CONDITIONED SUPPRESSION OF BAR-PRESSING BEHAVIOR BY STIMULI ASSOCIATED WITH DRUGS¹

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Ten naive male albino rats were trained to press a bar under a variable-interval 30-sec schedule with water as the reinforcer in two experiments. This behavior was disrupted by chlorpromazine in Experiment I (two rats) and by lysergic acid diethylamide (LSD) in both Experiment I (two rats) and Experiment II (six rats). The administration of the drug was paired with an originally neutral white light. After several pairings with either drug, the light also depressed behavior. When the light was no longer paired with drug, the depression effect extinguished much faster than is usually observed in conditioned suppression studies.

Wikler (1965) offered a conditioning hypothesis to explain the often-observed "relapse effect" of narcotic addicts who are no longer physically dependent on the drug. Many environmental stimuli become associated with the lack of the drug so that when an addict returns to his home environment after being removed for incarceration or detoxification, the cues associated with withdrawal precipitate a withdrawal response, which may induce the addict to procure the drug again.

This hypothesis has recently been tested in monkeys. Goldberg and Schuster (1967) found that a tone paired with injection of nalorphine, a morphine antagonist, elicited many of the same "withdrawal" symptoms that the drug elicited from morphine-dependent monkeys (*e.g.*, bradycardia, emesis, excessive salivation, suppression of on-going appetitive behavior). Hence, Wikler's hypothesis—that withdrawal symptoms can be conditioned to environmental stimuli, in this case the tone—was supported experimentally. This effect persisted when the monkeys were no longer physically dependent on morphine (Goldberg and Schuster, 1970). In a third study, a stimulus

associated with a morphine injection became a conditioned reinforcer; addicted monkeys would work for a light that had previously been paired with morphine injection (Schuster and Woods, 1968).

The present research sought to examine the possibility that conditioning might occur with drugs such as the tranquilizer, chlorpromazine (CPZ) and the hallucinogen, lysergic acid diethylamide (LSD) which have potent, unconditioned behaviorally-disrupting effects but which do not produce physical dependence. A design very similar to that of Goldberg and Schuster (1967) was employed. Rats were trained with water as the reinforcer under a variable-interval (VI) schedule and were given doses of CPZ or LSD that completely suppressed bar pressing. Would a light associated with administration of either or both of these drugs also suppress bar pressing and, if so, would the duration of the suppression be related to the duration of the stimulus light?

METHOD

Subjects

Ten experimentally naive albino rats of the Sprague-Dawley strain from Charles River Breeding Laboratories (Wilmington, Massachusetts) were used in two experiments. They were approximately 120 days old when the experiments started, and weighed between 200 and 250 g. The animals were housed separately in a room that was maintained at constant temperature and humidity on a 12-hr light-

¹This research was supported by USPHS Research Grant MH-13,186-04, Illinois State Department of Mental Health Grant 102-13-RD, Research Scientist Development Award 5-KO2-MH-09355 (to J.B.A.) from the National Institute of Mental Health, and by PHS Training Grant HD-00001 from the National Institute of Child Health and Human Development (to O.G.C.). Reprints may be obtained from: James B. Appel, Ph.D., Department of Psychiatry, The University of Chicago, 950 East 59th Street, Chicago, Illinois 60637.

dark cycle (7:00 am to 7:00 pm). Food was always available in the home cages.

Apparatus

The experimental chamber was similar to one that is commercially available (R. Gerbrands, Co.). It was equipped with a single lever, a dipper that supplied 0.05 ml of tap water when the reinforcer was presented, a pair of dim red houselights, and a 28-v white stimulus light behind a translucent panel directly above the lever. The chamber was enclosed in a sound and light attenuating wooden shell that was equipped with a blower to provide ventilation. A second blower, outside the shell, was modified to produce a continuous staccato background noise. All experimental events were controlled by switching and timing circuits in another room; responses were recorded on a cumulative recorder and on electromagnetic counters.

