HEDONIC VERSUS PREDICTIVE INHIBITION OF
AVOIDANCE RESPONDING IN RATS

THESIS

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By

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Traditional two-process theory predicts that a conditioned stimulus (CS) paired with shock offset on Pavlovian trials will inhibit operant avoidance responding. Two explanations of the inhibitory mechanism involved were compared: contemporaneous pairing of CS with a hedonic relief reaction versus the predictive, discriminative relationship of CS to the non-shock interval. The pattern of avoidance inhibition associated with cessation CSs paired with electric shocks of constant duration was expected to be different from the pattern accompanying cessation CSs paired with shocks of variable duration. Mean rates of responding by the two groups were compared by analysis of covariance using baseline as the covariate. Neither CS displayed any reliably observable effects on avoidance rates. Possible procedural flaws and compatible improvements are discussed.
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Biologists have long recognized the binary homeostatic mechanisms which govern the physical state of most organisms. Recently, writers and experimenters in the physiological and perceptual fields of psychology borrowed that concept to explain the phenomena they have observed. A prime example is the functioning of the endocrine system, where blood-glucose, water retention, and body temperature levels, to name a few, are maintained at balanced, or homeostatic, levels from which dynamic departures can occur and be controlled. This balance is maintained via systemic pressures which are mutually excitatory and inhibitory, thus arousing and maintaining or terminating endocrine activity according to the biological needs of the organism (Selye, 1950; Young, 1955).

A similar analysis has often been applied to neural functions (Milner, 1970). Most descriptions of the neural spike have insisted on a homeostatic interpretation of the balance of sodium and potassium ions within and without the axon. From this balance of positive and negative chemical charges, occur dynamic departures in electro-chemical voltage, forced by the presence of the highly negative charges of the glutamate, aspartate, and fumarate compounds. The limbic system of subcortical neural structures, which
presumably governs emotive and motivational levels, functions, by mutual excitation and inhibition of neural activity, to maintain a homeostatic balance, or base-rate, in behavioral terminology.

The concept of homeostasis, so fruitfully applied to the phenomena cited above, was an assumption central to the opponent-process theory of sensory adaptation advanced by Hurvich and Jameson (1957). Their paper explained the negative after-images which occur at the termination of intense visual stimulation and which slowly fade out (example: green after-image to red stimulus). To account for their experimental results, Hurvich and Jameson described a secondary, unconditioned sensory reaction to the unconditioned stimulus, which serves to reduce the intensity of the response. Such an approach would, it was hoped, account for sensory adaptation phenomena. Thus, the concept of dynamic homeostasis has organized, with minor adjustments, numerous observations of glandular, neural, and sensory mechanisms, but, until recently, few molar behavioral mechanisms.

Solomon and Corbit (1974), crediting the Hurvich-Jameson model as an idea source, formed a theory of motivation which is homeostatic and hedonic or affective in character. It is essentially a heuristic device, calling on directly unobservable and unspecified underlying processes which govern emotive behavior. However, it could be a promising model
describing subcortical neural activity, specifically that of the limbic system, controlling emotive responses. An outline of the Solomon-Corbit opponent-process model of motivation is presented here, but for a full understanding of the model and its predictions, the reader is invited to examine the original report.

Initially, it is hypothesized in the model that hedonic or affective states are opposed by central nervous system (CNS) mechanisms which serve to reduce the intensity of the former, producing emotive homeostasis. In other words, these CNS mechanisms were postulated as forces which control and stabilize motivational levels or predispositions to respond in certain ways. These hedonic states and their control mechanisms were derived from two hypothesized underlying processes. The first, referred to as the \( a \) process, was described as an unconditioned response to an emotive stimulus, producing hedonic reactions—such as fear, joy, and anxiety—to stimulation, such as automobile accidents, love, and graduate study, respectively.

The \( a \) process automatically triggers a characteristic \( b \) process which is hedonically opposed to the \( a \) process eliciting it. This \( b \) process was described by Solomon and Corbit as a slave-process, "inaccessible to direct environmental inputs, but indirectly arousable via hedonic and affective processes elicited by environmental inputs" (Solomon & Corbit, 1974, p. 129). Thus, the unconditioned
reaction to any emotion-producing stimulus has two components: the \( a \) process, which arises immediately upon stimulus onset, stabilizes and dies at stimulus offset, and the \( b \) process, which is also aroused, indirectly, by the unconditional stimulus (US), and has a longer latency, a slower recruitment time, and a longer decay function.

