Pharmacology Biochemistry & Behavior, Vol. 9, pp. 283–286. Printed in the U.S.A.

Control of Polydipsic Drinking by a Taste Aversion Procedure

J. CHRISTOPHER CLARKE[1] AND R. F. WESTBROOK

University of New South Wales, Kensington, N.S.W., Australia 2033

(Received 10 February 1978)

CLARKE, J. C. AND R. F. WESTBROOK. *Control of polydipsic drinking by a taste aversion procedure.* PHARMAC. BIOCHEM. BEHAV. 9(3) 283–286, 1978.—Rats were given daily sessions with free access to food and saccharin flavored water. After fluid consumption had stabilized food was delivered once every minute. Water was always available in the home cage. All rats showed the marked increase in fluid consumption known as schedule-induced polydipsia. The rats were then poisoned with lithium chloride after each of three sessions in an attempt to condition a taste aversion to the saccharin. On recovery from the toxicosis all rats showed first a reduction and then a recovery in saccharin intake. To establish the nature of this effect, the rats were poisoned after saccharin consumption in the home cage. Again there was a marked reduction in polydipsic drinking in the experimental chamber. These results indicate that common incentive mechanisms govern normal and polydipsic drinking and stand in contrast to published results pointing to different drive systems in the brain mediating normal and polydipsic drinking.

Taste aversion Polydipsia LiCl

IF A hungry rat, kept on ad lib water, is placed in an experimental chamber and exposed to an intermittent food reinforcement schedule there will be a marked elevation in fluid consumption for the period of the animal's confinement in that chamber. This phenomenon, known as schedule-induced polydipsia, was first reported by Falk [2]. He discovered that rats bar pressing for food on a variable-interval 1-min schedule, consumed, on the average, 92.5 ml of water during the 3.2 hr session, whereas the normal intake for animals on a free food and water schedule was about 27 ml over a 24-hr period. Subsequent experiments have ruled out "dry mouth" and superstitious or adventitious conditioning as explanations of schedule-induced polydipsia. It is also possible to rule out the response requirement (e.g., bar pressing) as a causal factor because food delivered independently of the animal's behavior will also produce the effect.

Schedule-induced polydipsia is strong and persistent when contrasted with the drinking that occurs under ordinary conditions of free access to food and water. Even strong facilitating conditions such as extreme water deprivation, heat stress or osmotic loading techniques do not stimulate such levels of fluid consumption as are routinely obtained in scheduled-induced polydipsia experiments.

While it is a robust effect it is also a puzzling one. To date no adequate theory of schedule-induce polydipsia has been developed. As a first step in this direction Falk [3] has suggested grouping schedule-induced polydipsia and presumably related behaviors such as schedule-induced aggression and schedule-induced wheel running together as adjunctive responses. These behaviors, which he likens to the displacement activities studied by ethologists, are not instrumental in obtaining reinforcement and, he argues, are governed by mechanisms which will be shown to be different from those which regulate normal appetitive or consummatory behavior.

Support for this view comes from a recent experiment by Singer, Armstrong and Wayner [7]. They found that intrahypothalamic injections of norepinephrine blocked deprivation-induced, salt-aroused or carbachol-produced drinking in rats. These same injections, however, had no such effect on polydipsic drinking.

The results from the experiment of Singer *et al.* suggest that different systems in the brain are involved in the regulation of normal (or chemically-induced) drinking as opposed to polydipsic drinking. The question which arises next is whether this difference established for the biochemical (drive) systems extends to the mechanisms responsive to the palatability (incentive value) of the fluids employed.

One method of altering the palatability, or incentive value, of a fluid is to pair it with an illness-inducing agent such as lithium chloride. In recovered animals, this technique has been shown to produce a more profound and generalized reduction in the intake of that fluid than is produced by pairing of drinking with other noxious stimuli such as electric shock [5].

The present experiment was carried out to investigate the

[1]Requests for reprint should be forwarded to: J. Christopher Clarke, School of Psychology, University of New South Wales, P.O. Box 1, Kensington, N.S.W., Australia 2033.

sensitivity of polydipsic drinking to taste aversion procedures and to add to our knowledge of the incentive control of this form of drinking.

