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Journal of Comparative and Physiological Psychology
1980, Vol. 94, No. 4, 641-649

Sensory and Associative Effects of LSD in Classical Conditioning of Rabbit (*Oryctolagus cuniculus*) Nictitating Membrane Response

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Three experiments were conducted to determine the effects of LSD (30 nmol/kg) on the acquisition of the rabbit's classically conditioned nictitating membrane response. Experiment 1 revealed that LSD significantly enhanced the acquisition of conditioned responses [CRs], and control groups receiving unpaired conditioned stimulus/unconditioned stimulus (CS/UCS) presentations served to identify LSD's effect to be on learning. Accordingly, subsequent efforts were directed at determining whether LSD's enhancing effects could have arisen from its altering the sensory processing of the CS and/or the UCS. In Experiment 1, unconditioned response (UCR) amplitude to a 3-mA UCS was not significantly affected by LSD. Moreover, Experiment 2 revealed that LSD had no significant effect on the psychophysical functions relating UCS intensity to the frequency or amplitude of UCRs and that the drug did not affect the UCS-intensity threshold for evoking UCRs. On the other hand, Experiment 3 revealed that LSD significantly enhanced the frequency of CRs to an extended range of CS intensities in the psychophysical function relating CS intensity to CR frequency. Furthermore, LSD lowered the CS-intensity threshold. The drug's enhancement of the sensory processing of the CS was postulated to facilitate conditioning through both learning and performance mechanisms.

Recently, Gimpl, Gormezano, and Harvey (1978) employed the rabbit nictitating membrane response (NMR) preparation in a classical defense conditioning study designed to establish a dose-response function for LSD (1, 10, 30, 100, or 300 nmol/kg iv) on the acquisition of conditioned responses (CRs). The study revealed a biphasic dose-response function in which dosages of 1 to 100 nmol/kg LSD significantly enhanced CR acquisition, with a peak enhancement occurring at 30 nmol/kg, whereas at 300 nmol/kg LSD, CRs were retarded relative to those of vehicle controls. Moreover, the performance of control groups given unpaired CS-alone and UCS-alone (conditioned stimulus and unconditioned stimulus, respectively) presentations revealed that, regardless of dosage, the enhancing effect of LSD on the acquisition of CRs could not be attributed to such nonassociative factors as an alteration in the base rate of responding, sensitization, or pseudoconditioning.

The precise basis for the facilitating associative effects of LSD on the acquisition of CRs reported by Gimpl et al. remains to be delineated. However, in accounting for their findings, Gimpl et al. suggested that the effects of LSD on conditioning could be due to an alteration in the sensory processing of stimuli that enter into the conditioning process (i.e., CSs and/or UCSs). This hypothesis appears particularly plausible since, first, enhanced auditory and visual evoked potentials have been reported in the cat (Purpura, 1956) and rabbit (Burian, Flemming, & Featherstone, 1958) at low dosages of LSD. Second, the abducens nucleus, which directly activates the final neural pathway (the VIth cranial nerve) for eliciting membrane extension in the rabbit, in addition to receiving inputs from peripheral UCSs like paraorbital shock or

This research was supported by National Institute of Drug Abuse Grant DA 01759. The assistance of Ellen Aycock is gratefully acknowledged.

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 0021-9940/80/9404-0641$00.75

