# Alcohol Myopia

## *Its Prized and Dangerous Effects*

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*ABSTRACT: This article explains how alcohol makes social responses more extreme, enhances important self-evaluations, and relieves anxiety and depression, effects that underlie both the social destructiveness of alcohol and the reinforcing effects that make it an addictive substance. The theories are based on alcohol's impairment of perception and thought—the* myopia *it causes—rather than on the ability of alcohol's pharmacology to directly cause specific reactions or on expectations associated with alcohol's use. Three conclusions are offered (a) Alcohol makes social behaviors more extreme by blocking a form of response conflict. (b) The same process can inflate self-evaluations. (c) Alcohol myopia, in combination with distracting activity, can reliably reduce anxiety and depression in all drinkers by making it difficult to allocate attention to the thoughts that provoke these states. These theories are discussed in terms of their significance for the prevention and treatment of alcohol abuse.*

Alcohol abuse has become the nation's most costly health problem. This has happened largely because, as a threat to public health, alcohol is a two-headed beast; it causes behaviors that are destructive of others and society, on one hand, and holds millions in the grip of addiction, on the other. As a source of antisocial behavior, alcohol is implicated in nearly 70% of fatal automobile accidents, 65% of murders, 88% of knifings, 65% of spouse battering, 55% of violent child abuse, 60% of burglaries, and so on, causing the National Commission on the Causes and Prevention of Violence (1970) to conclude that "no other psychoactive substance is associated with violent crimes, suicide, and automobile accidents more than alcohol" (p. 641). For some individuals, of course, drinking alcohol becomes something they cannot control—the other head of the beast. In 1985, nearly 10.5 million people in the United States were addicted to alcohol (Williams, Stinson, Parker, Harford, & Noble, 1987). We hasten to point out, however, that alcohol is not all bad. Most drinkers know that it can be a social lubricant, and as we show later, even a spur to altruism. Still, the price we pay for these benefits, as individuals and as a society, is frequently disastrous. When the cost of lost production, crime, and accidents due to alcohol are totaled and added to the cost of treating alcohol addiction—both heads of the beast—the bill comes to over $117 billion a year (U.S. Department of Health and Human Services, 1987).

In explaining how alcohol has these socially significant effects, a straightforward idea has dominated the thinking of laymen and scientists alike: Such effects stem directly from the pharmacological properties of alcohol, much the way relaxation stems from the pharmacological properties of valium. We know, for example, that people often drink alcohol to get the effects they assume it will directly cause: relaxation, a better mood, courage, social ease, and so on (e.g., Goldman, Brown, & Christiansen, 1987; Leigh, 1989; Maisto, Connors, & Sachs, 1981). This idea explains both heads of the beast; some of these direct effects, such as aggression and hostility, can be socially destructive, and others, such as relaxation and tension reduction, are reinforcing enough to make alcohol a potentially addictive substance. In recent years we have learned that drinking can have effects that are mediated not by alcohol but by the self-fulfillment of expectations about alcohol's effects or by the use of drinking to excuse reprehensible behavior (cf. Critchlow, 1986; Marlatt & Rohsenow, 1980). Still, we know that alcohol has profound social psychological effects that are independent of expectancy effects—a fact demonstrated throughout the literature (cf. Hull & Bond, 1986; Steele & Southwick, 1985) and in the self-destructiveness of many real-life alcohol effects. In explanation of such true alcohol effects, the idea persists that they stem *directly* from the pharmacological properties of the drug.

As research has accumulated, however, this idea has had to face a frustrating fact: Alcohol's effects on human social behaviors and emotions vary widely and are highly irregular. Studies show that alcohol intoxication can make us frighteningly aggressive (e.g., Zeichner & Phil, 1979, 1980) yet more altruistic (e.g., Steele, Critchlow, & Liu, 1985); it can relieve stressful anxiety and tension (e.g., Levenson, Sher, Grossman, Newman, & Newlin, 1980; Polivy, Schueneman, & Carlson, 1976) yet also increase anxiety and tension (e.g., Abrams & Wilson, 1979; Keane & Lisman, 1980); it can inflate our egos (e.g., Banaji & Steele, 1989) yet lead to "crying-in-one's beer" depression (e.g., Josephs & Steele, 1990; Steele & Josephs, 1988); and so on. Some of this variability could stem from alcohol having different effects on different people, that is, from individual differences in reactivity to alcohol. Distinctive physiological (cf. Schuckit, 1987) and personality-based (e.g., Hull, Young, & Jouriles, 1986) reactions to alcohol have been documented. But because alcohol can affect the social behaviors and emotions of all drinkers, not just those with special reactivities, such differences cannot explain the variability of these effects in the vast



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majority of drinkers. Nor can such differences explain the variability in these effects within drinkers (i.e., that alcohol has these effects on any one person only irregularly). In their influential book, *Drunken Comportment,* MacAndrew and Edgerton (1969) put it this way:

> The same man, in the same bar, drinking approximately the same amount of alcohol, may, on three nights running, be, say, surly and belligerent on the first evening, the spirit of amiability on the second, and morose and withdrawn on the third. (p. 15)

Thus, a basic puzzle remains: How can this single drug, aside from the effects of drinking expectancies and individual differences in reactivity to alcohol, have such varied and irregular social psychological effects?

In addressing this puzzle, Steele and his colleagues (e.g., Steele & Josephs, 1988; Steele & Southwick, 1985) have been led consistently to a particular kind of explanation. They see these effects as stemming from alcohol's general impairment of perception and thought—an effect of alcohol that occurs in every person every time alcohol is consumed. In the theories that follow, alcohol intoxication is viewed as affecting social behavior and emotion largely through an interaction of the *myopia* it causes—the short-sighted information processing that is part of alcohol intoxication—and the nature of the cues impinging on the person during intoxication. These are mixed models in the sense that alcohol's social and emotional effects are attributed to both pharmacological and environmental influences. In the present article, we describe the evolution of this idea in the field and in our work and offer what we hope is a broadened view of how alcohol has the effects it has.

The work of the Steele group has focused on three classes of socially significant alcohol effects: (a) *drunken excess,* alcohol's tendency to make social actions more extreme or excessive—the transformation, for example, of socially hesitant persons into friendly backslappers, or a person well informed about the health risks of promiscuity into a sexual risk taker; (b) *drunken self-inflation,* its ability to inflate our egos and enable us sometimes to view ourselves through rosier glasses; and (c) *drunken relief,* its ability, under some conditions, to relieve psychological stresses such as depression and anxiety. These effects of the drug underlie both heads of the alcohol beast; that drunken excess is the source of much of the social destructiveness caused by alcohol and that the other two effects, drunken self-inflation and relief, are powerful reinforcers that may underlie, to a significant degree, alcohol's addictiveness. We turn first to alcohol's effect on social behavior.

