Pharmacology Biochemistry and Behavior, Vol. 43, pp. 589–595, 1992
Printed in the U.S.A.

0091-3057/92 $5.00 + .00

Effects of Oral Ethanol Self-Administration on the Inhibition of the Lever-Press Response in Rats

MARC A. PALLARÉS,[1] ROSER A. NADAL AND NÚRIA S. FERRÉ

Area de Psicobiologia, Departament de Psicologia de la Salut, Facultat de Psicologia, Universitat Autònoma de Barcelona, Barcelona, Spain

Received 28 March 1991

PALLARÉS, M. A., R. A. NADAL AND N. S. FERRÉ. *Effects of oral ethanol self-administration on the inhibition of the lever-press response in rats.* PHARMACOL BIOCHEM BEHAV 43(2) 589–595, 1992.—The effects of ethanol on the inhibition of a learned response were examined in adult, male Wistar rats from two treatment groups: oral self-administration of alcoholic solution (10% ethanol and 10% glucose in distilled water) and oral self-administration of sweet solution (10% glucose in distilled water). Subjects were food deprived and alcoholic or control solutions were available 1 h per day during 15 days. After this period, rats were tested in a two-bottle paradigm during 1 h per day and placed in the operant chambers immediately afterward. This phase went on for 19 days. Subjects were trained to lever press for food and were tested in a continuous reinforcement schedule, operant extinction, successive discrimination, and two-stimuli tests. Alcohol impaired the ability to inhibit previously reinforced responses but only in situations indicated by exteroceptive stimuli. Ethanol intake did not impair the lever-press behavior neither in the acquisition of the response nor in the continuous reinforcement schedule. These data suggest that the sedative effects of alcohol at this dose were not apparent in reinforcement situations, in contrast with extinction situations.

Wistar rats Alcohol Self-administration Two-bottle paradigm Shaping
Continuous reinforcement schedule Operant extinction Successive discrimination
Two-stimuli test Inhibitory stimulus control

SEVERAL studies of ethanol's effects on learned behavior in maze tests have reported that when alcohol is administered rats show an impaired ability to change their response when the contingencies paired with the stimuli change (2,4,24). This impairment in the flexibility of the response has also been reported in rabbits (11,12) in a pavlovian conditioning situation (eye-blink conditioning). Also, this effect of alcohol has been shown in rats both in runway (37) and in operant chambers (38).

Some authors (2) have proposed that this phenomenon could be considered as a "proactive interference" and it would be due to a memory problem. To support this, said authors reported that rats that had been administered ethanol and showed a learning impairment also showed a decrease in the number of neurons in hippocampus and mammillary bodies (2). Later, the same authors reproduced the same neuronal injuries with kainic acid and reported that their rats showed the same learning problems (1).

Our experimental hypothesis proposes that ethanol produces a specific impairment of the subjects' capability to inhibit their previously reinforced responses, and the previous results could be reflecting this effect. We performed the present experiment to show the negative effect of ethanol on the inhibition of lever-press behavior.

This present investigation involves a between-groups study in Wistar rats, and the inhibitory learning was tested by means of operant extinction, successive discrimination, and two-stimuli tests it the Skinner box.

Operant extinction can be considered as an inhibition process of a previously learned response, where the only exteroceptive stimulus is the absence of reinforcement (10,22,23). However, operant discrimination usually involves a positive stimulus indicating the reinforced situation (23,34) and a negative stimulus indicating the extinction situation (5,14,34), which can be presented successively. The two-stimuli test, proposed by Pavlov (28) as a test for inhibitory stimulus control, was adapted to the operant conditioning (31) and may be considered a good measure of the inhibitory control of a stimulus paired with the extinction (10).

We achieved oral intake of a sweet alcoholic solution by

[1] Requests for reprints should be addressed to Marc Pallarés, M. D. Area de Psicobiologia, Departament de Psicologia de la Salut, Facultat de Psicologia, Universitat Autònoma de Barcelona, Ap. no 46, 08193 Cerdanyola, Barcelona, Catalonia, Spain.

means of food deprivation and access to the solution limited to once per day. Sweet solutions (19), food deprivation (19,20,26), and limited access (18,19,25) have been used successfully by several investigators.

METHOD

Subjects

Fifty-five male Wistar rats were individually housed in a temperature-controlled environment on a 12 L : 12 D cycle with lights on at 8:00 a.m. Subjects has continuous access to food and water in their home cage, except where otherwise noted, and weighed 270–452 g at the beginning of the experiment. Thirty rats were assigned at random to the experimental group and 25 to the control group.

Apparatus

Behavioral testing was conducted in two operant chambers (Ralph Gerbrands Co., Arlington, MA) encased in sound-attenuating cubicles. The chambers were fitted with a center lever placed 7.5 cm from the floor. The food cup was connected to a pellet dispenser that delivered 45-mg food pellets (Noyes). A 12-V light was situated 3 cm above the lever and 4 cm to the right. A 6-V buzzer (60 dB) was situated 3 cm above the lever and 4 cm to the left.

Procedure

All rats were food deprived until they reached less than 80% of their free body weight (minimum 75%, maximum 80%). This phase went on for 10 days.

