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Schedule induced polydipsia: control by taste aversion learning.

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SCHEDULE INDUCED POLYDIPSIA:

CONTROL BY TASTE AVERSION LEARNING

A Thesis Presented

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Abstract:

Conditioned taste aversions, acquired in the home cage, can control schedule induced polydipsia. Rats can acquire aversions to tastes ingested in schedule induced polydipsia sessions. Aversions acquired to tastes ingested in schedule induced polydisia sessions can come to control later polydipsia. Taste aversions appear to control the magnitude of schedule induced polydipsia more than its patterning. These data contradict the previous finding that polydipsia is very difficult to control through taste aversion learning. They also suggest that the notion that schedule induced polydipsia is insensitive to punishment requires serious reconsideration. Schedule Induced Polydipsia is considered as a tool for studying taste aversion learning.

In most environmental contexts, a water sated rat will drink 2 ml of water for every gram of food it eats (26); in the absence of food, drinking behavior is negligible (3). However, when small quantities of food are intermittently delivered to a hungry rat, it will no longer balance its food and water intake; instead, it increases sharply its water consumption, drinking 5, 10 or more ml of water for every gram of food (3). This phenomenon, schedule induced polydipsia (SIP) is not limited to the rat; it has also been demonstrated in a number of other species (16,21,24).

Difficulty in controlling SIP through standard operant techniques has been reported. Falk has consequently suggested that SIP might be insensitive to punishment (7). Roll, Schaeffer and Smith (18) exposed schedule induced water polydipsia (water-SIP) experienced rats to a large dose (100 R Co^{60}) of ionizing radiation immediately following the rats' first saccharin-SIP session. A single pairing of saccharin with a 60 R radiation exposure is usually found sufficient, in subsequent tests, to wipe out virtually all saccharin consumption in 48 hour water deprived animals (12). In the SIP session following their single pairing of saccharin with radiation induced malaise, Roll, et al detected no change in saccharin-SIP. Later, they irradiated these same animals following another saccharin-SIP session, this time using a 200 R Co^{60} exposure. And although a small transient decrease

was noted after this second pairing, the rats still remained fully polydipsic. These results support Falk's suggestion that SIP might be insensitive to punishment procedures.

Roll, et al suggested that the insensitivity of SIP to taste aversions must be due to some characteristic of SIP. If so, the insensitivity may be due to either (a) the inability of taste aversions to control SIP, (b) the inability of rats to form aversions to SIP ingested tastes, or (c) both of these factors. These three alternatives are considered in Experiments One, Two and Three respectively.

E X P E R I M E N T O N E

Subjects: Three individually housed, male albino rats (400-500 gram free feeding weight) were used. Their previous experimental history is discussed below.

Equipment: SIP sessions were conducted in Grason-Stadler rat operant chambers installed in sound attenuating aluminum picnic boxes. One wall of the chamber contained a Lehigh Valley Electronics retractable bar assembly (left), a Grason-Stadler rat food cup (right) and a 9.5 mm hole (center). A standard drinking tube (2.55mm orifice) was mounted directly behind the center hole, 6.4mm from the rear plane of the wall. Two incandescent lamps were located directly above the response lever and food cup respectively; these lamps were illuminated only before session start and after session termination (bar retracted). A third lamp was mounted outside the operant chamber but within the sound attenuating box. It was illuminated only during the experimental session (bar extended). White noise and ventilation were continuously provided. Events were programmed and data were recorded by standard electromechanical apparatus located in an adjacent room.

Procedure: With respect to prior experimental history, subsequent to shaping barpressing up to a Fixed Interval (FI) 150 second schedule (10) for 45 mg standard formula Noyes pellets, the rats were run in 4 twelve hour barpressing sessions (70-72% free feeding weight) with a 1% w/w saccharin