Drugs

The fluid used for injections and drug dilutions was 0.9% sodium chloride and 0.9% benzyl alcohol in distilled water. A solution of 10 mg/ml of CPZ HCl was prepared from ampules containing 25 mg/ml of chlorpromazine hydrochloride (Thorazine) obtained from Smith, Kline and French, Inc., Philadelphia, Pa. LSD was injected from ampules containing 0.1 mg/ml of LSD-25 (Delysid) manufactured by Sandoz Pharmaceuticals, Hanover, New Jersey and obtained from the National Institute of Mental Health, Center for the Study of Narcotics and Drug Abuse. Injections were given intraperitoneally. The doses used produced approximately equal suppression: behavior was almost completely eliminated for at least 45 min, while no residual effects could be detected 24 hr after injection. It is unlikely that tolerance effects would be a significant factor with the doses used and the number and schedule of drug administration employed in this study (Freedman, Appel, Hartman, and Molliver, 1964).

Behavioral Procedures

Preliminary training. Four days before shaping, no water was available for approximately 48 hr. Then, for two days, subjects were given 15- to 20-min access to water at the time they were later to be run. The experimental sessions were scheduled five days per week (Mon-

day to Friday) during which time no water was available except that which was consumed in the chamber (4.0 to 6.0 ml). Because the animals' weights were observed to fall, unlimited access to water was permitted for 18 hr after the Friday sessions. Then, no water was available until the Monday session.

All subjects were magazine trained, given two daily sessions in which every bar press was reinforced, and stabilized on a variable-interval 30-sec schedule (VI 30-sec) for five days. The distribution of inter-reinforcement intervals was rectangular, with a range from 0 to 60 sec. A fairly high and constant baseline rate of bar pressing was obtained. Each daily session was almost 1 hr, by the fifth day all animals were obtaining approximately 100 reinforcements per session.

Habituation procedures were then given in order to minimize (1) any stimulus change or novelty effects of the light, (2) disruption produced by the intraperitoneal injection, and (3) conditioning of the light to any aversive properties of the injection procedure. The stimulus light (white light on, red lights off) was presented for 10 min during six consecutive sessions. By the sixth session, no subject showed any decrement in behavior during the light. Then, the light was presented and an intraperitoneal injection of saline was administered for nine sessions. The temporal relationship between the light and the injection was random. By the ninth session, all subjects demonstrated an immediate return to baseline after being returned to the chamber following injection. (The injection procedure involved removing the animals from the chamber for no more than 20 sec.)

The third step involved arranging a constant temporal relationship between the light and the injection. The point in the session at which the light was presented remained random (but excluded the first and last 10 min), but now the injection was always given 3 min after light onset. By the fifteenth day, no subject showed any significant disruption of bar pressing at the time of light onset or at any time during the 10 min during which the light was on.

EXPERIMENT I.

In the first experiment, the fourth and final step involved conditioning sessions using the

Table 1
Outline of Drug Conditioning Regimen (Experiment I)

Drug	Animal	Dose mg/kg	No. con- ditioning sessions	No. of inter- spersed baseline days	Dose mg/kg	No. of sessions	No. of saline sessions	Dose mg/kg	No. of sessions	No. of saline sessions
CPZ	773J	4	6	5	10	2	1	10	4	3
	775J	4	6	5	8	2	1	8	4	3
LSD	774J	0.2	7	6			1	0.2	4	3
	776J	0.2	6	5			4	0.2	5	1

drug in place of saline. The procedure was identical in all ways to step three (above) except that Subjects 773J and 775J received CPZ (4 mg/kg), while 774J and 776J received LSD (0.2 mg/kg) instead of saline.

Table 1 outlines the conditioning regimen for Experiment I. Conditioning and baseline sessions were randomly interspersed during the initial conditioning phase. In baseline sessions, no drug or light was presented. For animals receiving CPZ the dose being used initially did not appear to be producing a significant conditioning effect, so two additional sessions were given at new, increased doses. This was not necessary for animals receiving LSD. Then, sessions in which saline was given (four sessions for 776J and one for the other animals) were given to test for conditioned effects. The conditioning regimens of Subjects 773J, 774J, and 775J are similar except for the change in dose for 773J and 775J. Animal 776J was treated somewhat differently.

