Subject-to-subject trial sequence, odor trails, and patterning at 24-h ITI¹

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It was possible to effect single-alternation patterning at 24-h ITIs, and then to disrupt it, and then to reinstate it in three successive phases of an experiment. The results support the hypothesis that rats exude odors when rewarded and/or nonrewarded which can serve as discriminative cues for the differential responding of other rats.

The results of some recently reported experiments (Ludvigson & Sytsma, 1967; McHose, Jacoby, & Meyer, 1967; McHose & Ludvigson, 1966) suggest the possibility that rats exude odors in the goal box of a runway when nonrewarded (N) which are discriminably different from odors on rewarded (R) trials, and that the odor trails of one S can serve as a cue for differential responding by another on a subsequent trial if a squad of Ss is run in rotation. (Conceivably, also, an S might pick up his own odor trail and use it as a cue under highly massed or free-responding conditions.) Furthermore, the data so far indicate that the appearance of differential responding based on such a presumed cue is confined to the goal area, where the odor hypothesis would naturally predict the cues to be strongest.

Results and interpretations such as these would seem to have relevance for the question of single alternation (SA) patterning at 24-h ITI (McHose, 1967). In two experiments in which we have been unable to demonstrate patterning at 24-h ITIs (Surrige & Amsel, 1966, 1968) we unwittingly prevented the operation of what McHose calls "odor trails" as cues for differential responding. On any one day, half our Ss received R trials and half N trials in a random sequence of presentation. Thus, neither "R odors" nor "N odors" were predictive of the state of events in the goal S was approaching.

If the odor hypothesis is correct, it should be possible to produce patterning, or to disrupt it at will, simply by manipulating the within-day sequence of trial administration. The study we report attempts to do just this.

METHOD

Ten male albino rats, 60-70 days of age on arrival in the laboratory, were maintained on a 23-h, 10-g food-deprivation schedule for 34 days prior to experimental training,

during which time they were handled and tamed. They were housed in individual cages which were distributed randomly throughout a 42-cage rack in the colony room, the restriction being that no two Ss were housed side-by-side.

The straight, 5-ft 3-in. runway was the same as that used by Surrige & Amsel (1968). The entry box and start box were painted flat grey, while the runway was painted flat white on the left half, flat black on the right half. Five 1-ft time measures of performance were provided by means of a microswitch on the start door and five photobeams located in the runway. The food pellet used as reward was placed in a pellet dispensing device located outside the

end wall of the goal box and was delivered when S broke the last photobeam.

All Ss ran a total of 168 trials in a single alternating schedule of R and N trials, one trial a day. The experiment was conducted in three phases, with a 6- or 7-day period of no running between phases. The food-deprivation schedule was maintained throughout. Reward was one 500-mg Noyes pellet. The experiment began with an R trial for each S.

The S was taken from its home cage in the colony room to the experimental room and placed in the entry box. The entry box door was then raised, and lowered when S entered the start box. The start box door was raised approximately 2 sec later. When S had interrupted the final photobeam the goal box door was lowered. S was removed from the goal box after eating (approximately 30 sec) on R trials, after 120 sec on N trials, and placed in a waiting cage. On R trials S spent approximately 90 sec (after consuming the reward) in the waiting cage; on N trials S remained in the waiting cage long

Fig. 1. Response speed on R and N trials across three phases.

enough for the apparatus to be reset for the next trial before being returned to the home cage. The daily ration of food was given to all Ss 30-40 min after the testing of the last S.

On Days 1-72 (Phase 1) all Ss received their R trials on odd days and their N trials on even days. One animal (S1) was always run first, while Ss 2-10 were run in random orders from day to day.

On Days 73-112 (Phase 2) the N and R trials were mixed within days, and half of the Ss received R trials, half N trials, on each day. In order to do this and maintain the SA schedule, half of the Ss began this phase 1 day before the remainder, which necessitated a 6-day interval between phases for some Ss and a 7-day interval for others. Again S1 was always run first. The sequence of running for the remaining nine Ss was such that within each block of 4 days, each S ran two trials when the trial for the preceding S was R and two trials when the trial for the preceding S was N. In each case S ran once to R and once to N when the preceding S's trial was R, once to R and once to N when the preceding S's trial was N.

On Days 113-168 (Phase 3) the trial sequence reverted to that used in Phase 1; on any given day, all Ss ran either to R or to N, and there was no mixing of trials within a day.

