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Resistance to Alcohol Impairment of Visual-Motor Performance II: Effects for Attentional Set and Self-Reported Concentration

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GEORGE, W. H., J. O. RAYNOR AND T. H. NOCHAJSKI. *Resistance to alcohol impairment of visual-motor performance II: Effects for attentional set and self-reported concentration.* PHARMACOL BIOCHEM BEHAV **36**(2) 261–266, 1990.—There has been little or no direct inquiry into the feasibility of nonbiological processes for attenuating alcohol effects. In an initial study, we found that an instructional *Set* (to concentrate) presented at each trial facilitated visual-motor performance among moderately intoxicated subjects. We extended this work in the present study by varying density of the Set presentation across trials, by varying Set onset and offset, and by assessing self-reported concentration levels. After dosing, subjects participated in two pairs of performance trials separated by a rest period. Six groups of subjects differed with respect to the number and sequence of Set presentations received across the postbeverage trials. We found that more Set presentations yielded better performance. Also, onset of Set presentation led to improved or sustained performance, whereas offset led to diminished performance. Finally, consistent with the possibility that concentration serves as a mediating variable, self-reported concentration correlated with performance. Implications of the obtained effects are discussed in the context of tolerance research and practical considerations.

Alcohol impairment Alcohol performance Alcohol resistance Attention Tolerance

THOUGH alcohol reliably impairs visual-motor performance, most drinkers recognize that the effect is subject to some degree of volitional control. A moderately intoxicated drinker, if so motivated, can actively attenuate (or exaggerate) alcohol-induced motor impairment. This observation fosters a proposition that has been largely ignored in the scientific literature: Alcohol impairment can be resisted via psychological (nonpharmacological) procedures and processes that are predominantly motivational in nature. The importance of volitional alcohol resistance lies in both its basic and applied implications. If alcohol resistance is a reliable and valid phenomenon, then the basic understandings of alcohol's motor effects and of tolerance will merit reexamination and possible revision. Furthermore, evidence for alcohol resistance would support the feasibility (though not the advisability) of developing interventions aimed at counteracting alcohol effects.

ALCOHOL RESISTANCE

In an initial study of alcohol resistance, we examined two motivational aids: an instructional *Set* ("to concentrate very hard") and an auditory feedback *Signal*. We found that moderately intoxicated subjects who received either aid outperformed unaided subjects on a modified pursuit-rotor task (5). Subjects who received both Set and Signal outperformed all other intoxicated subjects and performed equivalently with unaided placebo subjects. Analyses of the Trial 1 data and use of habitual drinking as a covariate provided confidence that the findings were not due to acute or chronic tolerance.

The subjects seemed to comply with a demand to exert more attentional effort than they otherwise would. The demand was direct and explicit in the Set condition and was presumably implicit in the Signal condition. Despite alcohol's pharmacological capacity as a CNS depressant to alter all motor responding, the fine and gross motor capabilities required to perform the task remained largely within subjects' volitional control. Therefore, heightened effort enabled subjects to resist alcohol's otherwise debilitating motor effects and to behave as though less intoxicated. Increased concentration was suggested to be the critical mediating factor.

The present study examines two hypotheses that postulate modulation of the attentional Set effects observed in the earlier study. In addition, self-reported concentration levels were assessed to examine the correlation between concentration and performance.

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1. Set density hypothesis: does modulation in density of the Set presentation across postbeverage trials yield varying degrees of alcohol resistance? More frequent Set presentation should yield better intoxicated performance or resistance.

2. Set onset versus offset hypothesis: Once postbeverage performance trials have begun, does onset or offset of the Set presentation alter performance? Generally, onset of the Set should improve or sustain performance while offset should diminish performance.

TOLERANCE

As with the proposed alcohol resistance phenomenon, functional tolerance (not due to altered metabolic processes) also involves reduced alcohol impairment. With tolerance, however, the reduced impairment develops over time, either within a single drinking episode (acute tolerance) or across multiple episodes (chronic tolerance). By definition, "the occurrence of tolerance is inferred when the readministration of a drug dose yields a weaker effect, or when a higher dose is required to reinstate the initial effect" (13). Unlike tolerance, alcohol resistance is not time dependent and therefore does not require the demonstration or inference of multiple and sequential assessments of an alcohol effect across time. Alcohol resistance can therefore be demonstrated at a single assessment in a between-subjects design using subjects who do not differ in chronic tolerance.

