisms, we would posit that the stress-response-dampening effect will be limited to those aspects of the cardiovascular system's response to stress that are mediated primarily by beta-adrenergic action. Additional research is needed to determine whether a *stress-response-enhancing* effect will be found for all alpha-adrenergically mediated vascular responding,[3] or if this effect will be found only in extreme peripheries such as the finger tips.

[3] An astute and anonymous reviewer of this article commented that the reduced beta-adrenergic responsivity produced by alcohol (which we have postulated as the basis for the stress-response dampening of IBI and EPTT) could be involved in the FPA findings as well. To the extent that beta-adrenergic drive produces vascular dilation in the peripheral arteries, its reduction by alcohol could allow alpha-adrenergically medi-

Law of initial values. To those familiar with the tenets of psychophysiology, the impact of the law of initial values on these findings needs to be addressed. Simply stated, the law of initial values recognizes the existence of floor and ceiling limits on levels of physiological functions and warns that as initial levels approach these limits, physiological reactivity can become attenuated in magnitude or even reversed in direction. The concern here would be that differences in prestressor physiological levels between the 0 g/kg and 1 g/kg dose groups would be responsible for observed differences in reactivity found for these two dose conditions. In the case of heart rate, alcohol increases prestressor heart rate, thus moving it toward its natural ceiling. Thus according to the law, this could be partly responsible for the finding that alcohol reduces the magnitude of heart rate increase to stress. Two points need to be made about this concern. First, only modest changes in prestressor physiological levels are produced by the 1 g/kg dose of alcohol (e.g., approximately a 5-bpm increase in heart rate); these levels do not come close to biological limits. Second, in the case of EPTT, the effect of alcohol on prestressor levels is to lengthen nonsignificantly EPTT. Unlike heart rate, this prestressor effect is in the direction opposite that of the effect of the stressors on EPTT (i.e., shorter EPTT in response to stress). Thus the finding that alcohol reduces the EPTT response to stress cannot be explained in any way by the law of initial values. In terms of stress-response dampening of IBI and EPTT in the present experiment, the law of initial values, although worth heeding, is unlikely to be of great consequence.

Modulating Role of Risk for Alcoholism

Although this study provided a considerable amount of information on the psychophysiological effects of alcohol, its main intent was to determine whether the often observed individual differences in the effects of alcohol were related to variables thought to indicate heightened risk for future alcoholism. A number of these relations were found, indicating that individual differences in the effects of alcohol are organized around factors meaningful to the future development of alcoholism.

More pronounced stress-response-dampening effect of alcohol in high-risk subjects. On the basis of our previous findings, we hypothesized that subjects at heightened risk for alcoholism were predisposed by nature or by nurture to receive an added increment of the stress-response-dampening effects of alcohol. In these earlier studies (Sher & Levenson, 1982) high-risk male subjects matching the prealcoholic personality profile received more of the stress-response-dampening effects from alcohol on IBI (in two experiments), EPTT (in one experiment), and ANX (in one experiment) than did their low-risk counterparts. The present study offered an opportunity to both replicate and expand these findings.

Table 5 lists each effect of alcohol on reactivity to the stressors that was unique to a risk group in the present study. The previous finding that alcohol dampens IBI responding to the stressors for personality risk male subjects was replicated for a third time. IBI responding to the end of the countdown was also found to be dampened by alcohol for personality risk female subjects. In the present experiment, as well as in the previous experiments, alcohol did not produce these stress-response-dampening effects in the comparison subjects who did not have this personality risk factor.

For the first time in our research program, a second kind of alcoholism risk was examined: risk associated with parental alcoholism. The results revealed that a greater stress-response-dampening effect of alcohol was associated with this risk factor as well. In high-risk subjects with a history of parental alcoholism, alcohol produced diminished EPTT responding to the stressors. Among high-risk male subjects during the shock stressor, alcohol produced diminished ACT responding as well. In the comparison group of subjects without parental alcoholism, alcohol did not produce these stress-response-dampening effects.