METHOD

Animals

The subjects were three experimentally naive female Wistar rats, 160–180 days old at the beginning of the experiment (approximately 300 grams). The rats were maintained at 80% (± 10 g) of their ad lib weight for the duration of the experiment. They were housed in individual cages with a normal daylight cycle and a temperature of 22° ± 2°C. A standard test chamber (48×48×48 cm) was employed. Food reinforcement was delivered by a 45 mg pellet dispenser. On one wall a drinkometer spout protruded into the chamber; on the same wall and 20 cm to the left was the food aperture. Licks at the drinkometer, pellet deliveries, and house lighting, were recorded or controlled by standard electromechanical equipment located in a room adjacent to the experimental area. All drinking bottles had stainless steel spouts fitted with ball bearings to reduce spillage. Separate bottles and spouts were used for the different fluids employed in the experiment. Additional food was supplied after each experimental session so as to maintain the appropriate body weights.

Procedure

Each rat's body weight was reduced to 80% of normal over a two-week period. During this time the rats were given ten 15-min adaptation periods in the test chamber. The experiment proper consisted of 6 stages: baseline; polydipsic condition (SIP–1); polydipsic condition and poisoning (SIP+LiCl); polydipsic condition (SIP–2); home cage poisoning (HC+LiCl); and the final polydipsic condition (SIP+3). The baseline condition consisted of ten daily 75-min sessions. During these baseline sessions the rats were given their daily food allowance and continuous access to a saccharin flavored solution (4% saccharin). Water was always freely available in the home cage. The next stage, SIP–1, was run for 15 sessions and was designed to establish polydipsic drinking. In these daily 75-min sessions food pellets were delivered once each minute and saccharin flavored water was continuously available. This schedule was in force for all succeeding stages of the experiment with the exception of stage HC+LiCl, home cage poisoning. The next stage, SIP+LiCl, was run for 6 days. On Days 1, 3 and 5 of this stage the rats were given an intraperitoneal injection of lithium chloride (2% of body weight, 0.15M) immediately after the conclusion of the 75-min polydipsic session. The rats were allowed to recover in their home cages on the days following the injections (2, 4 and 6). Thus, there were only 3 polydipsic sessions in this stage of the experiment. Next came stage SIP–2 which differed from the first polydipsic condition in that access to saccharin flavored water (Days 1, 3, 5 and 7) alternated with plain water sessions (Days 2, 4, 6 and 8). The next stage, HC+LiCl, was carried out in the home cage. It began with a 5-day period of adaptation to a daily 20-min drink period of plain water. On the sixth day the rats were given the saccharin flavored solution for the 20 min period. This was followed immediately by an intra-peritoneal injection of lithium chloride. (The solutions and injections were the same as those used earlier.) Two recovery days followed during which time the rats were put back on, and kept on, the ad lib water schedule. The last stage of the experiment, SIP–3, was run exactly as SIP–2 except that the drinkometer contained water for the first three days and saccharin for the last five.

RESULTS AND DISCUSSION

The fluid consumption data expressed as drinkometer licks are presented for each rat in Fig. 1. By the end of the first polydipsic stage, SIP–1, the consumption of saccharin flavored water had reached three to six times the average baseline consumption of the same solution for all three rats ("X" mark on Fig. 1). These figures are consistent with other published reports of schedule induced polydipsia [2,3].

When, in the next stage, SIP+LiCl, the rats were poisoned after each of the three sessions the polydipsic effect was abolished and drinking returned to baseline levels by the third of these sessions. While these data appear to indicate the sensitivity of polydipsic drinking to taste aversion procedures there are other explanations for the reduction which need to be considered. The sharp decline in drinking in the third and last session of SIP+LiCl may have resulted from an unintended taste aversion conditioned to the food pellets eaten in the experimental chamber. In other words, some or all of this decrease may have been due to the rats eating less of the food and, therefore, consuming less of the fluid. Support for this account comes from the food consumption patterns of Rats 1 and 3 in this session. They left uneaten the last 32 and the last 15 pellets, respectively, suggesting that the taste aversion procedures used in this study may have affected the food but not the saccharin-flavored water consumed prior to illness. There are, nevertheless, grounds for challenging this line of reasoning. First, Rat 2 ate every pellet in that session and yet showed the same decline as the other two rats. Second, there is the further reduction in intake of saccharin of all 3 rats during the next session, the first of SIP–2, where no food was left. These facts argue against the possibility that the reduction in saccharin was the indirect outcome of a conditioned food aversion. What is less clear, however, is the reason why all rats drank as little as they did during the following session (in SIP–2), when water was substituted for saccharin and where, once again, no food was left uneaten. (The last session of SIP+LiCl was the only one where food was left.) Taste aversion procedures, it will be recalled, affect the incentive value of the taste stimulus paired with illness and do not, in recovered animals, lead to a change in the drive or motivation to drink. Hence the very low levels of water consumption call for an explanation.

