ALCOHOL AND TENSION REDUCTION: THE IMPORTANCE OF INDIVIDUAL DIFFERENCES

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INTRODUCTION

Few research questions have generated more contradictory findings than the question "does alcohol reduce stress?". The research on this question has been frequently reviewed in recent years.[1-6] Although each review has arrived at somewhat different conclusions, there appears to be a consensus on two points: 1) much of the literature is confusing, and 2) there are few robust relationships. This chapter, which will focus on relevant human research, attempts to reduce the confusion by emphasizing the importance of individual difference variables in understanding the alcohol/stress literature. Particular emphasis is given to the authors' research on individual differences in the effect of alcohol on stress response. The general conclusion reached in this review is that alcohol can be considered stress reducing for certain individuals. In addition to making the alcohol and stress literature more comprehensible, the individual difference perspective sheds light on the relevance of stress reduction for understanding the etiology of drinking problems.

INDIVIDUAL DIFFERENCES IN THE TENSION REDUCING EFFECTS OF ALCOHOL

An appreciation of the importance of individual differences in drug effects may lead to an understanding of why the literature on alcohol and stress response is so confusing and contradictory. As has been pointed out previously by Claridge,[7] the average effect of a drug is as much a function of subject characteristics as of the pharmacological properties of the drug. As the following discussion should illustrate, subject characteristics do appear to be an important determinant of alcohol's stress reducing effects.

Gender. Research by Wilson and Abrams[8,9] and Sutker et al.[10] has examined differences in the stress reducing function of alcohol in men and women using balanced placebo designs. Although the methodologies and results of these investigations were different, in both studies it appeared that the expectancy for alcohol was more arousal inducing for women than men while the pharmacological effects of alcohol, when present, were comparable across sexes. These studies suggest that cognitive factors may be responsible for some observed differences between males and females in tension reducing effects of alcohol.

Anxiety proneness. Although not extensive, there are some data concerning how anxiety proneness relates to alcohol effects and drinking behavior. Post hoc analyses of the experiments reported by Sher and Levenson[11] showed that although trait anxiety was related to the magnitude of response to the experimental manipulations, the magnitude of stress reduction following alcohol consumption was independent of trait anxiety. In an experiment with socially anxious young men, Keane and Lisman[12] failed to find tension reduction effects attributable to alcohol. Finally, in a stress induced drinking experiment, Holroyd[13] found that high trait anxiety subjects drank less alcohol than low trait anxiety subjects. These experimental findings are consistent with data from the national drinking surveys[14,15] which showed that anxiety was not a very potent predictor of heavy or problem drinking status. Correlations between reports of "guilt" and "tension" and problem drinking measures were found to be low. Thus, the data summarized here do not suggest that anxiety proneness is a particularly important individual difference variable in determining either stress related drinking behavior or the effect of alcohol on anxiety.

Tolerance on standing stability. Lipscomb et al.[16] examined the relationship between tolerance to a challenge dose of alcohol and the tension reducing effects of alcohol consumption. Subjects who showed a smaller effect of alcohol on their standing stability showed less stress reducing effect of a high dose of alcohol on their heart rate (HR) response to a stressor than subjects whose standing stability was more affected by a challenge dose of alcohol. Simply put, the degree to which a person's standing stability is disturbed by alcohol predicts the degree to which alcohol is found to be stress reducing for him. The effect of alcohol on standing stability might represent a useful marker for persons who are susceptible to reinforcing properties of alcohol and additional research using this index is warranted.

Problem vs. non-problem drinking. In an experiment comparing problem drinking and non-problem drinking female subjects, Eddy[17] found that problem drinking women experienced greater stress reduction from alcohol than non-problem drinking women. These experimental findings are consistent with the findings from representative national surveys that tension reduction from alcohol is associated with problem drinking status.[14,15] It does not seem likely that these differences are attributable to differences in acquired tolerance since that argument would predict less stress reduction to a given dose of alcohol in heavier drinkers. Miller et al.'s[18] finding that alcoholics are more likely than nonalcoholics to increase alcohol consumption when stressed is certainly consistent with the notion that alcohol is especially stress

reducing for persons with drinking problems. While there is a substantial body of literature demonstrating that chronic alcoholics report increased anxiety and other dysphoric states after prolonged alcohol consumption,[19-21] those findings should not be interpreted as indicating that alcohol is not stress reducing for alcoholics. Those prolonged drinking studies lack an explicit stressor manipulation and so the question of stress reduction is not addressed. As pointed out by Levenson et al.,[22] the effect of alcohol on resting state is distinct from the effect on stress response.