These findings that risk for alcoholism was associated with greater stress-response-dampening effects of alcohol received additional support from the internal correlational analyses of data from subjects whose parents completed the MAST. Increasing levels of paternal alcoholism were associated with a greater stress-response-dampening effect of alcohol for EPTT. As was the case in the full group data, this effect was found for EPTT during the stressors, and in addition, it was found for EPTT during the end of the countdown to the speech. Several other aspects of this internal analysis produced significant results. Increasing levels of paternal alcoholism were associated with greater stress-response dampening of the IBI response to the shock. In addition, the effect of alcohol on FPA response to stress (i.e., increased vasoconstriction) became more pronounced as paternal MASTs increased. Finally, there were no relations between either parent's MAST and responses to stress in the sober condition, thus these relations only emerged when alcohol had been consumed.

The findings from this experiment, together with those from the previous experiments, provide strong evidence that both personality risk and parental risk are associated with greater stress-response-dampening effects of alcohol on certain physiological responses to stress. Thus far, this phenomenon has been limited to two measures of cardiovascular functions (IBI and EPTT) and one noncardiovascular physiological measure (ACT). Individual differences in other measured cardiovascular functions either have not been related to the two risk factors studied thus far (i.e., FPTT) or have shown a relation only in an internal analysis of paternal alcoholism (i.e., FPA). The remaining noncardiovascular physiological measure (SCL) has been unrelated to these risk factors. Results for the single nonphysiological measure (ANX) have been inconsistent. In one experiment there was evidence that alcohol produced reduced ANX responding to stress only in personality risk subjects (Sher & Levenson, 1982, Experiment 1). In the present study there was evidence of alcohol producing a quite different effect, increased ANX responding at the end of the countdown only in personality risk subjects. In the third study, no relation was found between personality risk and alcohol's effect on ANX responding (Sher & Levenson, 1982, Experiment 2).

Specificity and additivity of risk. At first glance, there appears to be some evidence of a specific relation between the type of

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ated vasoconstrictive responses even greater predominance in response to stress.

risk and the particular cardiovascular function for which alcohol produces greater stress-response dampening. In group data, personality risk was related to alcohol's reduction of IBI response to stress, whereas parental risk was related to EPTT. This level of specificity would be a very interesting finding if it proved to be reliable; however, other available evidence suggests that this will not be the case. Analysis of parental risk based on parents' own responses on the MAST indicated that both IBI and EPTT response were related to paternal alcoholism. In addition, in an earlier study (Sher & Levenson, 1982, Experiment 1) personality risk was also found to be associated with both IBI and EPTT response. On the basis of these findings, it seems likely that parental risk and personality risk will ultimately be determined to have the same relation with stress-response dampening (i.e., both risk factors will be associated with greater stress-response dampening in both IBI and EPTT).

Finally, no evidence was found for an additivity of effects associated with multiple risk. High-risk subjects who met both parental and personality risk criteria did not appear to receive any additional increment of the stress-response-dampening effects of alcohol.

Greater Reinforcement From Drinking for Those at Risk?

We will now consider the extent to which the differential effects of alcohol associated with personality risk and parental risk support the assertion that high-risk subjects receive incrementally greater reinforcement from drinking than do their low-risk counterparts. The notion that differential reinforcement from drinking might play a role in alcoholism is not without precedent. For example, speculation that low incidence of alcoholism among Asians may be due to their exaggerated peripheral vascular response to ethanol (e.g., Wolff, 1972) could be recast in terms of Asians' receiving greater punishment from drinking. Although in the present experiment the emphasis is on differences in *positive* reinforcement, the principle is the same.

This discussion will focus on alcohol's effects on *reactivity to stress* because it was there that the most pronounced differences between risk groups were found.[4] The findings for the effects of alcohol on physiological reactivity in the variables of IBI, EPTT, and ACT are quite consistent. In terms of these variables, alcohol is a more potent minimizer of the physiological perturbations produced by stress for high-risk subjects than it is for low-risk subjects. But should this be considered "greater reinforcement"?

At the simplest level, anything that minimizes disruption, reduces the impact of stressful environmental events, and makes the world more manageable can be considered reinforcing. This is an old notion that has parallels both in drive theory and in the tension-reduction model of alcohol consumption. Taking this model to its next logical step, people who are predisposed to obtain an extra increment of reinforcement from drinking would be more likely to repeat the antecedent drinking behavior than would people for whom drinking delivers less reinforcement or no reinforcement at all. Thus these individuals, by drinking more, are likely to move more rapidly along the path to increased tolerance, addiction, and alcoholism than are those who receive less reinforcement from drinking. It is in this way that the reinforcement value of a short-term effect (i.e., greater stress-response dampening produced by alcohol) provides a mediative bridge between individual difference factors (i.e., parental risk and personality risk) and a long-term effect (i.e., alcoholism).