Two different interpretations of this problem can be outlined. According to the first, the unexpectedly low water consumption can be attributed to the novelty of the water in that context. Prior to Session 2 (of SIP–2), the drinkometer had always contained saccharin and the "surprise" at encountering water for the first time may have caused the rats to be tentative in their approach to and consumption of the water. The three poisoning episodes, it could be argued, would have accentuated still further the rat's natural caution in the face of novel or unexpected events [1]. A second and simpler interpretation assumes that the administration of lithium chloride immediately after the end of Sessions 1, 2 and 3 produced a conditioned taste aversion to the saccharin and, in addition, a generalized aversion to the background or contextual stimuli of the experimental chamber. If so, very little drinking would be expected regardless of the fluid in the drinkometer. While this remains a possibility the weight of

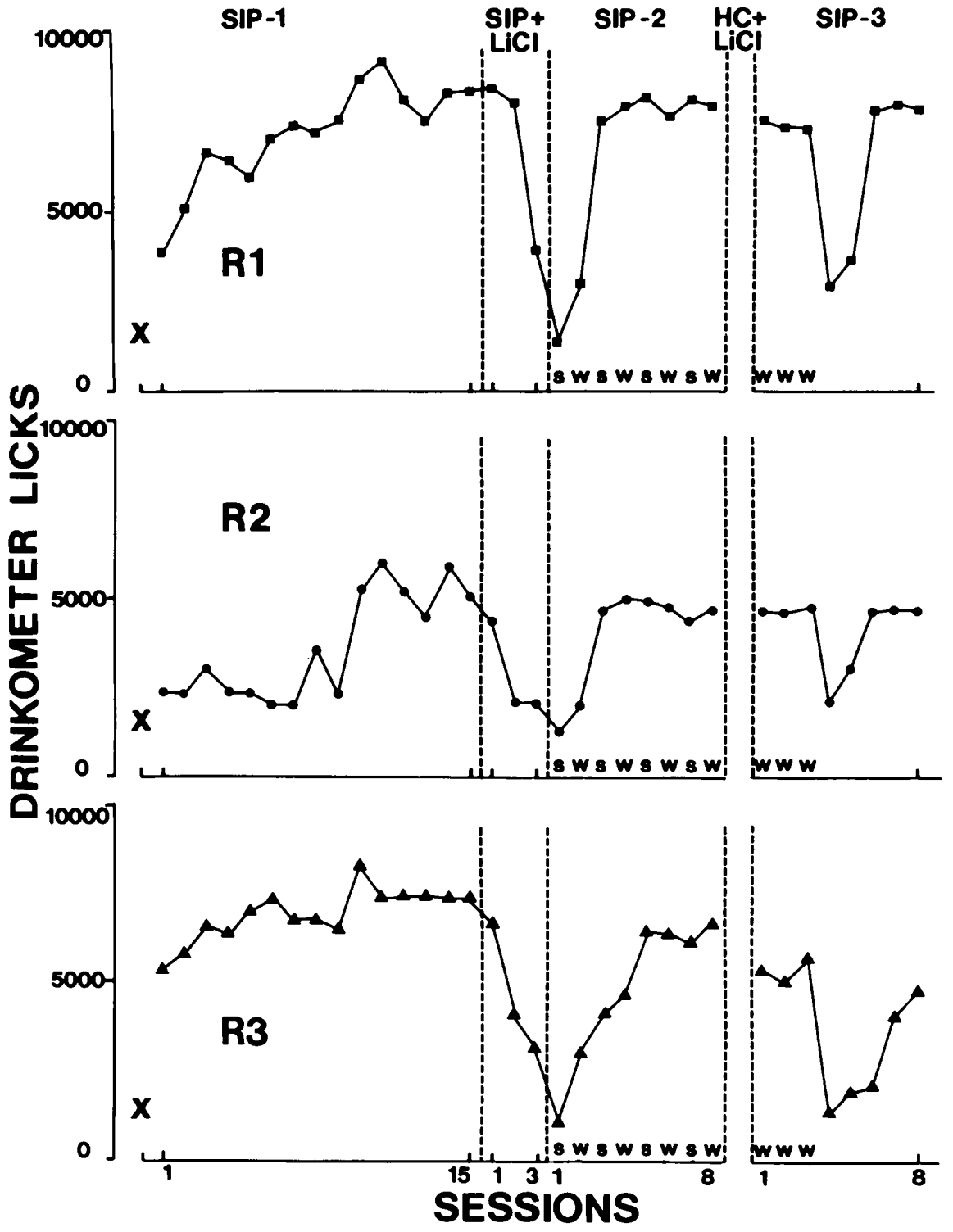

FIG. 1. Saccharin consumption, in licks, across the six stages of the experiment. (Abbreviations: X=average baseline consumption; SIP=schedule-induced polydipsia; LiCl=Lithium Chloride; HC=home cage; S=saccharin flavored water; W=tap water.) From the first session of SIP-1, except where indicated in the figure by W, saccharin flavored water was the fluid available in the experimental chamber.

the experimental evidence is decidedly against such an interpretation. It is a well established fact that in rats, illness based aversions condition very poorly, if at all, to non-taste stimuli [4]. The non-associative or novelty interpretation outlined earlier is a more plausible explanation of the low water consumption observed in the second session of SIP–2. If, in addition to this cautious reaction to water, it is also assumed that a taste aversion had indeed been conditioned to the saccharin then it is possible to account for the consumption patterns of all rats in SIP–2.

The last stages of this experiment were designed to provide more evidence for the effect of taste aversion procedures on polydipsic drinking. The rats had saccharin paired with lithium chloride in the home cage to ensure that if any conditioning of background cues to the illness did occur the contextual stimuli from the experimental chamber would not be implicated.

