Risk for Alcoholism and Individual Differences in the Stress-Response-Dampening Effect of Alcohol

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In two experiments, individual differences in the effects of a 1 g/kg dose of alcohol on physiological and affective responses to stress were found to be related to characteristics thought to relate to risk for alcoholism. In the first study of 95 male nonalcoholics, subjects considered to be at heightened risk for alcoholism on the basis of high scores on the MacAndrew Alcoholism scale (MAC) of the Minnesota Multiphasic Personality Inventory (MMPI) were found to show much more pronounced reduction of their cardiovascular and affective responses to stress when they consumed alcohol than did their low-risk controls. In the second study of 82 male nonalcoholics, a similar finding for cardiovascular responses to stress was observed when risk was predicated on the basis of low scores on the Socialization (So) scale of the California Psychological Inventory (or on the basis of combined risk on both the MAC and So). These results are interpreted as indicating that the outgoing, aggressive, impulsive, and antisocial individuals who are identified by these measures (and who have been shown to have a high incidence of alcoholism in prospective studies) may find alcohol consumption particularly reinforcing by virtue of their obtaining a greater amount of alcohol's stress-response-dampening (SRD) effect when they drink. These findings along with those concerned with the effects of alcohol on prestressor physiological and affective levels are discussed, and an etiological model of alcoholism that incorporates individual differences in the SRD effects is proposed.

Of all the purported properties of alcohol, perhaps none has received as much lay acceptance or scientific investigation as has "tension reduction." In spite of the common assumptions that alcohol can reduce tension and that alcohol is often consumed for this desired effect, research seeking to determine the exact relationship between alcohol and tension reduction has produced equivocal and contradictory findings (Cappell, 1975; Cappell & Herman, 1972). There appear to be both methodological and theoretical reasons for this apparent lack of harmony in the

data. Tension (or stress) has been equated with a wide range of experimental manipulations and dependent variables, including behavioral and self-report measures of anxiety and other affective states (e.g., Marlatt, 1976), autonomic arousal and reactivity (e.g., Naitoh, 1972), and muscular tension (Steffen, Nathan, & Taylor, 1974). Typically, the correlations among these different categories of measures are only modest. Additional complexity derives from the dose dependency of the tension-reducing properties of alcohol, with considerable stress response dampening (SRD) reported at higher doses (Levenson, Sher, Grossman, Newman, & Newlin, 1980; Wilson, Abrams, & Lipscomb, 1980), but more equivocal findings at lower doses (Wilson et al., 1980). To this must be added further complications associated with subjects' expectancies (Rohsenow & Marlatt, 1981), limb of the blood-alcohol curve (B. Jones, Parsons, & Rundell, 1976), the nature of the stressor (Higgins & Marlatt, 1973, 1975), and individual difference factors, all

of which may influence the tension-reducing effects of alcohol.

Individual Differences and the Effects of Alcohol

The notion that individuals differ in their susceptibility to the effects of alcohol is widely held, and the question of who is most susceptible has been approached in a number of ways. For example, individual differences in introversion/extraversion (Claridge, Canter, & Hume, 1973; Eysenck, 1957; Franks, 1964; McDougall, 1929), perceptual style (Petrie, 1967), race (Schaefer, 1981), and electroencephalographic subtype (Propping, Kruger, & Janah, 1980) have all been studied in relation to various effects of alcohol. Studying individual differences in relation to the tension-reducing effect of alcohol would seem to be a useful approach considering the potentially reinforcing nature of this effect. In the few existing studies that have addressed this relationship, the results have been promising.

Although tension reduction from alcohol among women may not be a robust phenomenon (e.g., Abrams & Wilson, 1979), Eddy (1979) found that a small dose of alcohol reduced state anxiety more in female problem drinkers than in non-problem-drinking females. Lipscomb, Nathan, Wilson, and Abrams (1980) reported that the heart rate (HR) response to a stressor following a moderate dose of alcohol was more pronounced in subjects who showed little tolerance to the effect of alcohol on standing stability. Wilson and Abrams (1977; Abrams & Wilson, 1979) demonstrated that the magnitude and direction of HR change in response to a psychological stressor was a function of both the expectancy for alcohol and sex of subject. Whether these differences in the tension-reducing properties of alcohol are etiologically significant in the genesis of alcohol problems is not clear. Eddy's (1979) findings of greater anxiety reduction among problem-drinking women would seem to suggest this. However, an etiological role cannot be inferred with certainty because greater anxiety reduction from alcohol consumption might result from physiological changes (e.g., tolerance phe-nomena) associated with a history of abusive drinking, as opposed to a predisposition antedating the drinking problem.

Risk for Alcoholism

Research has indicated that certain individuals are predisposed to developing alcohol problems. One predisposing or risk factor that has received empirical support is having alcoholic relatives (Goodwin, 1979), with some recent suggestions that genetic risk may be mediated through inherited differences in ethanol metabolism (Schuckit & Rayses, 1979). In the present research we studied another predisposing factor: alcoholism risk associated with certain personality characteristics.

Several prospective studies have shown that prealcoholics (i.e., individuals without current drinking problems who later become alcoholic) can be characterized as a group as being outgoing, aggressive, impulsive, and frequently antisocial (M. Jones, 1968; McCord & McCord, 1960; Robins, Bates, & O'Neal, 1962; Schuckit, Gunderson, Heckman, & Kolb, 1976). These studies relied on a variety of official records and on behavioral ratings for their assessment of problem drinking and its antecendents. In a "follow-back" study employing a widely used assessment instrument (the Minnesota Multiphasic Personality Inventory [MMPI]), Hoffmann, Loper, and Kammeier (1974) found that prealcoholics scored higher on the MacAndrew (1965) Alcoholism Scale (MAC) than did their college classmates. The MAC is an empirically derived scale composed of 49 items designed to detect alcoholics in a psychiatric outpatient population. Because of its ability to detect future alcoholics (Hoffman et al., 1974) and young problem drinkers (MacAndrew, 1979), and its stability over time (Apfeldorf, 1978), it has been suggested that the MAC is a measure of potential for developing alcohol problems. Consistent with this notion are recent findings that nonalcoholic offspring of alcoholics score higher on the MAC than nonalcoholic offspring of nonalcoholics (Saunders & Schuckit, 1981). However, the failure of the MAC to differentiate alcoholics from drug addicts (e.g.,

Lachar, Berman, Grisell, & Schooff, 1976) and its sensitivity to cigarette smoking within an alcoholic population (Willis, Wehler, & Rush, 1979) has led to some conjecture that the MAC may be a measure of a generalized propensity toward addiction (Lachar et al., 1976).

The question of exactly what the MAC is measuring has generated some empirical research. Based on their content analysis of the MAC, Finney, Smith, Skeeters, and Auvenshine (1971) characterized high scorers on the MAC as "bold, uninhibited, self-confident, sociable people who mix well with others. They show rebellious urges and resentment of authorities. They tell of carousing, gambling, playing hookey, and generally 'cutting up' " (p. 1058).

More recently, MacAndrew (1981) described the high MAC scorer as someone who is "moving (with 'boldness') into the world, albeit in a sometimes rancorous and ill-considered fashion, with little regard for future consequences" (p. 618). Factor analytic studies (Finney et al., 1971; MacAndrew, 1967; Schwartz & Graham, 1979) help to characterize further the item content of the MAC. For example, Schwartz and Graham (1979) isolated six factors, several of which were consistent with the description of the alcoholic person as outgoing (Interpersonal Competence, Extraversion and Exhibitionism), impulsive (Risk Taking), and aggressive (School Maladjustment). Earlier factor analyses (Finney et al., 1971; MacAndrew, 1967) also support the notion that the MAC is generally sensitive to antisocial behaviors (Graham, 1978). Our integration of the literature concerned with the prealcoholic personality and with the MAC led us to hypothesize that the MAC was measuring the same constellation of traits (e.g., outgoing, aggressive, impulsive, and antisocial) found to characterize prealcoholics in the prospective studies. Although a definitive prospective study using the MAC has not been done, we believe the MAC would serve as a useful measure of presumed risk for alcoholism.