Even though there is support for the notion that alcoholics receive greater stress reducing effects from alcohol consumption and may be more prone to stress induced drinking phenomena than nonalcoholics, drawing etiological inferences from comparisons of alcoholic and nonalcoholic samples is problematic. When differences are found between alcoholic and nonalcoholic samples, it is not possible to determine if these differences precede the development of drinking problems in the alcoholic sample or result from a history of problem drinking or excessive intake. Differential effects of alcohol that precede the development of drinking problems are much more intriguing and potentially important for understanding the genesis of alcoholism. Comparison of persons thought to be at high and low risk for alcoholism is one way of examining the potential etiological relevance of stress reducing effects of alcohol.

Presumed risk for developing drinking problems. In two experiments in our laboratory,[23] personal characteristics previously found to be related to risk for alcoholism were found to be predictive of stress response dampening (SRD) effects of alcohol.

In the first experiment, the effect of alcohol and its expectancy were investigated on stress response to both a physical stressor (receiving a painful electric shock) and a social stressor (making a self-disclosing speech).[22] Moderate to heavy social drinking young men consumed a moderately high dose of alcohol (1 g/kg) or a nonalcoholic beverage in a balanced placebo design. Following beverage consumption, subjects were instructed to sit quietly until digits appeared on a display in front of them and then to watch these digits count down from "360 to 1" at which time they either received a shock or gave a self-disclosing speech. Throughout the procedure subjects monitored their subjective tension on an "anxiety dial" (ANX) and a number of physiological measures were taken. These physiological measures included HR (as determined by the inter-beat-interval; IBI), skin conductance (SC), pulse transit time to the ear (EPTT), pulse transit time to the finger (FPTT), respiration rate, and an index of gross motor activity.

Findings during the prestressor phase of recording indicated that alcohol consumption was related to increased HR (i.e., shorter IBI) and SC, decreased ANX, and longer FPTT. This pattern of physiological activity was noted to be complex in that it incorporates indices of both arousal (increased HR and SC) and relaxation (longer FPTT and lower ANX). In regards to stress response, alcohol was found to dampen the IBI, EPTT, and ANX response to the stressor manipulation. No effects attributable to expectancy were found either on prestressor measures or on measures of stress response.

In order to further investigate the potential etiological relevance of alcohol's effects on stress responsiveness, subjects were subdivided into groups presumed to be at high and low risk for alcoholism.[23] Risk designation was made on the basis of the MacAndrew alcoholism scale (MAC).[24] The MAC is composed of 49 MMPI items which were found to statistically differentiate outpatient alcoholics from other psychiatric outpatients. The idea that the MAC is a measure of future risk for alcoholism derives from a number of lines of evidence including: 1) an archival study using the MMPI that found college-age prealcoholics scored higher on the MAC than their classmates;[25] 2) studies showing the temporal stability of the MAC in alcoholics following treatment and abstinence;[26] 3) evidence indicating the MAC is sensitive to drinking problems in youthful populations;[27] and 4) the finding that nonalcoholic sons of alcoholics score higher on the MAC than nonalcoholic sons with a negative family history of alcoholism.[28]