Following this period, experimental subjects received a bottle containing a sweetened alcoholic solution (10% ethanol + 10% glucose in distilled water) and control subjects received a sweet solution (10% glucose in distilled water) in their home cage during 1 h per day. When this bottle was removed, subjects received a single water bottle and the daily maintenance food. This phase went on for 15 days.

After this period, a two-bottle phase was conducted for 19 days. In this phase, subjects received the bottle containing alcoholic or control solution (depending upon the group) and the water bottle during 1 h per day (with the same concentrations as in the induction phase). The two bottles were situated at random each day to avoid place effects. Immediately after this hour, subjects were placed in the operant chambers.

The learning program went on for 19 days and was made up of the bar-pressing shaping (1 session), continuous reinforcement schedule (4 sessions), operant extinction (2 sessions), successive discrimination (10 sessions), and two-stimuli test (2 sessions).

The bar-pressing shaping was performed using an automatic procedure (6,7). If the subject did not emit 10 responses (acquisition criterion) in the first session, it underwent a second one (this session replaced the first one of continuous reinforcement schedule).

The continuous reinforcement sessions were stopped after 30 min or when subjects had obtained 120 pellets. The lever-press shaping and the continuous reinforcement schedule were performed with the light on.

The 30-min operant extinction sessions were conducted with the light off.

The operant discrimination alternated successively reinforcement periods (discriminative stimuli = light on + buzzer off) with extinction periods (negative discriminative stimuli = light off + buzzer on). The sessions were always started and finished with the positive stimulus. The duration of the 10 sessions was: 48, 24, 33, 37, 27, 28, 36, 24, 22, and 30 min. The positive discriminative stimulus duration range was between 48 s and 5 min 16 s, and the negative discriminative stimulus between 2 min 26 s and 7 min 6 s. The ratio between the two situations ranged from 60%(S+) −40%(S−) to 43%(S+) −57%(S−).

The two-stimuli sessions went on for 30 min. In this test, the two discriminative stimuli (positive and negative) were present (light and buzzer on) and there was no reinforcement (extinction situation).

For data analyses, we used the SPSS^X package. In the first place, for the mixed analyses of variance (MANOVAs) (groups × dependent variable ranks) we used the MANOVA program. In all cases (continuous reinforcement, extinction, successive discrimination, and two-stimuli tests), there was not homogeneity of variances between groups. Thus, we used a MANOVA for the evolution of the dependent variable across the sessions within each group (MANOVA/POLYNOMIAL). For the comparison of the dependent variable each day between groups, we used Student's *t*-test or the nonparametric Kruskal–Wallis test (χ^2) instead of the *t*-test when there was not homogeneity of variances or when some variable did not show a normal distribution.

RESULTS

Solution Intake

In the experimental group, all subjects ingested high amounts of ethanol, which produced ataxic effects (average dose of the 15 sessions: mean = 2.9 g/kg, SE = 0.18). Ethanol intake increased significantly through the 15 days of this phase. [MANOVA/POLYNOMIAL/contrast 2, $F(14, 0.05) = 63.7$, $p < 0.001$]. See Table 1 for the means of the 15 sessions.

In the two-bottle period, experimental subjects ingested ataxic amounts of ethanol as in the last days of the induction phase (average dose of the 19 sessions: mean = 3.6 g/kg, SE

TABLE 1

MEAN ETHANOL DOSE (g/Kg) IN THE INDUCTION PHASE

Sessions	Mean	SD	SE
1	1.38	1.02	0.19
2	2.05	1.27	0.23
3	2.33	1.23	0.22
4	2.68	1.22	0.22
5	2.94	1.36	0.25
6	2.79	1.18	0 22
7	3.02	1.52	0.28
8	3.16	1.33	0.24
9	2.82	1.45	0.26
10	3.18	1.19	0.22
11	3.10	1.38	0.25
12	3.48	1.24	0.23
13	3.40	1.58	0.29
14	3.48	1.33	0.24
15	3.63	1.33	0.24

Values are reported from the experimental group (n = 30).

TABLE 2

CALORIC VALUE OBTAINED FROM THE SOLUTION (Kcal.)

	Experimental Group			Control Group		
Sessions	Mean	SD	SE	Mean	SD	SE
1	11.72	4.52	0.83	11.04	4.02	0.80
2	11.44	4.49	0.82	11.04	3.82	0.76
3	11.78	4.92	0.90	11.66	3.55	0.71
4	10.90	4.01	0.73	11.92	3.10	0.62
5	11.41	4.33	0.79	11.98	3.78	0.76
6	11.41	4.89	0.89	11.87	3.14	0.64
7	11.84	4.00	0.73	10.91	4.24	0.85
8	12.94	4.80	0.88	11.18	2.61	0.52
9	10.25	4.04	0.74	11.29	3.12	0.62
10	11.72	4.50	0.82	11.66	3.92	0.79
11	12.38	4.51	0.82	11.73	4.03	0.84
12	12.69	4.72	0.86	11.07	3.63	0.73
13	13.54	4.22	0.77	11.75	3.08	0.62
14*	13.35*	4.04	0.74	11.36*	2.82	0.57
15	12.60	4.26	0.78	11.30	2.67	0.53
16	13.22	4.64	0.85	11.28	3.19	0.64
17*	13.41*	4.27	0.78	10.91*	2.71	0.54
18	13.19	4.64	0.85	11.52	2.81	0.56
19	11.53	3.78	0.69	10.74	3.03	0.61

Experimental group (n = 30). Control group (n = 25). *$p < 0.05$ (Statistically significant).