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## Psychology of Drunken Excess: Role of Inhibition Conflict

Alcohol intoxication frequently makes people aggress more, self-disclose more, gamble more, be more amorously or socially assertive, and so on, than they would if they were sober. That alcohol can cause these effects is reliable fact (cf. Hull & Bond, 1986; Pernanen, 1976; Steele & Southwick, 1985). These are the prized and dangerous effects of the drug. And as we noted earlier, it is possible to rule out or rule as incomplete, several explanations for this drunken excess. Alcohol cannot be a direct cause of such effects because if it were, they would occur everytime anyone takes a drink. Also, having a special reactivity to alcohol could not be the sole cause of such excess inasmuch as alcohol has these effects in all drinkers, not just the small subpopulation of drinkers with special reactivities. Finally, all drunken excess could not be mediated by drinking expectancies (i.e., the self-fulfillment of how one believes alcohol will make one behave or the use of drinking to excuse excessive behaviors). Research shows that alcohol has these effects even when expectancies are controlled for (cf. Hull & Bond, 1986; Steele & Southwick, 1985); also, it just does not make sense that such real-life drunken excesses as gambling away one's fortune, engaging in risky sex, or assaulting and even killing people one loves, could occur from drinking expectancies alone. As these possibilities are ruled out, however, the basic question reemerges: Through what process or processes does alcohol itself cause such excess?

In answer to this question, a line of reasoning began to emerge: Perhaps alcohol can cause excessive social behaviors indirectly, by somehow preventing the drinker from responding normally to impinging cues. In this view, the kind of behavior that occurs during intoxication reflects, in largest part, the nature of the external and internal cues impinging on the person, rather than on a specific pharmacological capacity of the drug or on some special reactivity of the drinker. Thus, whether the same drunk is surly and belligerent one night, and the spirit of amiability the next depends significantly on the cues that influence behavior and emotion during intoxication, cues that vary from person to person, occasion to occasion, and culture to culture. But what exactly is the nature of the alcohol impairment that essentially lets circumstantial cues have freer reign over behavior?

The answer seems to be an impairment of perception and thought. That alcohol intoxication impairs these functions is documented in the experience of even casual drinkers and in research showing that alcohol intoxication impairs nearly every aspect of information processing: the ability to abstract and conceptualize (e.g., Kastl, 1969; Tarter, Jones, Simpson, & Vega, 1971), the ability to encode large numbers of situational cues (e.g., Washburne, 1956), the ability to use several cues at the same time (Medina, 1970; Moskowitz & Depry, 1968), the use of active and systematic encoding strategies (Rosen & Lee, 1976), the cognitive elaboration needed to encode meaning from incoming information (e.g., Birnbaum, Johnson,

Hartley, & Taylor, 1980), and so on. But two general impairments are most critical in this line of thinking.

1. Alcohol intoxication consistently restricts the range of cues that we can perceive in a situation. When we are drunk we simply attend to and encode fewer available cues, internal as well as external.

2. Alcohol intoxication reduces our ability to process and extract meaning from the cues and information we do perceive. When we are drunk we are less able to elaborate incoming information, to relate it to existing knowledge, and thereby to extract meaning from it.

(See Huntley, 1973, and Schneider, Dumais, & Shiffrin, 1984, for particularly good illustrations of these two alcohol impairment effects.) Like the bar mitzvah food in an old Woody Allen joke, the information we receive when we are drunk is bad and there isn't enough of it. Alcohol makes us the captive of an impoverished version of reality in which the breadth, depth, and time line of our understanding is constrained. It causes what we have called an *alcohol myopia,* a state of shortsightedness in which superficially understood, immediate aspects of experience have a disproportionate influence on behavior and emotion, a state in which we can see the tree, albeit more dimly, but miss the forest altogether.

The first researchers to explain a social effect of alcohol in these terms were interested in the relation of alcohol to human aggression (cf. Pernanen, 1976; Taylor & Leonard, 1983; Zeichner & Phil, 1979). Their argument was as follows: Alcohol limits one's perceiving and thinking so as to leave one still able to respond to salient, immediate cues, but less able (than if one were sober) to respond to more peripheral cues and embedded meanings. Therefore, when the salient cues elicit violence and the peripheral ones inhibit it, alcohol intoxication releases violence.

Here is a laboratory example: Zeichner and Phil (1979) recruited male subjects, for a pain-perception/reaction-time experiment and after allowing them to ingest either alcohol or placebo drinks, they gave each subject a noxious tone (through earphones) that subjects believed was delivered by another "partner" subject. The actual subject was to stop the tone by giving the partner an electric shock as fast as possible. The intensity and duration of this retaliation measured subjects' aggression. The partner, of course, was not a real person but a computer that in the critical condition, matched the subjects' shock with a second noxious tone of equal intensity and duration—an "eye-for-an-eye". Clearly, the smart thing to do in this condition would be to give one's partner only a mild shock, and then one would get only a mild tone in return. But to be smart, one has to be mindful of the tone contingency. In a nutshell, the sober subjects played it smart, giving very little shock in this eye-for-an-eye condition, whereas the intoxicated subjects plunged ahead, giving nearly three times as much shock. Presumably, the myopia experienced by the intoxicated subjects allowed them access to the provoking stimuli, because of their immediacy and salience, but blurred their appreciation of the delayed inhibiting contingencies, allowing them to be more aggressive than their sober cohorts by a factor of 7 standard deviations.

If this reasoning could explain the relation of alcohol to human aggression, Steele and his colleagues (cf. Steele et al., 1985; Steele & Southwick, 1985) reasoned that it might explain alcohol's effect on other social behaviors as well. Alcohol might foster helping, for example, not through any special capacity to make people helpful and warm toward their fellow man, but simply because it places people under the control of immediate cues capable of eliciting helpfulness. Through the myopia it causes, alcohol may tie us to a roller-coaster ride of immediate impulses arising from whatever cues are salient.

But something is missing from this picture. It explains how alcohol can have varied social effects, but it doesn't explain when they will occur. What is missing is the identity of some factor, or set of factors, that determines the occurrence of drunken excess. To address this problem, Steele and his colleagues (Steele et al., 1985; Steele & Southwick, 1985) tried to identify the kind of situation in which alcohol myopia would lead to excess. This led to the hypothesis that it would do this in situations that, if the person were sober, would involve a certain response conflict, *inhibition conflict,* in which a response provoked by salient, strong cues is also inhibited by other strong cues that require further processing to grasp. The aforementioned aggression procedure illustrates this conflict. The provoking tone, at the moment it occurred, was more salient to the subject than the idea that his aggression would be retaliated against, an idea that at that moment required further processing to access. Our reasoning is this: If one is sober in this kind of situation, the salient, provoking cues can move one to respond, yet one can also search out, become aware of, and understand other now-relevant cues that might inhibit the response (i.e., possible negative consequences, relevant inhibiting standards of conduct, inhibiting cues in the situation, other more appropriate responses, etc.) As a result, one can hold back the response. If one is intoxicated in this situation, however, the resulting myopia allows the influence of salient provoking cues but reduces the influence of inhibiting cues and meanings, many of which require further processing to access. In this sense, the inhibition conflict is preempted, or blocked. As a result, alcohol leads to excess. What we can't see or appreciate, we can't use, and when alcohol intoxication prevents us from using cues that might otherwise inhibit a response, we respond more extremely.

The other equally important part of this idea is the implication that alcohol intoxication does not generally lead to excess in situations that do not involve this kind of conflict. In situations without these conflicting response pressures—in which cues provoking the response are weak, or cues inhibiting the response are weak, or both sets of cues are weak—the myopia that alcohol causes will not change the balance of pressures bearing on the response. (It would only block inhibiting cues that are already weak or weaken inhibition against a response tendency that was weak to begin with.) In these situations,

drunk people should behave no more extremely than sober people. The point here is that whether alcohol intoxication results in drunken excess can depend in large part on the situational strength of a simple condition—inhibition conflict.

