Single Alternation with Water Reward: Effects of Sodium Amobarbital on Two Strains of Rats Selectively Bred for High and Low Emotionality

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The action of sodium amobarbital is studied during the patterning effect in rats selectively bred for low and high emotionality. The drug disrupts patterned running in the goal section of the alley in Maudsley non-reactive but not in Maudsley reactive rats for the last five trial-pairs. Sodium amobarbital also affects the behavior of these strains in a differential manner, in start and run sections, during the first trial-pair. The results show that a function-related physiological change has taken place in the Maudsley strains of rats.

There are many previous reports of the behavior of rats rewarded for running in the straight alley on a single alternating schedule of reward (R) and nonreward (N). When a sort inter-trial interval (ITI) is used, it is uncontroversial that the animal develops patterned running, i.e. it runs fast on R trials and slow on N trials. Recently, it has been shown the existence of sex and strain differences in patterned running (9), the authors pointed out that male Wistar rats displayed a greater patterning effect than females in the goal and start sections of the alley. They also showed strain differences between Maudsley Reactive (MR) and Maudsley Non Reactive (MNR) rats, the former displayed a greater patterning effect in the run section of the alley.

On the other hand sodium amobarbital disrupts the patterning effect in Sprague-Dawley male rats (6) in such manner that it was suggested an explanation of patterning in which the after-effect of reward becomes a conditioned stimulus

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for the elicitation of anticipatory frustration.

In the present experiment was studied the effect of sodium amobarbital on patterned running in the MR and MNR strains, which differ in a broad spectrum of behavioral tests in a manner which is consistent with the hypothesis that MR rats are more fearful than MNR rats (2). The aim of this experiment was: to advance a hypothesis on the behavioral mechanism which control patterned running in MR and MNR strains and, to get some knowledge on the form of action of sodium amobarbital.

Materials and Methods

Subjects. 14 male rats, from the colony of the Department of Experimental Psychology, University of Birmingham, 7 each from the 51 generation of MR and MNR, 180 days old at the beginning of the experiment, caged individually, were put on a 23-h water-deprivation schedule 22 days before training began. Food was ad libitum throughout.

Apparatus. A black straight alley, 1.7 m long, 22 cm wide and 36 cm high, was placed in the experimental room. The alley was of the kind described elsewhere (8), with only the following changes. In addition to run and goal times, start time was measured. For this purpose, the black lucite startbox door was modified as to be automatically opened by the release of a solenoid. Upon its release the timer for the start section began to count. This timer was stopped when the rat broke a photobeam located 9 cm in front of the startbox door. Run time was measured between this photobeam and one located 99 cm in front of the startbox door, and goal time between the latter photobeam and a final one located 2 cm from the rear goal box wall, directly above the water-cup. All times were to the nearest 0.01 s.

Breaking the final photobeam caused the delivery of 0.5 ml of water into the watercup on R trials. On N trials no water was delivered; all apparatus sounds were identical on N and R trials.

Procedure. Each rat was first handled for 5 min/day over 10 days. Pretraining consisted of 3 days of individual exploration of the alley for 30 min/day, followed by 3 days of 3 rewarded confinements in the goalbox and 4 days of 2 rewarded trials/day. The order of running alternated between strains, beginning with an MNR rat. Each rat run at the same time each day, and was given 1 hour free water commencing 30-45 min after the termination of its daily session. During training each rat was run individually, 12 trials/ day regularly alternating between N and R trials, with the first trial of each day always N. The ITI was 10-30 s. The startbox door was opened 5 s after the rat was put in the startbox. On R trials the rat was removed from the goalbox as soon as it had consumed the reward; on N trials goalbox confinement lasted 10 s.

The experiment was divided in two phases: the acquisition of patterned running and the drug study. The first part took 31 days of consecutive training; at the end of this period the data of the Ss was reciprocally transformed and multiplied by distance in metres to give speed scores. These gave satisfactory homogeneity of variance, and the transformed scores were submitted to analysis of variance for each subject individually. Since it was found that the behavior on the first pair of trials of the day was systematically different from that observed on the five trials-pairs (8, 9) the data were analysed separately for the first trial-pair and the remaining 10 trials. After these analyses 1 rat of each strain were discarded because they did not show any signal of patterning.

In the drug period training continued

as in the first phase with the exception that each rat received an intraperitoneal (i.p.) injection of 1 ml/kg isotonic saline or 20 mg/l ml of sodium amobarbital i.p. 15 minutes before the session. These lasted 8 days, divided into two blocks of 4 which 2 were assigned to drug and 2 to saline. The order of drug (D) and saline (S) treatment was: S, D, D, S; D, S, S, D. The data of the drug period was transformed as in the first phase and submitted to analysis of variance.