corneal air puffs (Cegavske, Thompson, Patterson, & Gormezano, 1976), also receives inputs from the auditory and visual systems (Borg, 1973; Grantyn & Grantyn, 1976; Rasmussen, 1946). Third, it has been demonstrated that both NM CRs and UCRs (unconditioned responses) result from the activation of abducen motor neurons (Cegavske et al., 1976). Fourth, while tone CSs do not elicit unconditioned NMRs (Gormezano, 1966, 1972; Gormezano, Schneiderman, Deaux, & Fuentes, 1962), tones do increase the "excitability" of NM UCRs as measured by an increase in the amplitude of the UCR to subsequent application of peripheral UCSs (Ison & Leonard, 1971; Young, Cegavske, & Thompson, 1976) or direct electrical stimulation of the abducens nucleus (Young et al., 1976). Hence, not only does the abducens nucleus receive inputs from stimuli that can serve as CSs and UCSs for conditioning, but these stimuli also interact with each other. Fifth, and finally, the empirical manipulation of the intensity of CSs and UCSs, upon which conditioning was based, has been amply documented to affect the formation of CS–CR connections (cf. Gormezano & Moore, 1969). Accordingly, the behavioral laws of conditioning and the anatomical organization of the reflex pathways of the NMR have led us to examine the possibility that the enhancing associative effects of LSD on the acquisition of NM CRs (Experiment 1) could be due to alterations in the sensory processing of the UCS (Experiment 2) and/or the CS (Experiment 3).

Experiment 1

The purpose of the present investigation was to confirm the observation by Gimpl et al. (1978) of enhanced CR acquisition under the LSD dosage (30 nmol/kg) previously shown by them to produce peak enhancement, and to confirm that the source of such enhancement is associative. Furthermore, determinations were made of the effects of LSD, relative to vehicle control, on (a) the initiation (rate) of conditioning, (b) conditioning to tone and light CSs, and (c) UCR amplitude.

Method

Subjects. The subjects were 48 male and female albino rabbits obtained from Morrison Rabbitry, West Liberty, Iowa. They were 80–100 days old, and each weighed about 2 kg on arrival in the laboratory.

Apparatus. The conditioning chambers and transducers for recording the NMR have been described by Coleman and Gormezano (1971), who detailed departures from earlier specifications (Gormezano, 1966; Gormezano et al., 1962). In brief, the transducer for recording the NMR consisted of a resistive photocell assembly, which generated a signal that was recorded on an Offner oscillograph operating at a paper speed of 250 mm/sec. The conditioning chambers were fabricated from legal-sized, fireproof filing cabinets to which individual blowers, exhausts, and stimulus panels had been added. Two CSs were employed. One CS, an 800-msec, 75-dB (SPL), 1000-Hz tone, superimposed upon a 70-dB background ventilation noise, was delivered through an 11.4-cm speaker mounted in the center of the stimulus panel. The other CS consisted of a 10-Hz, 800-msec flashing of two 6-W, 24-V house lights, one mounted on each side of the speaker. The flashing of the house lights caused a change in illumination, measured at the animal's eye level, from approximately 32.0 lx to 8.0 lx. The UCS was a 100-msec, 3-mA, 60-Hz shock delivered to electrodes positioned 10 mm apart and 15 mm posterior to the dorsal canthus of the rabbit's right eye.

Procedure. All animals received 1 day of preparation, 1 day of recovery, 1 day of adaptation, and 10 days of acquisition training. On the preparation day, a small loop of surgical nylon (Ethicon 00) was sutured into the rabbit's right NM, the surrounding hair was clipped, and the remaining hair was removed by the application of a chemical depilatory. The UCS electrodes (Autoclip wound clips) were than applied. On the adaptation day, the animals, secured in a Plexiglas restraining box, were positioned in the conditioning apparatus for a period equal to the length of the subsequent conditioning sessions. No stimuli were presented during the adaptation session, although to obtain an index of base rate of responding, the data records were scored for responses occurring at times corresponding to the usual CS–UCS observation intervals.

Following adaptation, all subjects received 10 days of acquisition training in which two experimental groups (n = 12) received 60 CS–UCS paired conditioning trials daily at a CS–UCS interval of 800 msec. The conditioning trials were composed of 30 tone and 30 light CS–UCS pairings presented in a randomized sequence within 10-trial blocks with the restriction that not more than three tone or three light CS trials could occur consecutively. Trials occurred at intertrial intervals of 50, 60, and 70 sec, with a mean of 60 sec. In addition, two control groups (n = 12) were exposed to explicitly unpaired CS and UCS presentations in which each daily session consisted of 30 tone CS, 30 light CS, and 60 shock UCS trials presented in a randomized sequence within 20-trial blocks with the restriction that no more than three tone, light, or shock trials could occur consecutively. Trials in the unpaired controls occurred at 25-, 30-, and 35-sec intervals, with a mean of 30 sec.