In both of the experiments reported here, we will be examining the relation between presumed risk for alcoholism and individual differences in the magnitude of the SRD effect of alcohol. If subjects at presumed high risk for developing alcohol problems were to show more pronounced SRD after alcohol consumption than subjects presumed to be at low risk, this would suggest that alcohol is more reinforcing for the high-risk subjects. Such an effect would implicate a specific mechanism of how this risk for alcoholism might be mediated, that is, through increased susceptibility to a positively valued effect of alcohol.

Experiment 1

In this first experiment the pharmacologic and expectancy effects of alcohol on physiological and affective responses to stress were examined using a balanced placebo design. Type of stressor (electric shock or giving a self-disclosing speech) was also manipulated in order to assess the hypothesis that alcohol or expectancy of alcohol might be stress reducing for a social stressor but not for a physical stressor (Higgins & Marlatt, 1973, 1975). A detailed description of this study and its results has been reported previously (Levenson et al., 1980). Additional data collected at the time of the original study permitted us to reanalyze the results of this experiment by dividing subjects into presumed high and low risk groups on the basis of their scores on the MAC.

Method

Subjects

A newspaper advertisement was used to recruit males between the ages of 21 years and 30 years for research involving "alcohol and stress." Respondents to the advertisement were screened by telephone to select those who (a) drank at least once per week and consumed more than one drink per occasion and (b) had no history of drinking-related problems. Subjects meeting these criteria were told that the experiment would involve either giving a speech or receiving an electric shock, having physiological responses measured, and possibly drinking alcohol. Subjects who expressed a desire to participate were asked to come to the psychology department to complete a number of questionnaires (including more precise quantity/frequency measures and the MAC) and were scheduled for the laboratory session.

Ninety-six subjects participated in the experiment and received a $7.50 payment. Subjects were asked to refrain from consuming alcohol or other drugs for 24 hours, and to fast for 4 hours prior to the experiment.

Apparatus

Data were collected using a system consisting of a PDP 11/10 computer and a Grass Model 7 polygraph

that enabled on-line detection and averaging of physiological measures. Physiological measures included (a) cardiac interbeat interval (IBI)—Beckman miniature electrodes placed on opposite sides of the chest detected the electrocardiogram; the computer timed the interval between successive R waves at a resolution of 1 msec. Changes in IBI reflect changes in heart rate, which is one of the basic ways the heart can regulate its output of blood to the body. Heart rate is usually controlled by the parasympathetic branch of the autonomic nervous system (ANS) but may come under control of the sympathetic branch under certain stressful conditions. (b) Cardiac IBI variability—the standard deviation of the IBI (resolution = 1 msec) was used as an estimate of variability. (c) General somatic activity—a motion sensor attached to the underside of the platform on which the subject was seated produced an electrical signal with an amplitude that was proportional to the amount of bodily movement; this signal was electrically integrated by the polygraph and monitored by the computer. (d) Skin conductance level (SCL)—a constant voltage was passed through Beckman large electrodes on the medial phalanges of the second and fourth fingers of the subject's right hand using Beckman Electrode Paste as the conductive medium; the computer determined SCL at a resolution of 1 μmho. SCL obtained from the fingers reflects the activity of sweat glands thought to be associated with emotional responding. Sweat gland activity is controlled by the ANS but is unusual in that only sympathetic branch activity is involved, and the neurotransmitter substance is acetylcholine instead of the usual sympathetic transmitter (i.e., norepinephrine). (e) Pulse transmission time to the finger (FPTT)—a Grass photoplethysmograph (Model PTTI) was used to detect the arrival of the pulse wave at the finger; the computer timed the interval between the R wave of the electrocardiogram and the arrival of the finger pulse at a resolution of 1 msec. (f) Pulse transmission time to the ear (EPTT)—a Hewlett Packard (Model 780-16) photoplethysmograph detected the arrival of the pulse wave at the ear; the computer timed the interval between the R wave and the arrival of the ear pulse at a resolution of 1 msec. FPTT and EPTT reflect two kinds of cardiovascular activities (i.e., changes in the force of the heart's contraction and changes in the distensibility of the arteries between the heart and the peripheral site at which the pulse is detected), both mediated by the sympathetic branch of the ANS (Newlin & Levenson, 1979, 1980). Shorter pulse transmission times are indicative of greater sympathetic cardiovascular arousal.

In addition to these physiological measures, a continuous self-report of perceived anxiety (ANX) was obtained by asking the subject to adjust a small dial with his left hand as often as necessary so that the dial pointer always indicated his current level of tension. The pointer moved along a 10-point scale (where 1 = extremely calm and 10 = extremely tense), with a computer monitoring the position of the dial by tracking changes in voltage applied across a potentiometer attached to the pointer.

The computer was programmed to calculate the mean of each physiological variable and ANX every 30 sec during the experiment. In addition, the computer controlled a light-emitting diode (LED) numerical display located on a table in front of the subject. The display was used to signal him at various points in the experiment. A Smith and Wesson Model 900 Breathalyzer was used for determining blood-alcohol concentration (BAC).

Procedure

On arriving at the laboratory, the subject signed an informed-consent form and answered questions concerning when he had last eaten and consumed alcohol. An initial BAC reading was also obtained to verify that subjects had not recently consumed alcohol. A mood questionnaire (Nowlis, 1965) was administered, and then subjects were given instructions appropriate to the beverage they would be drinking. The information given to subjects as well as the procedure for administering the beverages can be found in Levenson et al. (1980). In the alcohol condition subjects consumed a beverage consisting of one part Popov's Vodka to four parts Sunrise tonic at a dose of 1 g ethanol/kg body weight. In the no-alcohol condition they consumed an equivalent amount (for their weight) of an all-tonic beverage. Expectancies for alcohol and no alcohol were manipulated independently of true beverage content in a standard 2 × 2 balanced placebo design. Subjects were given 45 minutes to consume their beverages (which had been divided into three equal portions), and an additional 40 minutes to allow for absorption. A second mood questionnaire was administered 20 minutes into the absorption period, and BAC was determined at the end of the period.

Subjects were taken to another room for the stressor portion of the experiment. There the various physiological recording devices[1] were attached and the subject was instructed as to the use of the anxiety dial. At this point the subject was told which of the two stressor conditions he was assigned to: electric shock or self-disclosing speech. Subjects in the speech condition were told that they were to make a self-disclosing speech that would be recorded by a video camera and later rated by graduate students for openness and honesty. Regardless of condition, the experimental procedure was essentially the same. Seven minutes of prestressor physiological recordings were obtained; then the LED display signaled the subject to pick up a clipboard that provided additional information. In the shock condition the subject learned that he would receive a shock at the end of a 6-minute period (which would be counted down by seconds on the display). In the speech condition he learned that he would have 6 minutes to compose a 3-minute speech on the topic, "what I like and dislike about my body and physical appearance," with the 6 minutes counted down on the display. After the 6-minute countdown, he either received a single electric shock or delivered the 3-minute speech (the end of the 3 minutes was signaled on the display) according to condition. The physiological and ANX recordings were obtained for 10 additional minutes, the first three of which contained the speeches in the speech condition. In our analyses of these data, we found the effects of alcohol to be the same in the two stressor conditions.

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[1] Thus, the recording of physiological measures did not begin until after the beverage manipulations. It would have been desirable to obtain a predrinking baseline, but this would have tied up the polygraph and computer for periods of up to 3 hours per subject.

Following this 23-minute procedure, subjects were administered a third BAC reading. A postexperimental questionnaire that assessed their perceptions concerning how much alcohol they had consumed and how drunk they felt served as a manipulation check for the expectancy conditions. Subjects were debriefed and transportation home was arranged for all subjects who had consumed alcohol as well as for subjects who had not consumed alcohol but needed assistance with transportation.