Results

Drug study. First it will be examined the final 5 trial-pair of each day.

In the start and run sections, the main effect of drug was not significant (F < 1); there were highly significant interactions between drug and reward (the last variable is R versus N trials), F (1.631) = 13.3 and 16.0, respectively, p < 0.001; and the interaction between drug, reward and strain were non-significant. Inspection of the drug \times reward interactions (figure 1) shows that the patterning effect was present in both saline (start, t=10.44, df = 631, p < 0.001; run, t = 10.59, p < 0.001) and drug (start, t = 5.29, p < 0.001; run, t = 4.91, p < 0.001) conditions, thoug clearly smaller in the



Fig. 1. Effects of sodium amobarbital on N and R trials in start and run sections for the last five trial-pairs.



Fig. 2. Strain differences induced by sodium amobarbital between MNR and MR strains, in the goal section, for the last five trialpairs.

latter than the former. The drug produced this change, however, not by increasing speed on N trials, but by decreasing speed on R trials (drug-saline difference in start, t = 3.63, and in run t = 4.12, p < 0.001). The difference between N speeds on drug and saline days, respectively, was non-significant.

In the goal section, the results were somewhat different. There was again a significant drug \times reward interaction, F (1.631) = 76.3, p < 0.001; but there was also a significant drug \times reward \times strain interaction, F (1.631) = 19.8, p < 0.001, and the drug \times reward effect was no longer significant when tested against this three-way interaction (fig. 2). The amobarbital increased goal speed on N trials. This effect was clearly significant in both the MNR (t = 9.49, df =631, p < 0.001) and the MR (t = 5.13, p < 0.001) strains. Within the MNR strain the patterning effect was no longer significant in the drug condition, but it continued to be highly significant in the MR strain under the drug (t = 7.62, p = 0.001). (In both strains, of course, the patterning effect was highly significant on saline days). With regard to the effect of the drug on R trials, this is negligible in MR strain, but there is a significant decrease in R goal speeds in the MNR rats (t = 3.25, p < 0.01).

Turning to the analyses of the first trial-pair, the results in the start and run sections were again similar, both yielding significant three-way interactions between strain, drug and reward, F(1.67) = 10.9 and 4.0, p < 0.01 and 0.05 respectively. These interactions are presented in figure 3, from which it can be seen that they arise largely because amobarbital, which reduced speeds in all other conditions, failed to do so in MR strain on the first N trial; indeed, in the start section there was actually an increased speed under the drug condition, t = 3.54, p < 0.02.

The first-trial effect, noted in earlier experiments (8, 9) is present in all conditions, but is noticeably reduced in the start section in MR rats under amobarbital.

In the goal section the two findings of note were significant interactions between



Fig. 3. Differential effects of sodium amobarbital on MNR and MR strains during the first trial-pair, start and run sections.

strain and reward, F(1.67) = 6.1, p < 0.05, and between drug and reward, F(1.67) = 6.4, p < 0.05. The strain \times reward interaction merely reflected the continuing slowness in the goal of MNR rats on the first trial of the day. The drug \times reward interaction was of more interest. It reflected the fact that amobartital slowed goal speeds more on the second, R, trial than on the first, N, trial thus reducing the first-trial effect. It is noteworthy that the direction of the effect of amobarbital on the first N trial (a reduction in goal speed) is the opposite of that found on subsequent N trials (an increase in goal speed: figure 2).

Discussion

The major finding in this experiment is that sodium amobarbital affected patterning quite differently in the two Maudsley strains than in previous experiments with Sprague-Dawley rats (6). In Sprague-Dawley, sodium amobarbital reduced patterning in all sections of the alley by increasing speeds on N trials; the drug had only small effect on R trials and these were in the direction of an increase in speed. In the two Maudsley strains, in contrast, the effect of drug varied with the section of the alley. In the start and run sections, the patterning effect was reduced, but this was due to decrease in speed on R trials, only non-significant effects being produced on N trials. In the goal section the effect of amobarbital was more comparable to its effect in Sprague-Dawley rats. Speeds on N trials were significantly increased, thus reducing the patterning effect. Even in the goal, however, the patterning effect was abolished (as occurred in Sprague-Dawley) only in the MNR strain: it cotinued to be highly significant, albeit reduced in size, in MR strain. Furthermore, the abolition of patterning in the goal in the MNR strain was in part due, not only to increased N speeds under the drug, but also to significantly *decreases* speeds on R trials; thus it seems that the mechanism of patterning in the Maudsley strains differs from that in Sprague-Dawley in one of two ways: either the psychological processes are different, and not so dependent on anticipatory frustration, which sodium amobarbital is known to antagonise (7): or the physiological mechanism which mediate these processes are resistant to the action of the drug. Both these possibilities have some merit.