Thirty minutes prior to each acquisition session, all

animals in one experimental and one control group were injected with *d*-lysergic acid diethylamide tartrate (LSD, molecular weight 430.5), obtained from the National Institute of Drug Abuse. The drug, dissolved in sterile distilled water, was injected into the marginal ear vein at a dosage of 30 nmol/kg. Animals in each of the remaining experimental and control groups were injected in the marginal ear vein with sterile distilled water. Animals were injected with the vehicle or drug in a volume of .4 ml/kg at a rate of 3 ml/min by a Harvard infusion pump. Throughout the experiment, a CR was defined as any NMR extension exceeding .5 mm and occurring during the 800-msec presentation of either CS. In addition, for the unpaired control groups, a response occurring during the 800-msec interval preceding each UCS was recorded as a baseline response, and the amplitude of the UCR (greater than .5 mm) was recorded for each of the 60 daily UCS presentations.

Results

Figure 1 presents the percentage NMRs to tone and light CS trials combined across the 10 days of acquisition training for each of the four groups. The figure reveals that under the paired CS–UCS condition, LSD, relative to the vehicle control, substantially enhanced the overall level of CRs as well as the rate of acquisition of CRs. These descriptive aspects of the percentage CR data for the paired CS–UCS groups were corroborated by a repeated measures analysis of variance which revealed significant effects of groups, $F(1, 22) = 18.78$, $p < .01$, days, $F(9, 198) = 60.92$, $p < .01$, and Groups × Days, $F(9, 198) = 2.34$, $p < .01$. The acquisition of CRs to tone and light CSs were highly similar—the mean levels were 57% and 59%, respectively—and the analysis revealed that modality was not a significant source of variation and that it did not enter into any significant interactions. To obtain a finer grain assessment of the effect of LSD on the acquisition of CRs, we made determinations of the number of trials to the first CR as well as to successively more stringent criteria of 2 to 10 consecutive CRs. The mean number of trials to each of these criteria revealed that LSD facilitated not only the initiation of the first CR (21.8), relative to vehicle control (42.0), but also the attainment of each successive criterion. Thus, for example, under LSD the mean numbers of trials required to attain criteria of 5 and 10 consecutive CRs were 80.2 and 86.0, respectively, and under vehicle they were 148.7 and 190.2, respectively. An analysis of variance of the number of trials to each of the 10 criteria revealed a significant effect of groups, $F(1, 22) = 16.94$, $p < .01$, criterion, $F(9, 198) = 38.91$, $p < .01$, and Groups × Criterion, $F(9, 198) = 4.78$, $p < .01$. Moreover, a subsequent Tukey honestly significant difference (HSD) analysis (see Winer, 1971) of the significant groups contrast revealed that LSD-injected animals required significantly fewer trials than vehicle controls at each criterion (all ps $< .05$).

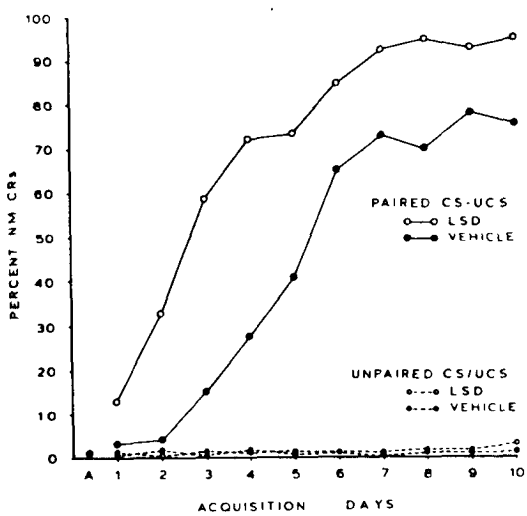

Figure 1. Mean percentage of response during each of the 10 days of acquisition training under LSD or vehicle for groups receiving paired CS–UCS and unpaired CS/UCS trials. (NM = nictitating membrane; CRs = conditioned responses; CS = conditioned stimulus; UCS = unconditioned stimulus.)

