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Reinforcement Reduces Behavioural Impairment Under an Acute Dose of Alcohol

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HAUBENREISSER, T. AND M. VOGEL-SPROTT. *Reinforcement reduces behavioural impairment under an acute dose of alcohol.* PHARMACOL BIOCHEM BEHAV **26**(1) 29–33, 1987.—Two experiments employed a total of 25 male social drinkers who learned a complex psychomotor task (Tracometer) and subsequently performed it 20 times under alcohol (0.60 g absolute alcohol/kg) while blood alcohol concentrations (BAC) rose and fell. In each experiment, one group received reinforcement for drug-compensatory performance (RP) and one received no reinforcement (P). The BACs associated with the onset and offset of behavioural impairment under the dose were measured, and these thresholds were significantly higher in RP than P groups; reinforcement delayed the onset and also hastened the offset of drug effects. The accelerated recovery from impairment was considered to imply that reinforcement may facilitate the adaptive process involved in acute tolerance. Since this same reinforcement treatment accelerates the development of tolerance to repeated doses of alcohol, the results of the present research suggest that the behavioural effect of acute and chronic doses may both be similarly influenced by environmental learning factors.

Alcohol Acute tolerance Learning Humans

ACUTE behavioural effects of a single dose of alcohol have a characteristic pattern. During absorption, the blood alcohol concentration (BAC) increases and drug effects are observed to intensify. When absorption is complete, elimination processes reduce the BAC and the drug effect abates. However, this elimination process cannot totally account for the diminishing drug effects because they typically diminish more rapidly than the decline in BAC. This phenomenon was first reported by Mellanby [11] who administered an acute dose of alcohol and measured the BAC at the first observable onset and offset of impairment in the gait of dogs. He concluded that the BAC offset threshold was typically higher than the onset threshold.

The consistent occurrence of a higher BAC for the offset of alcohol-induced impairment was subsequently demonstrated in the performance of a variety of tasks by humans whose drinking habits ranged from light to heavy [6]. This research also indicated that the actual BAC thresholds for onset and offset of drug effects varied with the type of task and with drinking habits. Heavier drinkers were generally less susceptible to alcohol, and attained higher BAC thresholds for the onset and offset of impairment in task performance. Since detection of BAC thresholds for the onset and offset of the effect of an acute doses of alcohol necessitates repeated monitoring and testing of performance, Goldberg [6] and others [4] suggested that this procedure might provide a learning opportunity which could improve performance and explain the accelerated recovery from impairment during declining BAC. This has prompted the use of other experimental designs which exclude learning factors by administering a single test at selected matching BACs on each limb of the alcohol curve. This procedure has successfully demonstrated the phenomenon of acute alcohol tolerance in a variety of tasks in animals and humans [8,15]. While such research indicates that acute alcohol tolerance is not simply a learning artifact, it does not test the possibility that learning may actually have some important role in determining the effect of an acute dose of alcohol.

Although this learning hypothesis has attracted scant attention, its investigation could be of theoretical interest. Acute tolerance to a single dose, and chronic tolerance to repeated doses, are both characterized by a diminished responsiveness to alcohol, and many investigators have suggested that those two phenomena may be influenced by some similar factors [2, 5, 6, 10, 14, 15]. Considerable research has already demonstrated that learning affects chronic tolerance. When a dose of alcohol is repeatedly administered, subjects who practice a task under drug develop chronic tolerance faster than those who receive equivalent, but separate practice and drug exposures [17]. An additional important tolerance-facilitating factor in task practice under drug appears to be the presence or absence of reinforcement for drug-compensatory performance. The importance of reinforcement was originally suggested by Schuster *et al.*

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[13] and its effect on drug-compensatory performance under a moderate dose of alcohol has been explored in a series of studies with social drinkers [1, 9, 12]. Comparison between practice groups with or without reinforcement consistently revealed more tolerance in the reinforced groups.