Consider this real-life example. You have just had an angering argument with your landlord, the kind of argument that makes you want to take his head off on sight. As you are milling around at a neighborhood cocktail party that evening, you turn from the eggplant dip to the wine table and encounter him face to face. If you are sober in this situation, you should experience the kind of response conflict we are describing. The salient cue of his presence will provoke in you an impulse to tell him off on the spot, yet with just an instance's further consideration you realize that if you do this you will cause a terrible scene, embarrass yourself, and maybe even get evicted the next day. Thus, you grit your teeth and hold in your tirade. Our point is that this kind of situation—characterized by such conflicting response pressures—can trigger drunken excess. Thus, if you should encounter your landlord after being three or four drinks into the evening, his presence, as a salient cue, should still provoke your anger, but the myopia that alcohol causes should reduce your access to the negative consequences of telling him off. As a result, a legendary scene erupts and the next day you are homeless.

Let us say now that you are not angry at your landlord or that you are vacating your apartment anyway and nobody is left at the party when you encounter him. Neither of these situations would involve inhibition conflict. Either there is no salient cue provoking your anger or there are no consequences to be accessed that would inhibit your anger if you wanted to express it. In situations like these, the myopia that alcohol causes will not change the balance of pressures bearing on the response and you should behave pretty much the same way drunk or sober. If you are not angry at him, the mere myopia of alcohol intoxication would not make you tell him off for no reason; or if you are angry at him but have nothing to lose from a tirade, you would probably tell him off even without the aid of alcohol.

To summarize, the central assumption of this reasoning is that alcohol myopia restricts attention and thought to the most salient cues in a setting, whatever they may be, and that this is a general process through which alcohol influences social behavior. In this process, the primary determinants of social behavior during intoxication, as during sobriety, are the internal and external cues that become salient to the actor (rather than specific pharmacological effects of alcohol or special alcohol reactivities of some drinkers). Sometimes these will be cues that provoke only a weak response, and not much will happen; sometimes these will even be cues capable of inhibiting a response;[1] and sometimes, of course, these will be strong response-provoking cues. Even then, alcohol intoxication may add little to the extremeness of the response. If there are few inhibiting pressures that further processing would access, then alcohol's impairment of this processing will do little to make the response more extreme. But when further processing would access inhibiting pressures, the myopia of alcohol intoxication should occlude these pressures, disinhibiting the response. It is this latter kind of situation that we call *inhibition conflict.*

One more consideration: The amount one drinks should also make a difference; 12 drinks would probably have greater effect on one's behavior at the cocktail party than would 3. This is because alcohol's impairment of perceptual and cognitive functioning—that is, alcohol myopia—increases with dosage (e.g., Jones & Vega, 1972). The greater the myopia, the more thoroughly peripheral cues and embedded meanings will be occluded from awareness and the greater should be alcohol's disinhibiting effect in conflict situations.

If this reasoning is correct, it should be possible to find support among existing studies of alcohol's effect on social behavior. To this end, Steele and Southwick (1985) identified every study, published or unpublished, that had ever tested the effect of alcohol on some human social behavior. They came up with 34 studies in all, and from these they compiled a set of 121 comparisons of treatment conditions in which all factors were equal, except that one group in the comparison got alcohol and the other one did not. Their expectation, of course, was that in comparisons for which the social response was under strong inhibition conflict, alcohol would have a large ef-

[1] We do not assume that cues provoking a response will always be more salient than cues inhibiting it, or that alcohol myopia somehow makes this so. Consider the example in which cues inhibiting aggression (e.g., the presence of several policemen standing in front of an individual at the bar) are more salient than cues provoking it (e.g., a hissed insult from an antagonist at the back of the barroom). Here, alcohol myopia may actually reduce aggression by narrowing the drinker's attention to the more salient inhibiting policemen, causing him or her to miss the more remote, provoking insult (a possibility brought to our attention by friends and colleagues, Jay Hull, David Kuykendall, Barbara Leigh, Kipling Williams, Ron Van Treuren, and Mark Zanna). Although in this way the logic of alcohol myopia suggests the possibility of drunken inhibition (see Brown, Mansfield, & Skurdal, 1980, for evidence of this effect in animal conflict learning paradigms) as well as drunken excess, it is the latter effect that occupies researchers in this field, as it is through this effect that alcohol has its most important social effects, both prized and dangerous.

One might also ask whether there are strong inhibition conflicts in which both motivating and inhibiting meanings are so salient that alcohol myopia would not impair the influence of inhibiting cues and thus would not disinhibit the response. Although this is ultimately an empirical question, we doubt that such situations arise very often. Once a strong response tendency is aroused, inhibition of that tendency requires further processing even when inhibiting cues are highly salient. One's attention and thought must get past the immediate cues arousing the tendency, past the tendency itself and the orientation it forces on perception and thought, and access other cues and standards relevant to the response and grasp their inhibitory significance. All of this takes processing beyond that required for the response to be aroused in the first place, even when cues and meanings of potential inhibitory significance are highly salient. It is this processing that we argue is impaired by alcohol. Thus, even when cues that would inhibit a response are highly salient—policeman-at-the-elbow situations, as they are known in British jurisprudence—we would expect alcohol myopia to weaken their influence, allowing a motivated response to be more extreme.

fect, in the sense that intoxicated subjects would respond more extremely than the sober subjects in the comparison, but that under weak conflict conditions, alcohol would have little or no effect—and also, that this tendency would increase with the level of alcohol in subjects' blood.

We believe that anyone who is familiar with the conflict idea could glance at the method sections of these studies and tell that inhibition conflict varied considerably from one experimental condition to the next. In the aggression studies, for example, many conditions established intense inhibition conflict of the sort we have described, but others established clearly weak conflict, as when subjects were neither provoked to aggress nor inhibited from doing so. In gambling studies, betting was sometimes under the strong conflict of large possible gains and large possible losses, yet in other conditions it was under the weak conflict of large possible gains and only small possible losses (e.g., a cheap raffle ticket). Still, not anticipating our theory, none of these studies had explicitly manipulated conflict. Thus, the classification of alcohol effects, whether the response involved was under strong or weak inhibition conflict, had to be one of judgment. It turned out, however, that these judgments could be made with great reliability. Steele and Southwick (1985) agreed in their ratings of conflict for 96% of the comparisons. And for a subsample of 60 comparisons, their judgments agreed with 83% of those made by independent judges who, blind to the hypothesis and the results of the studies, used their own coding rules. One more technical point: The dependent variable for each comparison was how much more extreme the behavior of alcohol subjects was compared with that of the no-alcohol subjects. To use a scale common to all comparisons, these differences were expressed in standard deviation units—in essence, how many standard deviations more extreme were the intoxicated subjects than were their sober control subjects.