The writers hypothesized a summative mechanism which determines the sign and intensity of the hedonic state of the organism. To unconditioned stimuli that are positive in nature (appetitive conditioning), the \( A \) state is positive in sign and pleasurable. The \( B \) state, which occurs at US offset and the subsequent disappearance of the \( a \) process, has a negative hedonic sign and is unpleasurable. The reverse is true for hedonic responses to aversive stimuli (affectively negative). The preceding was merely an extension of the ideas of Miller and Dollard (1941), Pavlov (1955), and Young (1955). These hedonic states can be inferred, their sign and intensity determined, by their behavioral manifestations, in consonance with the statements of Kimmel (1971) as well as Rescorla and LoLordo (1965). Thus, when a stimulus is presented, the \( a \) process rises quickly above baseline, peaks, and subsides to a steady level, still above baseline, as the \( b \) process begins to recruit. Then, at termination of the US, the \( a \) process dies, the \( A \) state disappears, leaving only the \( b \) process and its attendant \( B \) state, characterized by a drop below, and gradual recovery to, the pre-stimulus level.
Thus, the \( b \) process controls the intensity of any affective reaction, in a manner conceptually similar to the anxiety reduction notions of "conservation of anxiety" (Solomon & Wynne, 1953), "fear management" (Kimmel, 1971), and "adaptive habituation" (Hilgard & Bower, 1966).

To account for the effects of extended exposure to a stimulus, Solomon and Corbit (1974) described the \( b \) process as subject to strengthening through use and weakening through disuse. When a novel stimulus appears in an organism's environment, the \( a \) process is aroused at full strength and remains relatively stable, but the \( b \) process is weak and nearly undetectable. However, with repetition of the \( a \)-eliciting stimulus, the \( b \) process is indirectly aroused and strengthened, whereupon certain quantitative changes occur. While the \( a \) process is largely unaffected by any exposure parameters, the \( b \) process, now labeled \( b' \), has become more intense, its latency shorter and its decay function longer. By summation with \( a \), the \( b' \) process has relegated the \( A \) state to a weaker version (\( A' \)) and has made the \( B' \) state more powerful and longer lasting than the unaltered \( B \) state. Conversely, disuse weakens the \( b \) process and the \( A' \) and \( B' \) states revert to their original strengths.

The model is essentially non-associative, describing unconditioned neural and bio-chemical responses to emotive stimuli; yet, by utilizing certain two-process learning theory assumptions, it can account for a large body of
conditioning data. Kimmel (1971) and Overmier and Seligman (1967) reported experimental results (i.e., inhibition of emotional behavior and acquisition interference, respectively) from inescapable shock presentations, which, apparently, were not effected by stimulus exposure parameters. Although more molecular than Solomon's, these conceptualizations are similar to his in that they describe non-associatively acquired motivations which affect instrumental responding.

Solomon and Corbit asserted, from a stance characteristic of two-process learning theorists, that many conditioning phenomena can be organized or predicted by their non-associative model. To reach that end, the theory assumes that both A and B states (or A' and B' states) can "be brought under the control of previously neutral stimuli as a consequence of experience" (Solomon & Corbit, 1974, p. 133). Presentation of a stimulus repeatedly paired with the A state, for example, would evoke a conditioned response having emotive characteristics similar to the unconditioned A state. State-conditioning was stressed here, as opposed to process conditioning, which Solomon and Corbit did not postulate. The model holds, then, that a CS+ (conditioned stimulus paired with the onset of US and the A state) would evoke a conditioned A state, and its offset, like that of the US, would terminate the A state, leaving only the B state to peak and decay. However, a CS- (conditioned stimulus paired with US offset) would be paired with the peak of the B state.
and could, in a test session, elicit a conditioned B state, without having been preceded by the US. To add cohesiveness to the conditioning predictions, the authors assumed a contemporaneous conditioning process, associating an event with the characteristics of the concurrent hedonic state. In fact, the authors specifically rejected drive-reduction or expectancy notions in favor of conditioned hedonic reactions, controlling response topography and rate (Solomon & Corbit, 1974, p. 135).

As mentioned earlier, the opponent-process model offered certain predictions about conditioning paradigms. These involve the non-associative alterations in the intensity of the b process (dependent on US exposure parameters), the establishment of conditioned stimuli which trigger the onset of hedonic states, and the effect of these acquired motivational states on instrumental responding. A CS+, in immediate contiguity with the onset of a US and the a process it elicits, then, would evoke a conditioned A state during extinction trials, and would be followed, at its offset, by the appearance of a B state, gradually decaying to baseline in time. However, if the b process has been strengthened through repetition of the US, the summation of a with the strong b' process would reduce the intensity of the A state reaction to a level very near baseline, attenuating the excitatory function of CS+. In contrast, the
"pure b" state (B') would be much more intense and longer-lasting.