EXPERIMENT II

This experiment was concerned with (1) replicating the LSD-induced conditioned suppression effect using more subjects, and (2) relating the duration of suppression to the duration of the light (see results of Experiment I, Subject 776J).

After initial training and habituation (above), with some variation in the number of sessions under each condition, six rats were divided into three groups of two subjects each. In all groups the injection was given 3 min after light onset. In the first group, the light was on for 20 min; in the second group, 10 min; and in the last group, 5 min. Habituation procedures were continued for five more ses-

sions using the various light durations instead of the 10-min light initially used for all subjects. Next, eight conditioning sessions using LSD (0.2 mg/kg) were randomly interspersed with six baseline sessions. Finally, for eight consecutive sessions, saline injections were given to test further for conditioned and extinction effects.

RESULTS

In all figures that contain cumulative records, the actual performance is represented by the diagonal solid line. The vertical solid lines represent light onset and offset, and the vertical dashed line represents the point at which the injection was given.

Experiment I

The effects of CPZ can be seen for Subjects 773J and 775J in Figure 1. The records from the last light-saline habituation session are shown at A. The rate of bar pressing decreased when the light was presented after eight pairings of light and drug (B). Suppression also occurred on the next day, when a saline injection was given instead of a drug injection (C). Because only one light-saline pairing attenuated the amount of suppression, four light-drug reconditioning days were given. On the day after this reconditioning, saline was again given in place of the drugs; suppression again occurred as soon as the light appeared (D). Then, two further light-saline pairings produced almost complete attenuation of suppression for Rat 773J and at least partial attenuation for Rat 775J (E). The response rate usually returned to baseline immediately after injection on days when either drug or saline was given, even though the light was on for

Fig. 1. Cumulative records of the behavior of Rats 773J (10 mg/kg of CPZ) and 775J (CPZ—8 mg/kg). A—last habituation day, B—eighth drug day, C—first saline day after eight drug days, D—first saline day after 12 drug days, E—third saline day after 12 drug days. The light duration (between vertical solid lines) is 10 min; the vertical dotted line marks the injection (approximately 3 min after light onset).

7 min after injection (except for Rat 775J, record C; and 773J, record D).

The effects of LSD are shown in Figure 2 (Rat 774J) and Figure 3 (Rat 776J). The record of the last habituation session for 774J is shown in Figure 2 at A. The rate of bar pressing for 774J decreased when the light appeared on the first day in which saline was given after seven light-drug pairings (B). After four additional light-drug pairings, suppression again occurred at light onset (C). Two light-saline pairings partially attenuated bar pressing (D).

The results with LSD were similar to those obtained with CPZ, but, on days on which saline was given, the rate did not return to baseline immediately after injection, as it usually did for animals receiving CPZ. For instance, only five responses occurred in the 20 min after light onset on the first saline day

after reconditioning (Figure 2, C). In the same length of time, more than 100 responses occurred during the first half of the session.

The performance of Rat 776J is shown in Figure 3. The last habituation day is again shown in record A. The rate of bar pressing decreased when the light appeared on the sixth light-drug pairing (B), on the first (C), and the second (D), light-saline pairing. This suppression was partially attenuated by the third presentation of the light in the absence of the drug (E) and was almost completely attenuated by the fourth presentation (F). Then, after five more pairings of the light with drug, the rate was again suppressed in the presence of the light (G); this effect was also seen on the following day on which saline was given (H).

The suppression that occurred with Rat 774J after saline injection (Figure 2, C) also

Fig. 2. Cumulative records of the behavior of Rat 774J (0.2 mg/kg of LSD). A—last habituation day, B—first saline day after seven drug days, C—first saline day after 11 drug days, D—third saline day after 11 drug days. The light duration and time of injection are the same as in Figure 1.

occurred with Rat 776J, but was not observed in every session in which saline was given, and seemed to be related to the duration of the light. For example, on the first day (Figure 3, C) there was partial suppression for approximately 7 min after saline injection (until light offset) and then a return to a near baseline level of responding. On the following day (Figure 3, D), the result was the reverse of that observed on the first day. After injection, the rate returned almost to baseline, but then decreased after light offset. By the third day, the rate returned to baseline immediately after injection, and remained at that level after the light was turned off (Figure 3, E). These results suggest that the suppression effect might be related to either the duration of the light or the occurrence of the light offset (see Experiment II).