solution (SACC) available to drink. Under these conditions, all 3 subjects emitted polydipsic behavior, consuming SACC at rates in excess of 20ml/hr. Similarly deprived animals fed the same amount of food at the start of a 12 hour period of access to SACC consumed SACC at rates of less than 5 ml/hr. Cumulative records of licking and barpressing for the first and third subjects' fourth SIP session are presented in Figure 1 (upper half). After a hiatus of one month, the 3 rats were run for 30 minutes in one final SACC-SIP session to insure that they would emit characteristic barpressing and SIP in the current experiment. Following this 30 minute session, the rats were placed on a 23.5 hour fluid deprivation schedule. After 3 days of adaptation to this schedule, the subjects were given a coffee solution (COF: 0.75% Sanka Decaffinated Freeze Dried Coffee, w/w) in place of the usual tap water. After 30 minutes access to COF, this novel tasting solution was removed and the rats were injected intraperitoneally (IP) with 8ml/kg of LiCl solution (0.55M; 4.4mM Eq/kg). Henceforth, the rats were given free access to tap water. Pairing a 4.4mM Eq/kg dose of LiCl with a novel taste is usually sufficient to suppress virtually all drinking of the flavored solution in a subsequent one bottle test (12). COF was used because it is a relatively neutral taste (13). After one safe exposure to COF, rats consume virtually the same amount of COF ($\bar{X}$ = 25.15; σ = 3.25; n = 24) as tap water ($\bar{X}$ = 25.10; σ = 2.76; n = 24) (1). Several days later, the rats were run in one 7.9 hour

COF-SIP session (FI 150").

Results: Cumulative records of licking and barpressing have been reconstructed for two of the three subjects. A comparison of these records (Figure 1, lower half) with records from an earlier SACC-SIP session (Figure 1, upper half) demonstrates that a learned taste aversion may exert substantial control over SIP. The effects of the taste aversion can be examined with respect to both the patterning and magnitude of SIP. That drinking occurs after every pellet is a characteristic of developed SIP. This feature is clear in the upper portions of Figure 1, where 385 of the 386 reinforcers are followed by drinking.

In the current experiment, despite their massive aversion to COF, both subjects initially emitted typical SIP patterns of drinking. Since the magnitude of licking was low during at least the early segments of the post-aversion session, a heavy line (broken) has been drawn over each of the 2 cumulative records in the bottom portion of Figure 1. The line's presence indicates that one or more licks followed the delivery of the pellet, i.e. the line signifies SIP patterning. Subject 1 consistently drank COF after each of the first 8 pellets, subject 3, the first 11. For the first half hour of the session, both rats continued to drink after every pellet. SIP patterning broke down over the next 2 and 3.5 hours for subjects 1 and 3 respectively, that is, after initially emitting SIP patterning,

the rats stopped being polydipsic. Control over SIP patterning developed slowly, and for subject 1, this control was incomplete. Subject 1 still intermittently drank after a pellet. Further, control over both subjects' SIP was shortlived. Within 2.75 hours of session start, subject 1's SIP patterning permanently reappeared. And while for subject 3, control of patterning was more complete, SIP patterning appears reinstated over the last third of the session. With respect to patterning, taste aversions can affect SIP, but the control they exert is slow to evidence itself. With the parameters used, they do not exert effective control over one entire 7.9 hour session. The slow development and temporary nature of control over SIP patterning cannot be ascribed to small US magnitude since the doses of LiCl used were as large as any previously reported in the literature.

With respect to SIP magnitude, suppression of SIP in subject 1 was virtually complete during the first 2.5 hours of the session, even during those intervals when SIP patterning was evident. And although subject 3 emitted a large number of licks following each of the first 8 pellets, for the next 3 hours virtually no licking occurred.

These results cannot be ascribed to the transition from SACC-SIP to COF-SIP for 3 reasons. First, rats treat COF as a neutral, rather than innately aversive taste. Second, Segal reported that a transition from a preferred taste to a neutral taste does not significantly effect SIP, even during the first

hour with the neutral solution (22). Third, even the transition to an aversive solution (e.g. quinine) does not result in drastic decreases in SIP and whatever small disruption results from the introduction of quinine disappears before the second hour of quinine-SIP (22). Experiment Three, as a systematic replication of Experiment One, provides additional support for this position. A massive taste aversion acquired in the subjects' home cages appears capable of controlling SIP's patterning and magnitude.

