Pergamon

Physiology & Behavior, Vol. 57, No. 6, pp. 1221–1224, 1995

0031-9384/95 $9.50 + .00

0031-9384(94)00371-8

BRIEF COMMUNICATION

Food-Delay Duration and the Development of Schedule-Induced Polydipsia in Rats

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Received 21 July 1993

LAMAS, E. AND R. PELLÓN. *Food-delay duration and the development of schedule-induced polydipsia in rats.* PHYSIOL BEHAV **57**(6) 1221–1224, 1995.—Twelve rats were exposed to a schedule that delivered a food pellet every 60 s (fixed time 60 s). The development of schedule-induced polydipsia was measured in terms of the water consumed and the licks per interpellet interval. Every lick by master rats initiated an unsignalled delay of 2 or 50 s in food delivery. Yoked-control rats received food at the same time as their masters, being unaffected by their own licking. Schedule-induced polydipsia developed in master rats exposed to 2-s delays, but more slowly and to a lesser extent than control animals. The development of polydipsia was prevented in master rats exposed to 50-s delays, however. When these delays were discontinued, polydipsia was obtained by master rats. The finding that the effect of the delays was modulated by their duration supports the view that the development of schedule-induced polydipsia is sensitive to control by its environmental consequences.

Schedule-induced polydipsia Acquisition Unsignalled-delay duration Licks Rats

FOOD-DEPRIVED rats exposed to an intermittent food-reinforcement schedule normally drink excessive amounts of water if they have the opportunity to do so (2). The development and maintenance of this schedule-induced polydipsia can be controlled by consequences programmed on the rats' drinking. For example, well-established patterns of polydipsic drinking can be punished by lick-contingent shocks (1) or delays in food presentation (5).

The acquisition of schedule-induced polydipsia is also sensitive to lick-dependent delays in food presentation, but these effects were different when the delays were signalled or unsignalled (4,6) With signalled delays the development of schedule-induced polydipsia was not attenuated, and the effects of these delays were subsequently seen when the high levels of drinking were reduced (6). Unsignalled delays were more effective than signalled delays in attenuating the development of schedule-induced polydipsia. Studies that varied the delay duration have found that lick-dependent delays were either effective or not in preventing the development of schedule-induced polydipsia (4), but there was no clear evidence of a gradation in the effects of the delays as a function of their durations. Unsignalled lick-dependent delays of 1 min or 4 min prevented the development of schedule-induced polydipsia; however, delays of 10 s or 30 s did not. These results are difficult to interpret: the lick-dependent delays were not discontinued in a second phase of the experiments to see whether the rats then drank more. This experimental arrangement appears to be critical to attribute adequately the effects observed to the programmed delays, rather than merely to the individual characteristics of the animals.

In a previous study (6) we included a phase where the delays were discontinued, and we showed that 10-s unsignalled delays led to a lower development of schedule-induced polydipsia in master than yoked-control rats, but these delays did not prevent completely the acquisition of schedule-induced polydipsia. Furthermore, our study also showed that by discontinuing these delays the master animals generally increased the measures of schedule-induced polydipsia. The purpose of the present experiment was to investigate whether lick-dependent unsignalled delays shorter and longer than 10 s would have differential effects on the acquisition of schedule-induced polydipsia in rats. The experiment to be reported was therefore designed to complement previous findings by Pellon and Blackman (6). As before, the present experiment included a phase where the delays were discontinued, and appropriate yoked-control conditions.

METHOD

Subjects and Apparatus

Twelve naive male Sprague–Dawley rats (IFFA-CREDO, Lyon, France) served as subjects. They were 90 days old at the

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start of the experiment, with a mean body weight of 445 g (range: 417–473 g). Rats were housed individually in an environmentally controlled room (22°C temperature, 60% relative humidity, and 8 a.m./8 p.m light/dark cycle), and they were gradually reduced to 80% of their free-feeding weights by controlled feeding. Water was continuously available in the home cages.

The experiment was conducted in four identical Letica Instruments (Barcelona, Spain) LI-836 standard rodent test chambers, 29 × 24.7 × 35.5 cm. Each chamber was contained inside a ventilated sound-attenuating chest. No operant levers were installed. A water bottle was mounted on the outside of the right wall of the chambers, with its spout accesible through a hole 3.2 × 3.9 cm situated 20 cm from the front wall and 7 cm above the grid floor. The spout was positioned 2 cm behind the hole. Licks at the spout were sensed by a photocell beam situated in the hole 1 to 2 mm from the spout. Two 3-W houselights illuminated the chambers. The ambient noise produced by the ventilation fan was 60 dB, which served as masking noise. A Letica Instruments pellet dispenser was located behind the front panel, and it delivered 45-mg pellets of standard rat food to a receptacle in the center of the front wall of the chamber, situated 3.7 cm from the grid floor. The scheduling and recording of experimental events was achieved by a BBC microcomputer programmed in SPIDER.