Panel A of Figure 2 presents the frequency of baseline responding during the 800-msec prior to UCS delivery under the unpaired CS/UCS condition for LSD and vehicle groups over the 10 days of training. The figure indicates that the levels of baseline responding were low and undifferentiated by drug condition as revealed by mean levels of responding of 1.2% and 1.0% for LSD animals and vehicle controls, respectively. The mean level of baseline responding during adaptation when neither CSs nor UCSs were presented and no drug had been injected was 1.3%. Hence, as is characteristic of the rabbit NMR preparation (cf. Gormezano, 1966, 1972), the presentation of CSs and UCSs did not synergistically interact to alter

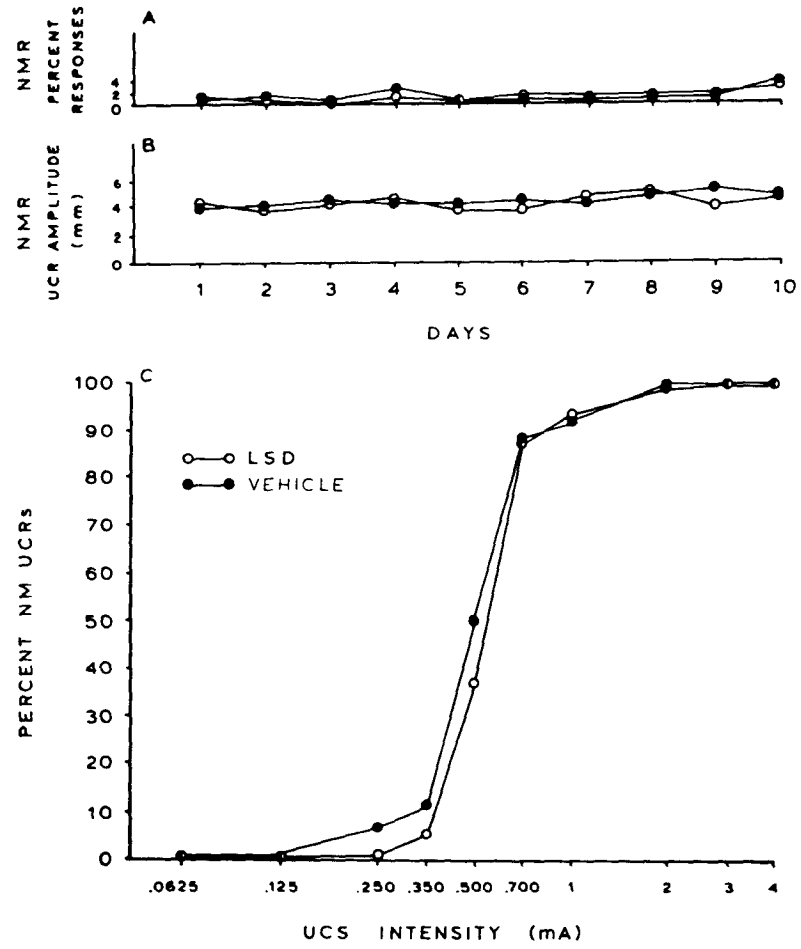

Figure 2. Frequency of baseline responding (panel A) and UCR amplitude (panel B) under LSD or vehicle for groups receiving unpaired CS/UCS trials, and frequency of UCRs under LSD or vehicle as a function of UCS intensity. (NMR = nictitating membrane response; UCR = unconditioned response; CS = conditioned stimulus; UCS = unconditioned stimulus.)

the base rate of NMRs. In addition, as revealed by a reexamination of Figure 1, under the unpaired condition the levels of responding to the CSs were low, never exceeding 2%, and LSD, relative to vehicle, showed no significant or systematic effects on CS responding. Accordingly, the enhancing effects of LSD on CR acquisition cannot be attributed to the drug's effects on such nonassociative factors as base rate of responding, sensitization, or pseudoconditioning.