E X P E R I M E N T T W O

Roll's failure to control SIP through a taste aversion may have been due to the inability of the rat to acquire a massive aversion to a taste ingested in a SIP session. To test this possibility, three more male albino rats were employed. Except as noted, procedures and apparatus were the same as those of Experiment One. These subjects had barpresing and SACC-SIP experience similar to that of the Experiment One subjects, i.e. barpressing on an FI 150" schedule for 45 mg pellets in 5 twelve hour SACC-SIP sessions followed by one 30 minute SACC-SIP refresher session. After the refresher session, these subjects were placed in a 30 minute COF-SIP session (COF was novel to these rats). Immediately upon session termination, they were injected (IP) with a 4.4 mM Eq/kg dose of LiCl. After 3 days of adaptation to a 23.5 hour water deprivation schedule, they were given COF in place of the usual tap water. COF intake over a 30 minute interval was recorded.

Results: During their COF-SIP session, all 3 rats engaged in polydipsic behavior, drinking sizeable quantities of COF after each pellet. In contrast, following a home cage COF-LiCl pairing, 2 of the 3 animals in Experiment One drank virtually no COF during the first half hour of their COF-SIP session. In their subsequent home cage test, the Experiment Two rats

consumed 4.0, 1.5 and 1.0 ml of COF during the 30 minute interval. These intake figures conform with other observations of massive taste aversions following the pairing of a taste with a 4.4 mM Eq/kg dose of LiCl (1,15). In the absence of previous poisoning and upon second exposure to COF, rats will normally consume 25.15 ml of COF in an identical test (1). The data support the conclusion that rats can acquire massive aversions to tastes ingested in a SIP session.

E X P E R I M E N T T H R E E

This experiment examined the ability of a taste aversion, acquired in a SIP session, to control later SIP. Experiment One demonstrated that a conditioned taste aversion can come to control SIP (temporarily). Experiment Two demonstrated that rats can form aversions to tastes ingested in a SIP session. These two findings together would predict that rats should be able to form, during SIP sessions, taste aversions which could control subsequent SIP. This prediction is not borne out by Roll's data. There are two major differences between Roll's procedures and the current ones. One, Roll used a more preferred substance, saccharin solution (0.1% w/v) while the current experiments used a neutral taste (COF). Two, Roll used radiation poisoning to induce illness; the current experiments used LiCl injections. Differences in the effect of taste-illness pairings might be due to either taste preference effects or greater aversive control by LiCl induced malaise. To resolve these questions, three more rats (naive, male albino) were trained to barpress for 45 mg pellets on an FI 150" schedule. Immediately prior to each SACC-SIP session, the rats were allowed to drink SACC, in the experimental chamber, for 30 minutes. SACC-SIP sessions, 3 hours long, were run once a week for 6 weeks. Immediately after each SACC-SIP session, the rats were injected (IP) with a mild emetic, a 7 mg/kg dose of Apomorphine HCl (APO). APO was used to insure that

the results of Experiment One were due to neither aversion magnitude nor peculiar to Lithium poisoning. Unlike the subjects in the first two experiments, these rats had no experience with SIP prior to their first SACC-SIP+APO session.

Two types of data were collected: licking and fluid consumption. In each session and for each subject, licks were sorted into 5 categories: SACC intake during a pre-session test (30 minutes), and SACC intake during each 45 minute quarter of the 3 hour session. Fluid consumption figures reflect total SACC intake over both the pre-session test and the 3 hour session. In Figure 2, total fluid intake, total licks (pre-session and SIP) and SIP session licks are presented for each subject and each session. In Figure 3, the lick data are subdivided into 5 categories: pre-session SACC intake and SACC intake during each quarter of the session. Each of the 5 curves for each subject charts licking from one of these 5 categories (for example, pre-session intake) across all 6 sessions.

As is apparent from Figure 2, overall licks, SIP session licks and overall fluid consumption decreased from session 1 to session 2, i.e. a single APO induced illness following one SACC-SIP session resulted in decreased SACC intake, decreased SIP during the following SACC-SIP session. This should be even more apparent in Figure 3, where subject 1 and subject 2 showed decreased SACC-SIP intake across all 4 quarters of their second SACC-SIP session.

