The interaction of conditioned taste aversions and schedule-induced polydipsia: Effects of repeated conditioning trials

ANTHONY L. RILEY, RICHARD L. HYSON, and CORY S. BAKER
The American University, Washington, D.C. 20016

and

PAUL J. KULKOSKY
*Laboratory of Metabolism, National Institute on Alcohol Abuse and Alcoholism
Rockville, Maryland 20852*

In Experiment 1, rats poisoned following schedule-induced saccharin consumption showed a moderate reduction in the schedule-induced consumption of saccharin. With repeated poisoning, schedule-induced saccharin polydipsia was markedly reduced. Acquisition of conditioned aversion under the schedule-induced procedure was significantly slower than acquisition under water deprivation. In addition, recovery of consumption of the previously poisoned solution during extinction was more rapid under schedule-induced polydipsia. Experiment 2 revealed that schedule-induced polydipsia was less sensitive to suppression by conditioned aversions than a prandial drinking condition in which subjects were equally food deprived but were given a mass feeding instead of spaced pellet deliveries, suggesting that the relative insensitivity of schedule-induced polydipsia to conditioned taste aversions is not simply a function of different levels of food deprivation. This relative insensitivity is offered as a partial basis for the occurrence and maintenance of schedule-induced alcohol polydipsia.

Since Falk's (1961) original demonstration of schedule-induced water polydipsia, the schedule-induced intake of a wide range of solutions has been assessed, among them, acetone, alcohol, dextrose, quinine, saccharin, saline, and sucrose (Falk, 1964; Falk & Samson, 1975; Freed, Zec, & Mendelson, 1977; Hawkins, Schrot, Githens, & Everett, 1972; Samson & Falk, 1974).

That schedule-induced consumption of alcohol occurs, often to the point of intoxication (Falk & Samson, 1975; Hawkins et al., 1972), is surprising in that the rat avoids voluntary consumption of intoxicating and physical-dependence-producing amounts of alcohol under ad-lib conditions in the home cage (Falk & Samson, 1975; Kulkosky, 1979; Samson & Falk, 1974; Kulkosky, Zellner, Hyson, & Riley, Note 1). It has been suggested that this avoidance in the home cage may reflect an acquired aversion to the taste of alcohol as a result of the post-ingestional,

pharmacologically aversive properties of alcohol (Berman & Cannon, 1974; Deutsch & Eisner, 1977; Deutsch & Walton, 1977; Deutsch, Davis, & Cap, 1976; Deutsch, Walton, & Thiel, 1978; Eckardt, 1975).

That schedule-induced alcohol polydipsia occurs suggests that the aversive, postingestional effects of alcohol are not sufficient to suppress schedule-induced consumption of alcohol; that is, schedule-induced alcohol polydipsia is insensitive to conditioned taste aversions (Freed et al., 1977). In support and extension of this suggestion, Riley, Lotter, and Kulkosky (1979) demonstrated that rats poisoned following schedule-induced saccharin polydipsia show only a moderate suppression of schedule-induced saccharin intake. Further, recovery of saccharin consumption, that is, extinction of the aversion, was very rapid, with total extinction after only two nonpoisoned exposures to saccharin. This moderate suppression and rapid recovery of aversions in a schedule-induced design is in contrast to the markedly suppressed and long-lasting aversions seen under water deprivation in the home cage, suggesting that schedule-induced polydipsia in general is relatively insensitive to conditioned taste aversions (Clarke & Westbrook, 1978; Riley et al., 1979; Roll, Schaeffer, & Smith, 1969).

This relative insensitivity, which may further reflect

Requests for reprints should be sent to Anthony L. Riley, Department of Psychology, The American University, Washington, D.C. 20016, or to Paul J. Kulkosky, Edward W. Bourne Behavioral Research Lab, New York Hospital, Cornell Medical Center, Westchester Division, 21 Bloomingdale Road, White Plains, New York 10605.

a strong tendency to drink following food delivery, offers a basis for the occurrence of schedule-induced alcohol polydipsia. Although the taste of alcohol may be paired with the aversive aftereffects of alcohol consumption, the strong tendency to drink induced by intermittent food delivery overrides the conditioned aversion to the taste of alcohol.