For the first three days of SIP–3 water was available in the drinkometer and all rats showed polydipsic levels of drinking comparable to those shown in the final sessions of SIP–2. These results indicate that no aversion had been conditioned to the water or the chamber itself. It was not until Day 4 when the drinkometer contained saccharin that the rats showed a marked drop in drinking. It is reasonable, therefore, to conclude that an aversion had been conditioned to the saccharin.

The results of this experiment show that taste aversion procedures which have been used so effectively to control normal drinking can also modify polydipsic drinking. It would thus appear that while the drive systems subserving these two kinds of drinking differ, the incentive control systems share common features. It is also worth noting, however, that the rapid recovery of schedule-induced polydipsia, in all rats, in stages SIP–2 and SIP–3, testifies to the powerful drive operations of the schedule conditions employed in this experiment [3]. By the end of the final sessions of the last stage, rats with free access to water in their home cages were, nevertheless, consuming in the experimental chamber large quantities of a solution which they did not need and which had been paired repeatedly with a powerful poison.

ACKNOWLEDGEMENTS

Preparation of this manuscript was facilitated by Australian Research Grants award (ARGC A76/15714). The authors are grateful to Ms. Terry Hall for her assistance with data collection.

REFERENCES

1. Barnett, S. A. *The Rat: A Study in Behaviour*. London: Methuen and Company, 1963.
2. Falk, J. L. Production of polydipsia in normal rats by an intermittent food schedule. *Science* **141**: 195–196, 1961.
3. Falk, J. L. A theoretical review: the nature and determinants of adjunctive behavior. *Physiol. Behav.* **6**: 577–588, 1971.
4. Garcia, J., J. C. Clarke and W. G. Hankins. Natural responses to scheduled rewards. In: *Perspectives in Ethology*, edited by P. P. G. Bateson and P. H. Klopfer. New York: Plenum, 1973.
5. Garcia, J., B. K. McGowan and K. F. Green. Biological constraints on conditioning. In: *Classical Conditioning II: Current Theory and Research*, edited by A. H. Black and W. F. Prokasy. New York: Appleton-Century-Crofts, 1972.
6. Rozin, P. and J. W. Kalat. Learning as a situation-specific adaptation. In: *Biological Boundaries of Learning*, edited by M. E. P. Seligman and J. L. Hager. New York: Appleton-Century-Crofts, 1972.
7. Singer, G., S. Armstrong and M. J. Wayner. Effects of norepinephrine applied to the lateral hypothalamus on schedule induced polydipsia. *Pharmac. Biochem. Behav.* **3**: 869–872, 1975.