Data Analysis

Subjects scoring above the median on the MAC (scores of 22 or higher) were designated as being at high risk for alcoholism, whereas subjects scoring below the median (lower than 22) were designated as being at low risk. The 23 minutes of physiological and ANX data were structured as forty-six 30-sec averages. The first 7 prestressor minutes (14 periods) were used to compute change scores by subtracting their average from the remaining 16 minutes (32 periods). This enabled us to focus on responses to the stressor. These analyses were accomplished in a series of $2 \times 2 \times 2 \times 2 \times 32$—Beverage (alcohol vs. no alcohol) $\times$ Risk (high vs. low) $\times$ Expectancy (expect alcohol vs. expect tonic) $\times$ Stressor (shock vs. speech) $\times$ Period—analyses of variance (ANOVAs) using an unweighted-means solution to handle the unequal cell sizes.

Because our previous analyses (Levenson et al., 1980) found no differences in the pattern and magnitude of responses between the shock and speech stressors, and no effects attributable to expectancy, the comparisons of interest were computed on data collapsed across the stressor and expectancy factors. Combining the MAC risk groupings (which were determined a posteriori) with the two beverage conditions produced four experimental conditions roughly equivalent in size: alcohol–high-MAC ($n = 27$), alcohol–low-MAC ($n = 20$), no-alcohol–high-MAC ($n = 24$), and no-alcohol–low-MAC ($n = 24$). Data from one of the original 96 subjects were not used because of missing data on the MAC. For the t tests reported below, there were 79 degrees of freedom in the IBI and EPTT analyses because 16 degrees of freedom were "lost" to the group mean, the main effects, and the two-, three-, and four-way interactions associated with the between-subjects factors. There were only 67 degrees of freedom in the ANX analyses because data from 12 subjects were excluded as a result of equipment malfunction.

Results

In the original report of the results of this experiment (Levenson et al., 1980), we focused on the effects of alcohol on (a) prestressor levels of physiological and self-report variables and (b) physiological and ANX responses to the stressors. The major findings in the original report were that, compared to tonic consumption, alcohol consumption (a) significantly affected prestressor levels, producing shorter IBI, increased SCL, longer

FPTT, decreased ANX, and increased report of "cheerfulness" on the Nowlis inventory (a pattern indicating a mixture of relaxant and arousal effects), and (b) significantly reduced responses to stress in IBI, EPTT, and ANX (all of which indicate reduced arousal).

As indicated earlier, our manipulation of alcohol expectancy did not produce significant effects. This could have been attributable to an imperfect expectancy manipulation. Although there might be procedural changes that could lead to a more successful expectancy manipulation (e.g., using a 5:1 mixture of tonic to vodka as opposed to the 4:1 mixture used), our manipulation check (reported in Levenson et al., 1980) showed the success of our manipulation to be comparable to that of other expectancy manipulations at the 1 g/kg dose (e.g., Lang, Goeckner, Adesso, & Marlatt, 1975). The difficulties of achieving perfect deception in the balanced placebo design using the 1 g/kg dose were discussed in the original report (Levenson et al., 1980), and the interested reader is referred there. The following reanalysis of data based on MAC groupings is being reported for the first time.

To determine whether the effects of alcohol were different for nonalcoholic subjects presumed to be at high or low risk for alcoholism on the basis of MAC scores, we first computed planned comparisons (using t tests) testing the interaction[2] of Risk $\times$ Beverage on prestressor levels. Comparable analyses were performed to test the interaction of Risk $\times$ Beverage on the magnitude of response to stress using the period of peak response. When the analysis of the interaction was significant, the nature of the relation between alcohol and risk was further explored by examining the "simple effects" of the alcohol versus no-alcohol comparison for high-risk subjects and low-risk subjects using one-tailed t tests. We were particularly interested in determining if the main effects that we had found in the overall analyses held true for high-risk subjects but not for low-risk sub-

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[2] The test of a specific interaction, like any planned comparison, can be expressed as a t ratio. Lindman (1974) provides a comprehensive framework and techniques for calculating specific comparisons in complex experimental designs.

jects. Such a finding would indicate an effect of alcohol that occurred only in subjects presumed to be at heightened risk for alcoholism.

Looking first at the significant effects of alcohol on prestressor levels, we found that the pattern of differences between the no-alcohol and the alcohol conditions was not a function of risk designations, that is, all Risk × Beverage interactions on baseline periods were nonsignificant ($p > .05$, two tailed).

As Figures 1, 2, and 3 illustrate, the effects of alcohol on response to the stressors revealed a strikingly different picture for high-risk and low-risk groups. For two of the three responses that were found to be dampened
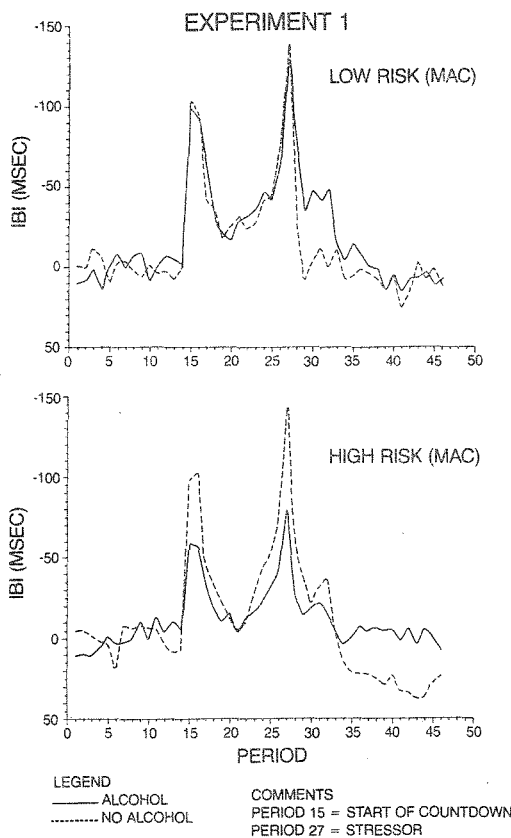

Figure 1. Interbeat interval (IBI) responses in the alcohol and no-alcohol conditions for high-risk and low-risk subjects. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates a higher level of arousal. MAC = MacAndrew Alcoholism Scale.)
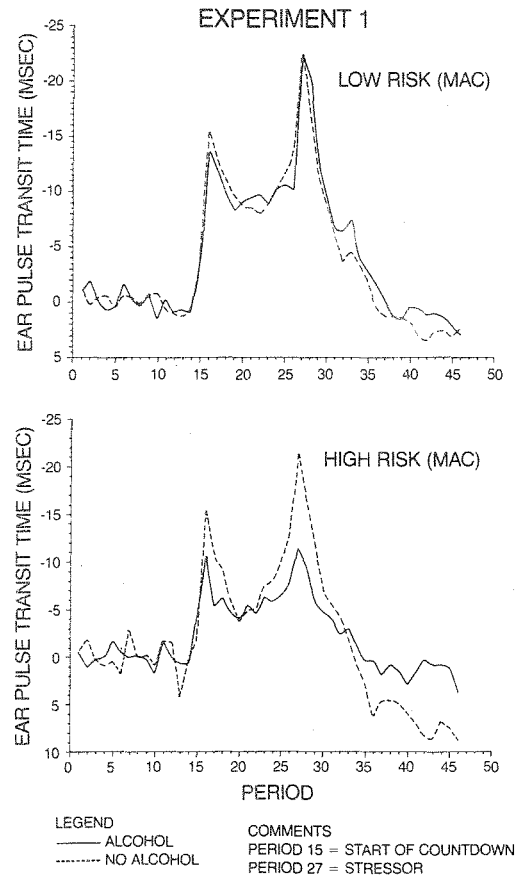

Figure 2. Ear pulse transit time responses in the alcohol and no-alcohol conditions for high-risk and low-risk subjects. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates a higher level of arousal. MAC = MacAndrew Alcoholism Scale.)

by alcohol in the overall analysis—IBI and EPTT—the interaction of Beverage × Risk was significant: IBI, $t(79) = 2.18$, $p < .05$; EPTT, $t(79) = 2.21$, $p < .05$. Simple-effect analyses for these variables revealed that a SRD effect of alcohol was found for high-risk subjects: IBI, $t(79) = 3.57$, $p < .001$; EPTT, $t(79) = 3.33$, $p < .001$. However, no evidence of an alcohol SRD effect was found for low-risk subjects: IBI, $t(79) = .50$; EPTT, $t(79) = .10$. Although the test of the interaction was not significant for ANX—$t(67) = .739$—simple effect analyses were performed to examine further the consistency of our findings. These analyses suggest that the ANX re-

