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BRIEF COMMUNICATION

Retrograde Enhancement by Alcohol of Delayed Free Recall Performance

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MANN, R. E., J. CHO-YOUNG AND M. VOGEL-SPROTT. *Retrograde enhancement by alcohol of delayed free recall performance.* PHARMACOL BIOCHEM BEHAV 20(4) 639–642, 1984.—Two experiments are reported in which retrograde enhancement of human memory by alcohol was observed. In both studies male undergraduate volunteers performed an immediate free recall task before and after consuming either alcohol (0.66 g abs alc/kg) or placebo. About two hours later, delayed free recall was tested when subjects were asked to write down as many words as they could remember from the free recall trials in the session. Subjects given alcohol recalled significantly more words from lists heard before drinking than subjects given placebo; this effect appeared more prounounced for words from the primacy portion of the lists. The possibility that this retrograde enhancement effect is due to alcohol's effects on brain reward systems is raised.

Alcohol Memory Humans Retrograde enhancement

THE considerable literature on the effects of alcohol on human learning and memory indicates that alcohol impairs acquisition and retention of new material (e.g., [10, 12, 15]). However, recent data suggest that alcohol, when administered immediately following acquisition of new material, enhances memory for that material [13,14]. This retrograde enhancement by alcohol stands in strong contrast to alcohol's impairment of learning and memory which occurs after consumption. Since the phenomenon may have great importance for understanding alcohol's effects on learning and memory, replication and extension of the finding would provide essential information on its robustness and conditions of occurrence.

Two experiments were performed to examine the effects of alcohol on free recall performance. In the first experiment repeated drinking sessions were scheduled, while the second experiment involved a single drinking session. The retrograde enhancement effect was observed in both experiments on a delayed free recall task.

EXPERIMENT 1

Method

The subjects were 12 male undergraduate volunteers, between the ages of 19 and 28, who reported no use of prescription medication and whose drinking fell within normal social limits as assessed by Vogel-Sprott's [17] questionnaire. The subjects were randomly assigned to alcohol (n=8) or placebo (n=4) groups; all agreed to eat nothing for four hours prior to each drinking session and to abstain from all drugs, including alcohol, for 24 hours prior to each session.

For for the free recall task, sets of five ten-word lists were constructed at random from the one and two syllable words in the Thorndike-Lorge [16] A and AA lists. During task performance the tape-recorded lists were presented to subjects at a rate of one word per second. Following presentation of each ten-word list subjects were allowed 50 sec for immediate free recall. Five lists were presented during each trial; thus five min were required to complete a trial.

The subjects first attended two nondrinking training sessions for task practice. Subjects then attended a series of six drinking sessions, which were separated by an average of four days, so that the effects of repeated exposures to alcohol could be examined. The scehdule of testing in each session was identical. Subjects in the Alcohol group were given a total dose of 0.66 g/kg absolute alcohol in each session. The alcohol (94.6% v/v ethanol; Consolidated Alcohols, Toronto, Ontario) was mixed 1:2 with a carbonated beverage (Wink®) and divided into three equal drinks; these drinks were served at 20 minute intervals and subjects were allowed five minutes to consume each one. Placebo subjects received an equivalent total volume of carbonated beverage on the same schedule, with a few ml of alcohol floated on top of each one. Blood alcohol content (BAC) was measured by an Omicron Intoxilyzer (Omicron Systems, California) at 20, 40, 60, 80, 110 and 140 min after the start of drinking. The first immediate free recall trial (presentation and testing)

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began 10 min before drinking started. The second immediate free recall trial began 61 min after the first drink, when peak BAC's were estimated to occur. The delayed free recall trial occurred 141 min after the start of drinking; subjects were given two min to write down any words remembered from the two immediate free recall trials in that session. New lists were used for each session.

Results

Alcohol subjects' BAC's were analyzed with a 6 (sessions) × 5 (trials) analysis of variance. Only the trials effect reached significance, $F(5,35)=154.27$, $p<0.05$, revealing a typical rising and falling BAC curve. The mean peak BAC (±SEM) over sessions of 76±2 mg/dl occurred 60 min after the start of drinking, and the mean BAC (±SEM) over sessions when the delayed free recall trial occurred was 44±3 mg/dl.