Learning Versus Motivational Factors

Learning factors have been implicated as important mediators of tolerance development [e.g., (1, 7, 8, 14, 15)]. This idea has received only mixed support in human studies of instrumental behavior. Some work showed that actual and simulated (mental rehearsal) task practice under alcohol facilitated pursuit rotor performance (12,13). Other work indicated that only task practice reinforced by auditory feedback, knowledge of results, and monetary reward facilitated tolerance (6,9). With acute tolerance, some studies have not detected single-session effects and others have yielded ambiguous evidence for tolerance reliant on monetary reward (10).

The uncertain viability of a learning account of human tolerance may be due, in part, to a methodological artifact remedied by the present paradigm. Specifically, earlier studies have confounded learning (skill acquisition) and motivational (skill expression) factors by using difficult tasks (e.g., 33–69% efficiency). In using a simpler task (90% efficiency), our alcohol resistance paradigm permits clearer explanatory analyses that more clearly restrict the interpretation to skill expression factors. Therefore, further support for alcohol resistance could have both conceptual and methodological implications for research on learning factors in tolerance. Specifically, it is possible that previous support for learning explanations may be due to strictly motivational factors.

METHOD

Subjects

Subjects were 24 male student volunteers. Ranging in age from 19 to 28 years (mean = 21.88, SD = 2.54), subjects were above the then current legal drinking age and were screened for medical contraindications to alcohol. Each subject was instructed to undergo a 24-hour abstinence from alcohol and drug consumption and a four-hour abstinence from food consumption before the scheduled lab session.

Apparatus

A modified pursuit rotor task was used to assess visual-motor performance. The standard pursuit rotor task involves tracking a target light (henceforth target) with a light-sensitive stylus while the target moves in particular pattern. For our work (5), the task was modified by attaching a transparent Plexiglas steering wheel so that the stylus moved in a circular pattern when the wheel was turned. The wheel with stylus was then mounted on a frame to incline the pursuit rotor display. When seated in front of the apparatus, the subject could track the target by turning the steering wheel. The movement pattern of the target was designed so that the target remained in the upper half (180 degrees) of the pursuit rotor apparatus. At the start of each 32-sec repetition, the target was centered in the middle of the pursuit rotor display. From this center position, the target movement corresponded to a fixed sequence of eight moves: 45 (degrees) to the right, 90 to the left, 45 to the right, 45 to the left, 90 to the right, 45 to the left, 45 to the right, and 45 to the left. This sequence constituted a 32-sec repetition and ended with the target repositioned at the center.

Scoring of visual-motor performance. An electronic timer was connected to the apparatus. It measured the total time-on-target for each 32 sec repetition. Scores were recorded to the nearest 1/100th sec. Subjects could not see the timer and never received time-on-target scores.

A performance trial consisted of four 32-sec repetitions of the 8-move pursuit rotor sequence. Each repetition within a trial was separated by a 15-sec interval. The score for a trial was the mean time-on-target in seconds for the four repetitions. To establish baseline performance, there were 3 prebeverage trials. Each postbeverage trial score was converted to a percentage-of-baseline score. To accomplish this, each postbeverage time-on-target score was divided by the final preverage baseline score, then multiplied by 100. This percentage-of-baseline performance score eliminates possible differences in postbeverage performance that are due to prebeverage differences in competence.

Experimental Design

Following three prebeverage training trials, there were four postbeverage performance trials. The Set manipulation was varied systematically across these postbeverage trials. The four trials were divided into two trial blocks which were separated by a longer rest period than the two trials within each block. Eight-minute periods separated the starts of Trials 1 and 2 and the starts of Trials 3 and 4; but a 16-minute period separated the starts of Trials 2 and 3. This distinction between the shorter trial-to-trial rest period and the longer block-to-block rest period enabled evaluation of the possibility that impact of a change in Set may vary with intertrial interval.