In general, there is a suspicion that some factor may similarly influence the degree of behavioural impairment evoked by acute and chronic doses of alcohol. In addition, there is evidence that reinforcement of drug-compensatory performance accelerates the development of chronic tolerance. Thus a similar reinforcement treatment may also diminish the effect of an acute dose, by delaying the onset of impairment and hastening recovery. This paper reports two experiments testing the effect of reinforcement on the onset and offset of behavioural impairment during an acute dose of alcohol.

EXPERIMENT 1

METHOD

Subjects

Eleven male university students, who replied to posters requesting volunteers for an alcohol experiment, served as subjects. Informed consent was obtained from subjects after an initial meeting which explained the requirements of the experiment (i.e., a four hour fast prior to an alcohol session and the use of no prescription or nonprescription drugs for 24 hours prior to a session). Subjects also completed a Personal Drinking Habits Questionnaire [16] which provided information on their age, weight, typical weekly frequency and duration of social drinking occasions and customary dose (g absolute alcohol/kg) administered on such occasions.

Apparatus

The Tracometer, a complex subject-paced psychomotor task developed by the National Research Council of Canada, was used to measure performance [3]. Reinforcement for drug-compensatory performance on this task has previously been found to accelerate the development of chronic alcohol tolerance [1]. The task consists of a steering wheel that controls a pointer which moves across a vertical board on the tracking unit. The board contains five dots, and when illuminated, a dot serves as a target. Subjects were required to align the pointer on the target. When the pointer was aligned, a new target was illuminated in another position. One trial presented 100 targets which occurred in a programmed order so that targets in all five positions occurred equally often. A different programmed sequence was presented on each trial. Performance was automatically measured by the time (sec) required to complete the 100 targets which comprised a trial.

BAC was measured from breath samples using an Intoxilyzer (Omicron Systems Inc.).

Procedure

Drug-free training. To ensure that the task was well learned prior to the administration of alcohol, all subjects attended two practice sessions which administered a total of 40 practice trials on the task. During the first session subjects had 24 practice trials, and their mean times to complete a trial at the conclusion of this session were used to assign subjects to one of two groups so that their performance efficiency was matched. These two groups were treated alike, except that one henceforth received monetary reinforcement for performance (RP) (n=6) and the other performed without reinforcement (P) (n=5).

Following the allocation of subjects to groups, a second drug-free session administered 16 trials to all subjects. These trials introduced the RP group to reinforcement, and the scores of the RP and P groups on these trials served to assess the consistency of performance under these conditions.

The reinforcement treatment was identical to that employed previously to hasten chronic tolerance [1]. Reinforcement consisted of showing the subject a graph containing a single horizontal line which represented the mean of his best eight scores obtained on the initial practice session. He was told that he would receive 25 cents for any subsequent score on the Tracometer task which was equivalent to, or better than, his mean score. A check mark on the graph line after each trial recorded the occurrence of this reinforcement, and the money earned was paid at the end of the study. In addition to the graph, an auditory beep sounded whenever the subject aligned the pointer with the target, and he was informed, five times during every trial, about the adequacy of his speed of response with respect to the reinforcement criterion. This information was presented by the experimenter after each set of twenty targets (e.g., 20, 40, 60, 80) by simply saying "Yes" or "No." No reinforcement was administered to subjects in group P. They were merely asked to work quickly and carefully, and try as hard as they could. They had no information about task performance, no auditory beep, and no opportunity to earn 25 cents.

Analysis of the scores of the last five trials of this final drug-free session obtained no significant group × trial interaction or main effects; drug-free performance was stable over trials and comparable in RP and P groups. The overall mean time score on these five trials was 141 sec (SE=3.5), but a subject's scores fluctuated somewhat from trial to trial and some better (faster) times were displayed prior to these final five trials. In order to provide a comparable estimate of each subject's "normal" drug-free variation in his best performance, the standard deviation (SD) of a subject's best eight scores was calculated. Subjects' SD values ranged from 1.6 to 5.0 sec, with an average SD value of 2.8 in the RP group and 3.9 in the P group.