EXPERIMENT 1

LOW RISK (MAC)

HIGH RISK (MAC)

LEGEND
———— ALCOHOL
·········· NO ALCOHOL

COMMENTS
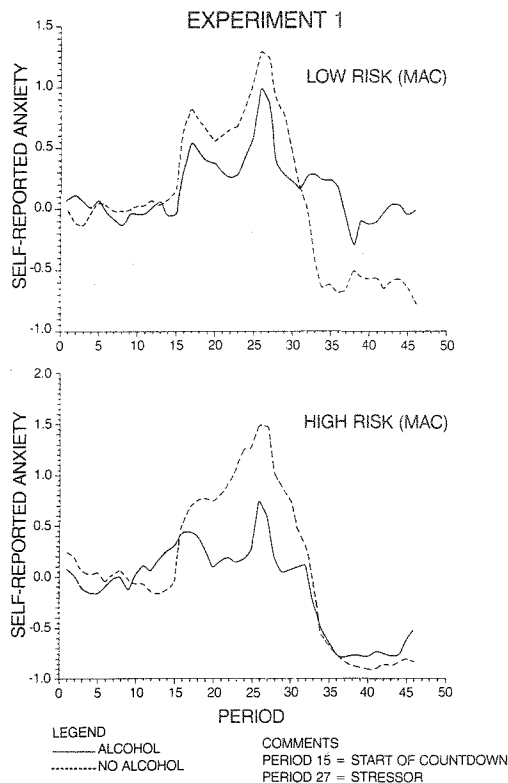PERIOD 15 = START OF COUNTDOWN
PERIOD 27 = STRESSOR

Figure 3. Anxiety responses in the alcohol and no-alcohol conditions for high-risk and low-risk subjects. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates a higher level of arousal. MAC = MacAndrew Alcoholism Scale.)

sponse to the stressor was dampened for high-risk subjects, $t(67) = 1.73$, $p < .05$, but not for low-risk subjects, $t(67) = .68$. Thus, in this sample of subjects, we conclude that the observed SRD effects of alcohol consumed at a fairly high dosage were much stronger in a group of subjects thought to be at heightened risk for alcoholism. As indicated earlier, these significance tests were carried out at the period of peak response to the stressor; Figures 1, 2, and 3 portray the IBI, EPTT, and ANX response profiles, respectively, in the various conditions and show that the effects were not limited to a single period.

Experiment 2

Experiment 2 was undertaken as an attempt to replicate the new findings from Ex-

periment 1. We felt the results from Experiment 1 were strong and clear-cut and that they had important implications for research on the SRD effect of alcohol, as well as for attempts at etiological model building for understanding alcoholism. However, because our analyses were all post hoc and there was no other research on the relation between the MAC and the SRD effect, replication was needed before we could place a high degree of confidence in these findings. Ideally, the replication would have taken the form of adding the high-MAC versus low-MAC factor to the design of the previous study. Unfortunately, we did not have the resources to launch a study of that magnitude; thus, several compromises were made. Because the previous study had failed to find any effects due to manipulating expectancy, or any differences between the shock and the speech stressors, we decided to drop those factors. In Experiment 2, all subjects were given a positive expectancy for alcohol (half actually received alcohol and half received tonic), and all were exposed to the speech stressor.

Method

Subjects

Approximately 200 males over 21 years of age were recruited by newspaper ads and posted notices to participate in "alcohol research." Respondents were preliminarily screened over the phone for quantity/frequency of drinking and for drinking-related problems, as in Experiment 1 (i.e., drank at least once per week and consumed more than one drink per occasion and had no history of drinking-related problems). Those meeting our criteria were scheduled for one of several group questionnaire sessions in which a number of instruments were administered, including the MAC. Subjects were paid $2 for attending the questionnaire session. Compared to Experiment 1, screening criteria for this study were somewhat stricter in terms of rejecting subjects with drinking-related problems: Subjects whose score on the Michigan Alcoholism Screening Test (Selzer, 1971) was greater than 9 were excluded. In addition, subjects who reported having an alcoholic parent were not used. We began contacting the highest and lowest MAC scorers to determine whether they would be willing to participate in an experiment that involved giving a speech, having physiological responses measured, and might involve drinking alcohol. Difficulties in contacting and scheduling subjects for the additional experimental session, due in part to the 2-month interval between screening and laboratory session and to limited laboratory availability, caused us to contact (or attempt to contact) all of the acceptable subjects from the initial screening. Thus, our original goal of using only subjects with extreme MAC scores was not realized.

Eighty-two subjects participated in the experiment and received a payment of $6. Subjects were instructed to fast for 4 hours and to abstain from drugs and alcohol for 24 hours prior to the experimental session.

Apparatus

All apparatuses for recording physiological data and ANX were the same as in Experiment 1. The computer was programmed to calculate the mean of each dependent variable every 20 sec (vs. 30 sec in Experiment 1).

Procedure

Informed-consent statements were signed, an initial Breathalyzer reading was obtained, and maintenance of the proscriptions against eating and drinking were verified. Then subjects were randomly assigned to one of two conditions depending on whether they consumed the alcohol beverage (1 g ethanol/kg body weight in a 4 parts tonic to 1 part vodka mix) or the pure tonic beverage. Beverages were administered and deception accomplished using the same procedures as in Experiment 1 (Levenson et al., 1980) except that subjects in Experiment 2 were all told they were consuming alcohol, and they were asked to rinse their mouth with Chloraseptic mouthwash prior to drinking to reduce taste acuity. The laboratory assistant who administered and supervised the drinking portion of the experiment was blind with respect to both actual content of the beverage and the subject's MAC score. Due to limited personnel, it was not possible to keep the experimenter who conducted the physiological portion of the experiment blind with respect to either beverage content or MAC score, but his interaction with the subject was limited to attaching the recording devices. By reducing the design in this manner, we were able to prescreen subjects and assign them to experimental conditions on the basis of their MAC scores.

The 23-minute sequence comprising a 7-minute baseline, 6-minute countdown, 3-minute speech, and 7-minute postspeech baseline used in Experiment 1 was followed. In Experiment 1 subjects were not told whether they were in the shock or speech condition prior to the attachment of recording devices; in Experiment 2 subjects all knew they would be giving a speech but, as in Experiment 1, did not know the topic until they picked up the clipboard at the start of the 6-minute countdown.

In Experiment 2 there were several changes in the paper-and-pencil measures used. Most important was the inclusion of a second measure of presumed risk, the Socialization (So) scale of the California Psychological Inventory. This scale was included because we felt it sampled the constellation of behaviors that had been found in prospective studies to characterize the prealcoholic (i.e., outgoing, aggressive, impulsive, antisocial). For example, the items of the So include aggressive content ("I go out of my way to meet trouble rather than try to escape it"), impulsive content ("I often act on the spur of the moment without stopping to think"), and antisocial content ("I used to steal sometimes when I was a youngster"). Gough (1969) described the So scale as indicating "the degree of social maturity, integrity, and rectitude which the individual has attained" (p. 10).

At this point in our research, it seemed important not only to find out whether our earlier findings would replicate but also, if they did, to be able to understand better what kind of person it was that was particularly vulnerable to the SRD effect of alcohol. We also replaced the mood inventory used in Experiment 1 (Nowlis, 1965) by an inventory developed by Mehrabian and Russell (1974) that allows scoring of three mood dimensions (pleasure, activity, and dominance). This mood inventory was administered shortly after the subject arrived at the laboratory and then again after the absorption of the beverages. At both administrations subjects were asked to complete the inventory twice, once in reference to their "current" mood and a second time in reference to their "anticipated" mood when asked to give the speech. At both administrations, subjects were asked to indicate how competent they felt they would be at giving the speech on a 1–100 scale.