With regard to the former, it was pointed out (6) that the application of CAPALDI's theory (4) to patterning, which holds that the behavior on R trials is under the control of the immediate aftereffect of the preceding N trial, might predict that sodium amobarbital would decrease speeds on R trials, by blocking this after-effect (5), while leaving speeds on N trials unaffected. AMSEL's theory (1), on the other hand, predicts that the drug will increase speeds on N trials, by blocking anticipatory frustration (7) while leaving speeds on R trials unaffected. Sprague-Dawley rats (6) behaved according to the frustration model (1). The Maudsley strains, in contrast, behave according to the derivation from CAPALDI's theory (4) in start and run sections of the alley (and it is in the early sections of the alley that CAPALDI's postulated processes are expected to be strongest). Thus it is possible that the Maudsley strains are more influenced by the immediate aftereffect of reinforcing events, while Sprague-Dawley rat is more influenced by conditioned frustration. The application of this analysis to the goal section is more complex. The MR strain behaves here like the Sprague-Dawley rat (i.e. according to the frustration model), but less completely than the latter. The MNR rat behaves in the goal section as though both processes were in play, with the frustration process the stronger.

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With regard to the possibility of a changed physiological response to sodium amobarbital in the Maudsley strains, there is other evidence pointing to this. Thus, the usual response to sodium amobarbital in the shuttlebox is an improvement in avoidance behavior (7), but Maudsley rats either show no response or an impairment (10, 11). A similar pattern is evident with respect to ethanol, which also facilitates performance in shuttlebox (7), but has not such effect in the Maudsley strains (3). Thus these strains may differ from other rats rather generally in their drug response.

The two possibilities mentioned in the preceding paragraphs have been distinguished principally for purposes of exposition. In any real case they are unlikely to be separate from each other. The results of the present experiment show that a function-related physiological change has taken place in the Maudsley strains.

With regard to first trial-pair, the results afford further evidence that behavior on these two trials is different in kind from that seen on the subsequent five trial-pairs (8, 9), since the effects of sodium amobarbital were different in these two conditions. Thus, in the goal section, the drug increased speeds on latter N trials, but reduced speed on first N trial of the day. In the start and run sections, in the MNR strain only, the drug again had opposite effects depending on which pairs of trials are considered: in later trials N speeds were unaffected but on the first N trial speeds were considerably reduced (fig. 3). Only in the start for the MR strain did the drug effect not differ depending on trial-pair (fig. 1). These results also suggest that the psychophysiological processes governing the first N trial of the day are strain-dependent: the drug had different effects on performance in the start and run sections in the MR and MNR strains.

Resumen

Se estudia la acción del amobarbital sódico sobre el efecto de patrón de carrera en ratas con índices bajos y altos de emocionalidad. La droga bloquea el patrón de carrera en la sección de meta del pasillo en la raza con índices bajos, pero no afecta a la discriminación en la raza de índices altos. La primera pareja de carreras está afectada por la droga de forma diferencial según la raza. Los resultados muestran que ha habido un cambio funcional con respecto al aprendizaje en ambas razas estudiadas.

References

- 1. AMSEL, A.: Psychol. Rev., 69, 306-328, 1962.
- 2. BROADHURST, P. L.: Behav. Genet., 5, 299-319, 1975.

- 3. BROADHURST, P. L. and WALLGREN, H.: Q. J. Stud. Alcohol, 25, 476-489, 1964.
- 4. CAPALDI, E. J.: Psychol. Rev., 73, 459-477, 1966.
- 5. CAPALDI, E. J. and SPARLING, D. L.: J. Comp. Physiol. Psychol., 74, 467-477, 1971.
- 6. FELDON, J., GUILLAMON, A., GRAY, J. A., DE WIT, H., and MCNAUGHTON: Q. J. Exp. Psychol., 31, 19-50, 1979.
- 7. GRAY, J. A.: In «Handbook of Psychopharmacology» (Iversen, L. L., Iversen, S. D. and Snyder, S. H., eds.) vol. 8, Plenum Press, New York, 1977, pp. 433-529.
- 8. GUILLAMON, A. and GRAY, J. A.: Rev. Psicol. G. Aplicada, 32, 581-592, 1977.
- 9. GUILLAMON, A., GRAY, J. A. and BROAD-HURST, P. L.: Rev. Psicol. G. Aplicada, 32, 800-816, 1977.
- 10. GUPTA, B. D. and HOLLAND, H. C.: Psychopharcol., 14, 95-105, 1969.
- 11. POWELL, B. J.: Proc. 75th Ann. Conv. Amer. Psychol. Ass., 1967, 69-70.