Alcohol session. Subjects fasted for four hours before returning in the late afternoon of another day for this session. Each subject performed two drug-free trials upon arrival at the laboratory, which yielded a mean trial score of 135.6 sec (SE=3.8) for the entire sample. To create a standard criterion of "normal" drug-free performance which controls for individual differences in achievement, this criterion for each subject was determined by the mean of his two trials on this session plus the SD of his scores from the preceding drug-free session. Any score within 1 SD of this mean was considered equivalent to his drug-free achievement. RP subjects received reinforcement for any trial score which was within this range, or better.

Subjects subsequently received 0.60 g absolute alcohol/kg body weight in the form of three drinks in a 1:2 ratio of alcohol and mix. Each drink was consumed within one minute, at 20 min intervals.

Fifteen minutes after drinking commenced, the subject performed the first Tracometer trial, and completed a total of six tests by the 60 minute period of the session (a rate of one trial every 7.5 minutes). Thereafter, and until the session concluded at 240 minutes, all subjects performed 14 more tests, which occurred at average rate of one test per 13 minutes.

TABLE 1

MEAN (SD) BAC AT ONSET AND OFFSET OF PERFORMANCE IMPAIRMENT IN RP AND P GROUPS

Groups	BAC (mg/100 ml) Onset Mean (SD)	Offset Mean (SD)
A. Experiment 1		
RP (n=6)	53.2 (22)	64.7 (14)
P (n=5)	18.6 (12)	49.0 (12)
Overall (n=11)	37.4 (17)	57.5 (13)
B. Experiment 2		
RP (n=7)	53.8 (24)	61.1 (15)
P (n=7)	29.5 (8)	47.9 (9)
Overall (n=14)	41.7 (16)	54.5 (12)

Measures. BAC was measured at regular intervals during the session and whenever the onset and offset of alcohol effects were observed. "Onset" and "offset" were defined with respect to a subject's own "normal" drug-free achievement (i.e., within 1 SD of his mean drug-free score immediately prior to alcohol). During rising BACs, the first test score that was impaired (i.e., slower) and outside a subject's normal range of achievement was considered to indicate the onset of drug effects. The first test score which fell within this range during declining BACs identified the offset of impairment. These onset and offset criteria thus identify a change in performance which is comparable for all subjects, and independent of individual differences in level of achievement on the task.

RESULTS

No significant differences between groups were obtained on age or any questionnaire measures of drinking habits. The entire sample (n=11) had a mean age of 22.0 years and reported an average of 1.9 drinking occasions per week with a mean duration of 3.82 hours. Their mean dose per occasion was 0.90 g absolute alcohol/kg. Assuming a body weight of 70 kg, this dose would represent 4.8 twelve oz (341 ml) bottles of 5% beer.

The alcohol treatment administered to RP and R groups was compared by analyzing nine BAC measures which were obtained at the same times for all subjects. This analysis obtained no significant group × time interaction or main effect of group ($p>0.40$). The change in BAC during the session was reflected in a significant main effect of time, $F(8,72)=58.99$, $p<0.0001$. A mean peak BAC of 68.2 mg/100 ml (SE=2.23) was observed 60 minutes after drinking commenced.

The average BACs when the onset and offset of impairment were observed in each group are presented in Table 1A, and an analysis of variance of these BACs yields significant main effects of groups, $F(1,9)=11.87$, $p<0.007$, and onset-offset, $F(1,9)=11.06$, $p<0.008$, with no significant interaction, $F(1,9)=2.25$, $p<0.20$. The main effect of onset-offset indicates the development of acute tolerance in both groups; the offset of impairment occurs at higher BACs than the onset. The main effect of groups demonstrates that the RP group has consistently higher BAC thresholds for the onset and offset of alcohol effects.