**Figure 1.**
*Alcohol Effect Sizes by Level of Inhibition Conflict and Blood Alcohol Level (BAL)*

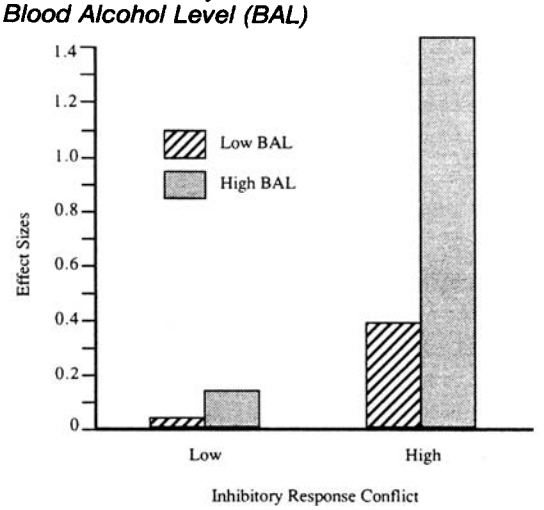



**Table 1**
*Effect Size Means by Level of Conflict and Type of Social Behavior*

| | Conflict level | | | |
|---|---|---|---|---|
| | High | | Low | |
| Type of social behavior | *M* | *n* | *M* | *n* |
| Aggression | 1.32 | 32 | 0.17 | 30 |
| Assertiveness | — | — | 0.54 | 1 |
| Human conflict | 0.89 | 1 | 0.01 | 3 |
| Drinking | 0.88 | 2 | −0.38 | 8 |
| Eating | 0.02 | 3 | 0.58 | 3 |
| Gambling | 0.38 | 2 | 0.05 | 6 |
| Mirth | — | — | 0.10 | 6 |
| Moral judgement | — | — | −0.17 | 2 |
| Risk taking | 0.48 | 2 | 0.66 | 3 |
| Self-disclosure | 1.34 | 3 | 0.07 | 4 |
| Sexual interest | 0.42 | 4 | −0.06 | 4 |
| Yielding | — | — | 0.29 | 2 |

*Note.* *M*s are not given for cells of the table for which there is only one effect size.

It will probably surprise no one to learn that intoxicated subjects were generally more extreme than their sober control subjects, 0.69 standard deviations over the entire set of comparisons. They gambled more, looked longer at sexual slides, gave more shock to their opponents, self-disclosed more, and so on. In strong support of our theory, however, these effects varied dramatically with the level of inhibition conflict of the response. Intoxicated subjects were a full standard deviation more extreme than their sober counterparts ($M = 1.06$) under strong conflict and only nonsignificant 0.10 standard deviation more extreme ($M = 0.14$) under weak conflict. Figure 1 presents mean alcohol effects broken down by conflict and degree of intoxication. Here the powerful mediating effect of conflict is even clearer. When conflict was strong and intoxicated subjects had blood alcohol levels (BALs) above .06, they were a full 1.4 standard deviations more extreme than their sober control subjects. In percentage terms they were, on average, more extreme than 95% of the control subjects. Even at low levels of intoxication, strong conflict led to significant alcohol effects ($M = 0.38$). Clearly though, it is the combination of strong inhibition conflict and higher BAL that caused whatever extreme drunken excess was evidenced in these studies.

Table 1 breaks down alcohol effect sizes by level of conflict and type of social behavior and shows that the pattern of bigger alcohol effects under strong conflict holds for all but two of the social behaviors studied.[2] Also, an analysis of expectancy effects in this literature—the difference between subjects who believed they had consumed alcohol and those who believed they had not, all other

[2] The studies measuring alcohol's effect on eating behavior used very low doses of alcohol and allowed almost not time for absorption. This may have contributed to the weak alcohol effects in these studies.

factors held constant—showed that unlike real alcohol effects, expectancy effects did not vary with rated conflict. This result, presented in Figure 2, makes an important point: Even though expectancy effects are substantial—people who believed they had consumed alcohol behaved almost 0.50 standard deviation more extreme than did people who believed they had not—they cannot explain the pattern of alcohol effects. That is, because expectancy effects do not vary with conflict, they could not have mediated, in any way, the observed relation between alcohol effects and conflict.

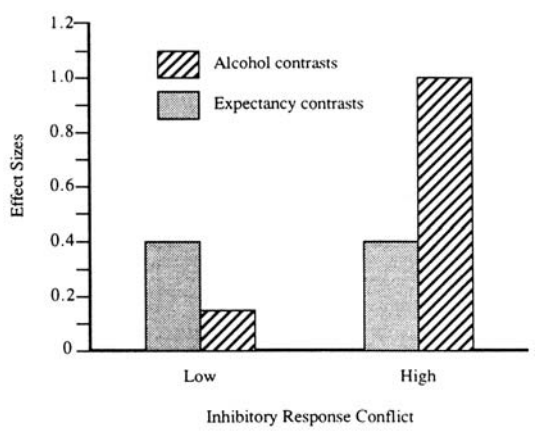
There is a gratifying thoroughness about meta-analysis; but in the end, largely because conflict had to be judged post hoc, this one could not stand as a test of the conflict idea, although it was encouraging. Also, aggression studies dominate this analysis, as they do the literature in general, raising some question about how well these findings represent alcohol's effect on the full range of social behaviors.

Thus, to test the idea experimentally and to examine its generalizability, Steele et al. (1985) tested the effect of alcohol on helping, a frequently conflictual prosocial behavior. They created a situation in the laboratory that was a little like being asked by a friend to stay and help paint after you'd already helped him move in all day. Subjects crossed out *a*s and *e*s in a paragraph of legal jargon for as many repetitions as they could do in 17 min. Then, just as they expected to relax for the remaining 25 min of the experiment, the experimenter appealed to each of them to help by doing more repetitions. This merciless procedure, it was assumed, would establish a strong conflict between the impulse to help (aroused by the immediate, urgent, face-to-face appeal) and a strong desire not to do any more of this boring task. A weak conflict condition was established by only weakly pressuring subjects to help (through a written appeal at the bottom of a questionnaire). One half of the subjects had consumed enough alcohol before the proofreading task to bring their BALs to .04 at the time of the request, whereas the other half had consumed only a placebo drink. It was expected, of course, that alcohol would increase helping in the strong conflict condition but not in the weak conflict condition, and that is exactly what happened. Over two experiments, this basic procedure was varied to test whether merely believing that one had consumed alcohol would increase helping—it didn't—and whether the effect of conflict was robust over slightly different BALs and operational definitions—it was.

Illustrating the real-world generality of the drinking–helping relationship, Lynn (1988) found that as patrons in a Columbus, Ohio restaurant drank more alcohol, they gave bigger tips—clearly an otherwise conflicted act of helping—even when the size of the check was held constant. As far as conflicted helping is concerned, alcohol is apparently a milk of human kindness.

We believe this evidence clarifies several things about social drunkenness. First, it identifies an important process through which alcohol contributes to these effects. *As far as drunken comportment is concerned, alcohol need not be a direct cause, a releaser of special alcohol reactivities (the devil's potion), or an inconsequential concomitant of drinking expectancy effects, but can affect social behavior by blocking inhibition conflict, that is, by freeing motivated responses from inhibiting cues.* And second, it identifies a pervasive condition under which alcohol causes drunken excess: in simplest terms, *whenever salient cues provoke a person to do something that if he were sober, remoter cues and thoughts would pressure him to inhibit.* This evidence makes drunken excess a more predictable phenomenon. If one can specify, even roughly, the degree of inhibition conflict a response is under in a setting, one can predict the extent to which alcohol intoxication is likely to make it excessive. The research we have described shows that, for the most part, this is a rather straightforward specification, both with regard to judging existing situations (the meta-analysis) and with regard to constructing situations that manipulate the level of this conflict (the helping experiments).[3,4]

**Figure 2.**
*Alcohol and Drinking Expectancy Effect Sizes by Level of Inhibition Conflict*




[3] We do caution, however, that inhibition conflict may be difficult to judge in situations in which it is difficult to judge the relative salience and strength of response-relevant cues. This was not a significant problem in the research we have reported (both the experiments and the studies in the meta-analysis) because these investigations were designed explicitly to test alcohol's effect on particular responses. Thus, the salience and strength of response-relevant cues was fairly straightforward to judge, and to the extent that any of this research generalizes to real-life (as we believe it does), we expect that much of the time these judgments will be reliable there as well. Nonetheless, there are settings in which these judgments are not straightforward. An example comes from research on alcohol's role in risky sex (i.e., sex in which the risk of contracting AIDS is high; e.g., Hasin & Martin, 1989; Stall, McKusick, Wiley, & Coates, 1986). Without specific information about the dispositions of the actors and the circumstances of their encounter, it may be difficult to judge whether cues leading to sexual arousal or to the fear of AIDS are more salient or stronger in a given situation. In such situations, although we believe that alcohol's effect is mediated by the level of inhibition conflict the relevant response is under, independent evidence of the relative cue salience and strength may be necessary before sound predictions from the theory can be made.