These data confirm and extend the findings from Experiment One. They suggest that control over SIP by a single taste-illness pairing is not dependent on the particular taste used (COF vs. SACC), the rat's preference for that taste (SACC is preferred; COF is treated as neutral), the magnitude of the illness (APO results in a much smaller aversion) the nature of the emetic (APO vs. LiCl) or the environmental context in which the aversion was acquired (home cage aversion training vs. SIP-illness pairing). The results also suggest that control over SIP is to some extent independent of SIP experience (48 hours prior experience with SIP vs. SIP naive).

Also apparent in Figure 2 is a further reduction in SACC-SIP with repeated SACC-SIP+APO pairings. Calculating the regression lines for SACC-SIP across sessions, their slope is negative (decreased SACC-SIP across sessions) for every quarter of every SIP session for every subject. The probability of this occuring by chance is 1.5×10^{-5}. While a single pairing of a mild emetic with a taste ingested in a SACC-SIP session is sufficient to reduce the magnitude of subsequent SIP, multiple pairings further reduce SIP magnitude.

A number of additional points should be considered. First, these results are not due to illness during the experimental sessions since sessions were spaced one week apart. A large number of investigators have reported that no cumulative illness effects exist when sessions are spaced 3 days apart (17). Second, these results are not due to any major changes in the

interpellet interval since no systematic changes in either barpressing or pellet deliveries were observed. Third, in the absence of poisoning or cumulative illness effects, SACC-SIP does not decrease over sessions (7,8). Existing data indicate that there are no successive across session decrements in SIP whether the fluid is neutral, naturally aversive or preferred. Fourth, these results do not represent control over only non-polydipsic drinking; the drinking that occured during the first session was clearly polydipsic. Subjects 1, 2 and 3 consumed 49, 28 and 25 ml of SACC per gram of food (session one) vs. 13 and 8 ml/g reported for Roll's rats. Roll's rats were polydipsic and the current animals even more so. The differences in SACC/food ratios reflect the fact that the 2.5 minute mean interpellet interval used herein generates much more SIP than a 1 minute interval (6). Fifth, the difference between the effects of one SACC-SIP+APO pairing in this experiment versus Roll's treatment cannot be attributed to the rat's greater preference for saccharin solution versus a neutral COF solution. In this experiment, a SACC solution more preferred than Roll's was employed and a consistent decrease in SACC-SIP was observed. Sixth, the difference following one pairing cannot be attribuuted to the use of a more aversive illness, since a 7mg/kg dose of APO usually results in a much smaller aversion than 60 R Co^{60}. Nor can it be argued that somehow the APO led to abnormally exaggerated illness effects since 3 non-polydipsic control animals, injected weekly after 30 minutes' access to SACC, showed only relative, and not absolute, suppression of SACC

intake; a suppression similar in form to parametric APO data from Garcia's lab (11). A 7 mg/kg dose of APO does not lead to a massive aversion (15). Apparently, just as taste aversions can control thirst induced drinking, SIP can also be controlled.

A final aspect of the data deserves consideration. In Experiment One, it was noted that both SIP patterning and SIP magnitude were disturbed by a massive taste aversion, but that SIP patterning was still present when ingestion magnitude was virtually zero. This suggests that SIP drinking patterns may be more resistant to the effects of a taste aversion than SIP magnitude. The results from Experiment Three, obtained using a smaller magnitude illness, reinforce this notion. Although there was in this experiment an obvious decrease in SACC-SIP magnitude across sessions (Figures 2 & 3), there was no discernable effect of the aversion on SIP patterning. Subject 2 is the best example of this lack of an effect. While its SIP magnitude dropped to one third its previous value, subject 2 emitted a drink after every pellet in every quarter of each of the first five SACC-SIP+APO sessions and after every pellet in the first three quarters of the sixth session. SIP magnitude appears more easily controlled by taste aversions than is SIP patterning.

Parenthetically, this paper has considered only Roll's first and second pairings of saccharin-SIP with Co^{60} induced malaise. Roll also examined the effects of sequential saccharin

SIP+Co^{60} pairings. However, these 50 R exposures were carried out on a daily basis. There are ample data to suggest that whatever SIP decrements were observed could have been due to cumulative radiation poisoning rather than taste aversion learning (17). Roll argued that if cumulative radiation poisoning had occured, then the rats should stop eating as well as drinking. Reporting that by the end of each SIP session, his rats had consumed all the delivered food pellets, Roll dismissed cumulative radiation poisoning as the variable controlling SIP. This decision was unjustified since Falk had already shown that the level of ongoing illness needed to suppress SIP was lower than the level of ongoing illness needed to suppress eating (5).