Subjects were randomly assigned to six groups that differed in the onset and offset of the Set manipulation across the four trials. The number and sequence of Set (S) and No Set (N) trials varied in correspondence with six arrangements: 1) NN-NN, 2) NN-SS, 3) SS-NN, 4) SS-SS, 5) NS-SS, and 6) SN-NN (hyphen indicates the longer block-to-block rest period). These particular six groups permitted conduct of specific planned comparisons for evaluating the Set density hypothesis and the onset-versus-offset hypotheses.

Procedure

Assessment of drinking practices. Drinking practices were assessed prior to the experiment using the Daily Drinking Questionnaire (DDQ). It is a brief version of the Drinking Practices Questionnaire (DPQ) (2), and has been shown to have convergent validity with the longer DPQ (4). Instructions for the DDQ, defined a drink as "either 12 oz. of beer, 4 oz. of wine, or a 1 oz. shot of liquor either straight or in a mixed drink." The DDQ required subjects to classify their regular drinking pattern on the basis of either weekly, monthly, or yearly consumption. From an item assessing average consumption for each day of a typical

week, we computed average total drinks per typical week. This consumption score ranged from 1 to 20 drinks (mean = 8.80, SD = 5.81). Four indicators of drinking practices were computed: total number of drinks per week, number of drinking days per week, number of drinks consumed per drinking day, and number of heavy (5 or more drinks) drinking days per week.

Assessment of blood alcohol concentration. Upon arrival at the lab, each subject was checked for proof of age and administered the consent form. An initial b.a.c. assessment was administered to insure that subjects were alcohol-free prior to beverage administration. Subsequent assessments were conducted immediately after each postbeverage trial, thus at 31, 39, 55, and 63 min after subjects began beverage consumption. All b.a.c. assessments were taken with an Alco-Sensor II intoximeter.

Assessment of self-reported concentration. After completion of all performance trials, subjects were asked to estimate their levels of concentration retrospectively for three particular points of time: 1) before beverage consumption, 2) after beverage consumption but before the extended rest period, and 3) after the extended rest period. The question "to what extent did you remind yourself to pay attention and concentrate on keeping the stylus on target?" was asked for each of the three points of time. The response scale for each concentration question consisted of the following options: none of the time, one-quarter of the time, one-half of the time, three-quarters of the time, all of the time. Concentration ratings were scored from 1 to 5, with 1 indicating lowest and 5 indicating highest concentration. A percentage-of-baseline concentration score was obtained for each subject by dividing each postbeverage concentration score (Prerest and Postrest) for that subject by the Prebeverage concentration score, then multiplying by 100. Possible differences in postbeverage concentration due to individual differences in prebeverage concentration are eliminated by this percentage-of-baseline score.

Distractor tasks. Three distractor tasks (magazine reading and digit spans, forward and backward) were administered at certain times to structure subjects' attentional focus and to minimize scrutiny of interoceptive intoxication cues. The tasks were administered during alcohol absorption, after each postbeverage performance trial, and during the extended rest period. Thirteen minutes of magazine reading followed completion of beverage consumption and 13 minutes of magazine reading occurred during the rest period. Digit spans were presented before drinking and then after postbeverage performance Trials 1 and 3.

Beverage administration. Each subject was weighed upon arrival to determine the amount of 80 proof vodka that was needed to achieve a b.a.c. of 50–60 mg% (dosage = 0.70 g ethanol/kg body weight). Drinks consisted of a one-part vodka to three-part orange juice preparation that was mixed within full view of the subject. Subjects in the six alcohol conditions observed vodka being poured and measured carefully from a legitimate vodka bottle (Boards) and a juice container into a calibrated cylinder. These procedures were the same for subjects in the placebo condition except that the vodka bottle contained only water. After mixing, the beverage was poured into two glasses dividing it into two equal portions. Subjects were instructed to pace their drinking evenly over a 7.5-min period for each glass. The 15-min consumption period was followed by a 13-min absorption period.