The results of the above control contrasts served to specify the enhancing effects of LSD on the acquisition of CRs as having arisen from its effects on associative processes. Yet, the locus of the associative effects of LSD remains to be specified since it may reflect the ability of the drug to facilitate directly the formation of a CS–CR connection and/or to alter the sensory processing of the CS and/or the UCS upon which conditioning is based. Unfortunately, no methods presently exist for determining the effect of drugs directly on the formation of a CS–CR connection since, in part, the neural pathway(s) linking the CS with the CR remains unknown. On the other hand, determining whether LSD affects the sensory processing of the CS and/or UCS is an operationally testable proposition. And, in fact, the outcome of one such test can be seen

from an inspection of panel B of Figure 2. The figure (and statistical analysis) reveals that under the unpaired CS/UCS condition the amplitudes of UCRs to the 3-mA UCS were virtually identical across the 10 days for LSD (M = 4.5 mm) and vehicle (M = 4.4 mm). Thus, it appears that LSD had no effect on the sensory processing of the UCS as revealed by the amplitude of UCRs elicited by the 3-mA UCS.

Experiment 2

Although in Experiment 1 LSD was observed to have had no effect on the amplitude of UCRs, the relatively intense UCS employed may have obscured the detection of LSD effects. Accordingly, the purpose of the present experiment was to examine the effects of LSD on UCRs under a wide range of UCS intensities. In particular, the present experiment determined the effects of LSD (30 nmol/kg) on the UCS-intensity threshold and on the psychophysical functions relating UCS intensity to the frequency and amplitude of UCRs.

Method

Subjects. The subjects were 24 male and female albino rabbits obtained from Morrison Rabbitry. They were 80–100 days old, and each weighed about 2 kg on arrival at the laboratory.

Apparatus and procedure. Unless otherwise specified, the apparatus, recording technique, and general procedure were identical to those of Experiment 1. The rabbits were assigned to two groups (n = 12), and on the day following a 50-min adaptation session, they were injected with LSD or vehicle 30 min prior to each of three daily experimental sessions. In each of the three daily sessions, all rabbits received 50 UCS-alone trials at randomized intertrial intervals of 50, 60, and 70 sec (with a mean of 60 sec). The 50 trials were composed of five 100-msec presentations at each of 10 UCS intensities: .0625, .125, .25, .35, .5, .7, 1.0, 2.0, 3.0, and 4.0 mA. Each of the 10 UCS intensities was presented once in a randomized fashion within each of five 10-trial blocks. The occurrence and amplitude of the UCR were recorded on every trial.

Results

Panel C of Figure 2 presents the psychophysical functions relating the frequency of UCRs to UCS intensity for LSD- and vehicle-injected animals. The figure clearly indicates that the psychophysical functions were virtually identical under LSD and vehicle. Furthermore, an analysis of variance revealed that while there were significant effects of UCS intensity, $F(9, 198) = 532.38$, $p < .01$, and days, $F(2, 44) = 7.75$, $p < .01$, LSD had no significant effect on UCR frequency and that there was no significant interaction of drug condition with UCS intensity or with days. In addition, a determination of the UCS-intensity threshold for each rabbit from its UCS-intensity–UCR-frequency function, by determining the UCS intensity at which UCRs would have occurred 50% of the time, revealed no significant effect. Finally, the psychophysical function relating UCR amplitude to UCS intensity (figure not shown) also revealed a significant UCS-intensity effect, $F(9, 198) = 110.34$, $p < .01$, but again no main or interactive effect of drug condition.