From the preceding analysis, the properties of CS+ in aversive conditioning and its effect on ongoing avoidance responding are easily drawn. A CS+, classically conditioned with electric shock onset, enhances or facilitates the avoidance rate. In other words, upon CS+ presentation, a weak relative of the A state is evoked and the avoidance rate rises sharply above baseline, levels off, drops sharply below baseline at offset, and gradually returns to baseline. A CS+, then, facilitates avoidance responding and results in a bi-phasic recovery to pre-stimulus levels. If, however, the b process has been strengthened, CS+ evokes state A' (i.e., CS+ is a weak fear elicitor), which is followed by the powerful B' state. Potentially, at least, an explanation has been found for such phenomena as "learned helplessness" (Overmier & Seligman, 1967), "interference of avoidance" (Kimmel, Kimmel & Silver, 1969), and "failures to learn" (Turner & Solomon, 1962).

A previously neutral stimulus, classically associated with the offset of an aversive US, has been labeled a Pavlovian, backward-conditioned stimulus, or CS-. According to Solomon and Corbit, a CS-, occurring immediately following offset of an electric shock, is paired with the peak of the B state, and is, therefore, a maximally powerful CS-, or CS_b, in the language of the model. Presentation of CS-
during ongoing avoidance responding should depress the rate of that responding, allowing a monotonic recovery to base rate. As the $b$ process strengthens, the aversiveness of the US attenuates and the positive hedonic qualities of CS- increase (Solomon & Corbit, 1974, p. 141), enhancing the power of CS- to function as a conditioned inhibitor of fear. The onset, then, of CS- takes on the safety signal characteristics of the offset of US or CS+, and may very well lead to identification of the locus of reinforcement in avoidance, a problem which has resisted theoretical solution for some time (Rescorla, 1968; Wiesman, Denny, & Zerbolio, 1967; Wiesman & Litner, 1969).

In support of the opponent-process model of motivation, Solomon and Corbit cited the observations of several researchers, a few of which are to be considered here. The initial question which arises concerns the accuracy with which the postulated $b$ process describes phenomena of acquired motivation. Rescorla and LoLordo (1965) reported the development of a sensitive index of Pavlovian conditioned inhibitor and facilitator control over avoidance responding, which required three stages: (a) Sidman (1953) unsignaled avoidance conditioning, (b) Pavlovian conditioning, and (c) observation of conditioned stimulus effects on Sidman avoidance. Any change in the avoidance rate during a stimulus presentation reflects the properties of that stimulus. In the first of three experiments, Rescorla and
LoLordo (1965) established a CS+ which signaled that either a shock or another tone, CS-, would follow at an interval of 2 to 8 seconds. CS- signaled an inter-trial interval (ITI) during which no shock would occur. When presented during Sidman avoidance, the rate of responding increased during CS+ and decreased at onset of CS-. Both departures from baseline were statistically significant, and both conditioned stimuli had effects which were predicted by the Solomon-Corbit model. Response rates showed a monotonic return to pre-stimulus levels after both stimuli, which was a predicted result for CS- effects. However, the effects of CS+ presentations did not follow the prediction of a bi-phasic recovery. Although Rescorla and LoLordo (1965) did not discuss the possibility, that result may have been due to the temporal effects of the trace conditioning procedure.

Rescorla and LoLordo (1965, Experiment 2) adjusted the shock parameters for their second experiment so that shock onset occurred at offset of CS+. The changes in avoidance rate during CS+ and CS- presentations occurred, as they had in the first experiment, according to the model predictions. Again, the decreased response rate during CS- monotonically returned to baseline, but CS+ offset, contrary to the previous results and in accordance with the model, depressed responding below baseline. In other words, CS+ presentations evoked a predicted bi-phasic departure in avoidance rate from pre-stimulus levels.
To observe the course of acquisition of the Pavlovian facilitators and inhibitors of fear, Wiesman and Litner (1969) replicated the shock parameters of the second Rescorla and LoLordo (1965) experiment, but measured the effects of conditioned stimuli after each of four conditioning sessions. During CS presentations, the rates of responding changed from the base rate, and the rate after CS- gradually rose toward baseline, all as predicted. Contradicting the model and Rescorla and LoLordo's (1965, Experiment 2) results, CS+ offset did not inhibit fear. One other result Wiesman and Litner reported was that the function of CS+ developed rapidly and remained stable across all test sessions, while CS- acquired its function much more gradually. This observation will be considered again, and in greater detail.