Set manipulation and visual-motor assessment. For initial training and prebeverage trials, the visual-motor task was introduced with the unadorned instruction that the object was "to keep the stylus on the moving light target." According to condition assignments described earlier, postbeverage performance trials either were accompanied or unaccompanied by a slightly revised version of the Set manipulation used in our previous study (5).

On Set trials, subjects were instructed as follows. "During the two-minute period, while performing the task, pay as close attention as you can to keeping the stylus on target. Concentrate as hard as you can to keeping the stylus on target. Remember, pay as close attention as you can to keeping the stylus on target." On No Set trials, the above instructions were not given and subjects merely performed the visual-motor task as they had during the prebeverage phase of the study. Thus, No Set trials were characterized by absence of the Set rather than by an active instruction not to concentrate.

Overall, the first postbeverage trial began 28 min after presentation of the first drink and the entire postbeverage visual-motor assessment lasted about 35 min. Placebo condition subjects were queried and debriefed about the effectiveness of the deception. All other subjects were driven to their local residence.

RESULTS

Manipulation Checks

Drinking practices. A one-way ANOVA was used to compare the six groups on self-reported indicators of drinking practices. There were no significant effects for either of the four indicators: total number of drinks per week, $F(5,15) = 1.33$, $p = 0.30$, number of drinking days per week, $F(5,15) = 1.56$, $p = 0.23$, number of drinks consumed per drinking day, $F(5,15) = 1.16$, $p = 0.38$, and number of heavy drinking days per week, $F(5,15) < 1$. These null effects indicated that there were no preexperimental differences in drinking habits and that random assignment had successfully controlled for such differences. The correlation between average weekly consumption and average performance further confirmed that variations in drinking practices were not influential in the current sample ($r = .12$, $p = 0.29$) and ruled out the viability of drinking practices as a covariate in subsequent hypothesis testing. Assuming a positive association between drinking practices and tolerance [e.g., (11)], the above findings are consistent with the contention that the six groups were equivalent in chronic tolerance. Again this equivalence would be expected from effective random assignment.

Blood alcohol concentration. Consistent with the administered dosage, a moderate b.a.c. level was achieved across all groups and across trials, mean = 0.052. A 6 × 4 group × trial ANOVA on b.a.c. failed to yield a significant group effect, $F(5,18) = 1.15$, $p = 0.37$, or interaction effect, $F(15,54) = 1.45$, $p = 0.16$. A trial effect, $F(3,54) = 2.26$, $p = 0.09$, indicated marginal variation in b.a.c. across trials (means = 0.050, 0.054, 0.053, 0.051). Surprisingly, there was no correlation between b.a.c. and average performance ($r = .01$). Evidently, there was not sufficient heterogeneity in b.a.c. in the current sample to document the standard negative relationship between intoxication and performance. This nonfinding also ruled out b.a.c. as a viable covariate in hypothesis testing.

Demonstration of alcohol impairment on performance. It was ascertained that alcohol intoxication impaired performance, in the absence of Set. This was accomplished by using half the subjects ($n = 12$) as their own control to compare sober with intoxicated performance. Using only those subjects who did not receive the attention Set on the first postbeverage trial, the analysis compared performance on the last prebeverage trial against performance on the first postbeverage trial and revealed a significant decline, $F(1,22) = 8.25$, $p = 0.009$. This established that the level of intoxication achieved in this study was sufficient to impair performance significantly.

Hypothesis Testing

To reiterate, the design consisted of six groups that differed in the number and sequence of Set (S) and No Set (N) trials: (1) NN-NN, (2) NN-SS, (3) SS-NN, (4) SS-SS, (5) NS-SS, and (6)

TABLE 1

MEAN (SD) PURSUIT-ROTOR SCORE ON FOUR PERFORMANCE TRIALS FOR SIX SUBJECT GROUPS THAT DIFFER IN THE SEQUENCE OF SET (S) AND NO SET (N) TRIALS BEFORE AND AFTER A REST PERIOD (–)