Experiment 3

The findings of Experiments 1 and 2 indicate that the enhancing effects of LSD on conditioning of the rabbit's NMR do not appear to be due to any effect of the drug on those UCS–UCR functions upon which conditioning is known to be based (cf. Gormezano & Moore, 1969). Therefore, the present experiment was addressed to the question of whether LSD might enhance conditioning by altering the sensory processing of the CS.

Method

Subjects. The subjects were 22 male and female albino rabbits obtained from Morrison Rabbitry. They were 80–100 days old, and each weighed about 2 kg on arrival at the laboratory.

Apparatus and procedure. Unless otherwise specified, the apparatus, recording technique, general procedure, and stimulus parameters were identical to those of Experiment 1. Thus, as in Experiment 1, all rabbits received 1 day of preparation, 1 day of recovery, 1 day of adaptaton, and 10 days of acquisition training. However, during each acquisition session, only tone CSs were employed for all 60 CS–UCS pairings, and no subject was injected with either LSD or vehicle. On the day after the last acquisition session, Phase 1 was initiated: Rabbits were randomly assigned to two groups (n = 11) and injected with either LSD (30 nmol/kg) or vehicle 30 min prior to each of three additional daily sessions of 60 tone CS–UCS pairings except that CS intensity was randomly varied over 12 values within each of five 12-trial blocks. The CS intensities were 0,

35, 40, 45, 50, 55, 60, 65, 70, 75, 80, and 90 dB, with responding during the 800-msec prior to UCS delivery, when no tone was presented (0 dB), providing a measure of baseline responding. After Phase 1, rabbits were given 3 days of rest followed by 3 days of retraining under the original acquisition conditions with no LSD or vehicle injected. In Phase 2, rabbits previously receiving LSD were injected with vehicle, and those previously injected with vehicle were injected with LSD 30 min prior to each of three additional sessions of exposure to the 12 CS intensities.

Results

By the last 4 days of acquisition training, all rabbits had achieved asymptotic levels of responding to the 75-dB tone CS which averaged about 95%. The panels of Figure 3 present the subsequently obtained psychophysical functions relating CS intensity to percentage CRs for LSD- and vehicle-in-