= 0.18). Ethanol intake remained steady throughout the 19 sessions of this period. The preference index for ethanol solution over water was high in all days of this phase (over 90%).

Subjects of the control group ingested high amounts of glucose solution, especially in the two-bottle phase (average volume of the 19 sessions: mean = 29.26 ml, SE = 1.26), and did so in greater amounts, $\chi^2(1, 0.05) = 39.75$, $p < 0.0001$, than the average alcoholic solution intake in experimental rats (mean = 13.02 ml, SE = 0.67). We calculated the caloric value of the solutions ingested by subjects of the two groups, considering that ethanol has 7 Kcal/g and glucose 3.87 Kcal/g. The average Kcal of the 19 sessions in the experimental group were mean = 12.17, SE = 0.63, and in the control group mean = 11.38, SE = 0.49. On average, there were no significant differences between groups. On the other hand, the caloric value was greater in experimental rats in the 14th session, $t(53, 0.05) = 2.14$, $p < 0.05$, and 17th, $\chi^2(1, 0.05) = 5.98$, $p < 0.05$, but there were no significant differences in the remaining ones. See Table 2 for the means of the 19 sessions.

Shaping and Continuous Reinforcement Schedule

Data analyses revealed that animals of the experimental group performed the 1st (mean = 3.2, SE = 0.99) and 10th (mean = 10.58, SE = 1.14) responses with shorter latency in seconds [1st, $\chi^2(1, 0.05) = 5.4$, $p < 0.05$, 10th, $\chi^2(1, 0.05) = 14.6$, $p < 0.001$] than control rats (1st, mean = 6.08, SE = 1.32, 10th, mean = 20.89, SE = 2.3), and the mean rate of response was greater, $t(53, 0.05) = 2.78$, $p < 0.01$, in experimental rats (mean = 31.33, SE = 2.66) than in control rats (mean = 21.64, SE = 2.25).

Continuous reinforcement data analyses revealed that experimental rats presented shorter latencies than control rats in performing the 1st response in the 1st session [experimental group (EG), mean = 0.26, SE = 0.06, control group (CG), mean = 0.85, SE = 0.21, $\chi^2(1, 0.05) = 17.16$, $p < 0.001$] and 2nd session [EG, mean = 0.04, SE = 0.006, CG, mean = 0.17, SE = 0.05, $\chi^2(1, 0.05) = 11.78$, $p < 0.001$] and the 10th response in the 2nd session [EG, mean = 0.53, SE = 0.04, CG, mean = 0.85, SE = 0.14, $\chi^2(1, 0.05) = 5.38$, $p < 0.05$] and 4th session [EG, mean = 0.48, SE = 0.06, CG, mean = 0.6, SE = 0.07, $\chi^2(1, 0.05) = 4.84$, $p < 0.05$]. Also, there were no significant differences between groups in the mean rate of response.

Operant Extinction

Data analyses showed that experimental rats presented shorter latencies than control rats in performing the 1st response in the 1st session (EG, mean = 0.023, SE = 0.005, CG, mean = 0.082, SE = 0.027, $\chi^2(1, 0.05) = 4.23$, $p < 0.05$] and the 10th response in the 2nd session [EG, mean = 1.22, SE = 0.14, CG, mean = 1.88, SE = 0.25, $\chi^2(1, 0.05) = 4.32$, $p < 0.05$]. On the other hand, control subjects performed significantly more responses than experimental subjects in the first, $t(53, 0.05) = 2.6$, $p < 0.05$, and second, $\chi^2(1, 0.05) = 6.31$, $p < 0.05$, sessions. These results are shown in Fig. 1.

Successive Discrimination

The discrimination index (DI) was obtained from the following formula:

DI = (total responses in presence of the positive stimulus/ total responses) × 100.

The average DI (10 days) was greater in control than experimental rats, $t(53, 0.05) = 2.42$, $p < 0.05$. The DIs differed