	Trial Means (SD)			
Groups	1	2	3	4
NN-NN (n=4)	98.15 (0.49)	97.06 (2.72)	92.68 (3.92)	93.15 (7.19)
NN-SS (n=4)	94.08 (4.57)	92.81 (4.07)	94.11 (0.43)	94.68 (1.15)
SS-NN (n=4)	101.80 (1.97)	99.51 (1.04)	98.32 (2.85)	96.48 (5.27)
SS-SS (n=4)	97.28 (2.91)	95.69 (3.25)	95.93 (3.38)	94.87 (3.49)
NS-SS (n=4)	99.90 (0.30)	100.31 (0.81)	99.38 (1.56)	98.64 (1.20)
SN-NN (n=4)	97.13 (1.69)	93.52 (2.93)	94.76 (0.87)	92.99 (1.94)

SN-NN. Table 1 displays the mean performance scores for each group on each trial. The experimental hypotheses were tested by conducting a series of analyses of variance (ANOVA) and planned comparisons involving alternate clusterings of the six groups.

Set Density Hypothesis

To test the hypothesis that more frequent presentations of Set would yield better performance (more resistance), the six groups (total n = 24) were collapsed into three groups based on whether subjects received a minority of Set trials (NN-NN and SN-NN groups), an equal number of Set and No Set trials (NN-SS and SS-NN groups), or a majority of Set trials (SS-SS and NS-SS groups). This design permitted a straightforward linear test of the Set density hypothesis using a one-way ANOVA with planned comparisons. Because Set density accumulates across trials, the hypothesized Set density effects should be evident in the second block of trials.

Analysis of average performance in the second trial block yielded the hypothesized linear effect of Set density, $F(1,21) = 5.64$, $p = 0.027$. Planned contrasts within the linear component revealed that the majority Set group performed significantly better than the minority Set group, $t(21) = 2.37$, $p = 0.027$. Neither the majority Set (mean = 97.21) nor the minority Set (mean = 93.39) group was distinguished from the equal Set (mean = 95.90) group which performed intermediately.

Set Onset Versus Offset Hypothesis

It was hypothesized that onset of the Set would generally improve or sustain performance while offset would diminish performance. This hypothesis was evaluated using two analytic groupings. The first utilized the four groups for whom Set presentation either changed or remained constant across the two trial blocks. For these four groups, Set presentation always remained constant within the two trial blocks. The second analytic grouping used the two remaining groups, for whom Set changed within the first trial block.

Onset versus offset across trial blocks. A 2 × 2 × 2 between-within ANOVA was conducted using four groups (total n = 16). The between-subject variables were presentation of Set prior to the extended rest period (prerest Set versus prerest No Set) and after the rest period (postrest Set versus postrest No Set). The within subject variable was trial block (prerest versus postrest block). The effects for prerest Set, trial block, and the postrest Set by trial block interaction were either significant or near significant.

The effect for prerest Set showed that subjects who received the Set in the prerest trial block did better across the blocks than counterparts who had not received the prerest Set, $F(1,12) = 4.62$, $p = 0.053$ (means = 97.49 versus 94.59). This suggests that Set presentation in the initial trial block not only generated better performance (consistent with findings from our previous study), but also produced effects that lingered across blocks regardless of subsequent constancy or offset of the Set.

The trial block effect showed that performance generally declined from the prerest block to the postrest block, $F(1,12) = 5.08$, $p = 0.044$ (means = 97.05 versus 95.03). However, this effect was qualified by a postrest Set by trial block interaction, $F(1,12) = 4.75$, $p = 0.050$. Postrest Set sustained performance across the trial blocks (means = 94.97 versus 94.89), whereas subjects in the postrest No Set condition exhibited a decline in performance (means = 99.13 versus 95.16). Therefore, irrespective of prerest Set, postrest Set enabled subjects to resist performance decrement.

Onset versus offset within the first trial block. A 2 × 2 between-within ANOVA was conducted using the two groups (total n = 8) who experienced a change in Set within the first trial block. The between-subject variable was group (NS-SS versus SN-NN groups) and the within subject variable was trial (trial 1 versus trial 2). There were significant effects for group, $F(1,6) = 17.39$, $p = 0.005$, and for trial, $F(1,6) = 12.05$, $p = 0.013$. These effects were qualified by a significant group by trial interaction, $F(1,6) = 19.19$, $p = 0.005$. For subjects who experienced Set onset (NS-SS), performance was sustained from trial 1 (mean = 99.90) to trial 2 (mean = 100.31). However, for subjects who experienced Set offset (SN-NN), performance deteriorated from trial 1 (mean = 97.13) to trial 2 (mean = 93.52). This pattern is consistent with the hypothesis.