Experiment II

Table 2 shows the mean daily rates of bar pressing during the light for the six rats of Experiment II. The uppermost group of figures are the rates (response per minute) from the last five habituation days, in which the light was of the same duration as in the conditioning sessions. The next eight scores (conditioning) represent the rates obtained on days in which LSD was paired with the light. These days were interspersed randomly with six baseline days, in which neither light nor drug was given. The final eight scores (extinction) are the rates during the eight consecutive sessions in which saline injection replaced the drug.

Figure 4 shows the means derived from the rates of the two animals in each group shown in Table 2. There was some variability in the rates that occurred in the five habituation days, but with one exception, these rates all exceeded those on any of the eight conditioning days.

The eight sessions in which the light and drug were paired demonstrate that conditioning had occurred. In all groups, the rates fell from the early to the late conditioning sessions (Figure 4). If tolerance had occurred, levels would be in the other direction (up from left to right instead of down); therefore, either tolerance did not occur, or the effect was masked by the much stronger conditioning effect.

A clear conditioned effect occurred on the first (extinction) day in which saline replaced the drug, but rate of bar pressing returned to normal after only one session in the 10-min light group, was not significantly suppressed in the 20-min group, and was unstable in the 5-min group (Figure 4).

Figure 5 contains cumulative records from the habituation, conditioning, and extinction sessions for Animals 907 (which had a 10-min light) and 910 (5-min light). A conditioned drug effect was more clearly seen in Animal 910 than in 907 with four conditioning sessions (Figure 5, C), but conditioning was observed by the eighth (last) session in both animals (D). Suppression was also seen in the first session after conditioning (E), in which saline was given; this effect is clearer in Rat 907 than in 910, but is present in both animals. By the third session in which saline was given, the effect was completely extinguished in 907 and partially extinguished in 910.

Fig. 3. Cumulative records of the performance of Rat 776J (0.2 mg/kg of LSD). A—last light-injection habituation day, B—sixth drug day, C, D, E, F—first through fourth saline days after six drug days respectively, G—fifth reconditioning day after four saline extinction days, H—first saline day after five reconditioning days. The light duration and time of injection are the same as in Figure 1.

The records of Figure 5 are representative of all subjects in this study. Occasional inappropriately high or low rates were observed, but both Table 2 and Figure 4 demonstrate that the overall trends were quite clear. The baseline rates sometimes dropped over conditioning sessions (*e.g.*, Rat 910) but the records show that, even in later conditioning sessions in which the rate was low, the light produced an even lower rate. This indicated that the decreases in rates observed during the light were not completely a function of a general decrease in baseline rate. Moreover, the decrease was not observed in all subjects and the conditioning effect was. Suppression by the light had ex-

tinguished in all subjects by the eighth saline test session, and low baseline rates had usually increased almost to pre-conditioning levels. The general decreases in baseline rates also indicated that tolerance effects were either absent or minimal.

The effect of light offset observed in Experiment I, Animal 776J (Figure 3, C and D) was not replicated in Experiment II, in which there were no systematic differences observed between any of the three groups in either rate of conditioning or rate of extinction. Suppression generally was maintained after light offset regardless of how long the period in which the light was present.

Table 2

Mean rates (responses per minute) of bar pressing on a VI 30-sec schedule in the presence of the light during the various phases of Experiment II.