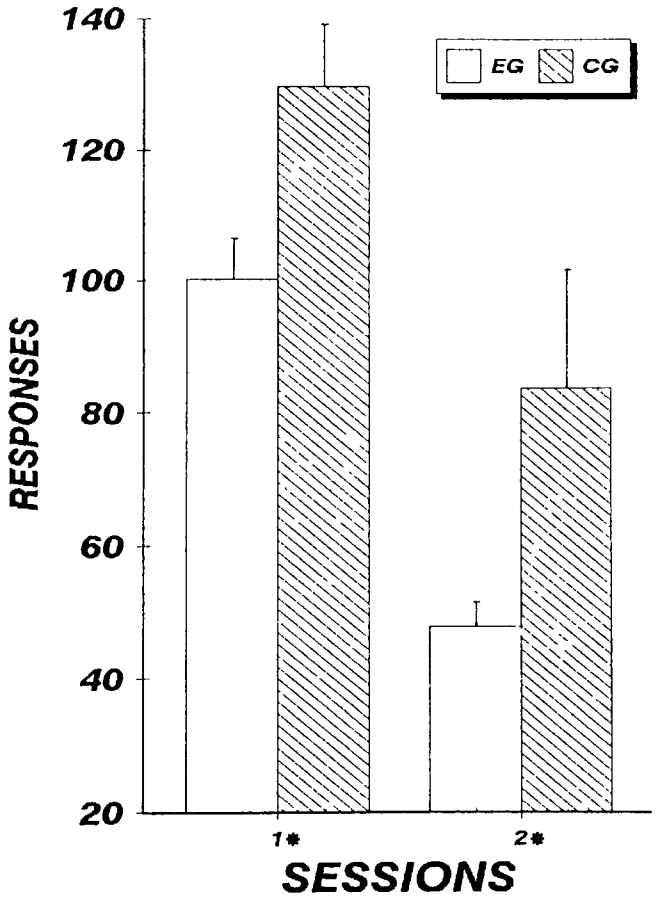

FIG. 1. Number of responses in the two sessions of the extinction phase. EG, experimental group; CG, control group. *$p < 0.05$.

partly because control subjects responded at a greater rate than experimental subjects during positive periods, in addition to their responding less than experimental rats during negative periods. Control subjects showed a significantly greater discrimination index than experimental subjects in the first, $t(53, 0.05) = 2.85$, $p < 0.01$, 8th, $\chi^2(1, 0.05) = 6.73$, $p < 0.01$, and 9th, $t(53, 0.05) = 2.48$, $p < 0.05$, sessions. In the remaining sessions, the discrimination index was always greater in control rats although it did not reach statistical significance. These results are shown in Fig. 2.

The number of responses in the presence of the negative discriminative stimulus did not show significant differences between the two groups, but experimental rats performed a significantly higher rate of response in the first, $t(53, 0.05) = 2.65$, $p < 0.05$, and eighth, $\chi^2(1, 0.05) = 3.95$, $p < 0.05$, sessions. In the remaining sessions, the number of responses was always greater in experimental rats although these differences were not significant. These results are shown in Fig. 3.

The average number of responses in the presence of the positive discriminative stimulus was greater in control than experimental rats, $t(53, 0.05) = 2.69$, $p < 0.01$. These differences were significant in the following sessions: $p < 0.05$ in the 2nd, $t(53, 0.05) = 2.4$, 4th, $t(53, 0.05) = 2.63$, and 8th, $t(53, 0.05) = 2.58$; $p < 0.01$ in the 5th, $t(53, 0.05) = 3.19$, 7th, $t(53, 0.05) = 3.26$, and 10th, $t(53, 0.05) = 2.95$; and $p < 0.001$ in the 6th, $t(53, 0.05) = 15.12$, and 9th, $t(53, 0.05) = 3.53$. These results are shown in Fig. 4.

Two-Stimuli Tests

There were no significant differences between the two groups as to the number of responses and the time needed to perform the 1st response, but experimental subjects presented shorter latency than control rats in performing the 10th response, in the 1st [EG, mean = 0.40, SE = 0.08, CG, mean = 0.65, SE = 0.11, $\chi^2(1, 0.05) = 9.27$, $p < 0.01$] and 2nd [EG, mean = 1.19, SE = 0.28, CG, mean = 2.5, SE = 0.4, $t(53, 0.05) = 2.74$, $p < 0.01$] sessions.

On the other hand, there were no differences between the number of responses in the first and second sessions in experimental subjects, but the number of responses in the first session was significantly greater than in the second session, $t(53, 0.05) = 3.85$, $p = 0.001$, in control rats. These results are shown in Fig. 5.

DISCUSSION

In the present experiment, we obtained an oral ethanol self-administration in our experimental rats, increasing as the days went by. In the induction phase, the highest average dose was 3.5 g/kg/h, and in the two-bottle phase alcohol intake