Data Analysis

Because little is known about designating risk for alcoholism on the basis of personality measures, the two personality measures thought to relate to prealcoholic personality (MAC and So) were examined separately and in combination. Examined separately, both MAC (median = 22) and So (median = 34) scores were divided into high-risk and low-risk groups on the basis of a median split. A composite variable, MAC–So, was constructed using both MAC and So scores. An individual was considered to be at low risk on MAC–So if he scored below the MAC median and above the So median, and to be at high risk if he scored above the MAC median and below the So median. Subjects scoring above or below the median on both MAC and So were not included in the MAC–So analyses.

The data-analytic procedures followed a similar strategy to that used in Experiment 1. First, using data from all 82 subjects, the effects of alcohol on prestressor levels of all physiological and self-report measures were determined. Then the effects of alcohol on physiological and ANX responses to the speech stressor were determined. The risk analyses followed. For any significant alcohol effect obtained using data from all subjects, we determined whether the Beverage × Risk interaction was also significant. When this was the case, simple-effects analyses were then computed to determine if the effect held for both low-risk and high-risk subjects. In this experiment risk was defined in terms of the MAC, the So, and the combined MAC–So; thus, three sets of analyses were performed. The ns associated with each of these analyses are given in Table 1. Two features of this analysis should be noted. First, it was very conservative insofar as we limited ourselves to examining risk effects only where significant overall alcohol effects had been found. Second, the number of subjects in the MAC–So analysis was fewer than in the other risk analyses due to exclusion procedures described above. To simplify matters and avoid the possibility of having different alcohol effects in the different risk analyses, tests of the interaction of Beverage × Risk in the MAC, So, and MAC–So analyses were performed contingent on finding a significant alcohol effect in the analysis of all 82 subjects.

The ANOVAs of change scores that were carried out

Table 1
Sample Sizes Associated With the Three Risk Designations in Experiment 2

	Beverage	
Risk	Alcohol	No alcohol
Low: Based on		
MAC	21	21
So	17	24
MAC–So	11	15
High: Based on		
MAC	20	20
So	24	17
MAC–So	14	11

for the physiological and ANX responses to the stressor were repeated in the analysis of Experiment 2. Taking into account that the second experiment used 20-sec measurement periods, the ANOVAs were 2 × 2 × 48—Beverage (alcohol vs. no alcohol) × Risk (high vs. low) × Period. Planned comparisons at the periods of peak response were again used for hypothesis testing.

Results

Manipulation Checks

BAC. Subjects who received alcohol had a mean BAC of .072% immediately following the absorption period and a mean BAC of .070% following the stressor portion of the experiment. Despite the fact that a similar dosage was employed in Experiments 1 and 2, subjects receiving alcohol in Experiment 2 did not reach BACs as high as did comparable subjects in Experiment 1 (.090%). Although we do not know the reason for this, the use of different scales to determine weight and the use of different Breathalyzers in the two experiments are plausible sources for the difference in BAC.

Subjects' ratings of intoxication and consumption. Following the stressor portion of the experiment, subjects were asked to rate on a "1 to 10" scale how drunk they felt (a) right after drinking, (b) during the countdown to the speech, and (c) right now. As Table 2 indicates, subjects who received alcohol rated themselves as feeling significantly more drunk than did no-alcohol subjects during each of these periods. Alcohol subjects also reported having consumed more ounces of vodka than no-alcohol subjects did. These findings reveal that subjects in the no-alcohol condition (who drank tonic but were told it was alcohol) were not completely deceived. As mentioned earlier, the problems of achieving complete deception at the 1 g/kg dosage were discussed at some length in Levenson et al. (1980).

Alcohol Effects on Prestressor Levels

Alcohol was found to significantly affect prestressor levels of a number of dependent variables. In the affective domain, subjects consuming alcohol had greater increases (from predrinking to postdrinking administrations) in the pleasure dimension of the mood questionnaire than did subjects consuming tonic, $t(79) = 3.56$, $p < .001$. The two other dimensions (activity and dominance) were not affected. Similarly, alcohol was associated with increased anticipated pleasure when subjects were asked to predict how they would feel while giving the speech, $t(79) = 2.42$, $p < .05$. Changes in anticipated

Table 2
Subjects' Ratings of Intoxication and Consumption in Experiment 2

	Mean perceived drunkenness[a]			Mean estimated ounces of vodka consumed
Group	Right after drinking	During the countdown	Right now	
Alcohol	6.92	6.18	5.18	7.98
No alcohol	2.88	2.15	1.83	4.02
$t(78)$	15.96*	15.80*	13.10*	4.70*

Note. $n = 41$ for each group.
[a] On a 1–10 scale.
* $p < .001$.

competence in giving the speech did not differ between the alcohol and no-alcohol condition, $t(79) = .11$.

Analyses of the averages of the prestressor periods for physiological variables and ANX revealed that subjects who consumed alcohol had shorter IBI (faster HR), longer FPTT, and higher SCL than subjects who consumed tonic. Means and significance tests for all variables are presented in Table 3. This pattern of alcohol effects is very similar to that found in Experiment 1 with the exception that ANX, which was significantly lower in the alcohol condition in Experiment 1, was lower, but nonsignificantly so, in Experiment 2 (the magnitude of ANX lowering in the two experiments was similar).

Alcohol Effects on Responses to Stress

The planned comparisons of periods of peak response revealed that the IBI response to the speech stressor was smaller in the alcohol than in the no-alcohol condition, $t(78) = 3.75$, $p < .001$. In Experiment 1 we found a similar SRD effect for peak responses in EPTT and ANX as well, but in Experiment 2 the SRD effects for these variables failed to reach significance in the conservative peak-periods analysis.[3] Thus, only the IBI effect was considered in the subsequent analysis with the risk groups.

Risk for Alcoholism

Differences in the effects of alcohol that were related to presumed risk for alcoholism were the results of primary interest in this research. Our manipulation check revealed one such difference in the prestressor phase of the experiment. High-risk subjects (based on So scores) in the alcohol condition estimated that they had consumed more ounces of vodka than low-risk subjects in the alcohol condition, $t(78) = 2.71$, $p < .01$. Because the actual number of ounces of vodka administered to subjects was based on body weight, the weights of these two risk groups were compared to determine whether weight differences might explain the differences in amount of estimated vodka consumption. There were no weight differences between these groups. Our risk groupings did not relate to any of the other prestressor measures

Table 3

Mean Prestressor Levels of Physiological Measures and Anxiety (ANX) in Experiment 2

| Measure | Group | | |
	Alcohol	No alcohol	$t(78)$
IBI (in msec)	733	794	-2.19*
ACT	2.795	2.762	.44
SCL (in µmho)	14.41	11.45	1.80*
FPTT (in msec)	227.9	219.6	2.11*
EPTT (in msec)	204.7	200.1	1.07
ANX	3.14	3.70	-1.33

Note. $n = 41$ for each group. IBI = interbeat interval. ACT = general somatic activity. SCL = skin conductance level. FPTT = pulse transmission time to the finger. EPTT = pulse transmission time to the ear.
* $p < .05$.

of mood, anticipated competence, BAC, or physiological state.

As shown in Figures 4 and 5, differences were found in the manner in which alcohol affected high-risk and low-risk subjects' IBI responses to the stressor when risk was designated on the basis of So—Beverage × Risk, $t(78) = 2.00$, $p < .05$—or MAC–So—Beverage × Risk, $t(47) = 1.99$,[4] $p < .06$—but not on the basis of MAC alone—Beverage × Risk, $t(78) = 1.03$. Low-risk subjects (defined either on the basis of So or MAC–So) did not show significant reduction of their IBI response in the alcohol condition compared to

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[3] Although the specific planned comparisons between the alcohol and no-alcohol groups during the period of peak response were not significant for EPTT and ANX, mean values for these two variables were in the predicted direction: attenuated stress response in the alcohol condition. Furthermore, significant Beverage × Period interactions for change-score ANOVAs on the 48 trials for both ANX, $F(47, 3658) = 2.60$, $p < .001$, and EPTT, $F(47, 3652) = 2.23$, $p < .001$, indicated the beverage manipulation had a significant effect on the pattern of response to the stressor for both ANX and EPTT.