EXPERIMENT 1

That a general insensitivity of schedule-induced polydipsia to conditioned taste aversions may underlie the occurrence of schedule-induced alcohol consumption is based on the fact that a taste paired with poison, for example, saccharin with LiCl, produces only a moderate and transient suppression of schedule-induced polydipsia (Clarke & Westbrook, 1978; Riley et al., 1979). However, schedule-induced alcohol polydipsia is maintained over repeated sessions (Falk & Samson, 1975). Over sessions, the taste of alcohol is repeatedly paired with the aversive aftereffects of alcohol. That alcohol polydipsia is maintained suggests that polydipsia is also insensitive to repeated taste aversion conditioning trials. To test this implication, the effects of repeated aversion conditioning trials on schedule-induced consumption were examined in the following experiment.

Method

Subjects

The subjects were 24 experimentally naive, female rats of Long-Evans descent (outbred, Charles River Crl: COBS (LE) BR), approximately 90 days of age at the beginning of the experiment. The subjects were maintained on a 12-h-light/12-h-dark cycle for the duration of the experiment.

Apparatus

The subjects were housed in individual wire-mesh cages. In the front of each cage were openings into which graduated Nalgene tubes were placed for presentations of water or .1% w/v sodium saccharin (Fisher purified) during conditioned aversion training. Six BRS/LVE experimental chambers were used during polydipsia training. A 3-cm hopper was located on the front wall 2 cm above the grid floor and 7 cm from the right side wall. A graduated drinking tube was located on the front wall 2 cm to the left of the food hopper. The spout of the tube protruded approximately 2 cm into the chamber and 3 cm above the grid floor.

Procedure

Schedule-induced polydipsia. Initially, 12 randomly selected rats were deprived of food and given ad-lib access to water. Following reduction to 85% body weight, each subject was removed from its home cage and placed in the experimental chamber for a daily 1-h polydipsia session. During this session, food was presented to the subject independently of its behavior on a variable-time (VT) 60-sec schedule. Water was continuously available throughout the session. Intakes were measured at the conclusion of the 1-h session. If necessary, supplemental feedings of Purina Rat Chow were given to subjects in the home cage 10 min after each daily session to maintain body weights at 85% of initial values. This procedure was continued for 30 consecutive days. On Day 31, a novel saccharin solution was presented in place of water during the daily free-food presentations. Differential treatment was then administered to two

groups of randomly selected subjects. Group SIP-P, $n = 6$, was given an intraperitoneal injection of 1.8 mEq/kg, .15 M LiCl 15 min following access to saccharin. Group SIP-W, $n = 6$, was given an ip injection of distilled water. On the following day, water was again made available during the 1-h session. This procedure of alternating conditioning trial and water recovery was continued until each subject had received five complete cycles.

On the day following the fifth water-recovery session, all subjects were given saccharin during the daily free-food presentations. During this extinction phase, however, no injection followed this access to saccharin for either group. On the following day, the subjects were given access to water during the 1-h session. This procedure of alternating extinction trial and water recovery was repeated until each subject had received nine complete extinction cycles.

Water-deprivation-induced drinking. Initially, 12 randomly selected subjects were deprived of water and given ad-lib access to food. On the next day, these subjects were given 20-min access to water in the home cage. This procedure was repeated daily for 30 consecutive days, at which point all subjects were approaching and drinking from the tube within 2 sec of its presentation. Differential treatment was then administered to two groups of randomly selected subjects. On Day 31, Group WD-P, $n = 6$, was presented with saccharin for 20 min followed 15 min later by an ip injection of 1.8 mEq/kg, .15 M LiCl. Group WD-W, $n = 6$, was given access to saccharin, followed 15 min later by an injection of distilled water. On the following day, all subjects were given 20-min access to water. This procedure of alternating conditioning trial and water recovery was continued until each subject had received five complete cycles.

On the day following the fifth water-recovery session, all subjects were given 20-min access to saccharin. During this extinction phase, however, no injection followed access to saccharin for either group. On the following day, the subjects were given 20-min access to water. This procedure of alternating extinction trial and water recovery was repeated until all subjects had received nine complete extinction cycles.