[4] We assume that alcohol's blocking of inhibition conflict is a general

With respect to the generalizability of these conclusions, an interesting fact about alcohol's effect on rat learning is worth noting: Alcohol seems to impair this learning only in paradigms that involve response conflict (cf. Brown, Mansfield, & Skurdal, 1980; Cappell & Herman, 1972; Gray, 1978). In the passive-avoidance paradigm, for example, in which the rat must learn to inhibit a tendency to approach a punishing goal area, or in the extinction paradigm in which the animal must learn to inhibit dominant responses so that new ones can be learned, alcohol consistently retards learning by impairing inhibition. Drunk rats in these paradigms plunge ahead, following their immediate impulses, regardless of the consequences. Sober rats play it safe. Yet in nonconflictual, instrumental learning paradigms in which the animal simply has to initiate a response to gain reward or avoid or terminate punishment, it is surprising that alcohol has no consistent effect. Thus, just like its effect on human social behavior, alcohol seems to impair rat learning through the particular effect of impairing the animal's ability to hold back motivated responses in light of conflicting pressures. We have no idea whether this impairment happens in rats the same way it does in people. For the rat data, Gray preferred the physiological explanation that alcohol changes the electrical activity in an area of the brain specifically linked to inhibitory control. Brown et al., however, offered evidence that alcohol has this conflict-reducing effect by reducing the influence of the weakest response tendency in these conflicts, regardless of whether it is excitatory or inhibitory. They show that alcohol can actually increase inhibition in circumstances in which inhibition cues are stronger than excitatory cues, and among several possible explanations, suggest that alcohol "interferes with the organism's ability to attend simultaneously to sets of cues demanding incompatible behaviors, so that only the more salient ones are effectively processed" (p. 430)—a view indistinguishable from our notion of myopia. Thus, the important suggestion of these data, which supports the generalizability of our findings and theory, is that alcohol affects rat conflict behavior in much the same way it affects human conflict behavior. It frees responses initiated by the most salient cues from the conflicting pressures of less salient cues, and it may do this in both species through the myopic processing it causes.

mediator of drunken excess in the sense that it can cause excess in all drinkers who have consumed alcohol to the point of intoxication. This assumption, plus the finding that alcohol intoxication caused no excess in the absence of inhibition conflict in any of the research reviewed, might suggest that disinhibition of conflict is the only process through which alcohol causes excess. Here we offer a caution. Most of this research used normal and heavy social drinkers as subjects, mostly college students. It is thus conceivable that it undersampled subcategories of problem drinkers who because of special reactivities to alcohol (cf. Schuckit, 1987) might show drunken excess even in the absence of inhibition conflict. We know of no evidence linking such reactivities to drunken excess. But because of this sampling limitation, we fall short of concluding that alcohol's disinhibition of conflict is the only route to drunken excess. We do conclude, however, that it is a general route that can cause drunken excess in all drinkers, even if in addition to its effect, other processes can cause this effect in some drinkers.

Another point worth stressing is the demonstration in the present work of alcohol's own influence on human social behaviors. This may seem painfully obvious. Nonetheless, in recent years, estimates of alcohol's role in causing drunken excess have trended downward. When tests of these effects are aggregated without regard to conflict, the overall social impact of alcohol appears surprisingly modest. This could be seen in our own meta-analysis. And the same result in several recent reviews of the balanced-placebo literature (Critchlow, 1986; Hull & Bond, 1986; Marlatt & Rohsenow, 1980; Reinarman & Leigh, 1987) led reviewers to conclude that alcohol has consistent effects on nonsocial behaviors (cognitive and motor performance) but that drinking affects social behavior more through the effects of drinking expectancies. As we hope we have shown here, a vastly different picture emerges when alcohol's social effects are broken down by the level of inhibition conflict the response is under. Under conditions of strong conflict, even doses of alcohol that typify just moderate social drinking (i.e., BALs between .06 and .14) have massive effects on social behavior, making intoxicated subjects more extreme than 95% of their sober controls (all of this with drinking expectancies held constant). Under the right conditions, then, very common conditions, alcohol has quite profound influences on these behaviors, influences that explain perhaps more plausibly than do expectancies alcohol's relation to extreme, self-threatening behaviors such as violent crime and risky sex.

## Drunken Self-Inflation: Alcohol as the Ego's Elixir

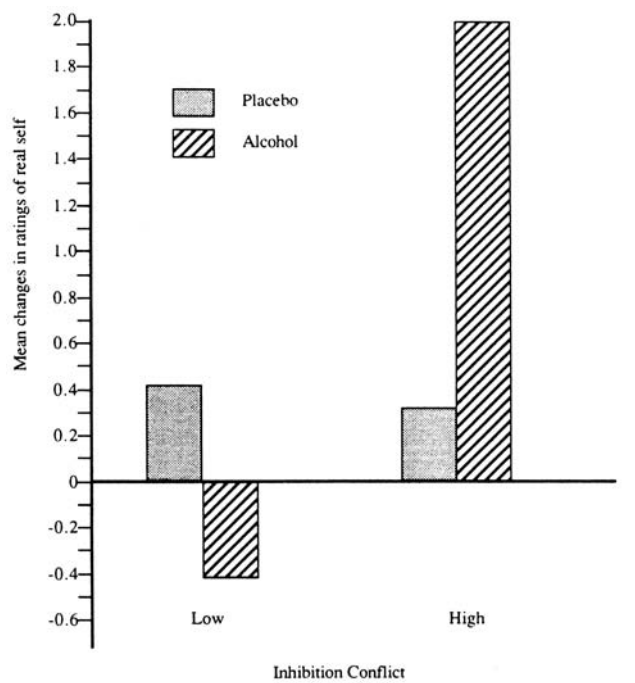
It is possible to make another generalization of alcohol's conflict-blocking effect, one that connects this work more closely to the other head of the alcohol beast, alcohol addiction. Not long ago, Banaji and Steele (1989, 1990) considered whether this effect of alcohol would generalize from the case of simple response conflict to the higher realm of intrapsychic conflicts—in particular, to conflicts over how well to evaluate oneself. Their reasoning began with the long-known fact that people have a powerful need to think positively of themselves, especially along dimensions that are important to them (e.g., Greenwald, 1980; James, 1915; Steele, 1988; Tesser, 1988). Yet, even when one is in full possession of one's faculties, it can be disappointingly easy to call to mind information that contradicts these desired self-images—establishing, in a real sense, a self-evaluative conflict. Consider the aspiring classical pianist, for example, who wants deeply to view himself as having a great talent but, whenever he begins to think this way, readily accesses deficiencies in his playing and comparisons to others that inhibit this evaluation. This kind of conflict, in some form or another, at one time or another, haunts us all. Banaji and Steele (1989) proposed that through the myopia it causes, alcohol may disinhibit these self-evaluative conflicts. That is, when immediate cues (such as topic of conversation) arouse a strong impulse for a favorable evaluation along some important dimension of the self-concept, alcohol myopia should allow us to experience the impulse and at the same

time impair access to more remote, inhibiting information. In this way, alcohol intoxication may inflate our self-evaluations, especially along important dimensions. Thus, when the question of talent arises during intoxication, our aspiring pianist, having less access to his deficiencies, ranks himself as worthy of a recording contract.