In all of the experiments cited previously, CS+ elicited fear and CS- inhibited fear, assuming avoidance responding rates to be an accurate reflection of fear intensity. Also, the gradual return to baseline after the sharp drop in avoidance frequency during CS- was as expected from the opponent-process model predictions. However, while the offset of CS+ inhibited fear in one paradigm reported by Rescorla and LoLordo (1965, Experiment 2), it did not reduce avoidance rates in the remaining paradigms of Rescorla and LoLordo (1965) or of Wiesman and Litner (1969). It is, of course, possible that the results of CS+ presentations were
unreliable and contradictory because the conditioning trials establishing the excitatory stimuli did not accurately fit the model. So the question remains: Does the B state manifest itself at offset of CS+ or US, as predicted by Solomon's hedonic analysis? Due to the unreliability of CS+ results and the difficulty of arranging a CS+/US relationship that closely adheres to the opponent-process model, observations of the function of CS- may hold more promise than those of CS+.

The observations by Rescorla and LoLordo (1965) and by Wiesman and Litner (1969) of the effects of CS- presentations have paralleled several predictions by Solomon and Corbit of B state conditioning. In all paradigms reported, CS- inhibited avoidance, evoked a monotonic recovery, and acquired its function more slowly than CS+. The theorists took this as an indication that a CS, paired with the peak of the B state, acquired the stimulus properties necessary to trigger the onset of a weaker relative of the B state. Also, by its gradual growth curve, as contrasted by the more rapid growth of CS+ functions, CS- function observations mirrored the "strength by use" property of the postulated b process, and its attendant quantitative change of B to B'. The acquired functions of CS- in the third paradigm reported by Rescorla and LoLordo (1965) and the first of Wiesman and Litner (1969) also paralleled the opponent-process model. In both experiments, CS- inhibited avoidance behavior and displayed a
gradual recovery of responding rate to pre-stimulus levels. CS- presentations, however, always occurred alone during Pavlovian conditioning, following the previous stimulus presentations by at least 90 seconds. Due to the long ITI between CS- and offset of the previous US, CS- could not have been consistently paired with the peak of the B state, as Solomon and Corbit described it.

The writers recognized that the backward CS in the two previous experiments could not "have been regularly paired with the peak of B, because the peak of B would have occurred very shortly after the last shock termination" (Solomon & Corbit, 1974, pp. 134-135). To explain the incongruous result, Solomon and Corbit asserted that after so many elicitations of the a process, the b process would have become extremely powerful and long-lasting, thus pairing CS- with a level of the B' state still far below baseline. Also, the authors assumed, as was mentioned earlier, "contemporaneous conditioning, an event-state conditioning process" (Solomon & Corbit, 1974, p. 135), which they admitted is central to the hedonic model and, for which, they admitted no empirical support. These are assumptions to be questioned. First, if the b process had been strengthened sufficiently for the peak level of the B state to have sustained itself for 90 seconds or more, the A state should have been relegated to its A' condition, where CS+ would have been a weak fear elicitor. However, Wiesman and Litner (1969) reported
no attenuation of CS+ effects on responding, and Rescorla and LoLordo (1965) reported none in their first two experiments, where subjects received roughly the same number of shocks as subjects in the third.

CS- was obviously capable of inhibiting fear and, thus, fulfilled the predictions of the opponent-process model. Yet, the means by which its inhibitory function was acquired was not explainable by that model. Moskovitch and LoLordo (1968) sought to discover the properties actually contributing to CS- inhibition by comparing the effects of backward CSs (CS onset occurs after US offset) to cessation CSs (CS onset occurs after US onset but prior to US offset). During the test sessions, backward CS- presentations resulted in decreases in avoidance rates, followed by a gradual recovery to baseline. To establish such a CS-, the experimenter presented a shock (US), the offset of which was followed by a tone (CS-) one second later. According to the analysis of Solomon and Corbit, this procedure paired the onset of CS- with the peak of the B' state, creating a maximally powerful fear inhibitor. Thus, both the conditioning trials and the test results fit the hedonic model, offering it rather unambiguous support.

Subjects in Group C of Moskovitch and LoLordo's first experiment received cessation conditioning (i.e., tone onset occurred at one second prior to shock termination). Konorski (1948) and Mowrer (1960) predicted that a CS which
predicts shock termination and the concurrent onset of a safety interval should produce a greater decrement in avoidance rate than other Pavlovian conditioned inhibitors. During test sessions, however, the decrease in rate during cessation CS- presentations was negligible, while CS- offset was accompanied by a drop in rate equal to that of the post-CS periods in the backward conditioning group. In other words, CS- offset produced greater inhibition than did its onset. Since the cessation CS- onset was paired with the hedonic A state and its offset with the peak of the B state, the function of CS- was expected by Solomon and Corbit. In the affective framework, CS- onset triggered a relative of the A state, and its offset evoked a conditioned B state, presumably observed as avoidance rate fluctuations.