Experimental Condition	Animal no.	Light Duration					
		20 min		10 min		5 min	
		909	931	907	932	908	910
Habituation	Days						
	1	39.55	21.10	24.20	94.90	12.00	103.20
	2	36.75	9.35	19.60	108.40	10.00	99.20
	3	22.30	15.65	25.10	79.60	9.80	75.80
	4	46.70	31.75	32.20	80.70	4.40	59.80
	5	55.00	15.90	30.20	94.10	11.00	79.60
Conditioning	1	16.15	2.60	18.90	23.50	10.40	75.80
	2	30.45	0.75	14.30	28.90	7.80	60.10
	3	19.50	9.15	13.00	60.00	15.20	41.80
	4	16.90	8.50	9.20	53.70	9.80	17.40
	5	2.15	4.95	7.00	25.80	7.20	2.00
	6	5.15	3.95	3.90	8.70	5.10	19.80
	7	2.90	4.30	7.80	25.00	3.20	24.20
	8	3.40	3.40	1.90	24.00	3.80	4.40
Extinction	1	5.90	4.05	0.30	21.40	5.00	3.20
	2	22.70	27.15	20.00	76.90	15.20	6.20
	3	25.60	18.60	19.70	88.10	7.60	27.80
	4	11.50	10.30	30.40	59.60	7.60	33.60
	5	21.50	13.15	33.30	66.10	2.20	13.40
	6	33.50	3.60	26.70	84.60	27.80	31.20
	7	55.50	42.50	19.70	85.70	2.00	1.80
	8	60.00	26.60	32.50	92.80	10.80	63.00

DISCUSSION

The results of these studies (1) confirm the finding that the (unconditioned) suppression of bar-pressing behavior induced by the administration of certain drugs can be conditioned to an originally-neutral stimulus (Goldberg and Schuster, 1967), and (2) extend both the number and kind of compounds for which conditioning has been demonstrated. Since different unconditioned states are presumably elicited by hallucinogens, tranquilizers, and morphine antagonists, the present results also suggest that many drug-related responses or states, in addition to those associated with "withdrawal" (Goldberg and Schuster, 1967), can be conditioned; in any case, neither "withdrawal" symptoms nor physical dependency are necessary preconditions for conditioned suppression by drugs.

The compounds used in this study produced somewhat different patterns of conditioning. For example, with CPZ, but not LSD, the animals usually returned to their baseline rates of responding immediately after the injection of saline or drug. The reason for this difference

is not clear. It may be that the unconditioned state induced by the hallucinogen is more potent or may have a more rapid time of onset than that induced by the tranquilizer, but further parametric work is necessary before such statements can be empirically supported.

The results of Experiment II indicated that light off-set did not function as a significant stimulus in most conditioned animals. "Attentional" variables may account for the lack of effect. At light onset, presumably no state, conditioned or unconditioned, is present, but at offset, after conditioning, the state is presumably present and the animal may be "attending" to the internal state, rather than to the scheduled environmental stimulus change. Many studies (for example, those cited by Tecce, 1970, and Mirsky and Kornetsky, 1968) have demonstrated that drugs including LSD and CPZ can attenuate "attention."

The paradigm employed both in this study and those of Goldberg and Schuster (1967, 1970) is similar operationally to the conditioned emotional response (CER) first described by Estes and Skinner (1941). An originally neutral stimulus is paired with an

Fig. 4. Graphs of rates (mean responses per minute) obtained by averaging rates of the two animals in each group during the stimulus light (CS). Habituation: last five pre-conditioning light-saline sessions; conditioning: eight sessions in which the light was paired with drug; extinction: eight sessions in which saline replaced drug.

unconditioned stimulus in a respondent conditioning paradigm (*i.e.*, noncontingent on the animal's behavior) and the measure of conditioning is whether the disruption of some ongoing appetitive behavior such as bar pressing, which originally occurred to the unconditioned stimulus, begins to occur to the originally neutral stimulus. In the CER, the unconditioned stimulus is usually some supposedly aversive event, such as painful electric shock, and the "explanation" of the conditioned disruption is in terms such as "anxiety", "emotion", "fear", *etc.*

In the work of Goldberg and Schuster (1967), the unconditioned stimulus, a "withdrawal" state, may reasonably be considered "aversive", and an explanation of conditioned disruption in terms of "fear", *etc.*, may be appropriate. But, when the drugs used in this study serve as unconditioned stimuli, this explanation seems tenuous at best because there is little to indicate that the administration of either LSD or