To test this idea, Banaji and Steele (1989) had subjects rate the personal importance of 35 trait dimensions and their "real" and "ideal" standing on each dimension, both before and after they were made intoxicated or had consumed a placebo drink—and that was all there was to the study. They found that getting drunk significantly inflated the self, *but only on traits that were both important to subjects and for which, before drinking, they had acknowledged that their "real" self was considerably worse than their "ideal" self.* On these strong-conflict traits (i.e., those five with the largest and most important ideal–real discrepancies on the pretest) intoxicated subjects significantly bettered their ratings of their real selves after drinking, but for weak-conflict traits (i.e., the five smallest and least important ideal–real discrepancies) alcohol had no such effect. Placebo drinks caused no change on any traits (see Figure 3). Also, additional experiments replicated these results and showed that they did not occur because of alcohol's elevation of mood.

On the basis of these data, alcohol causes conceit in much the same way it causes a tirade at a neighborhood cocktail party. It impairs the processing capacity needed to inhibit strong impulses toward these things, impulses aroused by immediate, salient cues, external or internal.

**Figure 3.**
*Changes in Rating of the Real Self by Conflict Level of the Trait Dimension and Alcohol*




The importance of these data to our discussion is their demonstration of the generalizability of alcohol as a conflict disinhibitor and the fact that this capacity of alcohol extends to intrapsychic, self-evaluative conflicts as well as response conflicts.

Herein too is a suggestion of how people can come to have reliable drinking reactions of the sort we know in real life, as for example, a "mean drunk" or a "sentimental drunk," seemingly distinctive individual reactions to alcohol. The reliable experience of certain inhibition conflicts, even intrapsychic ones, in situations in which a person consumes alcohol can cause, through alcohol's disinhibition of these conflicts, reliable drinking reactions. For example, the reliable experience of strong ego needs at gatherings of his colleagues, disinhibited by alcohol, could make our aspiring pianist a reliably egotistical drunk in these settings. This would not occur because of alcohol's pharmacological capacity to cause egotism in general or in people with special alcohol reactivities, but because of alcohol's general ability, through the myopia it causes, to disinhibit otherwise inhibited impulses.

Also, the fact that alcohol can be an elixir for such conflicts suggests that when conflicts become chronic, they may entice alcohol use and, in this way, provide a groundwork for addiction. Alcohol may bring our aspiring pianist so close to his ideal state, for example, as to make the drug powerfully reinforcing psychologically, and if he continues to seek this reinforcement, even physiologically addictive. We now turn to the problem of alcohol addiction.

## Drunken Relief: Alcohol and the Allocation of Attention to One's Worries

As we began to focus on the problem of how alcohol affects psychological stress, we encountered a familiar pattern of highly varied and irregular effects. Alcohol affects a variety of psychological stresses (e.g., anxiety, depression, and fear), and as with social behavior, it does so only intermittently. Sometimes it even increases these stresses (cf. Cappell & Greely, 1987; Steele & Josephs, 1988; Wilson, 1988). But before tackling this puzzle, we offer a word about why it is important.

It should be kept in mind that alcoholism has several causes. Recently, evidence has suggested that some forms of alcoholism have a significant genetic basis (Cloninger, Bohman, & Sigvardsson, 1981; Goodwin 1976, 1979; Petrakis, 1985). The evidence is strongest for early onset alcohol addiction that is associated with a general disposition toward impulsive and antisocial behavior; however, it is important to stress that genes are not the only cause of alcoholism. Estimates of variation in the incidence of alcoholism due to genes—that is, the heritability of alcoholism—range from near 0 to 60%, and recent estimates are closer to 30% (see Searles, 1988, for a thorough review). The important implication, then, is that environmentally induced processes (even allowing for some incidence of alcoholism stemming from Gene ×

Environment interactions) have the most powerful influence on the development of alcoholism.

And this is where behavioral psychology enters the picture. It proposes that learning is an important source of alcoholism: Alcohol does something that is reinforcing to the individual; this effect increases the frequency of drinking; the drinking response then generalizes to other situations and conditions; tolerance for alcohol eventually develops so that more alcohol is needed to produce the same reinforcing effect; and as the addiction is fully developed, one experiences uncomfortable withdrawal symptoms if drinking is stopped (cf. Bandura, 1969; Marlatt, 1976). The principle reinforcing effect of alcohol is presumed to be relief from psychological stress, stated more generally as *tension* in Conger's (1956) classic tension-reduction hypothesis.

It is in this effect that the behavioral etiology of alcohol addiction is presumed to begin, and it is here that the evidence is disturbingly inconsistent. Relevant studies are about equally divided between those reporting a tension-reducing effect of alcohol, those reporting the opposite effect, and those reporting no effect on such varied psychological stresses as social anxiety, depression, and fear (cf. Cappell & Greely, 1987; Marlatt, 1976; Steele & Josephs, 1988; Wilson, 1988).

Thus, a familiar question presented itself: How can the same drug have such varied and irregular effects on psychological stress? Drinking expectancies (cf. Hull & Bond, 1986; Marlatt, 1976) and distinctive individual reactivities to alcohol (cf. Hull et al., 1986; Levenson, Oyama, & Meek, in press; Sher, 1987) again play some role in this variability, but alcohol can have powerful effects on these states even when expectancies are held constant and even among the vast majority of drinkers who have no special reactivity to the drug.

If alcohol myopia could help explain the variability of alcohol's effects on social behavior, might it do the same for alcohol's effects on psychological stress? We came to the idea that as far as the stresses that come from worry are concerned (e.g., anxiety, depression, fear), whether alcohol reduces them in human beings may depend to a significant degree on the single factor of whether the drinker is doing something while he is intoxicated. The reasoning here begins with the central things we know about alcohol myopia: that it restricts attention to the salient, immediate aspects of experience and that it reduces processing capacity so that a greater proportion of this capacity has to be devoted to the demands of immediate, ongoing activity. Thus, when the drinker is doing something that requires attention and thought, alcohol myopia pressures him to attend to and think about that activity over less salient worries. That is, during intoxication, one may simply not have the processing resources to engage in a salient, ongoing activity like watching TV and to brood over one's worries at the same time.

The same idea suggests one condition under which alcohol should increase worry. This should be the case whenever in the absence of distracting activity, troubling thoughts are highly salient and easily understood. Then alcohol's narrowing of perception should focus attention onto these salient worries, and its impairment of thought should make it more difficult to rationalize or in some other way cognitively defend against them, resulting in a crying-in-one's-beer effect.

