UNCONDITIONED STIMULUS INTENSITY AND CONDITIONED INHIBITION OF FEARFULNESS

by

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INTRODUCTION

Pavlov (1927) asserted that reflexes were "the elemental units in the mechanism of perpetual equilibration," and that they constituted the foundation of the nervous activities of both men and animals. He distinguished between those reflexes that were inborn or unconditioned and those which were acquired or conditioned during the life of the organism. Using the language of the classical physiologist, he suggested that reflexes could be either excitatory or inhibitory, and viewed excitation and inhibition as fundamental properties, the two most important manifestations of activity in living nervous elements. Despite the bias in American psychology in favor of research on excitatory associative processes, the application of the excitatory-inhibitory distinction to behavioral phenomena has weathered psychology's usurpation of the conditioned reflex, and this distinction remains very much a part of present-day learning theory (see Boakes & Halliday, 1972).

Conditioned excitation is often typified by Pavlov's finding that a behaviorally "neutral" stimulus event (such as a light or tone) will acquire the ability to produce an increase in salivation as a result of repeated pairings with food or acid in the mouth. In general terms, the conditioned stimulus (CS) is said to acquire the ability to elicit a conditioned response (CR) as a result of repeated pairings with an unconditioned stimulus (US). In contrast, conditioned inhibition (Pavlov's "internal inhibition") refers to an acquired tendency that is opposite that produced by the conditioned excitor, and is usually thought to result from a negative

relation between the CS and US. When presented together with a conditioned excitatory stimulus, a conditioned inhibitor presumably will actively suppress the excitatory response tendency.

The present dissertation is concerned with conditioned excitation and inhibition in Pavlovian fear conditioning, and in particular, with the role that excitatory strength plays in determining strength of inhibition during the learning process. Before considering that relation, however, the definition and measurement of acquired fear and its inhibition must be examined, as well as the conditions that are believed to affect the development of those tendencies.

The Definition of Conditioned Fearfulness and Conditioned Inhibition of Fearfulness

The early formulations of Mowrer (Mowrer, 1939, 1947; Mowrer & Lamoreaux, 1942, 1946) and Miller (1948, 1951) did much to promote the idea that the learning of emotional reactions might best be viewed in terms of Pavlovian conditioning. Following the Pavlovian schema, they suggested that by being paired with some aversive (painful) event, a formerly neutral stimulus could acquire the ability to evoke an emotional response (fear) in anticipation of the aversive event. In the context of their larger views of the interaction between associative and motivational processes, they suggested further that the internal stimulus consequences of the fear reaction had drive-arousing properties, and that a reduction in fear could reinforce a contingent response. This type of analysis was used to interpret behavior in signalled-avoidance paradigms and in "acquired-drive" paradigms, and has been extended

to a variety of other situations (see McAllister & McAllister, 1971; Rescorla & Solomon, 1967).

Although the formulations of Mowrer and Miller clearly implicated an aversive US and its paired relation to some antedating stimulus event, it was not clear that those conditions by themselves defined fear. The question arises, for example, whether a stimulus that has been paired with an aversive US, yet fails to exert a motivational effect, is still to be regarded as eliciting fear. Mowrer's (1947) suggestion that the fear response was a complex conditioned visceral reaction generated a good deal of research into the relationship among fear conditioning procedures, changes in a variety of physiological measures, and performance on tasks alleged to be dependent upon conditioned fear (see Rescorla & Solomon, 1967). This concern, however, only postponed consideration of the problem of definition. What if physiological response measures failed to correlate with behavioral response measures or with variations in conditioning parameters? Had fear been improperly defined by that measure or was fear simply not related to the target task?

In an attempt to reduce the confusion produced by this lack of clarity in the early treatments of the fear concept, McAllister and McAllister (1967) suggested that fear might be most usefully defined in terms of exposure to CS-US pairings. Moreover, they proposed that the task of "measuring" or "indexing" fear remain apart from that of defining fear. In this manner, a clear and reliable operational definition could be had, despite a lack of correspondence among the various response measures used to "index" fear.

Brown (1974) has reviewed the problem of the definition and measurement of fear and has suggested a different approach. First, rather than define fear per se, he proposes to define fearfulness. The word "fearfulness" is a label given to a property or characteristic that an individual may possess to varying degrees, or not at all. To determine whether an individual is "fearful," reference must be made to the criteria described in a standard definition. The worth of any given definition will depend upon the extent to which the concept of fearfulness (as it is defined) can be shown to relate to other concepts or behaviors in a lawful manner.