The heightened BAC offset threshold in the RP group indicates that reinforcement hastens recovery from the impairing effect of a dose of alcohol. Since the RP group also displays a higher BAC threshold for the onset of drug effects, reinforcement appears to create some resistance to drug effects by delaying their onset until a higher BAC is reached. However, this interpretation is clouded by the fact that marked individual differences in susceptibility to the effect of a moderate dose of alcohol are common, and subjects were assigned to groups randomly with respect to their behavioural sensitivity to the dose. Therefore, the delayed onset and hastened offset of drug effects in the RP group might be attributed to the chance allocation of less sensitive subjects to this group. Thus, a second experiment was performed to test the reproducibility of the evidence when behavioural sensitivity to alcohol was matched in groups before testing reinforcement treatment effects.

EXPERIMENT 2

This study involved two groups of 7 males each. It was a replica of experiment 1 in all important respects except that a test of initial sensitivity to alcohol was inserted between the first and second drug-free training day. A day or two after the first training session, subjects reported in the late afternoon after a four hour fast and performed two tests on the Tracometer. The average of these test scores provided a measure of the subjects' drug-free level of performance that day. Then each subject drank 0.60 g absolute alcohol/kg. The subject's BAC was monitored continuously and when it reached 60 mg/100 ml on the rising limb of the curve, he performed one test on the Tracometer. The difference between this test score and his mean drug-free trial score prior to drinking provided an estimate of his initial behavioural sensitivity to alcohol. Subjects then were assigned to RP or P groups on the basis of these scores so that there was no significant difference between groups in sensitivity measures, $t(12)=0.41$, $p>0.50$. The sensitivity measures obtained on this session showed that performance was impaired (slowed) by an average of 13.05 sec (SE=3.2).

The second training session followed a few days later, and provided the SD measures of the subjects' variations in their best performance. These ranged from an SD of 6.9 to 2.8 sec, with an average SD value of 4.3 in each group. The subsequent alcohol session was conducted in a fashion identical to experiment 1, and subjects' mean drug-free trial score just prior to alcohol was 137.6 sec (SE=2.5).

RESULTS

The subjects in this second experiment had a mean age of 20.3 years. They reported a weekly average of 1.3 drinking occasions with a mean duration of 3.85 hours. Their average dose per occasion was 0.99 g absolute alcohol/kg. The two groups did not differ significantly on any of these measures.

Analysis of the BAC measures obtained at regular intervals during the alcohol session obtained no significant time × group interaction or main effect of groups. A significant main effect of time, $F(8,96)=71.5$, $p<0.0001$, reflects the change

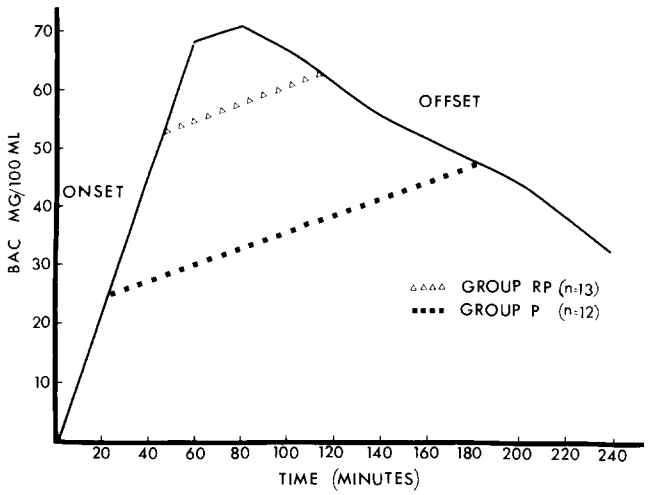

FIG. 1. Mean BAC curve from two experiments showing the mean BAC associated with the onset and offset of impairment in RP and P groups.

in BAC during the session. The peak BAC obtained was 74.9 mg/100 (SE=3.2).