Let us pursue an example. You get home from work one day worried about the poor raise you received and the continuing atmosphere of underappreciation you suffer. Because it is hot outside you fix yourself a tall gin and tonic to drink while you watch the nightly news. As the alcohol takes hold and as your processing resources become more occupied with the news broadcast, you will probably experience a fading away of your worries, but if you should decide to turn off the news and sit quietly, that same tonic might cause you to cry into it.

Here, then, we have an account of alcohol's effects on psychological stress that suggests how these effects can be so varied and irregular. In this view, alcohol can affect a variety of psychological stresses through its ability to screen out of awareness, in conjunction with activity, a common source of these states, that is, the thoughts that cause them. The variation in these effects, to an important degree, depends on what the drinker is doing while he or she is intoxicated. We have called this reasoning the *attention-allocation model* because it views alcohol and activity as affecting psychological stress by affecting how much attention one can pay to one's worries. It is not, of course, an explanation of alcohol's effect on all stresses, but rather of its effect on psychological stresses that arise from our thinking (e.g., those forms of anxiety, depression, and fear) that are rooted in disturbing thoughts and thought processes. Still, if this reasoning held up to test, it would identify a pervasive reinforcing effect of alcohol, one on which a learning model of addiction could rest quite comfortably. To find out, we tested the effect of alcohol and activity on both anxiety and depression. Because both sets of studies produced similar results and have been published elsewhere (Josephs & Steele, 1990; Steele & Josephs, 1988; Steele, Southwick, & Pagano, 1986), we summarize only the anxiety studies to illustrate the general pattern of findings.

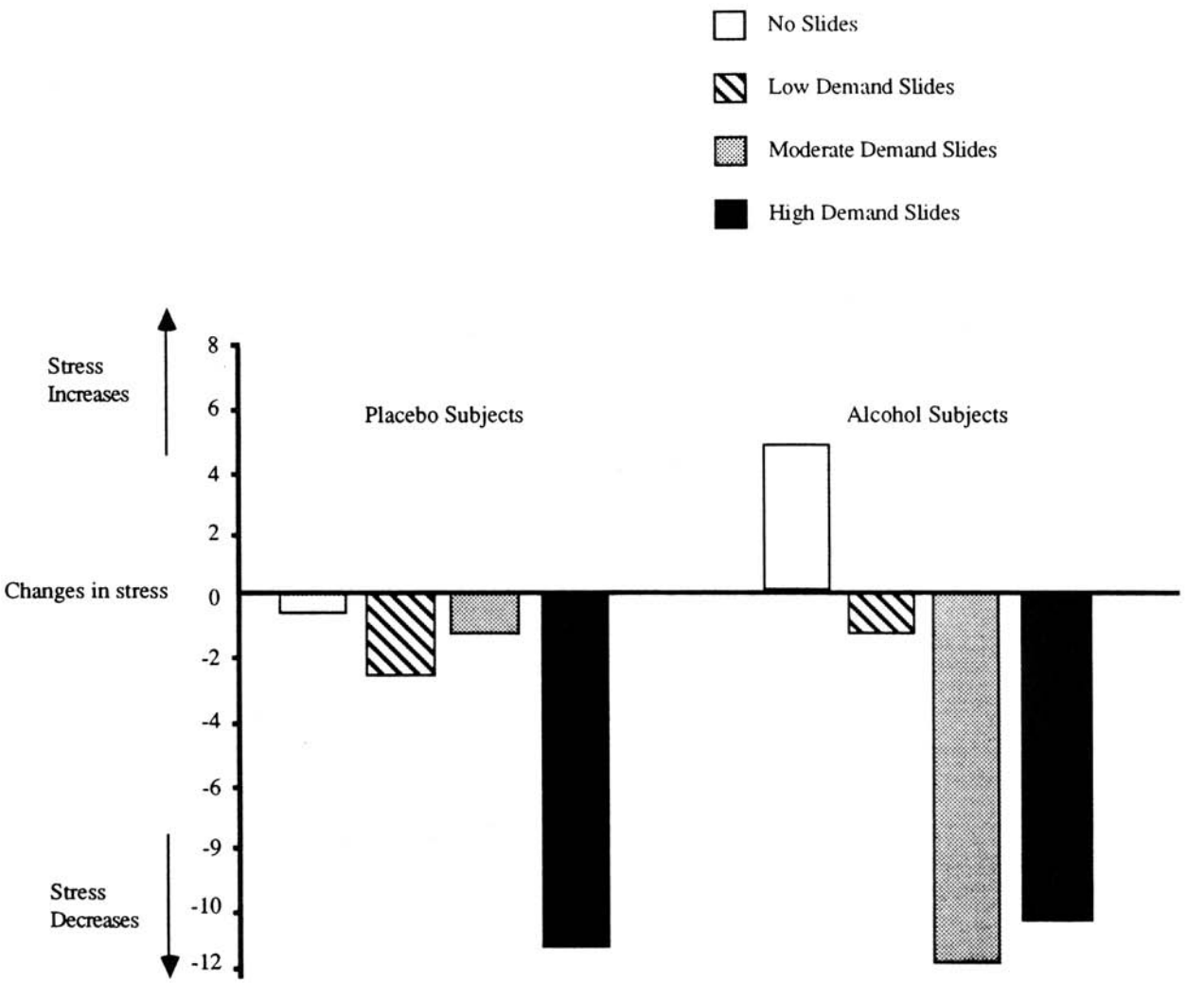
In the first of these experiments, intoxicated and sober subjects learned that immediately after a period of 15 minutes they would give a speech on "What I dislike most about my body and physical appearance" to be evaluated by psychology graduate students—a piece of news that, as one might imagine, raised anxiety in sober and intoxicated subjects alike. One half of the intoxicated and sober subjects then engaged in a distracting activity for the next 7 minutes (they rated the aesthetic features of art slides), while the other half did nothing for this period of time (hooked up to a false recording electrode, they sat quietly, ostensibly providing baseline physiological measures). Subjects' anxiety was measured again 7 minutes into this rating/waiting period, and then, after extensive debriefing, the experiment was over. The only subjects who experienced a reduction of anxiety during this period were those who were intoxicated and rated art slides. By the end of this brief period, these subjects

had recovered entirely from the anxiety caused by news of the speech (some even had to be reminded of the upcoming speech). It worsened among intoxicated subjects who did nothing during this period. For subjects in the other conditions, anxiety either did not improve or worsened.

This effect was replicated in a second experiment as well, further supporting the view that when the drinker is not distracted and stressful cognitions are salient, alcohol actually increases psychological stress—in this case, anxiety.

The argument here is that alcohol and activity reduce psychological stress through the one–two punch of pharmacologically weakening attentional capacities and then occupying them with immediate activity rather than worry. How much worry is reduced should depend on how much attentional capacity has been reduced by alcohol and on how much attention the distracting activity requires. Josephs and Steele (1990) tested this reasoning—and thus the hypothesized mediational processes—by varying the amount of attention required by the distracting activity. Over two experiments, using the anticipatory anxiety paradigm described earlier, subjects were exposed to a no-activity condition and to four conditions that involved increasingly demanding versions of the slide-rating task (i.e., these versions varied from the undemanding task of answering easy questions about the color and contents of the slides to the extremely demanding task of answering more difficult questions while the slides were continuously presented for only three-second exposures). Figure 4 presents the average changes in anxiety over the rating/waiting period for these conditions. Again, alcohol

**Figure 4.**
*Changes in Anxiety Over an Upcoming Stressful Speech by Level of Distraction and Alcohol*



without distraction significantly increased anxiety, illustrating the danger of drinking with nothing else to do. In the other conditions, the more demanding the distracting task, the more alcohol reduced anxiety, until in the condition with the most demanding task, the activity itself, without the aid of alcohol, was sufficient to reduce anxiety.