Using the McAllisters' "pairings definition," an organism might be said by Brown to be "fearful" if it had received so many pairings of such and such a CS with such and such a US under specified temporal conditions. Presumably, one could incorporate the notion of relative fearfulness into this type of definition by asserting that amount or degree of fearfulness was an increasing monotonic function of the number of CS-US pairings. Given this definition, the behavior scientist's task would then be to see whether fearfulness was a useful or significant concept in the sense of uniquely describing a characteristic that was lawfully related to other behaviors or characteristics.

Even though the McAllister's "pairings" definition provides an adequate and potentially significant definition of "fearfulness," Brown finds their separation of definition and measurement unnecessary. When one wishes both to define and measure, the statement of the definition and the statement of the measurement

procedures are inextricable. Because the McAllisters clearly wish to avoid a "response" definition of fear, the business of relating fear (defined in terms of antecedent conditions) to overt responses should not be characterized as somehow "indexing" or "measuring" fear. If the "number of pairings" definition described in the previous paragraph correctly reflects their position, "measurement" of fearfulness consists simply of counting CS-US occurrences. Relating the number of pairings to overt responses is more properly viewed as part of the process of determining and evaluating the significance of the defined concept.

Brown argues further that a definition of fearfulness that includes reference to the behavioral outcome of some standardized test (an "outcome" definition) may have certain advantages over one that refers only to antecedent conditions. Individual differences in "conditionability" or "sensitivity" to the CS or US (defined independently) might reduce the probability that a pairings definition would be as useful as a definition that included reference to some behavior. Moreover, the pairings definition would appear to be insensitive to "real changes" in response strength such as might occur as a function of time following extinction (spontaneous recovery). Finally, Brown proposes not only that an "outcome" definition might prove more useful, but that an outcome definition that excludes reference to antecedent conditions may be the most useful of all. If "pairings" remained a part of the definition, the concept would be restricted to a fearfulness that was learned, and learned only in the manner specified in the definition. Response outcomes that were dependent upon "innate" predispositions, or learning that occurred as a result of something other than

forward CS-US pairings could not be construed as reflecting fearfulness, even if those outcomes were identical to those produced by forward CS-US pairings. An "outcome-only" definition places no restrictions on the "source" of fearfulness and, in this respect, more closely resembles the procedures used to define other behavior characteristics such as intelligence.

Adoption of the outcome definition, however, is no guarantee of success. After choosing any given behavioral test or composite of tests for the definition, one must continually evaluate its worth by examining the types of behavior relations that appear as a result of its application. The McAllisters (1971) see two problems with response definitions of fear. First, different response measures may not be highly correlated with one another, and therefore, fearfulness defined by one behavior may not be the same as that defined by a different behavior. This problem, however, is as relevant to the pairings definition as it is to the outcome definition. Fearfulness defined by 12 pairings at one interstimulus interval (ISI) may be different from fearfulness defined by 12 pairings at another interval. Only comparisons among the types of laws that emerge from the careful use of each definition can show which is best. It may be that some composite or weighted average will provide the "best" definition.

The second objection that the McAllisters raise is that the response measures typically used to index fear are affected by variables unrelated to fear. This objection can only make sense if reference is made to some definition of fearfulness other than that being considered. Since their remarks occurred in the context of their distinction between

definition and measurement, presumably they were arguing that behavior on the fear-indexing task could be affected by factors unrelated to the pairing of the CS and US. However, if one accepts the outcome-only definition outlined above, logically there can be no variable that affects the response measure that is unrelated to fearfulness.

To summarize, it has been suggested that the concept of fearfulness must be adequately defined before we can decide whether it deserves to be included in the scientist's vocabulary. Although previous treatments of the concept appeared to contain implicit defining conditions, there was no clear distinction between the criteria that defined the concept and the outcomes predicted by theories relating it to overt behavior. Recent suggestions to provide precise definitions of fearfulness have focused on the nature of past experiences with CS and US and/or on performance on standardized tests.