Concentration and Performance

We examined the argument that concentration mediates alcohol resistance. First, using the three-group design, we tested the assertion that Set increases self-reported concentration. A one-way ANOVA revealed a marginal linear effect for Set density, $F(1,21) = 3.53$, $p = 0.074$. Planned contrasts revealed that the majority Set group reported more concentration than the minority Set group, $t(21) = 2.19$, $p = 0.040$. Neither the majority Set nor the minority Set groups was distinguished from the equal Set group which reported intermediate concentration. Second, we correlated retrospectively self-reported concentration with performance. Overall, concentration correlated modestly and significantly with average performance, $r = .392$, $p = 0.029$. Taken together, these two findings are consistent with the argument that Set increased concentration which, in turn, improved performance.

DISCUSSION

The present study replicated and extended the earlier finding that presentation of an attentional Set (to concentrate) enabled intoxicated subjects to resist alcohol impairment effects on visual-motor performance. The key new findings are that alcohol resistance varies with Set density and is characterized by demonstrable onset versus offset effects. Also, self-reported concentration correlated with performance. Random assignment and equivalence of the test groups on preexperimental drinking practices provided confidence that none of the obtained findings were attributable to artifacts involving chronic tolerance. Furthermore,

equivalence of the groups on b.a.c. levels across trials ruled out the possibility of artifactual effects involving acute tolerance or variable intoxication.

In support of the Set density hypothesis, subjects who received a majority of Set presentations across four postbeverage performance trials outperformed counterparts who received a minority of Set presentations. This is consistent with the earlier study where subjects who received the Set uniformly across four postbeverage trials outperformed No Set subjects. The present finding suggests a dose-like effect of the Set. That is, intoxicated performance improves linearly with more frequent Set presentations.

The Set onset versus offset hypothesis was examined in two ways. First, Set effects for before versus after an intervening rest period were evaluated independently and interactively. A postrest Set by trial-block interaction revealed that absence of Set during the postrest period resulted in diminished performance but presence of the Set resulted in stable performance across blocks. This is consistent with the onset versus offset hypothesis in that the effect of the postrest Set was to sustain performance that would have otherwise deteriorated. However, the lack of a triple interaction involving prerest Set indicates that this maintenance effect of the postrest Set occurred regardless of whether subjects had experienced Set onset or Set constancy in the second block. In a second and more pointed examination of the onset versus offset hypothesis, the effects of a trial-to-trial change in Set within the prerest period were evaluated. Consistent with the hypothesis, subjects who experienced Set offset exhibited a significant decline in performance from trial 1 to 2, while subjects who experienced Set onset did not. Overall, these findings indicate that when considered in the context of ongoing intoxicated performance, Set presentation enhances early performance and sustains later performance. Whether presented in early testing, late testing, or throughout testing (as in our previous study), the Set has a demonstrably positive impact on intoxicated performance.

The concentration data indicated two points. First, the Set was effective in increasing concentration. This indicates simply that people who were told to concentrate reported more concentration. Because of the demand to comply with instruction and the subjective nature of the measure, the veridicality of these self reports is suspect and cannot be ascertained. However, unsystematic anecdotal data were consistent with the obtained Set effects on concentration. Set subjects exhibited more behaviors indicative of greater concentration (leaning forward, furrowed brow, more continuous eye-to-target gaze, etc.) than No Set subjects. More definitive validation of Set effects on concentration will require more objective concentration measures (e.g., estimates by observers who are blind to condition assignment). Second, increased concentration was generally associated with better performance. It appears that the Set presentation led to increased concentration which, in turn, improved performance. Though not directly supportive, these data are at least consistent with the argument that concentration mediates between Set exposure and performance.

It is our contention that any viable account of the alcohol resistance effects observed in the previous and present studies must consider certain basic points about alcohol-behavior effects in humans.