Procedure

When each rat had stabilized at 80% of its free-feeding weight, a water-ingestion test was given on two sucessive days. Sixty 45-mg food pellets were placed together in a dish in the home cages and the amount of water consumed by each rat in 60 min was measured (massed-food test). The rats were then allocated to six pairs in terms of their water intake during the test. In each pair, one rat was assigned randomly as master and the other as yoked control. All rats were then exposed to the test chambers for 1 h, where they ate 20 food pellets.

After this pretraining, the experiment proper began. The water bottles were now filled with 100 ml of fresh tap water and installed immediately before each experimental session, conducted 7 days per week. Each session began with illumination of the houselights and delivery of one 45-mg pellet of food to the receptacle. In the first phase of the experiment (Delay), the rats were exposed to 42 sessions. In the absence of licks by a master rat, a single 45-mg pellet of food was delivered at regular 1-min intervals independently of the rat's behavior (an FT 60-s schedule) However, each lick by a master rat initiated a delay in the delivery of the next pellet of food. This delay was of 2 s for master rats 1, 3, and 5, and of 50 s for master rats 7, 9, and 11. Each lick by the master rat reset the delay. For the appropriate yoked-control animal, pellets of food were presented at the same time as they were given to the master rat. There was no programmed contingency between the licking of the control animals and the delays in food delivery. The houselights were turned off at the end of each session, which ended with the completion of the interval following the 60th presentation of food or 120 min after the beginning of the session, whichever occurred earlier. The second phase of the experiment (No Delay) consisted of 14 sessions. The licks of the master rats no longer had any programmed consequences. All rats were thus exposed to an FT 60-s schedule of food presentation.

The following measures were recorded for each rat each session: (a) the amount of water (ml) removed from the bottle; and (b) the total number of licks per session, which was converted to a measure of licks per interpellet interval. The number of interpellet intervals and the session duration were also recorded, which allowed for the calculation of the mean duration of the interpellet interval for each pair of rats each session.

RESULTS AND DISCUSSION

Table 1 summarizes the mean amount of water each rat drank in the two massed-food tests and in the last five sessions of the Delay and No-Delay phases. All master and yoked-control rats drank little water during the massed-food test, which was similar within each pair of rats. At the end of the Delay phase, yoked-control rats drank more water than they had in the test, and these high levels of polydipsic drinking were maintained when the delay contingency was removed in the second phase of the experiment (despite the decrease observed for rat 12). With master rats, the consumption of water at the end of the phase with delays was dependent on whether they were exposed to 2- or 50-s delays. Rats 1, 3, and 5 (with 2-s delays) showed an ingestion of water that was higher in the last five sessions of the Delay phase than in the massed-food test. Once the delay contingency was removed, rat 1 increased its final water intake to approximate the level of drinking shown by its yoked control. Rats 3 and 5 did not change the final amounts of water drunk from the first to the second phase, being similar to the levels of yoked controls 4 and 6 in both phases of the experiment, respectively. However, master animals submitted to 50-s delays (rats 7, 9, and 11) did not generally drink much more water with the delays than during the massed-food test, even though there was an increase for rat 9. When the delays were removed, these rats increased their mean water intake, but they did not reach the levels of water consumed by their yoked controls at the end of the No-Delay phase.

Table 1 also summarizes the mean interpellet interval duration for each pair of rats during the last five sessions of the Delay and No-Delay phases, calculated by dividing each session duration by the number of food pellets delivered (which was always 60). This interval was by definition 60 s in the No-Delay phase. In general, the mean interpellet interval was somewhat higher in the pairs of rats exposed to 2-s delays. This longer duration is the consequence of master rats 1, 3, and 5 drinking more than master animals exposed to 50-s delays, even though the delays initiated by the former were of a much shorter duration.

Figure 1 shows the mean daily number of licks per pellet for each rat, given as licks per interval (total number of licks divided

TABLE 1

MEAN WATER INTAKE (ml) AND MEAN INTERPELLET INTERVAL (s) IN THE LAST FIVE SESSIONS OF THE TWO PHASES OF THE EXPERIMENT. ALSO SHOWN IS THE MEAN WATER INTAKE FOR EACH RAT IN THE MASSED-FOOD TEST. LICK-DEPENDENT DELAYS WERE OF 2 s (MASTER RATS 1, 3, AND 5) OR 50 s (MASTER RATS 7, 9, AND 11).