Before leaving this discussion, some mention should be made of the findings indicating that personality risk subjects might metabolize alcohol differently from those not at risk. Within the limitations inherent in the breathalyzer methodology, our findings indicate that at a 1 g/kg dose, personality risk subjects do not reach as high a blood alcohol level immediately following drinking as do subjects without this risk factor. If this finding proves reliable, it could also have etiological significance. To the extent that subjective cues of intoxication are related to BAC levels, high-risk subjects might be inclined to engage in incrementally greater drinking because they would receive fewer immediate intoxication cues (subsequent BAC measurements indicated that this difference did not persist over time). In the natural environment, by the time BACs for high-risk subjects and low-risk subjects would have converged, high-risk subjects may well have engaged in additional drinking. Admittedly, this notion is speculative and rests on several untested assumptions regarding the relation between BAC level and intoxication cues. However, if it is true, and if intoxication cues are considered to be aversive, it could represent yet another way in which drinking would be more reinforcing (i.e., associated with fewer aversive intoxication cues) for personality risk subjects.

Limited Modulating Role of Sex

Considerable effort was expended to control for phase of menstrual cycle in female subjects in this experiment. BACs obtained in the study confirmed our pilot findings that sex differences in absolute levels of BACs reached with a weight-corrected dose and sex differences in rates of alcohol metabolism could be eliminated by scheduling female subjects in the 5-day window starting 5 days after the end of their menstrual cycle. Elimination of sex differences in BAC levels enables unconfounded tests of sex differences in the effects of alcohol and in the interaction of these effects with risk factors. For investiga-

[4] Excluded from this discussion are risk-group differences in the effects of alcohol on prestressor levels because these were limited in number and are difficult to characterize in terms of reinforcement (e.g., Is increased prestressor heart rate in personality risk subjects reinforcing?). Similarly, the finding from the internal correlational analyses that higher paternal MASTs were associated with pronouncement of the effect of alcohol on FPA response to stress (i.e., greater vasoconstriction) is not included because it was not found in the overall analyses. If this finding proves to be reliable, it would not fit with the notion of greater stress-response dampening. It would fit with the notion that the effects of alcohol (stress-response dampening and stress-response enhancing) are more pronounced in subjects at high risk for alcoholism. As to whether such an effect could be reinforcing, the case could be made on an evolutionary basis that greater vasoconstriction would be reinforcing by virtue of its value in reducing bleeding resulting from injury. However, this kind of reinforcement would be quite different from that being proposed for the stress-response-dampening effects of alcohol on IBI and EPTT.

tors wishing to minimize menstrual cycle related sex differences, the screening and sampling procedures we adopted in the present study appear highly effective.

Our findings suggest that with these kinds of carefully matched male and female samples, there is much more that is similar than is different. In addition to there being no differences in BAC levels, for those variables in which alcohol had an overall effect on prestressor levels or on responses to stress, these effects were consistent across sex. A few instances of sex differences in the effects of alcohol were found within higher order interactions involving the risk factors, and these were described earlier. Most important, nothing in these findings indicated that either men or women had an exclusive franchise on the relation between heightened risk for alcoholism and enhanced stress-response-dampening effects of alcohol. Thus both sexes may be equally vulnerable to any role that these factors play in the etiology of alcoholism.