Results of the risk analysis showed that the effect of alcohol on prestressor (basal) measures was similar for both high risk and low risk subjects. However, striking differences between high and low risk groups emerged when the data on stress response were examined. As illustrated in Figures 1, 2, and 3, alcohol consumption was associated with dampened IBI, EPTT, and ANX responsiveness for high risk subjects. In contrast with these strong findings for high risk subjects, alcohol consumption was found to be ineffective in altering stress response for low risk subjects. These results have potentially important implications for the etiological importance of tension reducing effects of alcohol. Most importantly, the data suggest that alcohol, when consumed in the context of a stressful situation, is more reinforcing for subjects thought to be at high risk for becoming alcoholic than for subjects thought to be at low risk. This heightened reinforcement value could be seen as a potentially important mediator of the presumed high risk of these subjects.
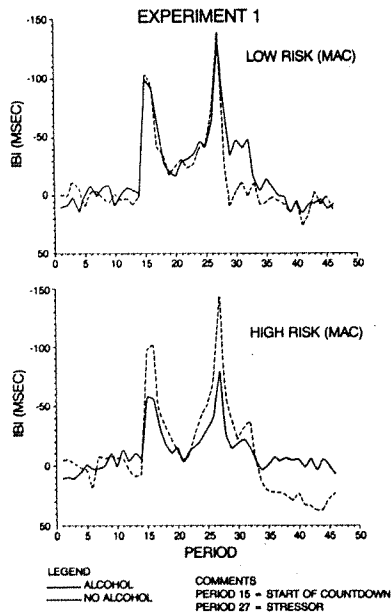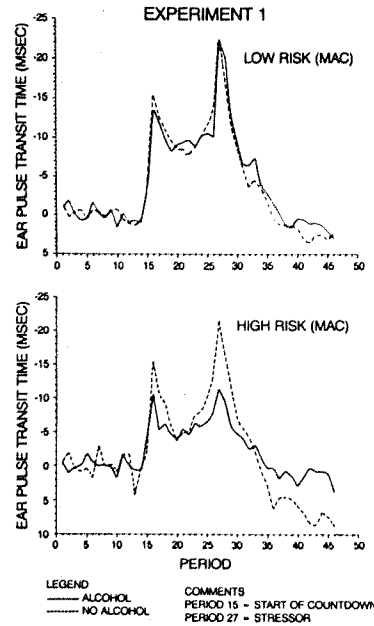
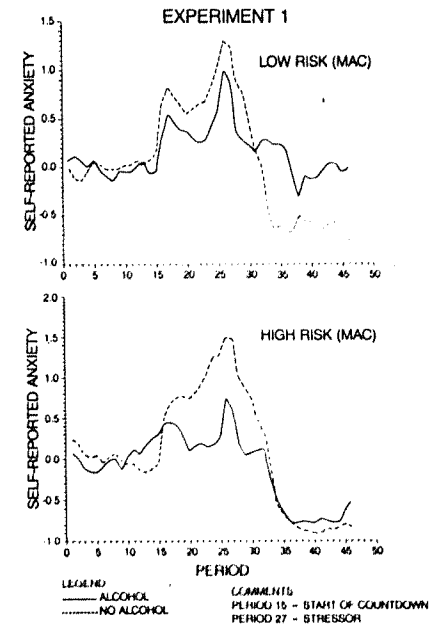**Fig. 1. IBI responses.**   **Fig. 2. EPTT responses.**   **Fig. 3. ANX responses.**

The above figures portray stress 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. (Copyright 1982 by the American Psychological Association. Reprinted by permission of the publisher and authors.)[23]

Since our analyses of these data were all post hoc and there was no other research on the relationship between the MAC and the SRD effect, replication seemed necessary before we could place a high degree of confidence in our results. Consequently, a second experiment was undertaken to replicate these findings.[23]

The design of the replication involved two major changes: 1) expectancy was not manipulated and all subjects expected to receive alcohol, and 2) all subjects were given the self-disclosing speech stressor. In addition, a second measure of presumed risk for alcoholism was included, the Socialization (So) scale of the California Psychological Inventory.[29] The decision to employ the So scale as a measure of risk for alcoholism was based on our impression that it seemed to sample the constellation of traits such as aggressiveness, impulsiveness, antisociality, and extroversion that a number of prospective studies found were predictors of the later development of alcohol problems.

Since little is known about designating risk on the basis of the questionnaire measures we employed, we decided to define risk in three alternative ways: 1) MAC alone - low scores on the MAC reflecting low risk, high scores indicating high risk; 2) So alone - low scores signifying high risk, high scores signifying low risk; and 3) MAC and So in combination (MAC-So) - high risk defined as scoring both high on the MAC and low on the So, and low risk as low on the MAC and high on the So.

As we had found in the previous study, alcohol consumption was associated with increased HR (shorter IBI), increased SC, and longer FPTT during the prestressor phase of the experiment. Unlike the previous study, alcohol consumption was not associated with significantly lower ANX ratings although the absolute magnitude of the difference in ANX ratings was similar in the two studies. Thus, the physiological effects of alcohol on basal functioning were found to be reliable across two independent samples although the finding regarding lower ANX ratings following alcohol consumption was less robust. The effects of alcohol on basal functioning were not related to subjects' risk status.