[4] Although the MAC–So × Beverage effect on IBI response just fails to reach the conventional significance level of .05, we chose to report it because this effect was just as pronounced as the statistically significant So × Beverage effect in absolute terms. This paradox arises because of the attenuated sample size associated with the MAC–So analyses. Perhaps of greater importance, the hypothesized simple effects of interest were highly significant in the predicted direction for high-risk but not low-risk subjects.

the no-alcohol condition: So, $t(78) = 1.17$; MAC–So, $t(47) = 1.04$. High-risk subjects (defined either on the basis of So or MAC–So) did show significant reduction of the IBI response in the alcohol condition compared to the no-alcohol condition: So, $t(78) = 4.00$, $p < .001$; MAC–So, $t(47) = 3.85$, $p < .001$. Thus, only high-risk subjects (defined in either of two ways) manifested the SRD effect for IBI following consumption of the 1 g/kg dose.[5]

To understand the relations between our risk variables (i.e., MAC and So) and the SRD effect of alcohol on IBI responses better, we computed the hierarchical multiple regression analysis reported in Table 4. The criterion variable used for this analysis was

Figure 5. Interbeat interval (IBI) responses in the alcohol and no-alcohol conditions for high-risk and low-risk subjects. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates a higher level of arousal. MAC = MacAndrew Alcoholism scale. So = Socialization scale.)
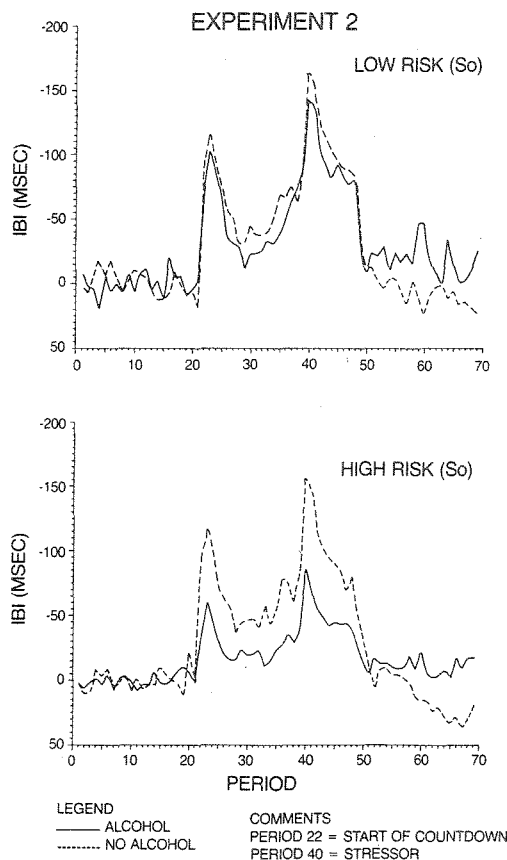

Figure 4. Interbeat interval (IBI) responses in the alcohol and no-alcohol conditions for high-risk and low-risk subjects. (Each data point represents the change from the mean of the prestressor periods. Data have been plotted so that the upward direction indicates a higher level of arousal. So = Socialization scale.)
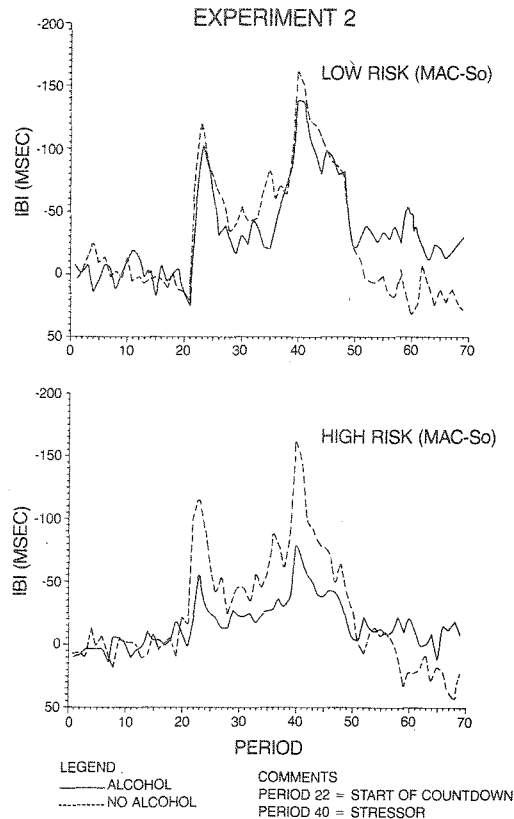
the change in IBI (calculated by subtracting the average IBI during prestressor Periods 1–21 from the period of maximal response to the speech stressor), with MAC scores, So scores, and the BAC level obtained following the absorption period serving as predictor variables. Only data from subjects who had consumed alcohol were used. The BAC level was entered into the regression first to determine if individual differences in BACs were related to the magnitude of the SRD effect; they were not significantly related. Then the

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[5] In the interest of completeness, we tested the Beverage × Risk interactions using each of our risk criteria for EPTT and ANX response, but none of these interactions approached significance.

So score was entered, which produced a significant R ($R = .43$). When the MAC score was added, it did not increase R at all. From this analysis we concluded that the relation between So scores and the magnitude of the SRD effect of alcohol on IBI was not mediated by differences in BAC levels and that the MAC did not provide additional predictive power beyond that provided by the So. Alternate versions of this analysis, in which the predictor variables were entered in other orders, did not alter this conclusion.

Additional correlational analyses were carried out to examine the relationship between the So and six subscales of the MAC derived by factor analysis (Schwartz & Graham, 1979). This analysis is presented in Table 5. Using our entire sample of 82 subjects, the correlation between the So and the overall MAC was significant but not large ($r = -.38$). However, the correlation between the So and the School Maladjustment subscale of the MAC was more impressive ($r = -.58$), especially when one considers that this subscale consists of only five items and thus is probably not very reliable. We studied the School Maladjustment subscale further by determining if, in subjects who consumed alcohol, it was related to differences in the SRD effect of alcohol on IBI response. Taken by itself, this subscale was significantly correlated with the SRD effect ($r = .36$), but it did not account for additional variance in a multiple regression analysis when the So score was entered first. From this we conclude that in our sample, there is a relatively strong relationship between the So and the School Maladjustment subscale of the MAC and that both account for similar variance in the SRD effect of alcohol on IBI response.

Table 4
Multiple Regression Analysis of Interbeat Interval Responses Using Blood Alcohol Concentration (BAC), the Socialization Scale (So), and the MacAndrew Alcoholism Scale (MAC)

Variable	R	r
BAC	.15	.15
So	.43*	-.42*
MAC	.43	.11

* $p < .05$.

Table 5
Correlations Between the Socialization Scale (So) and the MacAndrew Alcoholism Scale (MAC)

MAC	Total MAC	So
Total scale		-.38**
Subscales		
Cognitive Impairment	.13	-.36**
School Maladjustment	.54**	-.58**
Interpersonal Competence	.55**	-.02
Risk Taking	.53**	-.19
Extroversion/Exhibitionism	.32*	-.12
Moral Indignation	.31*	.03

Note. $N = 82$.
* $p < .01$. ** $p < .001$.

General Discussion

The original objective of our alcohol research program was to explore a subset of the physiological and psychological effects of alcohol in the context of a stressful laboratory experience. As the research progressed, we became increasingly interested in the individual differences in these effects that were observed and that were part of both popular and empirical lore. To demonstrate that all people do not respond to alcohol in the same way would not be a new or particularly interesting finding, but to be able to predict individual differences in one of the effects of alcohol on some independent basis would be extremely useful. The effect we chose to focus on was the SRD effect because relief or "insulation" from stress can be a powerfully reinforcing consequence of consuming alcohol that can lead to more frequent drinking behavior; thus, we wanted to search for the kind of person who would get more of this SRD reinforcer at a given dose. A logical starting point for this search was with nonalcoholics thought to be prone to alcoholism. If these high-risk individuals were found to receive more of the SRD effect of alcohol, then they would receive a relatively greater amount of this particular reinforcement when they drank. This would provide one understandable pathway (in the form of greater SRD reinforcement value associated with drinking) that could mediate between risk for alcoholism and subsequent alcoholism. The results from Experiment 1 indicated that sub-

jects at presumed risk for alcoholism on the basis of the MAC did in fact receive more of the SRD effect than their low-risk counterparts. Thus, it seemed that the MAC might be the kind of potent predictor of individual differences in the SRD effect that we were looking for. Experiment 2, in which the MAC only predicted these differences when it was combined with the So, complicated the picture. Thus, in both experiments we were able to relate individual differences in the SRD effect to independent factors thought to indicate heightened risk for alcoholism, but these independent factors differed in the two experiments.