CPZ is aversive in animals (or humans). There is some suggestive experimental evidence that LSD will be self-administered in animals (Schuster, personal communication) and the drug has been used frequently enough in at least some subcultures of humans to constitute a drug problem. Although there is no evidence that CPZ is self-administered in animals, it is certainly self-administered in therapeutic situations by millions of people. Moreover, there is no reason to believe that the doses used in this study were any more "aversive" than those normally given either experimentally or clinically or that they have any significant toxic side effects (Goodman and Gilman, 1970). Thus, an aversive unconditioned stimulus is probably not necessary to produce a "CER", and terms such as "emotional" are probably not adequate to explain this paradigm under all conditions. In this context, it should be noted that Azrin and Hake (1969) demonstrated that the non-response dependent

Fig. 5. Cumulative records for Rats 907 and 910 (0.2 mg/kg LSD). A—last habituation day, B—first light-drug pairing, C—fourth pairing, D—eighth pairing, E—first light-saline day after conditioning, F—third day (907—10-min light, 910—5-min light).

presentation of food, water, or intracranial stimulation will sometimes depress ongoing appetitive behavior, and that this suppression can be conditioned to an originally neutral stimulus in a "CER" paradigm.

An interesting and unexpected finding was the rapid attenuation (extinction) of the conditioned drug effect. The suppression to a saline "placebo" injection given after conditioning was completed was clearly demonstrable and stable for only one trial (Figure 4). Past studies, using shock as the unconditioned stimulus (Hunt and Brady, 1955; Hoffman and Fleshler, 1965) have shown that the suppression that occurs during the CER extinguishes very slowly, even slower than suppression induced by punishment.

There are at least three possible explanations for the observed difference between the present results and those usually reported. First, extinction in the usual case is produced by relatively massed trials (several per daily session) while, in the present experiments, only one trial was given each day. Second, the duration of the unconditioned stimuli was much longer in this study (at least 1 hr) than in the usual paradigm, in which brief shocks (shorter than 1 sec) are normally used. Hence, in extinction the duration of *no* unconditioned stimulus is also longer, and may functionally serve as several extinction trials. Third, the nature of the stimulus, drug state *vs.* shock-induced pain, may account for the differences in rate of extinction. (In the Hunt and Brady study, at least, the number of conditioning trials was comparable to the number used in these studies; therefore, the greater number of trials to extinction in the Hunt and Brady study was probably *not* a function of a stronger conditioned effect before extinction.)

The first and second explanations are, in a sense, opposite of each other, so that it is unlikely that both could be correct. But any one explanation or other combination of explanations could account for the observed differences in results. The present study does not allow for supporting one or another of these possibilities.

Whitney and Trost (1970), working independently of the authors, have recently obtained almost identical results, using a CER paradigm with a rhesus monkey in which intravenous amphetamine injection was the unconditioned stimulus and a tone the condi-

tioned stimulus. In their study, as in ours, eight pairings were sufficient to establish the conditioned suppressing effect, and in both studies the number of trials to extinction (1 to 3) was fewer than normally observed in the CER paradigm. While these authors were unsuccessful in establishing self-administration with the dosage used to produce the CER, they concluded (as we did with LSD and CPZ) that the conditioned suppression effect cannot be completely explained by any presumed "aversiveness" of the unconditioned drug state. These results (fast extinction) are also in contrast to the large number of trials required by Goldberg and Schuster (1967, 1970) to produce extinction, and may be a function of the presumed "aversiveness" of the withdrawal state induced in their studies *versus* the presumed "non-aversiveness" of the LSD, chlorpromazine, or amphetamine states.

The results of Goldberg and Schuster (1967, 1970), Whitney and Trost (1970), as well as those reported here, demonstrate the large number of drug types—narcotic analgesics, central stimulants, major tranquilizers, and hallucinogens—that can successfully function as unconditioned stimuli in a conditioned suppression paradigm. Most probably others, such as barbiturates (central depressants), could also function in this capacity.

One final point concerns the general decrease in baseline rate occasionally observed during conditioning trials. This may have resulted from conditioning not only to the light (conditioned stimulus) but also to other stimuli present in the operant chamber during conditioning (metallic walls, grid floor, lever, *etc.*)—an effect that has been discussed by Wagner (1969) among others.

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Received 2 September 1970.

(Final acceptance 30 August 1971.)