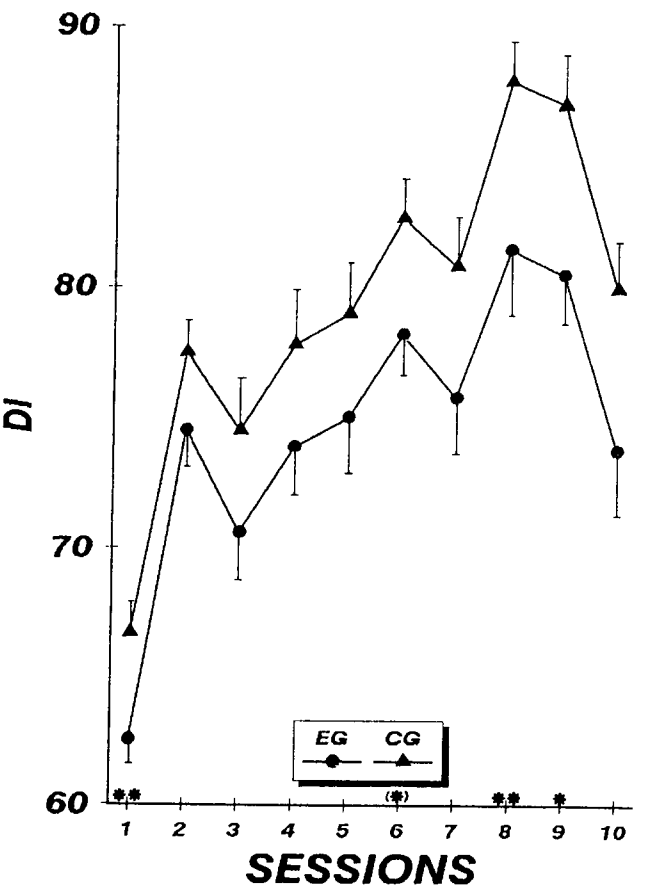

FIG. 2. Discrimination index (total responses in presence of the positive stimulus/total responses) ×100. DI, discrimination index; EG, experimental group; CG, control group. *$p < 0.05$; **$p < 0.01$; (*), a tendency to be significant.

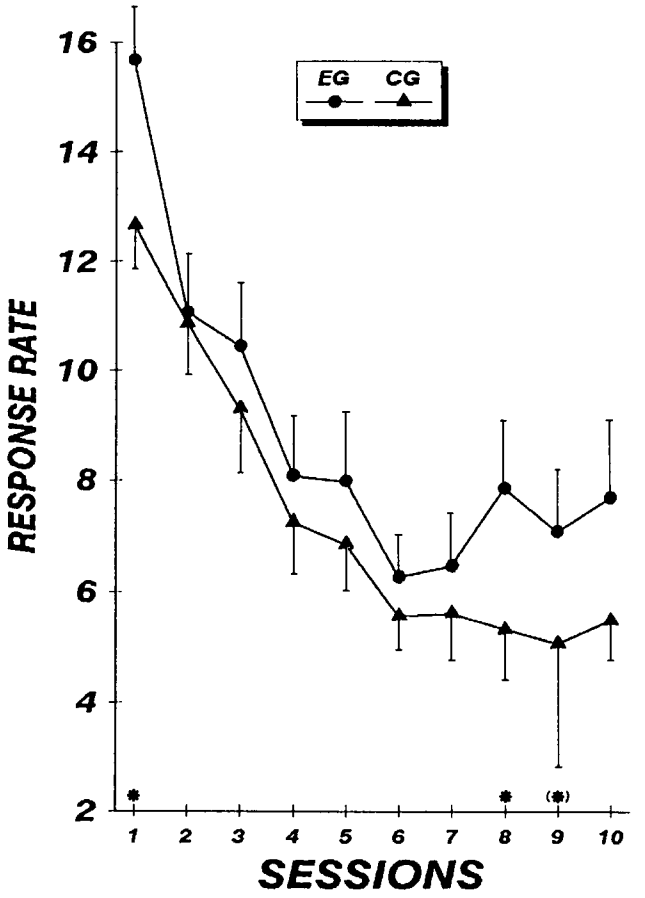

FIG. 3. Response rate in presence of the negative stimulus. EG, experimental group; CG, control group. *$p < 0.05$; (*), a tendency to be significant.

remained steady. Ethanol intake was high because experimental rats showed ataxic effects as activity decrease, muscular relaxation, and loss of fine motor control. However, lever-response acquisition, measured as the time needed to perform the 1st response, the time needed to perform the 10th response, and the rate of response, was not impaired. It seems that the alcoholic dosage ingested by our rats did not have any negative effect on the ability to acquire an operant appetitive response. Surprisingly, experimental subjects performed the bar-pressing acquisition (latencies and response rate) and the continuous reinforcement schedule (latencies) better than control subjects. This fact cannot be due to a motivational difference because the caloric intake obtained from the solutions was similar for both groups (see Table 2). Nevertheless, the phenomenon of "empty calories," well documented in human alcoholics (8,17) could be involved. Therefore, it has been proposed that in the rat the ingestion of alcohol together with an oral glucose load could trigger a reactive hypoglycemia (16). On the other hand, alcohol has anxiolytic effects (30,35,36) and this fact could have shortened the latency in performing the first lever-press response.

In the operant extinction, rats have to inhibit a response they made in a preceding situation of reinforcement. Considering the number of responses, Fig. 1 shows results opposed to the predictions of our hypothesis: Control rats made more responses than experimental rats. This could be due to the well-documented depressing effects of ethanol on behavior (15,21,29,32). Nevertheless, alcohol's depressing effects showed on the response rate did not appear in either the shaping session or continuous reinforcement schedule. The critical factor for the appearance of depressing effects seems to be the absence of the reinforcement because in situations where the reinforcement was available these effects did not appear.

On the other hand, results showed that experimental rats presented shorter latencies than control rats in making the 1st and 10th responses, just as in the shaping session and continuous reinforcement schedule.

Our results about the response rate in the operant extinction tests are opposed to the results of other authors (11,12) working in pavlovian conditioning under stimulus control, yet this is not so in instrumental extinction (21). Pavlovian extinction is under stimuli control, as well as the negative compound of the successive discrimination (23).

In a discrimination schedule, rats perform a response in the presence of a certain stimulus and inhibit their response in the presence of a different stimulus. So, the discriminative training provides excitatory control from the positive stimulus and inhibitory control from the negative stimulus because of the differential reinforcement (34).