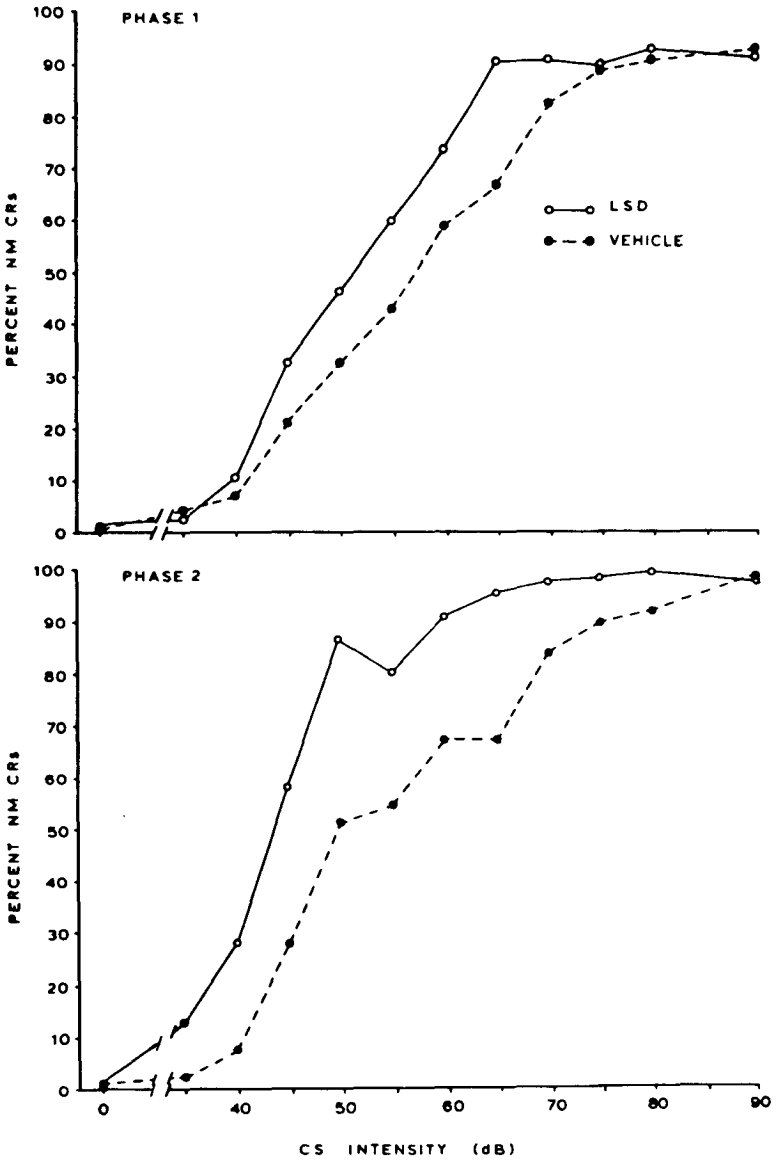

Figure 3. Mean percentage CRs in 15-trial blocks as a function of CS intensity under LSD or vehicle subsequent to 10 days of acquisition training (Phase 1) and under drug reversal conditions (Phase 2) initiated 6 days later. (NM = nictitating membrane; CRs = conditioned responses; CS = conditioned stimulus.)

jected animals in Phases 1 and 2. As expected from the results of an earlier study of CS-intensity effects (Scavio & Gormezano, 1974), the figure and statistical analyses revealed that for Phase 1, percentage CRs was a direct function of CS intensity, $F(11, 220) = 134.44$, $p < .01$, and a significant interaction of drug with CS intensity, $F(11, 220) = 1.93$, $p < .05$, appeared to reflect the enhancing effect of LSD at tone CS intensities between 40 and 70 dB. A subsequent Tukey HSD follow-up analysis to localize the source of the significant interaction revealed that LSD significantly enhanced responding at each of the CS intensities between 45 and 65 dB (all $ps < .05$). Furthermore, a CS-intensity threshold was determined for each rabbit in Phase 1 by calculating from its own psychophysical function relating CS intensity to CR frequency, the CS intensity at which CRs would have occurred on 50% of the trials. It was found that LSD significantly lowered threshold ($p < .01$) in that the mean ± *SE* for vehicle was 57.1 ± 2.7 dB and for LSD, 50.9 ± 2.3 dB.

In Phase 2, percentage CRs was also a direct function of CS intensity, $F(11, 220) = 250.90$, $p < .01$, the occurrence of CRs was significantly enhanced by LSD, $F(1, 20) = 32.22$, $p < .01$, and a significant interaction of drug with CS intensity, $F(11, 220) = 7.47$, $p < .01$, appeared to reflect the enhancing effects of LSD at all but the most extreme value of CS intensity. A Tukey HSD analysis of the significant interaction revealed that LSD significantly lowered threshold ($p < .05$); the mean ± *SE* for the vehicle group was 51.1 ± 2.2 dB and for LSD, 42.8 ± 1.8 dB. Although the rabbit's audiogram has not been reported, clearly these high threshold values would not be expected to correspond with the values that might be obtained under procedures directed at obtaining "absolute" threshold determinations. Nevertheless, they do reveal substantial effects of the drug on the sensory processing of the CS.

Discussion

The enhancing effect of LSD (30 nmol/kg) on the acquisition of NM CRs was observed to occur to both visual and auditory CSs with equal facility and could not be attributed to the drug's potentiating such nonassociative factors as base rate of responding, sensitization, or pseudoconditioning. Moreover, the pattern of LSD effects on CR acquisition was essentially the same before and after the occurrence of the first CR. Specifically, in addition to the enhancing effect of LSD on the overall acquisition of CRs, the drug markedly facilitated the initiation of conditioning, as revealed by LSD-injected animals requiring substantially fewer trials to make their first CR as well as by the more rapid attainment of each successive CR criterion up to 10. Accordingly, whatever may be the mechanism by means of which LSD enhances the acquisition of CRs, it is operative during the CS–UCS pairings prior to the first CR.