While IBI, EPTT, and ANX responses to the stressor were found to be significantly dampened by alcohol in the original study, only the findings for IBI were robust to replication in the second study. As can be seen in Figures 4 and 5, the dampened IBI response to the stressor was limited to high risk subjects when defined on the basis of either the So alone or the combined MAC-So criterion. Thus, for these risk designations, high risk subjects demonstrated the SRD effect and low risk subjects did not. However, when risk was defined
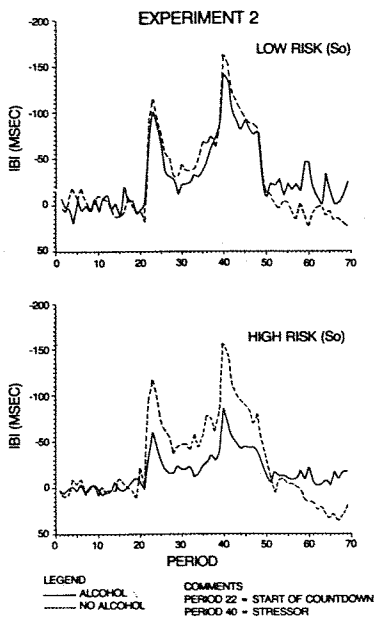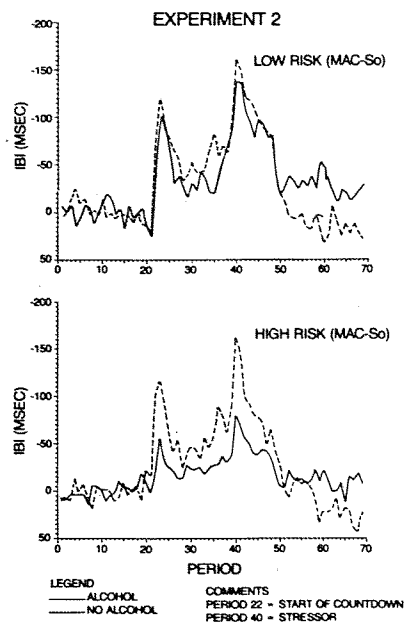
Fig. 4. IBI responses.   Fig. 5. IBI responses.

The above figures portray stress 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. (Copy-
right 1982 by the American Psychological Association. Reprinted by per-
mission of the publisher and authors.)[23]

solely in terms of the MAC, the magnitude of the SRD effects associated with

alcohol consumption did not differ significantly between high and low risk

groups. Additional correlational analyses revealed that a subset of MAC

items, those associated with reports of early school maladjustment did

significantly predict the magnitude of SRD effects from alcohol.

Despite the less than complete replication, we feel that we have been able

to demonstrate in two independent studies that: a) individual differences in

the SRD effect of alcohol do exist; b) these differences are statistically

significant and nontrivial in that one group of subjects manifests the SRD

effect and another does not; and c) these individual differences are related to

a set of characteristics that are sampled by measures such as the MAC and So.

Integration: Vulnerability to SRD effects. Although not yet conclusive,

evidence from both field and experimental studies suggests that alcohol consump-

tion is more stress reducing for alcoholics than nonalcoholics. For example,

compared to nonalcoholics, alcoholics are: a) more likely to report that alcohol is tension reducing for them,[14,15] b) likely to drink more when stressed;[18] and c) more likely to show diminished stress responsiveness following consumption of an alcoholic beverage.[17] It is not yet possible to determine whether alcohol related tension reduction predisposes one to alcoholism or somehow results from the addictive process. Even though the definitive prospective studies remain to be done, cross-sectional studies have found pronounced SRD effects in nonalcoholics presumed to be at risk for alcoholism,[23] suggesting that a tendency to experience SRD effects from drinking precedes the development of alcoholism. Given the potentially reinforcing nature of SRD effects, it may be that SRD effects mediate this risk for alcoholism.

Boundary conditions: Dose of alcohol and drinking history. As pointed out previously by Bandura,[1] tension reducing effects of alcohol appear to be dose dependent with greater tension reduction at higher doses. While stress reducing effects of alcohol have been observed at relatively low doses,[17,31-33] around .5 g/kg, there have been a number of negative findings at this and slightly higher doses.[8,9,12,30] One of the more consistent findings in the literature is that of attenuated HR responding following a 1 g/kg dose of alcohol.[22,23,30] The dose response relationships for stress reducing effects are best illustrated by Wilson et al.'s[30] finding of diminished HR responding at the 1 g/kg but not at the .5 g/kg dose.