Figure 2 shows that the discrimination index was always

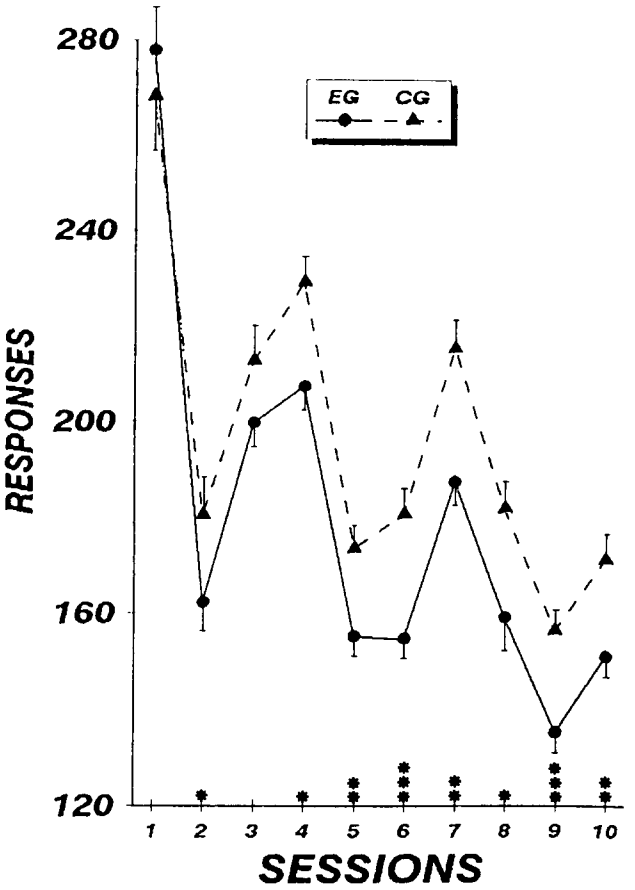

FIG. 4. Number of responses in presence of the positive stimulus. EG, experimental group; CG, control group. *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

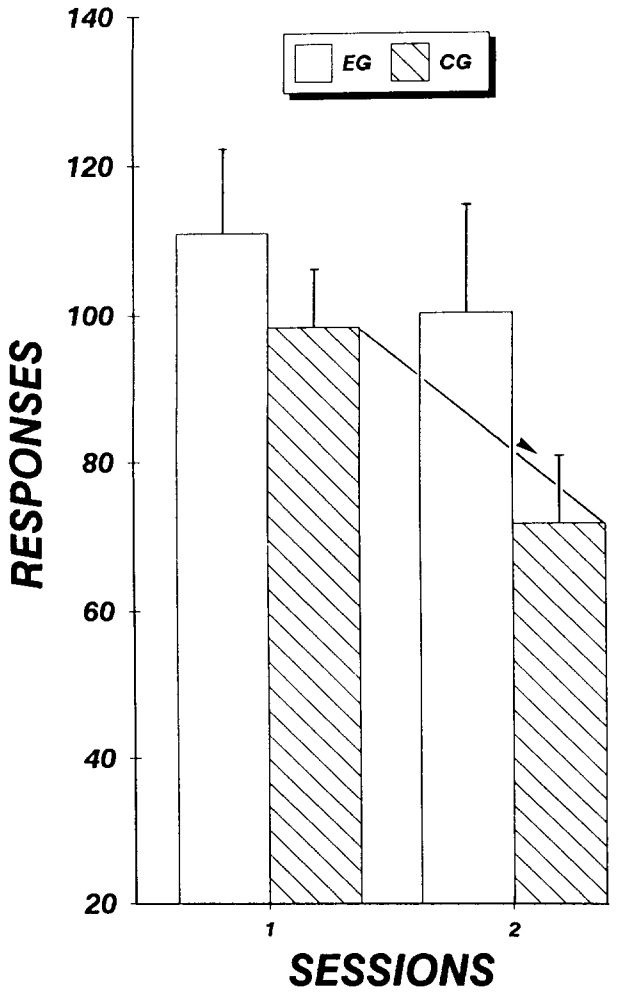

FIG. 5. Evolution of the number of responses in the two sessions of the two-stimuli test. EG, experimental group; CG, control group. →, significant evolution.

greater in control than in experimental subjects although this difference was statistically significant only in the first, eighth, and ninth sessions. This fact suggests that the differences between groups did not alter throughout the training. On the other hand, the results of the first day of discrimination schedule are in agreement with several studies (2,4,24) because the curves between the two groups look perfectly parallel, suggesting that the groups learned at the same rate but just started at different beginning levels. Nevertheless, according to our hypothesis this impairment in the change of contingencies learning could be due to an impairment in the inhibition of the previously reinforced response more than to a memory interference, which is proposed by the above-mentioned authors.

The impairment in the discrimination index is not due to alcohol's depressing effects because experimental rats made more responses than control rats in the presence of the negative discriminative stimulus (see Fig. 3). These results are different from the operant extinction results and could be due to the stimuli control. It seems that the negative effects of ethanol on the response inhibition only appear in tests that involve exteroceptive stimuli.