One area of great consistency in our findings has been the effects of alcohol at the 1 g/kg dosage on physiological and affective measures taken prior to the introduction of our formal stressful manipulations. Physiologically, these effects have consisted of faster HR, longer FPTT, and greater SCL (in both experiments). Affectively, we have found euphoriant effects in both experiments using two different measures ("cheerfulness" in Experiment 1 and "pleasure" in Experiment 2) and lowered reported anxiety (of similar magnitude in both experiments, but statistically significant only in Experiment 1). In an earlier paper (Levenson et al., 1980) we noted that the physiological changes were not readily classifiable using the labels "aroused" or "relaxed". Similarly, the affective changes we have found do not sort readily into either aroused or relaxed categories. To us, these findings make intuitive sense because neither category by itself seems to capture the nature of the intoxicated state. Our purely informal observations of the behavior of subjects who had consumed this dosage suggest that they are both relaxed and aroused; these informal observations are supported formally by our physiological and affective data. Consistent with these observations, a recent survey of the expectancies of college-age drinkers (Southwick, Steele, Marlatt, & Lindell, 1981) indicated that these individuals expected alcohol to both reduce tension and increase arousal.

Several additional considerations need to be made concerning these effects in prestressor levels. First, we have been observing subjects in an admittedly "unnatural" situation.

They are after all in a laboratory, with electrophysiological recording devices attached to their bodies. However, it is important to realize that despite this artificiality, the changes we have observed are in comparison to subjects in the sober state who are also in the same "unnatural" situation. Thus, except for the possibility of complex interactive relationships between alcohol and this particular situation, these physiological and affective changes can be seen as being largely the result of alcohol consumption. Second, our measures, although certainly not exhaustive, have been comprehensive.[6] Physiologically, we found that this dose of alcohol had no significant effects on prestressor levels of ACT, EPTT, systolic blood pressure, diastolic blood pressure, or oral temperature in either experiment. Affectively, we found no effects of alcohol on the other subscales of the Nowlis (1965) mood inventory in Experiment 1 or on the Activity and Dominance subscales of the Mehrabian and Russell (1974) inventory in Experiment 2. Finally, and most importantly, these prestressor effects have been the same for all designations of high-risk and low-risk subjects studied in either experiment. Thus, these prestressor effects have shown sufficient robustness to warrant accepting them as reliable effects of consuming alcohol at this dosage. Before leaving this point we should add one caveat. The effects we have been studying follow a 45-minute absorption period. Our pilot plots of BAC-based absorption curves for the 1 g/kg dosage suggest that we are studying effects during the plateau phase that occurs following the rapid BAC rise, during the "ascending limb" and before the "descending limb" begins. At this dose, compared to lower dosages, the plateau phase is a fairly prolonged one, and the descending

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[6] We have recently completed a third study in this series that included a measure of the effects of the 1 g/kg dosage of alcohol on a measure of central nervous system reactivity, the averaged evoked potential (an electroencephalographic measure) response to auditory stimulation (Meek & Levenson, Note 1). In this study we found alcohol to reduce the magnitude of this evoked cortical response by approximately 40%. In addition, we are preparing a report describing the results of our analyses of the effects of alcohol on the self-disclosing speeches given by subjects in all three experiments.

limb may be quite protracted. The extent to which the pattern of prestressor effects we have described holds for other portions of the BAC curve remains to be studied. Beyond these methodological concerns, the next task that lies before us is to explain the effects of alcohol on physiological and affective states in terms of underlying pharmacologic and biological mechanisms.

Less consistent than these findings on prestressor levels but much more interesting are the SRD effects of alcohol at this dosage. The basic effect—reduced cardiovascular response to stress (in HR and EPTT) and reduced self-report of distress (in ANX)—was stronger in Experiment 1 than in Experiment 2. Assuming that these changes are reliable, the cardiovascular SRD is most likely a function of ethanol decreasing beta-sympathetic neurotransmission.[7] HR increase under stress is generally considered to be beta-sympathetically mediated (e.g., Obrist et al., 1974). EPTT shortening under stress is likewise a beta-sympathetic effect by virtue of the relation between EPTT, cardiac pre-ejection period, and cardiac contractility (e.g., Newlin & Levenson, 1979, 1980). The beta-sympathetic model of SRD is also supported by findings in both experiments of no reduction in ACT response to stress associated with alcohol (cardiac–somatic nonparallelism, suggesting sympathetic rather than parasympathetic control of HR), and no reduction in SCL response[8] to stress (indicating something other than generalized sympathetic dampening).

The striking thing about our SRD data is that it now appears that even at our relatively high dose of alcohol, the SRD effect may only occur in certain kinds of people. In this regard it is quite unlike the alcohol effects on prestressor levels we observed, which were robust in relation to the individual differences we have studied. Our first hypothesis might be that the SRD effect is mediated more psychologically than pharmacologically. However, data obtained in Experiment 1 indicate that manipulating expectancy (the most potent psychological factor studied in the literature to date) does not affect the SRD effect. As an alternative we might postulate that our subjects, who differ in the extent that they manifest the SRD effect, might also dif-

fer in terms of their drinking patterns. However, our data do not support this hypothesis either: High-risk and low-risk groups (which differ markedly in the SRD effect) do not differ on quantity and frequency of drinking. Admittedly, our quantity/frequency data are subject to self-report biases; however, even if there are systematic self-report biases as a function of risk designation, we feel they would operate in the wrong direction. High-risk subjects (whether chosen on the basis of MAC or So) have the kind of personality profiles that would seem to be associated with the development of alcoholism. If anything, we would expect that these high-risk subjects would drink more than their low-risk controls. If this were true, we would expect them to show greater tolerance to the effects of alcohol, and thus, at a fixed dosage, to show less of the SRD effects (e.g., Lipscomb et al., 1980). Of course, our results show just the opposite: High-risk subjects show more of the SRD effect. Another line of reasoning might be that our stressors are "less stressful" for our high-risk subjects (who might be expected to be more outgoing, impulsive, and adenturesome). However, once again our data do not support this. In neither of these experiments have we found any systematic differences in response to our stressors between high-risk and low-risk subjects in the no-alcohol condition; the differences have only emerged in the alcohol condition.

What we are left with at the end of this process of reasoning is simply that subjects thought to be at heightened risk for alcohol-

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[7] Decreased beta-sympathetic neurotransmissions could be brought about by decreased release of norepinephrine, blocking of postsynaptic beta receptors, or yet some other mechanism. The beta-blocking model gains some support from findings that propranolol (a beta-blocking agent) will reduce alcohol craving and other symptoms associated with alcohol withdrawal (Carlsson, 1971; Carlsson & Johansson, 1971). Also, alcohol has been shown to reduce cardiac contractility, a beta-sympathetic function (Child, Kovick, Levisman, & Pearce, 1979).