The words recalled by subjects in the delayed free recall trials were sorted according to whether they originated in the primacy (serial positions 1–5) or recency (serial positions 6–10) portions of the pre-drinking or post-drinking lists. A 2 (groups) × 6 (sessions) × 2 (pre/post drinking lists) × 2 (primacy/recency words) analysis of variance yielded a significant groups × pre/post lists interaction, $F(1,10)=8.82$, $p<0.05$. Mean words recalled from the pre/post lists displayed both the retrograde enhancement effect and the impairing effect of alcohol on retention. Thus, simple main effects tests [18] demonstrated that Alcohol subjects recalled significantly more words from the pre-drinking lists than Placebo subjects ($p<0.05$; mean words recalled per group=1.8 and 1.0, respectively), while Placebo subjects recalled significantly more words from the post-drinking lists than Alcohol subjects ($p<0.05$; mean words recalled per group=1.9 and 1.2, respectively). As well, the interaction of groups, pre/post lists, and primacy/recency words approached significanace, $F(1,10)=3.74$, $0.05<p<0.10$. This interaction is summarized in Fig. 1, which presents the mean primacy and recency words recalled per session from pre-drinking and peak BAC lists by the two groups. The figure suggests that the interaction was due to a tendency for the retrograde enhancement effect to be more pronounced for primacy than for recency words. Neither the main effect of sessions nor any of the interactions of sessions with other factors reached significance (all p's>0.10), indicating that the phenomenon was consistently observed over repeated exposures.

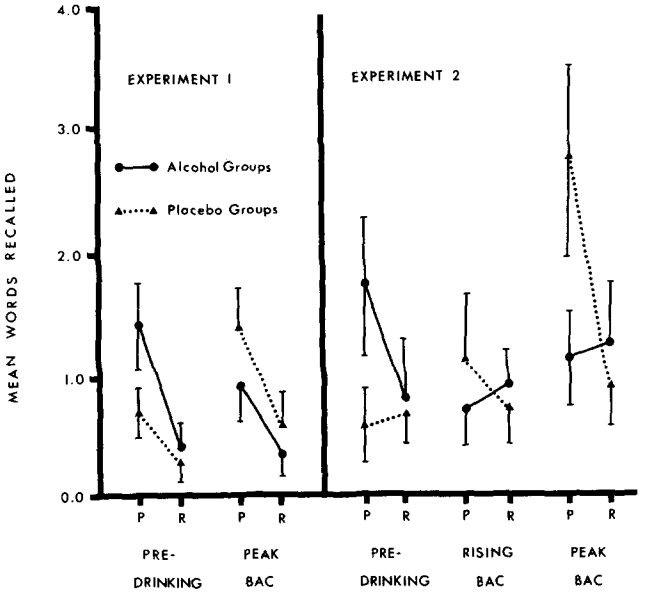

FIG. 1. Delayed free recall (±SEM) of primacy (P) and recency (R) words from pre-drinking and post-drinking lists, Experiments 1 and 2 (data from Experiment 1 averaged over six drinking sessions).

EXPERIMENT 2

Method

Twenty male volunteers between the ages of 19 and 38 were subjects in the second experiment, which was designed to replicate and extend the results of the first. A single drinking session was employed since the first experiment suggested that the phenomenon was observed consistently over sessions. Restrictions on subject selection, eating, and drug and alcohol consumption were identical to those of the first experiment. Subjects were randomly assigned to one of four groups (n=5 per group); two groups received alcohol and two received placebo. Subjects in one of each of the Alcohol and Placebo groups were required to repeat each word in the immediate free recall task when it was presented, while the remaining two groups did not repeat the words.

The word lists for free recall used in this experiment were chosen from among sets of five ten-word lists constructed for Experiment 1. The tape-recorded lists were presented to subjects at a rate of one word per two sec, and following presentation of each ten-word list 50 sec were allowed for immediate free recall.