The average BAC associated with the onset and offset of impairment in each group is shown in Table 1B, and the analysis of these BACs yields no significant interaction, $F(1,12)=2.00$, $p<0.20$, but significant main effects of groups, $F(1,12)=6.62$, $p<0.02$, and onset-offset, $F(1,12)=10.78$, $p<0.006$. The main effect of onset-offset again shows that the BAC at the offset of impairment is higher than that at the onset. The group effect is also consistent with experiment 1; the onset and offset of impairment occur at higher BACs in the RP group than in the P group. When the data of both studies are analyzed incorporating experiments as an additional factor, no significant interactions with experiments are obtained with group, $F(1,21)=0.36$, $p>0.60$, or with onset-offset, $F(1,21)=1.33$, $p>0.30$. There is no three way interaction, $F(1,21)=0.31$, $p>0.60$, and no main effect of experiment, $F(1,21)=0.11$, $p>0.70$. The experiments thus agree on their identification of comparable BACs for the onset and offset of impairment in each group. This combined evidence, illustrated in Fig. 1, shows the average BACs during the session in the two experiments, and identifies the mean BAC associated with the onset and offset of impairment in each group. The higher BAC onset and offset thresholds of the RP group occurred approximately 50 minutes and 120 minutes respectively, after drinking commenced, so the total duration of their impairment was 70 minutes. In contrast, these BAC thresholds in the P group were observed 20 minutes and 180 minutes after drinking, and their impairment had a duration of 160 minutes.

GENERAL DISCUSSION

These experiments administered an acute dose of alcohol to test the effect of reinforcement for drug-compensatory performance on BAC thresholds for the onset and offset of drug effects on behaviour. This particular treatment was chosen for investigation because it has been found to enhance the development of behavioural tolerance to repeated doses of alcohol, and because of the speculation that some similar factors may influence the responsiveness to acute and chronic doses of alcohol. The present research demonstrated that the application of this reinforcement treatment generated a reproducible and sizeable raise in the BAC required to induce and to recover from alcohol effects. By raising the BAC thresholds for impairment, reinforcement diminished the duration of the behavioural response to alcohol. These effects of reinforcement cannot be attributed to individual differences in behavioural sensitivity to alcohol, for this research controlled for these possible effects. The results also are not an artifact of different levels of achievement in the task because the same standardized criterion of onset and offset of impairment was applied to each subject and was based upon his own typical drug-free performance of the task.

Acute tolerance, a diminished responsiveness to drug during the course of a single dose, is indicated by a higher BAC threshold for recovery than for the onset of the drug effects. In the present research, groups with or without reinforcement all had higher BAC thresholds for the offset than the onset of drug effects. This observation implies that some process of adaptation to drug contributing to acute tolerance can occur independently of environmental conditions. Such a conclusion would be consistent with other research [8] demonstrating acute tolerance when environmental learning factors are excluded. However the observations in this study do not necessarily exclude the possibility that this adaptive process may also be accelerated by environmental reinforcement. Since the BAC offset thresholds were consistently higher in RP than in P groups, the evidence suggests that reinforcement may accelerate the recovery process involved in acute tolerance.

From this perspective, the findings parallel the evidence showing that reinforcement enhances the development of tolerance to repeated doses of alcohol [1, 12, 13, 17]. Some of this research [1] has also proceeded to separate the components of the reinforcement treatment (i.e., monetary incentive, information) and evaluate their individual effects on the development of chronic tolerance. To date, this evidence indicates that the conditions known to hasten the learning of a new response also accelerate the display of tolerance to repeated doses; information about performance facilitates tolerance but greater facilitation is obtained when incentive is added. In contrast, the provision of incentive and information unrelated to drug-compensatory performance has little detectable effect. Tests of the effect of these components of reinforcement during an acute dose of alcohol remain to be performed. By demonstrating that an environmental treatment can reliably influence the behavioural effect of a dose of alcohol, the present research provides an impetus for this line of investigation.

In summary, the present research indicates that the learning variable of reinforcement influences the behavioural impairment induced by a single dose of alcohol. Additional studies are needed to clarify the extent to which learning may determine the impairment evoked by a dose of alcohol, and influence the adaptive process of acute tolerance. Such research may also be of practical importance. Social drinkers perform many tasks, including driving, after drinking. It may be that reinforcement for drug-compensatory performance is an important factor determining the onset, duration and degree of behavioural impairment displayed by social drinkers under a moderate dose of alcohol.

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