With regard to the extinction phase, we used the light off to potentiate the response decrease. Nevertheless, we consider this phase a situation without stimuli control because it is generally assumed that this kind of control is acquired by means of a conditioning process (9,34) and requires the presentation of the negative stimulus paired with the contingency of no reinforcement (13), as well as the successive or simultaneous presentation of the positive stimulus paired with the contingency of reinforcement (33). In our experiment, there was no differential reinforcement in the extinction phase, in contrast with the successive discrimination phase. For these reasons, we consider that our rats did not acquire the stimuli control in this phase. On the other hand, with regard to the subsequent discrimination schedule acquisition a previous extinction with light on would have been worse than with light off.

Figure 4 shows that control subjects made more responses in the presence of the positive discriminative stimulus than experimental rats. These results differ from those obtained from the continuous reinforcement schedule and show a positive contrast phenomenon that is well described in operant discriminations (3,33,34). Subjects have approximately half the time to get to the reinforcement, and they increase their response in the positive periods. This greater increase in control rats shows that they reached higher acquisition levels than experimental rats in the successive discrimination.

The two-stimuli test involves the presence of both discriminative stimuli (positive and negative), and we would have to show ethanol's effects on the inhibition of response in accordance with the successive discrimination. In these tests, there were no significant differences between groups in the number of responses, but the two groups had a different evolution from the first session to the second one. Control rats showed a significant decrease in the number of responses from the first session to the second, as was expected, whereas experimental rats showed a slight decrease that did not reach statistical significance. Comparing Figs. 1 and 5, it can be observed that the very presence of the two exteroceptive stimuli implied an inversion of the results: In operant extinctions, control subjects performed more responses than experimental rats (these differences were statistically significant), but in two-stimuli tests experimental rats made more lever-press responses than control subjects (although these differences did not reach statistical significance).

We could consider that these results are due to a general deterioration of the memory because of the chronic ethanol intake. Nevertheless, the negative effects of alcohol have only been shown in the successive discrimination and two-stimuli test. If these results had been caused by a nonspecific memory worsening, we would have obtained an impairment both in the continuous reinforcement and extinction test (in all learning tests).

In summary, the results of this experiment suggest that oral ethanol self-administration specifically impairs the ability to inhibit a previously reinforced response, but only in situations indicated by exteroceptive stimuli. In absolute reinforced response situations, ethanol does not impair the acquisition of the lever-press response, whereas in extinction situations, not paired with exteroceptive stimuli, alcohol's depressing effects appear.

These results could explain the well-known desinhibitory effects of alcohol in humans (27), which could be due to this

impaired ability to inhibit previously reinforced responses indicated by exteroceptive stimuli, especially if we consider that, in most cases, human behavior is closely related to environmental cues.

ACKNOWLEDGEMENT

The authors thank the CIRIT (Generalitat de Catalunya), the Universitat Autònoma de Barcelona, and the Ministerio de Educatión y Ciencia for financial aid.