With respect to the original concern over conditioned fearfulness and conditioned inhibition of fearfulness, only a small part of the picture has been presented. The "outcome-only" definition explicitly ignores any distinction between conditioned and unconditioned fearfulness. In addition, although it has been implied that certain test outcomes will define greater degrees of fearfulness than other outcomes, no mention has been made of an inhibitory-excitatory dimension or whether, indeed, if such a distinction is desirable, the same test can be used to define both excitation and inhibition of fearfulness. In the remainder of this discussion, consideration will be given to what might be entailed in extending the basic definition of fearfulness to incorporate the notions of acquired fearfulness and inhibition of fearfulness.

The reader should be aware that although the following formulations will be expressed primarily in terms of a classical-conditioning model of acquired fearfulness, they need not be restricted to that model. As Kimmel (1974) has recently suggested, an instrumental-conditioning model of the acquisition of fearfulness may more accurately describe the learning process, particularly when fearfulness is likened to chronic anxiety as diagnosed by the clinician. The choice of the language of classical conditioning was made for convenience in presentation and because most of the data and theories to be discussed later were derived from a Pavlovian model.

In general terms, we say that a response tendency has been learned if there has been a measurable change in some aspect of the response (e.g., its frequency, latency or magnitude) that occurs due to particular stimulus-reinforcer (classical conditioning) or response-reinforcer (instrumental or operant conditioning) relations. The classical conditioning paradigm is usually distinguished by the independence between CS-US presentation and the organism's responding, whereas in instrumental or operant conditioning, presentation of the reinforcer is contingent upon the response. Although the classical CR has traditionally been characterized as being "similar" to the unconditioned response (UR) elicited by the US (cf. Gormezano & Moore, 1969, p. 124), the present formulation will place no restrictions on the types or behavior changes that may qualify as learned changes (CRs). For now, similarity or lack of similarity between CR and UR will be treated as an empirical or theoretical problem, not a definitional one (see Rescorla, 1969d, p. 78, for similar comments). Eventually, it may

prove useful to include restrictions in the definition of acquired fearfulness, but it seems unnecessary at this point, and might exclude consideration of a number of potentially useful test-outcome procedures (e.g., those involving anticipatory heart-rate changes).

Thus, to say that a response tendency has been learned, we require both that a change in behavior occur and that the change be attributable to a particular relation between the CS and US. The question arises, however, as to the exact nature of that CS-US relation. What are the boundary conditions or criteria that distinguish associative CS-US relationships from nonassociative CS-US relationships? We can ask further, whether, or to what extent, these boundary conditions ought to be included in our formal definition. Usually, a change that occurs concomitantly with repeated forward pairings is considered possibly to be due to learning. However, simply observing a change in behavior following forward pairings may not be sufficient if it can be argued that the change was due to such processes as sensitization (enhancement of the unconditioned reactions to the CS and/or US), adaptation (depression of those unconditioned reactions), maturation, or chemical or surgical intervention. It may also be desirable to distinguish "learned changes" from changes that occur following CS-alone presentations (habituation, "latent inhibition") or US-alone presentations (habituation, "pseudo-conditioning"). Although changes falling into these latter categories might be regarded as "learned" in some broader sense of the word, they will not be considered as such here, since they do not result specifically from exposure to <u>both</u> the CS and US.

There appear to be several ways in which the learning definition

can be formalized so as to incorporate the views expressed in the preceding paragraphs. First, the notion of "change" implies at least the first, and possibly both of the following: (a) There must be at least one occasion on which the response is shown to occur to a greater degree in the presence of the CS than in its absence (or vice versa), and (b) there must be a difference (positive or negative) between the response to the CS prior to CS-US exposure and the response to the CS following CS-US exposure ("response to the CS" may refer either to some absolute level of responding in the presence of the CS, or to some relative level of responding in the sense implied in [a]). The exact way that "change" enters the definition depends upon the criteria chosen to distinguish associative changes from nonassociative ones. At this point, it will be helpful to consider how those criteria might be applied, and the consequences of choosing any particular method.