[8] We are beginning to think that alcohol may actually increase the SCL response to stress; although we have not been able to demonstrate statistical significance for this finding, we have seen this pattern in several of our alcohol studies. If this finding proves to be true, it might be understandable in terms of other peripheral effects of alcohol (such as peripheral vasodilation leading to "flushing").

ism are receiving a greater amount of the potentially reinforcing SRD effect at a given dosage. Whether these high-risk individuals are biologically predisposed to respond to alcohol in this manner remains to be seen. Recent research on ethnic differences in sensitivity to ethanol among Orientals and American Indians (e.g., Schaefer, 1981) as well as research on differences in ethanol metabolism related to familial alcoholism (Schuckit & Rayses, 1979) certainly are supportive of a biological basis for individual differences in an effect of alcohol such as SRD. A biological model that accounted for our SRD effects would have to be related to the personality characteristics that define our risk groups. For example, if we postulate that individual differences in SRD are a function of individual differences in the rate of ethanol metabolism, we might postulate that our high-risk individuals (who score high on the MAC or low on the So) metabolize ethanol at a slower rate than our low-risk subjects and thus have a higher effective dose of ethanol in their systems when they encounter our stressors. Our BAC data do not support this hypothesis, but we did not obtain a sufficient number of BAC readings to provide an adequate test. There are other biological models that seem worthy of investigation. Ethanol or one of its metabolites such as acetaldehyde may have more pronounced effects on certain biochemical pathways, such as the metabolism of biogenic amines (Davis & Walsh, 1970), in high-risk subjects. A more direct model based on the beta-blocking model of the SRD effect of alcohol would be that high-risk subjects are more sensitive to beta-blocking drugs. Building biological models that attempt to bridge behavioral, "personality," physiological, and drug-response phenomena is not a foreign activity for psychologists, a good example of this being the work on the introversion–extraversion dimension (Claridge et al., 1973; Eysenck, 1957; Franks, 1964).

Tarter (1978) has proposed an intriguing theory that links prealcoholic traits to alcohol's tension-reducing effects. He hypothesizes that primary alcoholism, childhood hyperactivity, and psychopathy all share the same common diathesis: a deficit in maintaining an adequate arousal level. This dysfunction in arousal mechanisms is thought

to lead to heightened responsivity to stress. Alcohol is hypothesized to induce a more normal arousal level in inadequately aroused persons, which in turn is thought to lead to decreased responsiveness to stress. Other writers (e.g., Hare, 1970) have discussed ways in which low arousal could account for a variety of psychopathiclike behaviors. Using the theories of Tarter and of Hare, low arousal levels or poorly modulated arousal levels could be associated both with certain behavioral characteristics and with reduced responsiveness to stress following a dose of alcohol. Tarter's theory clearly has merit, particularly in its ability to integrate a body of diverse empirical findings. In certain respects the data reported in this article fit this theory quite well in that we found psychopathiclike traits to be associated with large SRD effects of alcohol. However, other aspects of our data are inconsistent with Tarter's theory, specifically our failure to find differences in either prestressor levels or responses to stress between our high- and low-risk subjects in the sober state. In addition, we are skeptical of the ability of a unidimensional concept of arousal to account for findings across different physiological response systems. Our research indicates that alcohol increases prestressor arousal in some physiological functions and decreases prestressor arousal in others, and that further cardiovascular and electrodermal responses to stress are differentially affected by alcohol. Although Tarter's (1978) theory may be a useful first attempt at integrating behavioral characteristics, alcohol consumption, and SRD effects, we are still far from understanding the mechanisms underlying these relationships.

In conceptualizing the model of the SRD pathway that is evolving in our research, there seem to be three basic components: (a) risk for alcoholism associated with certain characteristic traits, (b) individual differences in the magnitude of the SRD effect of alcohol, and (c) development of alcoholism. In our thinking to date, the evidence for a biological link between the first two components of the model (i.e., risk and individual differences in SRD) seems compelling. A psychological link between these two components remains a possibility, but our data have not supported the importance of expectancy

nor differences in the stressfulness of our manipulations, two prime candidates for such a link. On the other hand, we continue to view the link between the second and third components of the model (i.e., individual differences in SRD and alcoholism) in terms of "reinforcement," with individuals who receive more of the SRD effect deriving greater reinforcement for drinking and thus being inclined to drink more often. Once this reinforcement pattern has been established, the processes of addiction take over. These include increased tolerance that leads to greater consumption, unpleasant withdrawal symptoms that accompany attempts to stop drinking, and generalization of drinking behavior to a greater range of potentially stressful situations. Although the support for this model is far from complete at this juncture, we are confident of the utility of an alcoholism-research strategy that uses high-risk methodology and studies individual differences in an effect of alcohol (such as SRD) that bears a direct theoretical linkage to the addictive process.

Although persons who are at high risk for alcoholism do appear to get greater SRD effects from alcohol than low-risk persons, we do not see large-magnitude SRD effects as a necessary condition for the development of alcohol problems because it is clear that as a category, alcohol problems are etiologically heterogeneous (McCrady & Sher, in press). For example, while the low-So/high-MAC individual might be at risk for the SRD pathway, a depressed individual might be at high risk for a pathway that involves pronounced mood effects of alcohol (e.g., Mayfield, 1968). Even among our high-risk subjects who do show marked SRD effects, it is not likely that heightened reinforcement from alcohol is a sufficient condition for the development of alcohol problems, because in all likelihood many of these persons will not develop a drinking problem. Finally, two traits associated with the prealcoholic prototype—extraversion and impulsiveness—may be related to additional pathways to alcoholism that do not rely on SRD effects. For example, extraverted persons might be especially likely to place themselves in social environments where drinking takes place, and impulsive individuals might have difficulty refraining

from alcohol consumption when their drinking becomes problematic.

Up to this point we have emphasized that in both experiments, high-risk subjects manifested greater SRD effects of alcohol consumption. However, it is in the designation of risk between the two experiments that our major inconsistent finding can be seen. In Experiment 1, risk was predicated on the MAC and, for all practical purposes, the SRD effect was limited to high-risk subjects. In Experiment 2, risk predicated on the MAC did not relate to individual differences in the SRD effect unless the MAC was combined with a second measure—the So—and then the SRD effect for IBI was essentially only found for high-risk subjects. However, using the So alone provided similar results; thus, the importance of the MAC in Experiment 2 was questionable. We cannot resolve this discrepancy to our own satisfaction. Certainly there were procedural differences between the two experiments that might have influenced the findings (e.g., manipulation of expectancy and use of shock and speech stressors in Experiment 1). Screening out subjects with parental alcoholism in Experiment 2 could have eliminated some high MAC scorers who might have shown large SRD effects. Unfortunately, we have no way of evaluating this possibility; family history data were not obtained in Experiment 1, and the MAC scores from subjects excluded on the basis of a family history of alcoholism in Experiment 2 were not retained. Saunders and Schuckit's (1981) work indicates that there may be differences in MAC scores between young nonalcoholic males with and without familial alcoholism. Other explanations of the MAC versus So discrepancy do not readily emerge from consideration of these procedural differences. Taking another approach, it might be that the So is the more potent predictor of individual differences in the SRD effect; however, we did not administer it in Experiment 1 and thus have no way of knowing if it would have been the more potent predictor in that study. Our rationale for designating risk on the basis of these two measures was different. Unlike the MAC, which has been related to risk for alcoholism in a "followback" study (Hoffman et al., 1974), we considered the So to be related to this risk because it seemed to identify

the outgoing, aggressive, impulsive, and antisocial individuals who, identified on bases other than the So, have been shown to be at risk for alcoholism in prospective studies (M. Jones, 1968; McCord & McCord, 1960; Robins et al., 1962; Schuckit et al., 1976). We have attempted to resolve this discrepancy by characterizing subjects who manifest the greatest amount of SRD as being outgoing, aggressive, impulsive, and antisocial regardless of whether they have been identified on the basis of the MAC in Experiment 1 or the So in Experiment 2.

We do feel confident that we have been able to demonstrate on two occasions that (a) individual differences in the SRD effect of alcohol do exist; (b) these differences are statistically significant and nontrivial insofar as one group of subjects manifests the SRD effect and another, for all practical purposes, does not;[9] and (c) these individual differences are related to a set of characteristics that are sampled by measures such as the MAC and So. As regards this last point, we feel that the optimal method for predicting which subjects will show the most pronounced SRD effect of alcohol has yet to be determined.

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[9] This finding has important implications for research concerned with alcohol and stress. If a subject sample is constituted such that it excludes the kind of subjects we have designated as being at risk, our data would predict that the SRD effect would not be found, even at the 1 g/kg dose.

Reference Note

1. Meek, P. S., & Levenson, R. W. Ethanol effects on the auditory evoked potential. Paper presented at the meeting of the Society for Psychophysiological Research, Washington, D.C., 1981.

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