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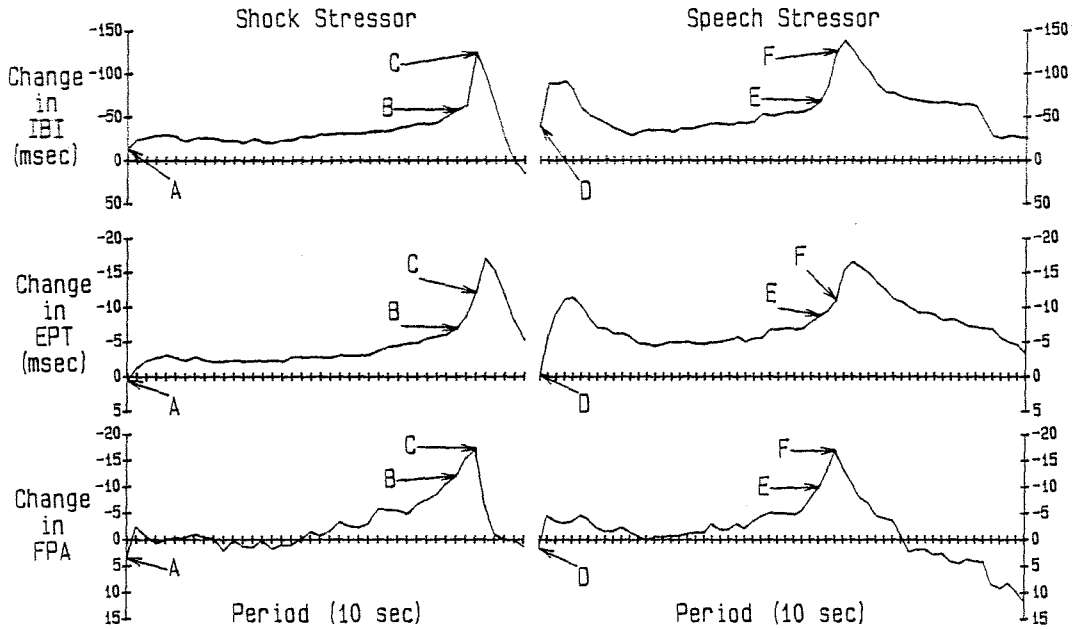
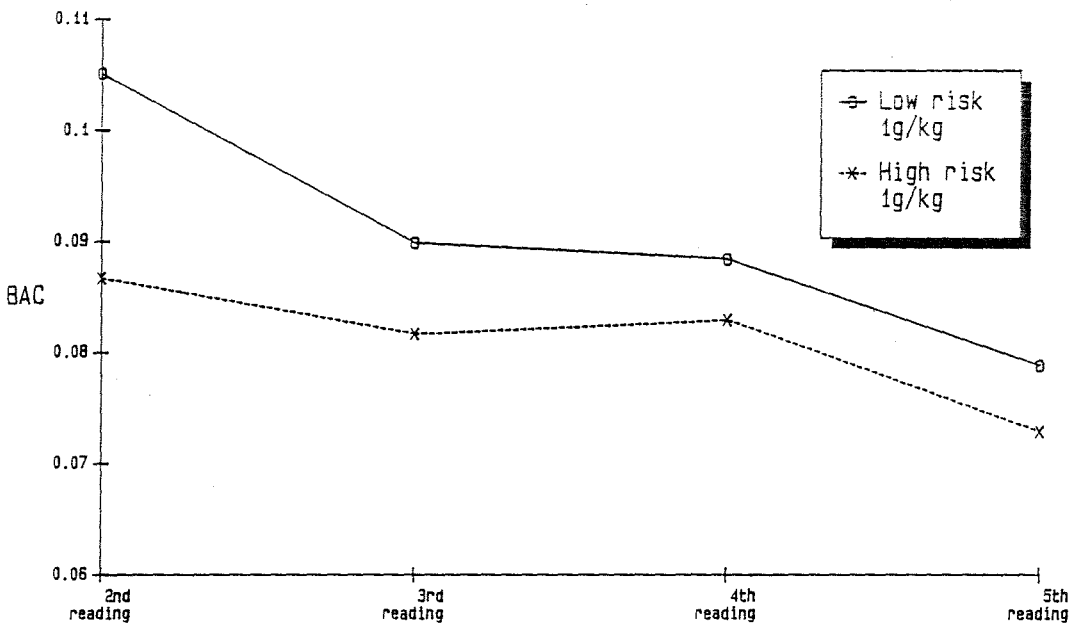

Shock Stressor
Speech Stressor
Change in IBI (msec)
Change in EPT (msec)
Change in FPA
A
B
C
D
E
F
Period (10 sec)
Period (10 sec)

54.5


0.11
0.1
0.09
0.08
0.07
0.06
BAC
Low risk 1g/kg
High risk 1g/kg
2nd reading
3rd reading
4th reading
5th reading