Results

All determinations of statistical significance were made at $p < .05$, two-tailed.

Schedule-Induced Polydipsia

On Day 1 of polydipsia training, each subject drank approximately 6 ml of water, which increased to approximately 20 ml on Day 30. On Day 31, all subjects showed a slight, but nonsignificant, increase in saccharin consumption above the amount of water consumed on Day 30. When water was reinstated on the following day, all subjects returned to their water polydipsia baseline.

Figure 1 presents the mean saccharin consumption for Groups SIP-P and SIP-W over the 14 treatment cycles (5 conditioning cycles followed by 9 extinction cycles). On the second exposure to saccharin (the first exposure following on-baseline aversion conditioning), Group SIP-P drank significantly less saccharin than Group SIP-W [$t(10) = 2.53$]. With repeated exposure to saccharin followed by poison, Group SIP-P further decreased consumption of saccharin until, following the fifth conditioning trial, it reached a level of only approximately 1 ml. On the other hand, Group SIP-W increased its consumption of saccharin over the same

Figure 1. Mean saccharin consumption (ml) for Groups SIP-P and SIP-W during conditioning (S-L) and extinction (S).

Figure 2. Mean saccharin consumption (ml) for Groups WD-P and WD-W during conditioning (S-L) and extinction (S).

treatment cycles. Consumption of saccharin increased in Group SIP-P over the nine extinction trials (Days 41-57). On the final extinction test, the difference between Groups SIP-P and SIP-W was small and statistically insignificant.

Water-Deprivation-Induced Drinking

On Day 1 of water adaptation, each subject drank approximately 8 ml of water, which increased to approximately 13 ml on Day 30. On Day 31, all subjects showed a small, but significant, decrease in saccharin consumption below the amount of water consumed on Day 30 [$t(11) = 2.55$]. When water was reinstated on the following day, all subjects returned to their water baseline.

Figure 2 presents the mean saccharin consumption for Groups WD-P and WD-W over the 14 treatment cycles (5 conditioning cycles followed by 9 extinction cycles). On the second exposure to saccharin (the first exposure following on-baseline aversion conditioning), Group WD-P drank significantly less saccharin than Group WD-W [$t(10) = 11.05$]. With repeated exposure to saccharin followed by poison, Group WD-P further decreased consumption of saccharin until, following the fifth conditioning trial, it reached a level less than .5 ml. On the other hand, Group WD-W increased its consumption of saccharin over the same treatment

cycles. Consumption of saccharin gradually increased in Group WD-P over the nine extinction trials. On the final extinction test, however, Group WD-P still consumed significantly less saccharin than Group WD-W [$t(10) = 6.32$].

Comparison of Schedule-Induced and Water-Deprivation-Induced Drinking

Because the specific baseline levels of consumption for the polydipsia and home-cage conditions were different, a direct comparison of the effects of conditioned taste aversions can not be made. Figure 3, however, presents a percentage shift from baseline transformation of the absolute data for individual groups that allows an indirect assessment of the effects of taste aversions on the two baselines.

While both Group SIP-P and Group WD-P decreased consumption of saccharin, the percent shift from baseline was significantly larger in Group WD-P, the home-cage condition [$t(22) = 3.59$]. With repeated conditioning trials, this difference in the degree of the aversion was no longer evident, as both groups showed almost complete avoidance of the saccharin solution; that is, a percent shift of 100. While Group SIP-P completely recovered saccharin consumption after nine extinction trials, Group WD-P showed only a partial recovery of consumption of saccharin, still avoiding saccharin consumption on the final extinction test. Both Group SIP-W and Group WD-W, the non-poisoned controls, maintained saccharin consumption over repeated exposures.

Discussion

It is clear from the data that schedule-induced polydipsia can be totally suppressed if schedule-induced consumption is repeatedly followed by poison. The acquisition of this suppression, however, was slower and the recovery of drinking was more rapid and complete in the schedule-induced drinking condition than under water deprivation. These differences suggest that, while schedule-induced consumption is

Figure 3. Percentage shift from baseline consumption of saccharin for subjects under schedule-induced polydipsia (Groups SIP-P and SIP-W) and water deprivation (Groups WD-P and WD-W) during conditioning (S-L) and extinction (S).

not totally insensitive to taste aversions, it is insensitive relative to water-deprivation-induced drinking (Clarke & Westbrook, 1978; Riley et al., 1979).