One might simply assert that any pre- to post-conditioning change that occurs when conditioning involves forward CS-US pairings (the exact limits of this relation would have to be specified) defines learning. If the specified training had been given, any change would have to be considered a learned change. Unfortunately, it could be argued that such factors as sensitization, adaptation, maturation, etc., equally well described such an outcome, and therefore, these concepts overlapped or were redundant. Moreover, any similar change that occurred following exposure to a CS-US sequence other than that specified by the definition would be excluded. Perhaps this last restriction could be lessened by modifying the definition to include not only changes that occur following forward pairings, but also any changes that occur

(following exposure to an equal number of CSs and USs) that are qualitatively the same as those produced by forward pairings (e.g., in the same direction). In essence, this latter type of definition was the one used by Pavlov to define excitatory conditioning.

To refine the definition of learning to eliminate the possibility of overlap with other concepts, it is necessary to include a "control-condition" comparison in the definition. The changes in responding observed under the conditions of interest (the experimental condition) are compared with those in some control condition. To assert that the response observed under the experimental condition was learned, there must be a difference between the changes in behavior measured under each condition. The comparison can be made either within the same group (e.g., CS+ vs CS- in differential conditioning) or between different groups (e.g., CS+ in Group 1 vs random-CS in Group 2). In either case, the stimulus used on the control-condition test cannot have been related to the US in the same way as the stimulus in the experimental condition. For a within-group comparison, the control-condition stimulus cannot be the same as the experimental-condition stimulus, whereas for a between-group comparison, the same stimulus may be used in both conditions.

The response observed under the control-condition sets a standard against which "learned responses" are evaluated. The difference between the scores under the experimental and control conditions after conditioning, or the change in the difference between those scores due to conditioning trials becomes critical to the assertion that the response was learned. If these differences are not obtained, no learning can be said to have occurred. When using the control-condition procedure,

administration of the experimental and control condition tests before conditioning may not be absolutely necessary. When they are not given, however, it is generally assumed that the response to the stimulus would have been the same in each condition.

The control condition, in essence, sets the boundary conditions for learning. The decision to use any one or set of control conditions is inevitably affected by empirical and theoretical considerations (see McAllister & McAllister, 1971; Rescorla, 1967b; Seligman, 1969). However, discussion of the types of controls that have actually been used to define learning will be postponed until the notion of inhibition has been evaluated, since the choice of control procedures may depend critically upon the manner in which inhibition is viewed (cf. Rescorla, 1967b).

Having outlined briefly what a definition of learning might entail, let us consider what is involved in asserting that fearfulness, or more generally, that some change in level of fearfulness is due to learning. First, it must be shown that the organism's level of fearfulness in the presence of a potential CS differs from its level in the absence of that stimulus. Assuming an "outcome" definition, the test specified by the definition must be given both in the presence of the putative CS and in its absence, and both before and after exposure to the experimental conditioning procedure. One's definition of learning might go on to demand comparison with an "appropriate" control condition as described previously. If the changes following stimulus onset meet the requirements of our definitions, the change in level of fearfulness can be said to be the result of learning.

How might the notions of excitation or inhibition apply to fearful-

ness? To begin, to say that a response tendency is excited or inhibited necessarily implies that there is some reference point ("zero") along the scale that defines response strength. A response tendency is either excited or inhibited with respect to that reference point. It should be noted that the direction of the change in response strength does not invariably determine whether the change is to be said to reflect excitation or inhibition for all possible responses. The decision to apply one label or the other in the case of a single change in behavior is arbitrary. As an example, in the conditioned-emotional-response (CER) paradigm (to be discussed shortly), a <u>decrease</u> in rate of barpressing during a conditioned stimulus may be used to define <u>excitation</u> of fearfulness. That same decrease in behavior also might be said to reflect <u>inhibition</u> of the barpress response. In any event, in order to apply the excitatory-inhibitory distinction, one must refer to a particular response and to some "zero" point along the scale that defines response strength.

In the case of fearfulness, one might use the average score obtained on the fearfulness test for some population (or any other score) as the "neutral point," defining fearfulness above that point as excited and below that point as inhibited. A single application of the test would be all that would be required to determine whether the fearfulness shown by an individual exemplified excitation or inhibition with respect to the population norm. This simple classification might have little value, however, if the effects of "fearfulness variables" were no different above and below the zero point.

A potentially more useful way of handling excitation and inhibition

might be to compare an individual's fearfulness score under the conditions of interest with his own score under some reference set of conditions. In this instance, to say that changes in fearfulness reflected either excitation or inhibition would require that the fearfulness test be given at least two times, once under the reference conditions, and again following some change in those conditions. The choice of reference conditions is completely arbitrary. At one extreme, they might be whatever conditions precede the stimulus change of interest. If the difference between the level of fearfulness just before and just after some stimulus change were in the direction of increased fearfulness, then the change could be said to be excitatory. If level of fearfulness decreased, the change would be inhibitory.