REFERENCES

1. Beracochea, D.; Jaffard, R. Impairment of spontaneous alternation behavior in sequential test procedures following mammillary body lesions in mice: Evidence for time-dependent interference-related memory deficits. Behav. Neurosci. 101:187–197; 1987.
2. Beracochea, D.; Lescaudron, L.; Tako, A.; Verna, A.; Jaffard, R. Build-up and release from proactive interference during chronic ethanol consumption in mice: A behavioral and neuroanatomical study. Behav. Brain Res. 25:63–74; 1987.
3. Bloomfield, T. M. Behavioural contrast and the peak shift. In: Gilbert, R. M.; Sutherland, N. S., eds. Animal discrimination learning. London: Academic Press; 1969:215–241.
4. Devenport, L.; Stidham, J.; Hale, R. Ethanol and spatial localization. Behav. Neurosci. 103:1259–1266; 1989.
5. Farthing, G. W.; Hearst, E. Generalization gradients of inhibition after different amounts of training. J. Exp. Anal. Behav. 11: 743–752; 1968.
6. Ferré, N. S. Un procedimiento rápido y eficaz para condicionar la respuesta a la palanca en ratas. Lat. Am. J. Psychol. 17:303–313; 1985.
7. Ferré, N.; Garcia-Sevilla, L. Conditionability and open field measures. Person. Individ. Diff. 8:193–200; 1987.
8. Gruchow, H. W.; Sobocinski, K. A.; Barboriak, J. J.; Scheller, J. G. Alcohol consumption, nutrient intake and relative body weight among US adults. Am. J. Clin. Nutr. 42:289–295; 1985.
9. Hearst, E. Some persistent problems in the analysis of conditioned inhibition. In: Boakes, R. A.; Halliday, M. S., eds. Inhibition and learning. New York: Academic Press; 1972:99–119.
10. Hearst, E.; Besley, S.; Farthing, G. W. Inhibition and the stimulus control of operant behavior. J. Exp. Anal. Behav. 14:373–409; 1970.
11. Hernandez, L. L.; Powell, D. A. Ethanol enhancement of pavlonian conditioning: Comparison with instrumental conditioning. Psychopharmacology (Berl.) 88:75–81; 1986.
12. Hernandez, L. L.; Valentine, J. D.; Powell, D. A. Ethanol enhancement of pavlovian conditioning. Behav. Neurosci. 100:494–503; 1986.
13. Honing, W. K.; Beale, I.; Seraganian, P.; Lander, D.; Muir, D. Stimulus and response reduction: Two aspects of inhibitory control in learning. In: Boakes, R. A.; Halliday, M. S., eds. Inhibition and learning. New York: Academic Press; 1972:41–71.
14. Jenkins, H. M.; Harrison, R. H. Generalization gradients of inhibition following auditory discrimination learning. J. Exp. Anal. Behav. 5:111–121; 1962.
15. Keane, B.; Leonard, B. E. Changes in 'open field' behaviors and in some membrane-bound enzymes following the chronic administration of ethanol to the rat. Neuropharmacology 22:555–557; 1983.
16. Larue-Achagiotis, C.; Poussard, A.-M.; Sylvestre-Louis, J. Does alcohol promote reactive hypoglycemia in the rat? Physiol. Behav. 47:819–823; 1990.
17. Lieber, C. S. The influence of alcohol on nutritional status. Nutr. Rev. 46:241–254; 1988.
18. Linseman, M. A. Consumption of alcohol compared to another bitter solution in a limited access drinking paradigm. Alcohol 5: 301–303; 1988.
19. Linseman, M. A. Effects of weight restriction and palatability on the apparent pharmacological regulation of alcohol consumption by rats in a limited access paradigm. Appetite 12:153–159; 1989.
20. Linseman, M. A.; Harding, S. Alcohol consumption following bidirectional shifts in body weight in rats. Psychopharmacology (Berl.) 97:103–107; 1989.
21. Lister, R. G. The effects of repeated doses of ethanol on exploration and its habituation. Psychopharmacology (Berl.) 92:78–83; 1987.
22. Mackintosh, N. J. Stimulus selection: Learning to ignore stimuli that predict no change in reinforcement. In: Hinde, R. A.; Stevenson, J., eds. Constraints on learning. New York: Academic Press; 1973:75–96.
23. Mackintosh, N. J. The psychology of animal learning. London: Academic Press; 1974.
24. Maier, D. M.; Pohorecky, L. A. The effect of ethanol and sex on radial arm maze performance in rats. Pharmacol. Biochem. Behav. 25:703–709; 1986.
25. Marcucella, H.; Munro, I. Ethanol consumption of free feeding animals during restricted ethanol access. Alcohol Drug Res. 7: 405–414; 1987.
26. Meisch, R. A.; Thompson, T. Ethanol intake as a function of concentration during food deprivation and satiation. Pharmacol. Biochem. Behav. 2:589–592; 1974.
27. Mello, N. K. Behavioral studies of alcoholism. In: Kissin, B.; Begleiter, H., eds. The biology of alcoholism. New York: Plenum Press; 1972:219–291.
28. Pavlov, I. P. Conditioned reflexes. London: Oxford University Press; 1927.
29. Prunell, M.; Boada, J.; Feria, M.; Benitez, M. A. Antagonism of the stimulant and depressant effects of ethanol in rats by naloxone. Psychopharmacology (Berl.) 92:215–218; 1987.
30. Rassnick, S.; Koob, G. F. GABA mediation of ethanol's anticonflict effect. Neurosci. Abstr. 16:135; 1990.
31. Rescorla, R. A. A pavlovian conditioned inhibition. Psychol. Bull. 72:77–94; 1969.
32. Sonderegger, T. B.; Ritchie, A. J.; Berney-Key, S.; Flowers, J. H.; Zimmermann, E. G. Microcomputer-assisted open field measures of rats given ethanol on postnatal days 1–8. Neurobehav. Toxicol. Teratol. 6:325–331; 1984.
33. Terrace, H. S. Stimulus control. In: Honing, W. K., ed. Operant behavior: Areas of research and application. New York: Meredith Co.; 1966:271–344.
34. Terrace, H. S. Conditioned inhibition in successive discrimination learning. In: Boakes, R. A.; Halliday, M. S., eds. Inhibition and learning. New York: Academic Press; 1972:99–119.
35. Thiébot, M. H.; Soubrié, P.; Simon, P.; Boissier, J. R. Dissociation de deux composantes du comportement chez le rat sous l'effet de psychotropes. Psychopharmacologia 31:77–90; 1973.
36. Vogel, R. A.; Frye, G. D.; Koepke, K. M.; Mailman, R. B.; Mueller, R. A.; Breese, G. R. Differential effects of TRH, amphetamine, naloxone, and fenmetozole on ethanol actions: Attenuation of the effects of punishment and impairment of aerial righting reflex. Alcohol Clin. Exp. Res. 5:386–392; 1981.
37. Wigal, T.; Greene, P. L.; Amsel, A. Effects on the partial reinforcement extinction effect and on physical and reflex development of short-term in utero exposure to ethanol at different periods of gestation. Behav. Neurosci. 102:51–53; 1988.
38. Zimmerberg, B.; Mattson, S.; Riley, E. P. Impaired alternation test performance in adult rats following prenatal alcohol exposure. Pharmacol. Biochem. Behav. 32:293–299; 1989.