	Mean Water Intake (ml)			Mean Interpellet Interval (s)	
	Massed Food	Delay	No Delay	Delay	No Delay
Rat 1 (master)	2.5	15.8	25.4	67.88	60.00
Rat 2 (control)	2.5	23.0	29.0		
Rat 3 (master)	5.5	21.2	22.8	71.03	60.00
Rat 4 (control)	5.5	23.0	16.8		
Rat 5 (master)	4.0	29.6	29.0	76.48	60.00
Rat 6 (control)	4.0	31.2	30.8		
Rat 7 (master)	5.0	5.8	9.0	64.89	60.00
Rat 8 (control)	5.0	19.9	19.0		
Rat 9 (master)	4.0	11.2	27.8	69.12	60.00
Rat 10 (control)	4.0	45.6	40.0		
Rat 11 (master)	7.0	5.2	18.0	64.60	60.00
Rat 12 (control)	6.5	54.6	33.6		

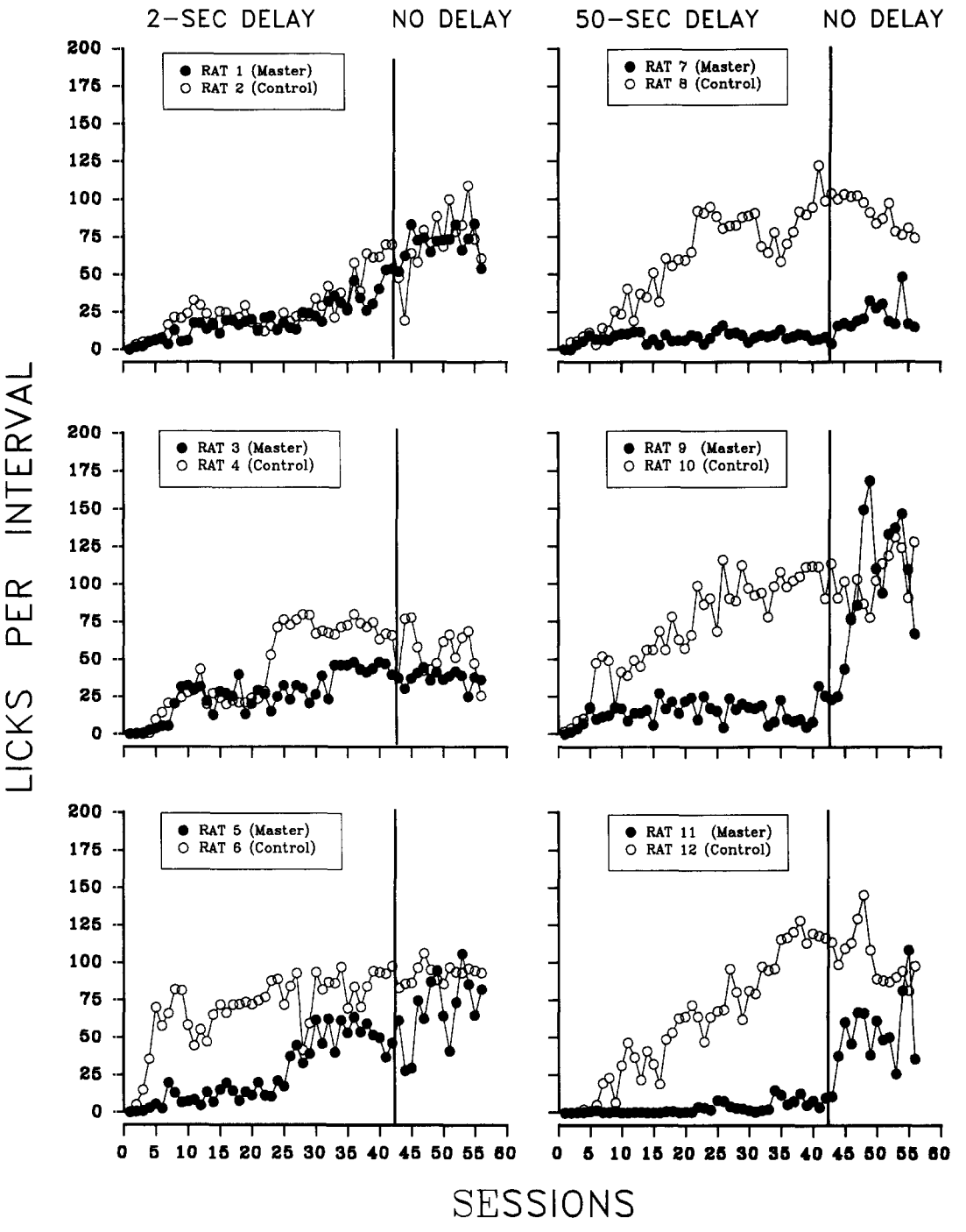

FIG. 1. Daily mean licks per interpellet interval for each rat.