Such flexibility, however, may not be desirable. Consider the following example. Suppose that a test for fearfulness were given during each of the 1-min intervals that preceded, coincided with, and followed the presentation of a 1-min tone. Suppose further that scores of 50, 100, and 50, were obtained during each minute, respectively. What could be said about the effect of the tone on fearfulness? The change from the tone-off condition (Interval 1) to the tone-on condition (Interval 2) could be said to have been excitatory. However, might it not also be reasonable to ask whether the change from Interval 1 to 2 was due to the removal of some inhibitory condition, rather than to the onset of an excitatory one? And what of the change that occurred following tone offset (Interval 3)? With respect to the second interval, tone offset was inhibitory. But, if the change is labeled inhibitory, has anything more been said than that an excitatory stimulus (defined

by the difference between Intervals 1 and 2) was removed?

The preceding example simply illustrates that, given a variable set of reference conditions or no reference conditions at all, an increase in level of fearfulness might be said to be due either to an increase in excitation or a decrease in inhibition. Similarly, a decrease in level of fearfulness might be due to an increase in inhibition or a decrease in excitation. The only way to use the terms excitation and inhibition uniquely to describe changes in level of fearfulness is to standardize the reference conditions. For instance, the score obtained on the fearfulness test in the absence of any discrete stimuli known to affect level of fearfulness (determined empirically) might be used to define the "neutral" (zero) point. The utility of any such arbitrarily chosen "zero" point would ultimately have to be evaluated in terms of the significance of the behavioral laws generated by its application.

Stimulus-produced departures in level of fearfulness from the reference could clearly be labeled excitatory (if the change were an increase in fearfulness) or inhibitory (if the change were a decrease in fearfulness). However, there would still be a problem in handling changes that originated from non-zero levels of fearfulness. Increases in absolute level of fearfulness from above or below zero might be due either to an excitatory stimulus change, or to the removal of an inhibitory stimulus. Decreases from above or below zero would be equally ambiguous.

How can the ambiguous cases be handled? It would seem that it is only when the conditions of which a given level of fearfulness is a function remain constant that one can hope to apply the labels excita-

tory and inhibitory in a meaningful way. Above-zero decreases in level of fearfulness might only be called inhibitory if it can be shown that the stimulus conditions that aroused the initial level of fearfulness have not been altered, either physically or in some (measurable) "psychological" sense (e.g., attentional increases or decreases).

The foregoing considerations may be especially important if the response used to define level of fearfulness were at a minimum (or maximum) strength in the absence of any alleged fear inhibitors or excitors. Because the level of fearfulness could change in only one direction from the reference point, only excitation or only inhibition could be directly defined by a change from the reference point. In order to find evidence for the opposite type of change under these conditions, fearfulness would have to be at some non-zero level to begin with, and the stimulus change of interest would have to bring the level of fearfulness closer to the zero point. And even then, one would need to be reasonably sure that this return toward zero was not simply due to a modification in the stimulus conditions that produced the original non-zero level of fearfulness. This procedure could be standardized for purposes of definition by always testing for the opposite type of change in the presence of a stimulus of known (excitatory or inhibitory) strength.

The preceding discussion has been noncommittal as to whether excitatory or inhibitory changes in level of fearfulness reflect the conditioned or unconditioned properties of the stimuli under consideration. Presumably, a stimulus can produce an excitatory (inhibitory) change in level of fearfulness whether or not the requirements of our

definition of learning have been met. Moreover, there appears to be no reason why a stimulus that has acquired inhibitory (excitatory) properties can not affect fearfulness due either to conditioned or unconditioned excitation (inhibition). Similarly, a stimulus with unconditioned inhibitory (excitatory) properties may affect either type of excitation (inhibition).