As an explanation of the unexpected failure of the cessation CS- to inhibit fear during its presentation, Moskovitch and LoLordo offered three observations. The first was that the constancy of avoidance rate during the cessation CS presentations could have been due to a stimulus generalization decrement since shock did not precede CS in test trials. A second observation, and one more relevant to the current considerations, was that the onset of CS- was a redundant predictor of shock termination in Pavlovian trials, and thus did not acquire inhibitory properties. Shock duration did not vary widely in that experiment, so the onset of shock could have provided the information about its
offset, which, theoretically, would have been attributed to 
CS- by an expectancy model. The third option was that the 
rate of responding during CS- did not change from pre-
stimulus levels because CS- had acquired some fear-inducing 
qualities from its temporal overlap with shock, or with the 
A state of the affective model being considered here. This 
interpretation, if substantiated, would lend weight to the 
predictions of Solomon and Corbit.

Moskovitch and LoLordo included a second experiment in 
their 1968 report in which they established two types of 
backward conditioned stimuli during Pavlovian conditioning. 
One group of subjects received a tone (CS-) which followed a 
shock of 15 seconds and always predicted a shock-free ITI of 
two or three minutes. The backward CS- in this experiment 
effected the same changes in avoidance rate as did the back-
ward CS- of Experiment 1, which followed US offset by one 
second. Again, the CS- presentation on Pavlovian trials was 
not concurrent with the peak of B, a pairing necessary for 
the acquisition of an inhibitory function according to the 
hedonic analysis. The objection here, though, is less sub-
stantial than in the cases of the first and third experiments 
reported by Wiesman and Litner (1969) and Rescorla and 
LoLordo (1965), respectively. A second group received a 
tone following shock termination by one second, and followed 
by an ITI which varied randomly from 0-15 minutes. Conse-
quently, the tone immediately followed shock termination and
temporally coincided with the peak of Solomon's B state, but
did not reliably predict safety from shock. During test
sessions, this CS- evoked no change in Sidman avoidance
rates.

In these experiments, backward-conditioned stimuli have
inhibited avoidance rates whenever they had reliably pre-
dicted safety intervals in Pavlovian conditioning. Two
groups of researchers (Rescorla & LoLordo, 1965; Wiesman &
Litner, 1969) deduced from their observations that a backward
CS- acquires its inhibitory function via its explicit pairing
with a period free from shock, and not necessarily its pair-
ing with shock offset. These writers qualified their
positions by stating that not only must it be paired with a
non-shock interval, but a CS-, to depress avoidance rates,
"must occur without shock against a background in which
shock does in fact occur" (Rescorla & LoLordo, 1965, p. 411).
Had the opponent-process model been revised to take all these
observations into consideration, it would have postulated
certain predictive qualities of US termination of CS- onset
to be the source of conditioned B state onset, rather than a
contemporaneous conditioning process as its source. In
other words, once shock has been experienced in a certain
stimulus background, the a process might not die until the
stimulus background which threatened shock has been removed
or negated in some fashion.
The results of cessation CS- presentations, however, did not prompt such an alteration of the hedonic analysis. If the predictive qualities, as suggested earlier by the backward CS results, were the critical aspect of an inhibitory stimulus, a CS which predicted extremely proximal shock termination should have acquired properties which inhibit fear during its presentation. That is not what happened. Instead, the cessation CS- seemed to have acquired the properties of the A state with which it was contemporaneously paired, lending support to the state-conditioning assertions of Solomon and Corbit. On balance, the forward-predictive qualities of CS- seem much more critical to its acquisition of inhibitory qualities, yet the effects of the cessation CS on instrumental responding have prevented a unilateral rejection of the notion of contemporaneous, hedonic state-conditioning. As will be remembered, however, Moskovitch and LoLordo pointed out that their results might have been confounded. To restate their explanation, the authors had attributed the relatively inefficient inhibition of fear by the cessation CS- to three possible factors. First, "The absence of large inhibitory effects during the cessation CS might be attributed to stimulus generalization decrement, due to the fact that the usual ongoing shock was not present during CS onset in test trials" (Moskovitch & LoLordo, 1968, p. 676). However, that experiment assessed the effects on avoidance responding of a cessation CS- and a backward CS-
under identical test conditions. Yet the result in question was not common to both inhibitors. Consequently, this explanation cannot be considered seriously as an explanation of the minimal inhibition during the cessation CS-. The second explanation, and a more viable one, pointed to the temporal overlap of the US and CS- onset. Mirroring the later statements of Solomon and Corbit, Moskovitch and LoLordo indicated that the onset of CS- might have acquired some fear-inducing properties due to the temporal contiguity of the two stimuli. Whether by a CS-US pairing, espoused by Tolman and others, or by pairing of CS and UR, favored by Solomon and other S-R theorists, such an explanation requires an association by a contemporaneous conditioning process. The third hypothesis indicted the redundancy of CS- onset, as a predictor of shock offset, for its relatively inefficient inhibitory qualities. Although Moskovitch and LoLordo varied shock durations by increments of one second, they regarded US duration, for purposes of interpretation, as a constant. Apparently, then, shock onset was the operative predictor of its own offset and, thus, the onset of a safety interval, limiting the importance of CS-onset in that capacity.