EXPERIMENT 2

While this relative insensitivity may be a function of the strong tendency to drink induced by the pellet delivery, it may simply reflect the degree of food deprivation under the schedule-induced drinking procedure. The production of schedule-induced polydipsia requires prior body weight reduction by food restriction, although water is available ad lib. No food restriction is necessary in water-deprivation-induced drinking, although food and water deprivation may naturally covary (see Bolles, 1967). Since treatments which increase hunger or food-motivated behaviors can attenuate conditioned aversions to specific foods (Gold & Proulx, 1972; Wise & Albin, 1973), it is possible that in Experiment 1 the food deprivation necessary for schedule-induced polydipsia resulted in a comparative reduction of the efficacy of taste aversion conditioning to saccharin, a solution whose consumption is increased by food deprivation (Smith & Duffy, 1957; Strouthes, 1973).

In support of this, Bond and Corfield-Sumner (1978) have shown *equal* suppression of saccharin consumption by conditioned taste aversions if hungry male rats drinking under schedule-induction are compared to *water-deprived* rats maintained under equal conditions of food deprivation but mass fed at the time of fluid access. A combined water and food-deprivation control, however, does not allow for the analysis of the contribution of hunger alone as a mitigating factor in the conditioning of aversions to saccharin under schedule-induction.

In the following experiment, the effects of taste aversions on saccharin consumption by food-deprived rats receiving either spaced feedings or a mass-fed meal are compared. Thus, schedule-induced polydipsia is contrasted with drinking induced by a single meal presentation, that is, prandial drinking. This comparison allows for the isolation of the motivating properties of intermittent food delivery alone in the interaction of schedule-induced drinking with conditioned taste aversions.

Method

Subjects

The subjects were 12 male and 12 female experimentally naive rats of the same age and strain as in Experiment 1. The rats were maintained on a 12-h-light/12-h-dark cycle for the duration of the experiment.

Apparatus

The subjects were housed in individual wire-mesh cages. All experimental conditions were run in the same six BRS/LVE chambers described in Experiment 1.

Procedure

Male and female rats were randomly assigned to either the schedule-induced polydipsia group (Group SIP, six males and six females) or the prandial drinking group (Group PD, six males and six females). Initially, all subjects were deprived of food and given ad-lib access to water. Following reduction to 85% body weight, each subject was removed from its home cage and placed in the experimental chamber for a daily 1-h feeding session. For Group SIP, a single 45-mg Noyes pellet was delivered to each subject on a variable-time (VT) 60-sec schedule until each subject had received a total of 60 food pellets. For Group PD, 30 45-mg Noyes pellets were placed simultaneously in the food hopper for each subject at the beginning of each session. Five minutes later, 30 additional pellets were presented simultaneously, such that each subject received a total of 60 food pellets in two massed meals. Water was continuously available to all subjects during both the spaced and massed feedings. Intakes were measured at the conclusion of the 1-h session. If necessary, supplemental feedings of Purina Rat Chow were given to subjects in the home cage 10 min after each daily session to maintain body weights at 85% of initial values. This procedure of daily 1-h sessions was continued for 20 consecutive days.

On Day 21, a novel .1% sodium saccharin solution replaced water during the daily free-food presentations. Fifteen minutes following this session, all subjects were given an ip injection of 1.8 mEq/kg, .15 M LiCl. On the following day (Day 22), water was again available during a 1-h water-recovery session. On Day 23, saccharin was again made available during the 1-h experimental session. As above, all subjects were injected with LiCl following this session. On the following day (Day 24), water was made available during a 1-h water-recovery session.

On the day following the last water-recovery session (Day 25), all subjects were given saccharin during the daily free-food session. On this extinction session, however, no injections followed the access to saccharin. On the following day, water was made available during the 1-h session. This procedure of alternating extinction trial and water recovery was repeated until all subjects had received six complete extinction cycles.

Results

All determinants of statistical significance were made at $p < .05$, two-tailed.