Although not well studied, the extent of a person's prior experience with alcohol would seem likely to be important in determining his/her response to alcohol. However, the degree to which drinking history will determine the extent of alcohol related tension reduction is difficult to determine. While heavier drinkers tend to report greater tension reducing effects than lighter drinkers in drinking surveys,[34,35] Wilson et al.[30] failed to find differences between light and heavy social drinkers on the tension reducing effects of a small and a large dose of alcohol. Additional studies are needed to further delineate dose response relationships and the importance of prior drinking history.

DISCUSSION

Aspects of stress response affected. Before we can consider individual differences in the effect of alcohol on stress response, we must be able to determine those components of stress response that are modified by prior alcohol consumption. First, it is clear that not all aspects of stress response

are equally affected by alcohol consumption. In several studies, cardiovascular responding was affected while electrodermal responding was not, indicating that something other than generalized sympathetic dampening is occurring.[22,23,30]

Although less consistent than the finding of dampened cardiovascular reactivity, the finding that alcohol often leads to decreased report of anxiety[17,22,31,33] in response to laboratory stressors is obviously of great interest. However, the number of negative findings at several dosage levels[8,9,12,23,30] tempers confidence in the robustness of this effect. The reason for the greater variability of findings for affective self-report measures might be a function of the different assessment strategies used, variation in the demand characteristics associated with different experiments, and probably other factors as well.

The finding that not all aspects of stress response are equally affected by alcohol consumption has important implications for theorizing about alcohol/ stress relationships. Clearly, a global tension reduction hypothesis is not supported. Alcohol consumption leads to a dampening of some aspects of stress response but not others. Given that alcohol does appear to dampen certain responses to stress, a reasonable question to ask is "how does alcohol effect these changes?".

Possible mechanisms of alcohol related tension reduction. It is not yet clear whether the SRD effects that we and others have observed are mediated centrally or peripherally, nor is it clear what psychological or pharmacological mechanisms are involved. We have previously suggested[22,23,36] that SRD effects could be brought about in a number of different ways including: a) a direct pharmacological effect on physiological responsiveness; b) increased distractibility (making the person attend less fully to the stressor); c) altered evaluation of the level of the threat (making the person feel the stress is less severe); or d) increased efficacy expectations leading the person to believe s/he will perform more competently in a stressful situation. Added to these speculations is Hull's recent suggestion[37] that alcohol interferes with the cognitive processes subserving the state of self-awareness.

Evidence bearing on each of these alternative mechanisms is just beginning to accumulate. Somewhat surprisingly, alcohol consumption has not been found to lead to heightened feelings of "competence"[23] or "skillfulness"[12] in coping with social stressors. However, alcohol can affect the appraisal of the anticipated affective response to a stressor[23] and a number of alcohol effects are consistent with the notion that alcohol consumption reduces self-awareness.[37]

Thus, there is some basis for the notion that some of the observed physiological effects can be mediated cognitively.

Certainly the data are consistent with a direct pharmacological effect on sympathetic activity, perhaps through blocking of beta-adrenergic receptors. This mode of action is supported by findings of decreased HR but not SC reactivity. Furthermore, alcohol has been shown to reduce cardiac contractility, a beta-adrenergic function.[38,39] Finally, craving and other symptoms associated with alcohol withdrawal can sometimes be reduced by the administration of beta-blocking agents.[40,41] Obviously, evaluation of this and other possible mediating mechanisms will require much additional research.

Basis for individual differences in tension reducing effects. Although we are confident that we have been able to demonstrate significant differences in SRD effects among groups thought to differ in their proneness to developing alcoholism, we are less confident in our ability to explain the basis of these differences. However, several possible mediators of these effects can be considered.

One hypothesis is that these differences in SRD effects are mediated by differences in the blood alcohol concentrations (BACs) achieved by study participants. Since it appears that SRD effects are most likely to be found at higher doses of alcohol, it might be expected that persons who obtain higher BACs from a standard dose of alcohol are more likely to show SRD effects than subjects who reach lower BACs. Although we did not sample BAC frequently enough to be able to definitively test this hypothesis, BAC obtained 15-20 minutes prior to exposure to the stressor did not correlate significantly with HR response and thus is not the most likely candidate for a mediating mechanism.