The present experiment was designed to assess the relative attractiveness of two of the explanations offered by Moskovitch and LoLordo of the results of their cessation conditioning procedure. Two groups of subjects received
unsigned two-way avoidance training followed by classical, cessation conditioning, the difference being that one treatment delivered Type I shocks which were constant in duration while the other treatment varied shock duration to a greater degree than had Moskovitch and LoLordo. Presentation, during operant avoidance responding, of a cessation CS-, previously paired with a shock of constant duration, was expected to trigger a gradual drop in avoidance rate, followed by a monotonic recovery to the pre-CS rate over a period 10-30 seconds after CS- offset. Such a result would have indicated consistency and cross-species generality of the effect being examined here, and was considered a necessary prerequisite for a viable test of the Solomon-Corbit hypothesis. Pairing of a cessation CS- with a shock of variable duration should have reduced or eliminated the redundancy of CS- onset, while retaining the temporal overlap. If that overlap of US offset and CS onset was the relationship critical to acquisition by CS- of safety-signal characteristics then the response pattern should be similar to the response patterns observed in the former group of this experiment and to those reported by Moskovitch and LoLordo. However, if the predictive characteristics of CS- were more important, then the asymptote of response inhibition should have been observed during CS- presentations.

It was hypothesized, then, that Group C should display minimal, and Group V, maximal, inhibition during test
presentations of CS- . Both groups should display maximal inhibition during the first post-CS interval, followed by similar patterns of recovery to the pre-CS rate.

Method

Subjects

Experimental subjects were 16 naive males obtained from the Sprague-Dawley strain of albino rats. Subjects were maintained in individual cages on ad lib. food and water, and were tested at an average weight of 200 grams.

Apparatus

A shuttle box was used for all conditioning and testing sessions. The two compartments, 14" x 10" x 8" each, were separated by a drop gate barrier which was adjustable in height. On Pavlovian conditioning days, the barrier prevented subjects from crossing to the opposite chamber, while it provided a 1.5" hurdle during avoidance training sessions. The floor consisted of 1/8" tubular, stainless steel grids which could be electrified by a Lafayette Instruments master shocker through a scrambling device. Momentary illumination of two 15-watt lamps above a white plexiglass ceiling served as conditioned stimuli. Electric shocks and light-intensity variations were programmed through a remote, automatic apparatus, while these events were recorded by an Esterline-Angus operations recorder.
Procedure

On Days 1-3, 5, 7, 9, and 11, subjects received a one-hour session of unsignaled, two-way avoidance training, as described by Sidman (1953). If a shuttle response was not emitted during the specified interval, a 0.5-second, 0.3-ma electric shock was delivered to the grid floor. In the absence of the appropriate avoidance response, the foot-shocks were delivered at 5-second intervals. Each successful response postponed the next shock presentation for 20 seconds. Thus, the US-US interval was 5 seconds, and the R-US interval, 20 seconds.

Pavlovian conditioning sessions were administered on Days 4, 6, 8, and 10. During each of these sessions, subjects were confined to one-half of the shuttle box, while experiencing 33 conditioning trials. The trial series followed a 10-minute warm-up/exploratory period at the beginning of each Pavlovian session. Each trial consisted of US (0.3-ma electric shock) presentation followed by a cessation CS- of 5 seconds duration, occurring at 1 second prior to US offset. Illumination of a 15-watt lamp served as the CS. The inter-trial interval (ITI) from CS- offset to US onset was of 60-, 90- and 120-second durations, with a mean ITI of 90 seconds. For the 8 subjects in Group C, US duration was constant at four seconds, while for Group V, US duration varied among 2, 4 and 6 seconds, with equal numbers of each type trial presented randomly.
During the test session on Day 12, shocks were presented as programmed for earlier Sidman sessions during the first 10 minutes of the session. After the 10-minute warm-up period, a series of 33 extinction presentations of CS- was initiated, as programmed in earlier Pavlovian sessions. No shocks were presented during the test session.

As in the Moskovitch and LoLordo (1968) procedure modified here, the effects of CS- were assessed for each subject by examining the mean jumping rates for the 15-second period before, the 5-second period during, and the 30-second period immediately following CS presentations. Each 50-second trial was divided into two 5-second blocks. The mean rate of jumping per block during the 15-second period immediately preceding CS presentation was taken as the base rate.