On Day 1 of water adaptation, subjects in Group SIP drank approximately 7 ml of water, which increased to approximately 23 ml on Day 20. Subjects in Group PD drank approximately 4.3 ml on Day 1, decreasing slightly to approximately 2.8 ml on Day 20. On Day 21, subjects in both groups showed a slight, but significant, increase in saccharin consumption above the amount of water consumed on Day 20 [$t(11) = 5.39$ and $t(11) = 2.96$ for Groups SIP and PD, respectively]. When water was reinstated on the following day, all subjects returned to their respective water baseline.

Figure 4 presents the mean saccharin consumption for Groups SIP and PD over the eight treatment cycles (two conditioning cycles followed by six extinction cycles). On the second exposure to saccharin (the first exposure following on-baseline aversion conditioning), both Group SIP and Group PD decreased their consumption of saccharin [$t(11) = 4.77$ and $t(11) = 4.16$ for Groups SIP and PD, respectively]. Following the second saccharin-poison pairing, both

Figure 4. Mean saccharin consumption (ml) for Groups SIP and PD during conditioning (S-L) and extinction (S).

Figure 5. Percent shift from baseline consumption of saccharin for Groups SIP and PD during conditioning (S-L) and extinction (S).

groups further decreased saccharin consumption, reaching a level of approximately .5 and 5.0 ml for Groups PD and SIP, respectively. Consumption of saccharin increased in Group SIP over the six extinction cycles, reaching the level of its prepoisoned saccharin baseline on the final extinction test. Group PD continued to avoid saccharin over the six extinction cycles, drinking less than 1 ml on the final extinction test. While there were no sex differences within groups during acquisition, within SIP, females extinguished significantly faster than males [$F(1,19) = 4.57$] (cf. Sengstake, Chambers, & Thrower, 1978).

As in Experiment 1, the specific baseline levels of consumption were different for Groups SIP and PD. Figure 5 presents a similar percentage shift from baseline transformation.

While both Groups SIP and PD decreased consumption of saccharin following poisoning, the percent shift from baseline was significantly larger in Group PD after both the first [$t(22) = 3.59$] and the second [$t(22) = 2.16$] conditioning trials. Although Group SIP fully recovered to baseline saccharin consumption over repeated extinction sessions, Group PD maintained its avoidance of saccharin during this period. These differing rates of extinction were highly significant [$F(1,119) = 214.5$].

Discussion

The data clearly show that both schedule-induced polydipsia and prandial drinking can be greatly disrupted by the presentation of a flavor paired with poison. This finding is consistent with previous demonstrations of the suppression of schedule-induced polydipsia by concomitant taste aversion conditioning (Bond & Corfield-Sumner, 1978; Clarke & Westbrook, 1978; Corfield-Sumner & Bond, 1979; Riley et al., 1979; Roll et al., 1969). However, when schedule-induced polydipsia and prandial drinking are compared on the basis of proportional suppression from initial consumption, schedule-induced polydipsia appears less disrupted after either one or two conditioning trials and recovers faster during extinction. These differences in the effects of conditioned taste aversions on schedule-induced polydipsia and prandial drinking closely resemble the differences between schedule-induced and water-deprivation-induced drinking (see Experiment 1), suggesting that the relative insensitivity of schedule-induced polydipsia to food aversions is not simply a function of the interaction between food deprivation and saccharin consumption (see also Kulkosky, Crutchfield, & Riley, Note 2, and Riley, Hyson, & Kulkosky, Note 3).

GENERAL DISCUSSION

It is clear from the data that schedule-induced polydipsia can be totally suppressed by repeated taste aversion conditioning trials. It is also clear, however, that the suppression was acquired significantly more slowly and recovered faster during extinction in the schedule-induced polydipsia subjects than in subjects maintained under water deprivation (Experiment 1) or under food deprivation but without receiving spaced meals, that is, the prandial subjects (Experiment 2). This difference in the rate of acquisition of the aversion and recovery of the aversion suggests a relative insensitivity in the schedule-induced polydipsia design. As described earlier, this insensitivity may reflect an increased tendency to drink following pellet delivery under the schedule-induced polydipsia procedure, a tendency that can override the display of a conditioned taste aversion to a specific solution (see also Grote & Brown, 1973; Riley & Lovely, 1978).