All subjects attended a brief nonalcohol practice session followed at least 24 hours later by the single drinking session. The dose of alcohol or placebo and the drink administration schedule were identical to those used in the first experiment. Subjects began the first (pre-drinking) trial on the immediate free recall task ten min prior to receiving the first drink. The second trial was administered 50 min after receiving the first drink, in order to test Alcohol subjects at rising BAC levels. The third trial was administered 61 min after receiving the first drink, while Alcohol subjects were at peak BAC levels. Since the fourth trial was designed to test Alcohol subjects at a falling BAC comparable to their rising BAC, the time for this test could vary for each Alcohol subject (resulting times of administration ranged from 85 to 130 min after receiving the first drink). Individual Alcohol and Placebo subjects were matched to determine when the fourth trial was administered to the latter subjects. The delayed free recall trial was administered 141 min after receiving the first drink, when subjects were given six min to write down as many words as they could remember from the lists heard in the drinking session. BAC's measured 49, 60 and 140 min after starting to drink, and at variable times between 85 and 130 min.

Results

Alcohol subjects' BAC's from the 49, 60 and 140 min measures were examined with a 3 (trials) analysis of variance. No significant effect was observed, although a rising and falling BAC curve was reflected in the data. The mean BAC's (±SEM) at these times were 56±3, 59±4 and 54±2 mg/dl.

The words recalled in delayed free recall were sorted according to the sets of lists (pre-drinking, rising BAC, peak BAC, falling BAC) and the portion of the lists (primacy and recency) they originated from. The results from the pre-drinking, rising BAC and peak BAC lists were analyzed separately from the falling BAC list data, since the former occurred at fixed times in the experiment while the latter occurred at variable times. Preliminary analyses revealed that the delayed free recall of subjects required to repeat or not repeat words did not differ significantly (all p's>0.10); thus this variable was not considered in subsequent analyses. A 2 (groups) × 3 (sets of lists) × 2 (primary/recency words) analysis of variance on the data from the baseline, rising BAC, and peak BAC lists demonstrated the retrograde enhancement effect in a significant drug × sets of lists × primary/recency words interaction, $F(2,36)=4.06$, $p<0.05$, presented in Fig. 1. Simple main effects tests [18] on the interaction revealed that Alcohol subjects recalled significantly more words from the primacy portion of the baseline lists, and significantly fewer words than the primacy portion of the peak BAC lists, than placebo subjects ($p<0.05$ for both comparisons).

DISCUSSION

The results of these two experiments are consistent with previous studies showing retrograde enhancement of human memory by alcohol [13,14] and extend the observations to the verbal free recall task. The effect is therefore not restricted to the pictorial recognition and incidental verbal learning tasks on which it has previously been observed. Since the retrograde enhancement effects in the present experiments were obtained while subjects still had substantial BAC's, the phenomenon apparently does not depend on processes occurring after alcohol has been cleared from the body, and instead seems to originate in the period shortly following alcohol consumption [14]. However, interpretation of these data must be tempered by the possibility that testing of subjects after BAC's had reached zero may have produced a different pattern of results.

An interesting observation was the specificity of the effect to the primacy words from the lists. As well, the alcohol-induced deficit on peak BAC lists seemed most pronounced for words from the primacy portions of the lists. An alcohol-induced deficit on primacy, but not recency, words in free recall has previously been reported [10]. At present there are several proposed models of human memory (e.g., [3, 6, 7]), but most seem to agree that recall of primacy words represents material from a more advanced memory stage than recall of recency words (e.g., [5,8]). The data from the present experiments then, suggest that both impairment and retrograde enhancement by alcohol may occur at a similar stage in the memory process.

The retrograde enhancement effect may be explained by the arousing effects of a low dose of alcohol, a decrease in retroactive interference due to the change of state, or a facilitation of physiological memory consolidation processes [1, 13, 14]. Recent animal studies have found similar retrograde enhancement effects with opiates (e.g., [4,11]) which have been attributed to the drugs' actions on the brain reward systems. Such an explanation might be applicable to alcohol as well. There is much data suggesting that alcohol can act as a reinforcer [9]; thus, retrograde enhancement of human memory by alcohol might also be due to its specific actions on brain reward systems [2,19]. Future studies of the phenomenon may profit from collection of other measures of alcohol's reinforcing effects (e.g., subjective estimates) for comparison purposes.

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