Results

In order to accurately assess the stimulus control by CS- over operant avoidance responding, the rate of that responding must, necessarily, be stable and relatively high. Consequently, it was decided that, to be retained for statistical manipulation, a subject must successfully avoid at least 75 per cent of all possible US presentations on Day 11. Subject #10 was dropped from Group V due to its failure to learn the appropriate avoidance response, and subject #7 was dropped from Group C due to equipment malfunctions. All remaining subjects met the arbitrary criterion on Day 11,
leaving seven subjects in each group for statistical manipulation.

Baseline was computed as the mean response rate of each subject per 5-second interval. This was accomplished by combining the response rates in each pre-CS block for all subjects and dividing that total by \( N \times 3 \). The dependent variable (i.e., number of responses per 5-second interval) was measured at seven points, during and after CS-presentations, and was totalled across trials. The resultant response totals were distributed among the 14 cells of a \( 2 \times 7 \) table and subjected to a repeated measures analysis of covariance employing the base rate as the concomitant variable. That procedure was designed to statistically eliminate the influence of any irrelevant factors, which were not controlled for procedurally, from the dependent measure. As an intermediate step in that process, an analysis of covariance generates adjusted means for each cell which are equated with respect to the concomitant variable, in this case, baseline. The adjusted means appear in Table 1.

The analysis of covariance generated a ratio of variances, \( F (6,71) = 1.799 \), which indicated no statistically significant differences occurring within groups. The probability that the differences among all means (within subjects) were chance fluctuations about a single population mean was at \( p = 0.1116 \). The \( F \) for between-groups variance,
Table 1
Summary of Adjusted Mean Response Rates

<table>
<thead>
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<th>Treatment</th>
<th>During- and post- CS intervals</th>
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<td></td>
<td>CS</td>
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\( F(1,11) = 2.5656, p > .05 \), allowed no empirical confidence in any ostensibly observable differences in rate changes between groups. These Fs were generated after compression of the cell means about a regression line, with slope \( (β = 0.7486) \), an adjustment operation which, apparently, had no substantial effect on the dispersion of cell means about that regression line. The inference from the above is that the variability of criterion means is largely explained by the variability of the covariate. The residual variance was minimal, \( F(6,71) = 0.9559, p > .05 \), suggesting virtually no lack of additivity of the main effects. Put more simply, the amount of variability of criterion measures left unexplained by the covariate dispersion was negligible. A summary of this analysis of covariance appears in Table 2.

In conclusion, the insignificant result of the test for main effects indicated that CS-presentations demonstrated
Table 2
Analysis of Covariance of Mean Response Rates

<table>
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<th>Source</th>
<th>df</th>
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<td>Between Subjects</td>
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<td>6.2104</td>
<td></td>
</tr>
<tr>
<td>Rows (A)</td>
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<td>14.0945</td>
<td>2.5656</td>
</tr>
<tr>
<td>Subject within Rows (C)</td>
<td>11</td>
<td>5.4937</td>
<td></td>
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<tr>
<td>Within Subjects</td>
<td>83</td>
<td>3.1222</td>
<td></td>
</tr>
<tr>
<td>Columns (B)</td>
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<td>5.3265</td>
<td>1.7991</td>
</tr>
<tr>
<td>Interaction (A &amp; B)</td>
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<td>0.9559</td>
</tr>
<tr>
<td>B x C</td>
<td>71</td>
<td>2.9606</td>
<td></td>
</tr>
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</table>

no consistently observable control over avoidance responding by either group. Secondly, the negligible interaction reflected the absence of any systematic differences between groups in their temporal patterns of response inhibition, and the fact that no distinct effects can be attributed to differences in treatments.

Discussion

The primary aim of this investigation was to identify which aspects of a US-CS relationship were critical to the establishment of a classical safety signal. Moskovitch and LoLordo had, in 1968, demonstrated inhibition of avoidance responding by a cessation CS-, but that inhibition displayed an unexpected temporal pattern. It was hoped that certain
manipulations of that US-CS relationship might be accompanied by changes in the temporal location of the lower asymptote of whatever inhibition might be observed. However, the anticipated replication by Group C, considered a necessary prerequisite for a test of the Moskovitch and LoLordo suggestions, did not occur. It is quite possible, of course, that the failure to replicate, by Group C, was due to a lack of species generality of the previously observed phenomenon. Regardless of the cause, the failure of the treatment administered to Group C to demonstrate any observable effect on avoidance rates precludes any critical examination of the primary hypotheses. The administration to Group V, of a treatment designed to assess the value of suggestions by Moskovitch and LoLordo, also failed to affect responding. That neither treatment produced effective safety signals allows no theoretical evaluation of the procedural suggestions of Moskovitch and LoLordo (1968) or of the theoretical assertions of Solomon and Corbit (1974). That all of the studies known to this author describing similar treatments reported successful inhibition of avoidance responding indicates that the apparent failure reported here was due to procedural flaws rather than to inaccuracies in previous formulations.