There is, however, at least one additional similarity

between Experiments 1 and 2 other than the spaced feedings in the schedule-induced procedures that could account for the apparent relative insensitivity within each study. In the present experiments, the level of schedule-induced saccharin consumption on the first conditioning trial was approximately 25 ml, whereas animals consumed only approximately 15 and 10 ml of saccharin under the water-deprivation and prandial drinking conditions. It is possible that the different levels of saccharin consumption on the conditioning trial resulted in different degrees of conditioning, that is, the relative insensitivity reflects a learning deficit, and not the aforementioned performance decrement. While there is evidence that aversions are weak if animals consume less than 3-5 ml during conditioning (Barker, 1976; Bond & Digiusto, 1975; Bond & Harland, 1975; Braveman & Crane, 1977), it is unlikely that this constraint affected any comparison since all conditioning groups in the present experiments consumed more than this minimum amount during the first conditioning trial. Recently, however, Braveman and Crane (1977) reported that as the amount consumed exceeds approximately 5 ml during conditioning, conditioned taste aversions become weaker. This report has been, in part, confirmed by Deutsch (1978), who also demonstrated that aversions were inversely related to the amount consumed during conditioning once the amount consumed exceeded 5 ml. This relationship between aversions and amount consumed was evident, however, only when the poison was delayed longer than 1 h following consumption. With shorter delays, for example, 15 min, there was no weakening of the aversion as the amount consumed during conditioning increased. (Note that the delay in the present series of experiments did not exceed 15 min). While each of these studies suggests that some of the insensitivity seen in the schedule-induced polydipsia subjects may reflect weaker conditioning due to the large intakes during conditioning, neither Braveman and Crane (1977) nor Deutsch (1978) examined the effects of the amount consumed when intakes exceeded 10 ml. In both Experiments 1 and 2, all subjects approximated or exceeded this level. Given the apparent nonlinearity in the relationship of amount consumed and subsequent aversion, it is unclear whether the aforementioned argument applies when the amounts consumed during conditioning approach 20-30 ml. A recent report by Archer and Sjöden (1979) reexamined this relation between the amount consumed and conditioned taste aversions, including intakes approximating the levels in the present studies. From their analysis, there was no evidence that the degree of the aversion was in any way dependent upon the amount consumed during conditioning; that is, aversions were equally strong, independently of amount consumed. A similar position had been noted earlier by Kalat (1976), although the range of intakes was substantially smaller. The data to date are clearly

equivocal, and, as such, the insensitivity of schedule-induced polydipsia to taste aversions may in part be a function of the amount consumed during conditioning.

Independently of the mechanism underlying the relative insensitivity of schedule-induced polydipsia to taste aversions, that is, the tendency to drink following pellet delivery or weak conditioning due to large amounts consumed during conditioning, it should be noted that the insensitivity is only relative. Acquisition of total suppression is simply delayed. With repeated trials, schedule-induced polydipsia was reduced to approximately 0 ml. That repeated conditioning trials can effectively attenuate schedule-induced drinking precludes the argument that schedule-induced alcohol consumption is *totally* a function of the relative insensitivity of polydipsia to taste aversions (Riley et al., 1979). It was described earlier that schedule-induced alcohol consumption occurs even though the taste of alcohol is paired with the aversive aftereffects of alcohol, suggesting that schedule-induced polydipsia was insensitive to conditioned taste aversions. That schedule-induced alcohol polydipsia is maintained despite repeated pairings of the taste of alcohol with its aversive aftereffects suggests further that schedule-induced polydipsia is insensitive to repeated conditioned aversion trials. As is evident from the present experiments, such a procedure can suppress schedule-induced polydipsia. Thus, other variables unique to the procedure of schedule-induced alcohol polydipsia, for example, the taste or calories of alcohol, the aversive effects of alcohol, dependence, etc. (Falk & Tang, 1977), must be operating in the maintenance of schedule-induced alcohol polydipsia. The relative insensitivity of schedule-induced fluid consumption to taste aversion conditioning may be only a single factor that combines with these specific variables to produce and maintain schedule-induced alcohol polydipsia.

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