by total number of intervals in the session). Note that this measure is preferred to licks per minute, as it captures better the postfood drinking pattern and because the mean interpellet interval varied between phases of the experiment. Each pair of rats is depicted in a separate panel, with the closed and open circles corresponding to the master and control rats, respectively. Master rats 1, 3, and 5 (left panels) increased steadily their number of licks throughout the sessions with 2-s delays up to about 50 licks per interval at the end of the first phase. Yoked controls 2 and 4 developed schedule-induced licking similar to their masters during the first 22 sessions; then rat 4 increased licks per interval to stabilize at values approximating 75. Rat 2 continued to increase the number of licks from session 22 to the end of the Delay phase in parallel to master rat 1. Yoked control 6 developed licking much more rapidly, and to a greater extent, than master 5. Although masters 1, 3, and 5 licked less than their respective yoked controls at the end of the Delay phase, there were no very marked differences between master and control animals. When the delay contingency was removed during the second phase of the experiment, master and control rats showed similar changes in licks

per interval (except pair 5 and 6). Rats 1 and 2 increased slightly the number of licks during the No-Delay phase, there were no changes in the licking of rats 3 and 4 with the removal of the unsignalled delays, and rat 5 increased licks per interval to reach the level of yoked control 6.

The right panels of Fig. 1 represent the results of the three pairs of rats exposed to 50-s lick-dependent delays. As can be seen, yoked-control rats 8, 10, and 12 developed a high number of licks as the sessions progressed until they reached about 100–125 licks per interval. Master rats 7, 9, and 11, however, never developed a substantial number of licks. In fact, 50-s lick-dependent delays prevented the development of schedule-induced licking in master rats 7 and 11, and they reduced considerably the number of licks in master rat 9 in comparison to its yoked control. At the end of the Delay phase control rats licked much more than master rats. When the delay procedure was discontinued in the second phase of the experiment, all master rats showed an increase in the number of licks per interval. This increment was moderate for master rat 7, but rats 9 and 11 increased considerably, reaching levels of licking that were similar to those of their yoked controls. All yoked controls showed no significant changes in licks per interval when the delays were removed, but minor decreases in rat 12.

The results of the present experiment show that unsignalled lick-dependent delays even as short as 2 s have effects on the acquisition of schedule-induced polydipsia. The present results also show that the effects observed with lick-dependent delays were a function of the delay duration. The development of drinking induced by the FT 60-s schedule was retarded and slightly attenuated when every lick by the master rats initiated a 2-s delay on the delivery of the next pellet of food. A higher attenuation of the asymptotic level of drinking was observed with longer delays, such as 10 s (6). Longer delays served in addition to abolish almost completely the normal development of schedule-induced polydipsia. This was shown with 1-min delays in previous work (4; but see 8), and with 50-s delays in the present study. Similar functions of the delay duration have been demonstrated on already established patterns of schedule-induced polydipsia (3).

The present results appear in general to support the view that the consequences of licking can modulate the acquisition of adjunctive polydipsia. This claim is in keeping with the finding that the acquisition of schedule-induced polydipsia can also be attenuated when extra food is programmed to occur for not licking (7). The acquisition of schedule-induced polydipsia is affected by its environmental consequences, though this is is not to say that it is established or maintained only by its consequences The development of schedule-induced polydipsia was prevented only when lick-dependent unsignalled delays in food delivery were relatively long, thus emphasizing once again the robustness of this behavioral phenomenon.

ACKNOWLEDGEMENTS

This work was carried out while Esmeralda Lamas was recipient of a research grant from Universidad Nacional de Educación a Distancia (Madrid, Spain). We thank Derek E. Blackman for his continuous support and for his comments on an earlier version of this article. Parts of these data have been presented to the 17th Annual Convention of the Association for Behavior Analysis (Atlanta, GA, 1991) and the Annual Conference of the Experimental Analysis of Behaviour Group (London, UK, 1993).

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