1) Acute alcohol intoxication impairs visual-motor performance. However, at low to moderate levels of alcohol intoxication, the fine and gross motor responses needed to execute a simple visual-motor task remain largely under the volitional control of the human behaver.

2) Provided that sober performance of the task has been mastered, the behaver can resist the normally impairing effects of moderate alcohol intoxication on the same visual-motor task by actively exerting more intensified effort at performing well. If motivated to do so, the behaver will exert such effort and will exhibit better intoxicated performance than he or she otherwise would.

3) Extrinsic conditions that influence the behaver's motivation to exert intensified effort will indirectly influence alcohol resistance. Consequently, extrinsic conditions such as incentive reward, informational feedback, and verbal praise and encouragement should enhance motivation and thereby enhance alcohol resistance. Thus far, we have demonstrated that an instructional set ("to concentrate as hard as you can") and an auditory feedback signal improved alcohol resistance. Moreover, modulation of the extrinsic conditions should yield predictable dose-like variation in alcohol resistance.

4) The proximal determinant of increased exertion of effort is an internal state that mediates the extrinsic conditions as well as intrinsic dispositions and states. Presently, we have conceptualized this internal state as heightened concentration or attention that is consciously experienced and willfully controlled.

Tolerance

Functional tolerance involves reduced alcohol impairment that develops over time either within the same drinking session or across drinking experiences. Because tolerance by definition requires within-subject time-dependent evidence of change, between-subject alcohol-resistance effects exhibited right after drinking cannot be easily explained by appealing to tolerance phenomena. Still, the present data can inform the literature on human tolerance.

The omission of uniquely human psychological processes from tolerance research has limited our understanding of human alcohol tolerance. Current understandings of human alcohol tolerance have been heavily shaped by animal research emphasizing biological (pharmacological/biochemical/genetic) and/or environmental (conditioning/learning) processes. Accordingly, human tolerance studies have tended to emphasize explanatory forces and analyses built on these processes. This trend has fostered minimization and exclusion of uniquely human psychological processes in explanations of tolerance. Without incorporation of these processes, analyses of human tolerance will remain incomplete. Specifically, constructs that capitalize on language and thought mediated influence over behavioral expression have been neglected. An implication of the present work is that incorporation of such constructs may promote a fuller understanding of human alcohol tolerance, at least with responses reliant on the voluntary musculature. For example, Set manipulations may increase the speed or otherwise alter the process of tolerance development.

Limitations, Caveats, and Conclusions

There are important limitations with this work on alcohol resistance. First, intoxication level was modest and was restricted to the ascending (previous study) and peak portions of the b.a.c. curve. Further study is needed to determine if the observed effects are particular to these conditions. The capacity to resist alcohol impairment no doubt diminishes with increasing dosage. Second, all subjects in the present study received alcohol. Therefore, it is not known whether Set effects would be different in placebo or no-alcohol control subjects. Larger Set effects for sober than intoxicated subjects would suggest that intoxication reduces the degree to which an individual can exploit Set effects. Further research is needed to clarify the possibility of important alcohol by Set interaction effects. Third, concentration level was measured through retrospective self-report. Therefore, it cannot be ascertained that concentration is a critical mediating variable. Finally, the observed Set effects were obtained with a behavior subject to voluntary control. It is unknown whether Set effects would extend to behavioral indices that are less controllable (e.g., the sway test).

An implication of this work is that it supports the feasibility of nonbiological interventions aimed at counteracting alcohol impairment effects. However, it is important to distinguish the scientific from the practical merits of this implication. Scientifically, the development of such interventions is vital for documenting heretofore unrecognized malleability in alcohol's behavioral effects and for thereby revising our understanding of alcohol's drug action. Practically, various ethical considerations limit the advisability of developing such interventions. A risk is that the existence of such interventions could inadvertently convey dangerous messages to the drinking public. Problematic messages might minimize negative behavioral sequelae to abusive drinking, lend explicit official sanction to the value of "holding one's liquor," and convey false confidence in the ability to drive or operate dangerous machinery after drinking. Such inadvertent messages could soften treatment motivation for abusers and could promote heavier drinking for both users and abusers.

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