Under the unpaired CS/UCS procedure, LSD had no significant effect on UCR amplitude to a fixed-shock UCS intensity. Furthermore, LSD did not affect the psychophysical functions relating a wide range of UCS intensities to the frequency or amplitude of UCRs, nor did LSD affect the UCS-intensity threshold for eliciting UCRs. Hence, LSD does not appear to exercise its affects on conditioning through an alteration of the sensory processing of UCS components involved in the UCR system. These results also indicate that the enhancing effects of LSD on the acquisition of CRs cannot be attributed to any alteration in the motor response system, since both CRs and UCRs involve the abducens nucleus, the final common pathway (via the VIth cranial nerve) for NMRs (Cegavske et al., 1976). It is important to note that while these conclusions are based on negative findings, the statistical power of the experiments was considerable, so if LSD does affect the sensory processing of the UCS, its effects could not be very appreciable.

Phase 1 of Experiment 3 revealed that LSD enhanced the frequency of CRs to an extended range of CS intensities and also lowered the CS-intensity threshold. These results unequivocally indicate that LSD can operate to enhance the sensory processing of an auditory CS. Moreover, to the extent that these functions were obtained following essentially asymptotic levels of CR acquisi-

tion, adherents of a learning-performance distinction would identify LSD's effects to be on performance. Further support for a performance interpretation of LSD's enhancement of the sensory processing of the CS comes from the ready reversibility of effects revealed in Phase 2 and from studies currently in progress which reveal that LSD (30 nmol/kg) potentiates the excitability of NM UCRs to tones. On the other hand, Experiment 1 clearly identified LSD's enhancing effects on CR acquisition to be associative. Although appearing to be contradictory, it has been well recognized in behavioral theories of conditioning that an enhanced or more intense CS may operate as a learning or performance variable or both. Thus, although all behavioral theorists have not adhered to a learning-performance dichotomy (e.g., Guthrie, 1935), the majority of theoretical accounts of CS-intensity effects in classical conditioning (Grice, 1968, 1972; Grice & Hunter, 1964; Hull, 1943, 1949, 1952; Logan, 1956; Perkins, 1953; Razran, 1957) contain such a distinction.

From among the theoretical accounts of CS-intensity effects, Hull (1943, 1949, 1952) has been the most explicit in postulating that CS intensity affects both the acquisition of CRs (through V_1) and the energizing of CRs once conditioning has occurred (through V_2). Specifically, in Hull's analysis, stimulus intensity dynamism (V), which is postulated to enter into a multiplicative relation with habit strength, is composed of associative (V_1) and nonassociative (V_2) components. As a determiner of habit (associative) strength, V_1 was considered to be a monotonically increasing function of the CS's "neural stimulus trace intensity" contiguous with the UCS/UCR, whereas the performance factor, V_2, was held to have a dynamogenic effect (i.e., response evocation was postulated to be proportional to the intensity of the CS). Thus, Hull's analysis assumes that associative strength (conditioning) accrues at the point of contiguity between the CS trace and the UCS/UCR and that the strength of conditioning will be proportional to the intensity of the trace (V_1) at the point of contiguity. Accordingly, the first CR is assumed to be conditioned at the point of CS-trace–UCR contiguity at a rate proportional to V_1, and although CR strength may continue to accrue at the point of contiguity, all anticipatory CRs are assumed to result from the generalization of association along an intensity dimension from the point of CS-trace–UCR contiguity to earlier portions of the trace at a level proportional to the intensity of the trace (V_2) at the point of generalized CR occurrence. Therefore, if, as appears to be the case, LSD enhances (or potentiates) the sensory processing of the CS, it appears to do so in a manner consistent with a Hullian learning-performance account of CS-intensity effects on the initiation of CRs, subsequent strengthening of CRs, and postasymptotic frequency of CRs.

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Received August 17, 1979 ■