These data, we believe, support an important conclusion: *Even modest accompanying distraction transforms alcohol intoxication from a sometime reducer of psychological stress into a strong, reliable one that consistently reduces this stress at even moderate doses for all drinkers.* This explains in a simple way how such common drinking experiences as a beer and TV sports or cocktails and conversation can have strong relieving effects that can underlie a behavioral etiology of alcoholism in all drinkers, even when the alcohol involved, taken without these activities, would not.[5] This is not to say that accompanying distraction is always needed for alcohol to reduce psychological stress. Clearly, extreme, near-ataxic doses of alcohol can prevent worry without distraction by preventing thought of any sort. Also, alcohol has been shown to reduce anxiety (most consistently on measures of cardiovascular activity) among some subtypes of subjects—for example, the children of alcoholics (Levenson et al., in press; Sher, 1987) and those individuals who are high in self-consciousness (e.g. Hull et al., 1986)—apparently without accompanying distraction (although variation in experimental procedures makes the role of distraction in these effects difficult to assess; see Wilson, 1988, for a more thorough discussion of these effects). Still, our findings show that even slight distraction greatly increases the magnitude and reliability of alcohol's stress-reducing effect for all drinkers.

The Steele and Josephs (1988) and Josephs and Steele (1990) studies have shown also that alcohol can increase psychological stress in the absence of distraction. This is not to say that this will happen every time one drinks without distraction. As we just noted, alcohol may reduce some forms of stress more or less directly for some people. Also, whether alcohol has an anxiogenic effect depends a great deal on how available and strong the stressful cognitions are during intoxication. We failed to get this effect in the Steele et al. (1986) studies when the stressor was a past event and was less believed by the subjects, but we got it reliably in the Josephs and Steele and Steele and Josephs studies when the stressor was strong, upcoming, and salient. Similarly, Wilson (1988) reported a clear anxiogenic effect of alcohol among a group of interacting alcoholics, but only when a strong social stressor was made the focus of their attention. Thus, without accompanying distraction a beer can make one cry into it, but only if one's troubles are obvious like the nose on one's face.

Such data, we would like to believe, suggest a new understanding of how alcohol can affect psychological stress. To an important degree, these effects are mediated indirectly through the conjunctive effects of alcohol myopia and the demands of ongoing activity. This view has several advantages: (a) It identifies an important and pervasive condition—a distracting activity—under which alcohol has the reliable tension-relieving effect that behavioral scientists have for so long seen as a central root of alcoholism. (b) It shows how alcohol intoxication can be reinforcing even though many of its physiological effects (i.e., nausea, confused thinking, impaired motor control, slurred speech, fatigue, and hangovers), especially during the descending limb of the BAL curve, are so stressful. The protection against psychological distress that one gets from alcohol and distraction may easily outweigh these discomforts. (c) It suggests that chronic psychological stress, whether it stems from the circumstances of one's life or from dispositional factors, is an important susceptibility to alcohol addiction. It creates the conditions under which drinking alcohol (and distraction) can be chronically reinforcing, suggesting that effective treatment of alcoholism would entail treatment of one's troubles as much as one's drinking.

A few comments in closing: Two facts about the effect of alcohol on social behavior and psychological stress have been particularly difficult to explain: (a) the variety of these effects, the fact that alcohol affects so many social behaviors and stresses, and (b) the irregularity of these effects, the fact that they occur only intermittently. In addressing these puzzlements, we have used a strategy focused on alcohol's impairment of perception and thought (the myopia it causes) rather than on other pharmacological properties of the drug, individual reactivities to the drug, or expectancies associated with its use. We offer three conclusions:

1. First, aside from the effects of drinking expectancies, alcohol itself can make human social behavior more extreme. It does this primarily by blocking a form of response conflict. When salient cues strongly motivate a response that, if one were sober, would be inhibited by further access to other cues and meanings (i.e., under inhibition conflict), alcohol myopia makes the response more extreme by reducing access to the inhibiting cues. Aside from such response conflict, alcohol does not appear to have much effect on social behaviors.

2. Alcohol can have profound effects on the depression and anxiety that arise from worries. This can depend on whether alcohol intoxication is accompanied by dis-

[5] Anyone familiar with Hull and Young's, 1983, self-awareness model of alcohol use might wonder how much our results stem from alcohol's reduction of self-awareness. Hull and Young have proposed that alcohol can reduce stress by reducing self-awareness, the higher order processing related to the encoding of information in relation to the self. Thus, alcohol may have reduced stress in these studies by reducing subjects' sensitivity to the self-relevant threat inherent in the stressors. This model does not seem to fit our findings; in particular, the fact that alcohol actually increased stress in reaction to the speech when subjects were not distracted does not fit. Whatever self-awareness-reducing effects alcohol had in this condition were overridden by its effect of focusing subjects' attention onto the stressful speech. The alcohol and activity condition may well have reduced self-awareness in the process of occupying subjects' attention with the task. But this is essentially the same argument as our own: The combination of alcohol myopia and occupying activity made it difficult for subjects to process stressful cognitions about the stressor or its relevance to the self.

tracting activity and on the processing demands of the activity. Together with distraction, alcohol can forge a highly reinforcing and reliable diversion tactic for all drinkers, a means of effectively keeping one's mind off one's worries. Without accompanying distraction, however, alcohol myopia can backfire as a palliative, restricting attention to salient troubles and worsening affect.

3. Alcohol is a reliable means of self-inflation; during intoxication one gets closer to the self of one's dreams. Whether alcohol has this effect depends on whether an important self-evaluative conflict has been made salient. When this happens, alcohol intoxication apparently leaves enough capacity to experience the need for self-regard but not enough to access the reasons for humility.

We offer with some confidence conclusions that describe the effects of alcohol and the conditions that mediate them; we offer more cautiously the conclusions that describe the mediational processes. We still lack a certain kind of direct evidence (a) that alcohol's disinhibition of response conflict is associated with less use of inhibiting cues, (b) that in conjunction with distraction, alcohol's effect on depression and anxiety is associated with variation in attention to one's worries, or (c) that alcohol self-inflation is associated with less processing of self-deflating information. Some of our findings get quite close to this type of evidence, for example, the finding that the effect of alcohol and activity on anxiety varies with the processing demands of the activity. Also, of course, we have been able to rule out alternative explanations we have come up with over the years. Nonetheless, whatever the fruits of these theories, they must still be taken as essentially working theories.

The most certain and practical fruit of this research so far is the identification of general conditions that mediate alcohol's prized and dangerous effects. We can now say that it is largely the nature and level of inhibition conflict that determines whether alcohol intoxication fosters behavioral excesses from altruism to aggression; also, it is whether a distracting activity accompanies alcohol intoxication that determines whether it will uniformly reduce tension or deepen despair. We hope this research underscores the importance of alcohol's social psychological effects in the effort to understand, prevent, and treat the ill effects of this drug.

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