The first of three potential flaws which have come to my attention is limited to the operant-avoidance training phase of the experiment. Several researchers (notably
Denny & Ratner, 1970; and Testa, 1974) have described the difficulty of learning the two-way avoidance task, and its ease of extinction. For whatever reasons, the shuttle response rates of subjects in this experiment were not uniformly high and stable as was the case in other investigations. If it is conceivable that the avoidance rate in this experiment was not an optimally accurate correlate of fear intensity, then any changes in fear level associated with CS presentations could not have been measured precisely. Thus, the effects of the CS associated with the B state would have remained covert. Remediation of this obstacle might be achieved by employing a more stringent performance criterion as a prerequisite to statistical treatment, or the use of an entirely different response measure.

The second potentially extraneous influence originates from the possibility that the light-CS was not a neutral stimulus, but held some unconditioned, innate stimulus properties. Although the aversiveness of intense light to white rats has been well documented (Hanson, 1951; Hefferline, 1950; Jerome, Moody, Connor & Fernandez, 1957; Kaplan, 1952, 1957; Kaplan, Jackson & Sparer, 1965; and Keller, 1941), only 10 per cent of the literature specifies the intensity of the light stimulus even when it was the primary variable (Lockard, 1964). Due to the unavailability of an illuminometer or of transformation formulae, this report, of necessity, must join the majority. In the interest of
specificity, it might be inferred, from what little information is available, that the light employed as a CS in this investigation was of sufficient intensity to be inherently aversive. Hanson argued in 1951 that the intensity threshold of light-aversion in rats was at 1.076 millilamberts (ml). In 1952, Kaplan obtained avoidance responses from several light intensities, specifying the luminosity of a 15-watt lamp in a white chamber at 111 ml. CS presentation in the treatments reported here consisted of the illumination of two 15-watt lamps, within a highly reflective, brushed aluminum chamber. This increment in direct and ambient brightness could easily have been sufficient to produce aversion in a subject having minimally adapted receptors. If such was the case, the relationship of a less than neutral CS to shock and the safe B state could conceivably have been negated by the unconditioned aversive qualities of intense light. A more neutral stimulus, then, would likely facilitate the acquisition of its intended function.

Popham (1967, p. 230) has suggested that the analysis of covariance technique is not as robust (i.e., is less able to tolerate marked departures from the requisite assumptions) as several other procedures. He went on to point out that it is nearly impossible to demonstrate that samples of similar size to those reported here satisfy the assumptions necessary to the appropriate use of covariance techniques (p. 234). It is conceivable then that those assumptions may
very easily have been violated. If such was the case, the lack of robusticity of the covariance technique would necessarily reduce its sensitivity to whatever effects the two treatments may have had. If any effects due to treatment were existent, they obviously were not large enough to be detected through observations of such small samples. A larger $N$ would have decreased the chance of a $\beta$ error and at the same time, while reducing $\alpha$ also, would have increased the accuracy and power of the statistic. The means of during- and post-CS measurements did not fluctuate as markedly or as consistently as those reported in studies cited earlier, although the number of subjects ($N = 7$) per group was approximately the same for all experiments. With the exception of the less robust character of the analysis of covariance technique, in comparison with those techniques utilized in the precedents of the current investigation, the power of the analysis ($1-\beta$) is probably not substantially different from that reported by earlier experimenters. It must be assumed, then, that acceptance of the null hypotheses was an accurate deduction as it applies to the current samples.

While the null hypotheses are probably true for these samples, the possibility certainly exists that they may be false in regard to the population. The reported results of similar treatments suggest that such is the case, since reported observations of fear inhibition attendant to CS-
presentations have been markedly consistent. The conclusion drawn here is that certain procedural flaws prevented these samples from demonstrating acquired inhibition of avoidance responding. The substitution of a more neutral CS, and the measurement of a more easily acquired response index may eliminate the barriers discussed earlier. In addition, a more stringent, operant-performance criterion may reduce the variability among base rates, and eliminate the need for the adjustment operation of covariance analysis. The subsequent improvement in homogeneity among responding rates would allow the use of a statistical technique which would be less susceptible to any violations of its assumptions. If employed, these suggestions may lead to a more productive examination of the assertions of Solomon and Corbit.
BIBLIOGRAPHY