The overall pattern of our physiological data suggests the cardiovascular SRD effects we have observed are most likely a function of decreased beta-adrenergetic neurotransmission. It therefore seems likely that individual differences in SRD effects from alcohol are mediated by differences in the effect of alcohol on beta-adrenergic systems. Presumably, these SRD effects can be mediated peripherally. However, we would not wish to rule out relevant CNS effects. Davis and Walsh[42] have speculated that ethanol or one of its metabolites such as acetaldehyde has effects on certain biochemical pathways such as the metabolism of biogenic amines. Perhaps individual differences in the tension reducing effects of alcohol can ultimately be related to differences in effects on central catecholamines.

Another possibility for a mediating mechanism might be related to the psychological dimension of augmentation-reduction (A/R). A/R refers to differences in perceptual experience with augmenters tending to increase perceived sensory input and reducers tending to decrease it. In her pioneering studies, Petrie[43] found that acute alcohol intoxication tends to make persons reducers and that alcoholics, who tend to be augmenters, show more of this reducing effect. Using evoked potential measures to assess A/R, Buchsbaum and Ludwig[44] have obtained roughly similar findings. It has previously been pointed out[45] that these A/R effects are consistent with data generated by Cutter and his colleagues[46,47] showing alcoholics are more sensitive to pain stimulation than nonalcoholics and that alcohol is more pain reducing for alcoholics than for nonalcoholics. Whether or not the dimension of A/R predicts the magnitude of SRD effects has not yet been studied.

An interesting theory relating risk for alcoholism and tension reducing effects of alcohol has been proposed by Tarter.[45] He hypothesizes that the person prone to primary alcoholism (as well as psychopathy and "minimal brain dysfunction") has a deficit in maintaining adequate arousal levels. This dysfunction in arousal mechanisms is thought to lead to heightened responsivity to stress. Alcohol is posited to have a normalizing influence on arousal levels in inadequately aroused persons, which in turn is thought to lead to decreased responsiveness to stress. In certain respects, data from our laboratory fit Tarter's model quite well in that large magnitude SRD effects are associated with psychopathic-like traits. However, other aspects of our data are inconsistent with Tarter's theory, particularly our failure to find differences in either prestressor levels or responses to stress between our high and low risk subjects in the sober state. Although Tarter's model is a useful attempt at integrating person variables and alcohol's tension reducing effects, we are still far from understanding the empirical relationships that we've observed.

CONCLUSIONS

If research on tension reducing effects of alcohol is to make sense, individual differences must be considered. In our review of the literature, individual differences were shown to be important predictors of tension reducing effects. In our own research, the SRD effect was shown to be largely an individual difference which was present in those thought to be at high risk for alcoholism and absent in those thought to be at low risk. There are a number of important implications of this finding. First, if a sample were

obtained in such a way as to exclude the most impulsive, extroverted, and aggressive individuals (e.g., sampling from graduate and professional student populations), SRD effects might not be found. Second, even if a broad range of personality types are sampled, SRD effects might be obscured unless some procedures are employed for identifying persons likely to show such an effect and partitioning the variance accordingly. Third, an etiological theory of alcoholism based on tension reducing effects must account for these important individual differences.

The ultimate determination of the etiological relevance of individual differences in SRD and other tension reducing effects will result from prospective studies where the magnitude of relevant alcohol effects in young social drinkers is correlated with the development and severity of later alcohol problems. If alcohol researchers begin to bank sufficient identifying information to permit a long term follow-up of research participants, we can begin to construct an empirical data base for testing etiological hypotheses of alcoholism.

SUMMARY

Evidence from a variety of sources demonstrates the importance of individual differences in the tension reducing effects of alcohol. The possible etiological importance of individual differences in tension reducing effects is suggested by studies demonstrating more pronounced SRD effects from alcohol in groups of problem drinkers and non-problem drinkers at high risk for alcoholism. These differences would not appear attributable to acquired tolerance since an explanation based on acquired tolerance would argue that these groups would show less of a tension reducing effect, not more. It is important to underscore that the data accumulated to date do not suggest that anxiety proneness is a crucial individual difference variable, at least in sub-clinical ranges. The key determinant of whether or not alcohol is stress reducing appears to be how susceptible someone is to the stress reducing property of alcohol, not how prone s/he is to anxiety and other tension states.

It appears that even when relevant individual difference variables are considered, SRD effects are found only for certain measures, and then only at high doses. Nevertheless, we believe these effects are of great potential importance. The challenge that lies before us is to explain the mechanisms underlying these pronounced individual differences in SRD and other tension reducing effects.

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