RESULTS

Time data from each measure were converted to speeds by reciprocal transformation, and were analyzed as a Reinforcement (R or N) by Rat by Block (of eight trials) within-S design. The unit of analysis is the mean of four R or four N trials per block per rat, and data for S1 and for Ss 2-10 are treated separately. Response speeds over the three experimental phases for the Start (first), Run 2 (third), and Goal (fifth) runway measures are presented in the top three panels of Fig. 1. The bottom panel presents the goal data for Trials 1-72 from the comparable group (N-confinement 120 sec) of Surridge & Amsel (1968), in which N and R trials were mixed within days, and which showed no evidence of patterning over 134 days.

During Phase 1 patterned responding for Ss 2-10 appeared, but only in the goal measure, both in the form of an overall difference [$F(1,8) = 15.85$, $p < .01$], and differentially over blocks of trials [$F(8,64) = 20.25$, $p < .01$]. Although the Reinforcement by Blocks interaction was significant in the Run 1 measure (not shown) [$F(8,64) = 3.99$, $p < .01$], there was no consistent performance difference over blocks on R and N trials. It should be noted that patterning did not occur in the comparable group from Surridge & Amsel (1968). Their animals were treated identically to our present ones except for the

sequence of trial administration.

In Phase 2, patterning in the goal measure disappeared completely [$F(1,8) = 1.07$, $p > .10$], and, as was to be expected, did not appear in any other runway segment.

Reverting to Phase 1 conditions in Phase 3 restored patterning in the goal region [$F(1,8) = 10.12$, $p < .01$], but not differentially over blocks. There was some evidence of patterned responding in the Run 1 measure during the final phase [$F(1,8) = 7.46$, $p < .05$], and there was a Reinforcement by Blocks interaction in the Run 2 measure [$F(6,48) = 2.77$, $p < .05$].

The reliability of the patterning phenomenon in individual animals was assessed by comparing speeds on adjacent R (odd-numbered) and N (even-numbered) trials for the goal measure. All nine "experimental" animals, running randomly over days from Positions 2 to 10, responded faster on R trials on at least 81% of trial pairs over the final 32 trials of the first phase. The "control" S that always ran first (S1) "patterned" at a chance level (i.e., 50%) as the speed data indicate. The consistency of patterning was somewhat less during Phase 3. Six of the nine experimental rats patterned on between 64% and 82% of trial pairs, while three were at a chance level, as was the "control" animal.

The R-N differences were not as large in the third phase as they were in the first. Eight experimental Ss had a smaller R-N difference in Phase 3 than in Phase 1, when the last 24 trials of the two phases were compared, and the mean decrease across animals was significantly greater than zero [$t(8) = 4.89$, $p < .01$].

DISCUSSION

The present results, taken together with the results of our previous studies (Surridge & Amsel, 1966, 1968), suggest that the sequence of R and N trial administration can be a powerful factor in the appearance of patterning at 24-h ITIs. In the absence of other obvious explanatory factors, our results support the contention of Ludvigson and of McHose that rats lay down odors related to reward and nonreward which can serve as cues for differential responding by other rats.

The fact that the very strong patterning produced in Phase 1 was completely disrupted in Phase 2 simply by altering the within-day presentation of rewards and nonrewards is perhaps our most impressive evidence that discrimination of R from N trials, in the present experiment, was based on differential cues from something like odor trails. Barnett (1967) has stressed the importance of odors to rats and has indicated that "rats can learn to associate food with particular odors, and even with an odor to which they had previously shown an aversion [p. 36]."

There is a ready explanation for the finding that discriminative patterning in Phase 3 was not as strong as it was in Phase 1. In Phase 2 training Ss 2-10 ran to nonreward in presence of R and N odor cues and ran to reward also in the presence of both cues. We know that exposing animals to nonpredictive experience with stimuli ($S_1 \pm S_2$) which are to become discriminative stimuli subsequently ($S_1 + S_2$) severely retards subsequent discrimination (Amsel & Ward, 1965). Phases 2 and 3 of the present experiment constitute such a sequence, where reward and nonreward odors are the S_1 and S_2 .

Finally, even though odor-trail factors may be a sufficient condition, they may not be a necessary condition for the appearance of SA patterning at 24-h ITIs. Capaldi & Lynch (1966) obtained one-trial-a-day patterning, and took great pains to control for "odor trail" by running half the N trials and half the R trials on each experimental day, in a random order. Their procedure most nearly corresponds to our Phase 2, in which we observed that patterning was eliminated. The discrepancy between their results and those of Phase 2 of the present experiment and our two earlier studies (Surridge & Amsel, 1966, 1968) cannot be accounted for simply in terms of an odor-trail hypothesis. The reasons for such a discrepancy must be sought elsewhere.

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NOTE

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