D I S C U S S I O N

Despite previous difficulty in controlling SIP through taste aversion learning, the current experiments demonstrate that SIP can be controlled in such a fashion. SIP can be suppressed by an aversion acquired in the home cage. Rats can acquire aversions to tastes ingested in a SIP session and such an aversion can come to control SIP. Data have recently been obtained in another very different punishment paradigm (lick contingent shock) which also demonstrate control over SIP by punishment (4). The conclusion that SIP may be insensitive to punishment appears premature.

That taste aversions can control SIP in a fashion not unlike control over other ingestive behaviors leads to a number of interesting corollaries. First, Stricker and Adair have reported that SIP has a number of physiological consequences, notably stomach distension and overhydration (25). Falk has suspected that SIP leads to dilutional hyponatremia and water intoxication (8). If SIP is physiologically aversive, why isn't it controlled by these consequences? In a series of pilot experiments, the taste of SACC, ingested in 12 hour long SIP sessions, has been paired with the physiological consequences of SIP. Thirty minute, one bottle preference tests indicated that a small aversion to SACC may develop, but no effect on subsequent SIP has yet been detected. Further research on control of SIP by its natural, aversive consequences is required.

Second, a number of investigators have been interested in SIP as an animal model of human alcoholism (14,22). Clearly, SIP provides a useful tool for studying the physiological effects of chronic alcoholism. One of the problems encountered in using SIP in a functional behavioral analysis of human alcoholism has been the apparent inability to control SIP with either electric shock (23) or taste aversion learning (8). Chronic human alcohol consumption can be controlled, albeit temporarily, with either procedure. Taken together, the work or Bond, et al (4) and the current experiments indicate that SIP is sensitive to these same punishment procedures.

Third, these results suggest alternate methods for analysis of taste aversion learning. Grote and Brown (13) report increasing water deprivation levels increases the rate at which an aversion to a fluid based taste extinguishes. Fluid deprivation, however, has a number of effects including hypovolemia, hyperosmolarity and an increased frequency/probability of drinking. If it is assumed that the extinction of a taste aversion follows the same general rules as the extinction of any other passive avoidance task, Grote and Brown's results may be interpreted in terms of increased frequency/probability of drinking. However, several researchers would question the assumption that taste aversion learning and extinction follow rules discovered in other learning paradigms. Rozin (19) has argued that taste aversion learning is an adaptive specialization and, congruent with descriptions

of rat behavior in the wild (2,20), a primary dimension controlling rate of extinction may be biologically important stimuli for thirst (hyperosmolarity, hypovolemia) as opposed to the probability of a drink. Using ordinary deprivation techniques, physiological stimuli for thirst cannot be separated easily from the behavioral aspects of thirst. However, using SIP, such questions can now be approached.

As a final methodological implication for the current results, much emphasis has been placed on understanding the acquisition of taste aversions over long taste-illness intervals, but little attention has been paid to the question of the minimal time course of extinction of taste aversions. Extinction occurs slowly when both a continuously safe and a previously poisoned substance are available, but the rate of extinction increases in the absence of a safe substance. The recovery of SIP (Figure 1) in Experiment One exemplifies the latter phenomenon. What is not yet understood are the specific learning variables underlying the extinction of a conditioned taste averion. While Garcia, Ervin and Koelling's data provide evidence of extinction of an illness induced aversion to saccharin after one safe thirty minute exposure to saccharin, the delay between the 1 safe exposure and the subsequent increase in intake was 72 hours (11). Rozin has speculated that in adapting to a new food, the rat learns the taste is safe if ingestion is not followed by aversive post-ingestional cues for a number of hours, i.e.

the rat's natural aversion to novelty (neophobia) extinguishes over time. Does the extinction of an acquired aversion also follow a similar temporal pattern? Using SIP to force the rat to come into contact with a taste previously associated with illness, this aspect of taste aversion learning may now be examined.

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