At this point, our formulations can be quite readily extended to encompass the notions of acquired excitation and inhibition of fearfulness. The general definitions of excitation and inhibition already include part of the idea of response change that is contained in the definition of learning. That is, the test must be given once before and once after the stimulus change of interest. All that is needed is the addition of the control condition to rule out nonassociative excitatory and inhibitory influences. The logic here parallels that described before. If the change in level of fearfulness produced by the alleged conditioned stimulus differs from that produced by the stimulus in the control condition, the change can be said to be due to learning. If that change is in the direction of increased fearfulness, the stimulus is called a conditioned excitor. If the change is a decrease, the stimulus is called a conditioned inhibitor.

To summarize all of the foregoing, it has been suggested that, in order to understand what is meant by the phrases "conditioned excitation of fear" and "conditioned inhibition of fear," it is necessary to consider carefully what it might mean to say that an individual is fearful, that a response tendency has been learned, and that a change in the strength of a response tendency reflects excitation or inhibition. It

was proposed that a definition of fearfulness that depends, in part, upon some behavioral outcome might prove more useful in the delineation of important behavioral relations than one which does not. Moreover, a definition that does not restrict the concept by including or excluding certain types of antecedent experiences in the statement of the definition may prove the most useful of all. Examination of the concept of a learned response tendency led to the suggestion that changes in level of fearfulness might be attributed to learning if certain requirements were met. Most important among these requirements were that the changes had to be attributable to CS-US exposure, and different from those shown in some control condition. Although specific control strategies have not yet been discussed, it was noted that the choice of the control condition, in effect, sets limits on the types of CS-US relations that would be said to produce associative changes in response tendency. Finally, in the discussion of excitation and inhibition, it was suggested that level of fearfulness could be said to reflect excitation or inhibition if there were a shift in level of fearfulness away or toward the level shown under a reference set of conditions. Only those shifts in fearfulness that could be attributed to stimulus changes that did not affect the conditions responsible for the original deviation from the reference level could reasonably be said to be either only excitatory or only inhibitory. To assert that a given change in level of fearfulness reflected <u>acquired</u> excitation or inhibition would require consideration of both the direction of the change with respect to the reference condition, and the nature of the change shown by an "appropriate" control condition.

Tests for Fearfulness and the Inhibition of Fearfulness

Hopefully, the preceding discussion has provided a conceptual framework for a more detailed consideration of two critical issues: (a) specification of the types of behavioral tests that might be used to define fearfulness and/or its inhibition, and (b) specification of the control procedures that might be used to assess whether changes in level of fearfulness are due to learning. Attention will be given to these problems in this section and the next section, respectively.

McAllister and McAllister (1971) have recently furnished an extensive review of behavioral measures of conditioned fear. They focused primarily on situations in which the conditioning of fear was presumed to be independent of its measurement. Specifically, they were interested in situations in which the US did not appear directly to elict the measured response. Thus, they were not concerned with avoidance conditioning or classical defense conditioning. They admitted that fear might well be conditioned in these types of situations, but preferred to avoid the complications that may arise in interpreting such measures. As noted before, the present analysis places no restrictions on the nature of the test used to define fearfulness. However, when changes in level of fearfulness are believed to be produced by conditioned stimuli, we shall consider only those instances in which the CS and US have been presented independently of any particular response during conditioning. This approach is consistent with our interest in a Pavlovian view of acquired fearfulness, and rules out tests in which the experimenter does not maintain precise control over the number, duration, and temporal distribution of CSs and USs. Thus, as the McAllisters suggest,

avoidance conditioning will be ignored. However, there appears to be no apriori reason to exclude consideration of traditional defense conditioning procedures.

The fear measures described by the McAllisters fall into three general categories: (a) changes in level of performance resulting from the response-contingent removal of a fear-arousing stimulus, (b) changes in performance resulting from the response-contingent introduction of a fear-arousing stimulus, and (c) changes in the strength of responses (learned or unlearned) that occur in the presence of a fear-arousing stimulus. A brief review of fearfulness tests that fall into these categories follows.

An example of the first method is the so-called "escape-from-fear" paradigm. The organism is simply placed into a situation and given an opportunity to escape the alleged fear-arousing stimulus. The frequency or speed with which the escape response occurs can be used to define level of fearfulness. Faster or more frequent responding is usually associated with higher levels of fearfulness. The classic experiments of Miller (1948) and Brown and Jacobs (1949) typify this procedure.

In the second category, fearfulness is defined by the ability of a given stimulus to punish responding. The more a response-contingent event weakens a response (e.g., increases its latency or decreases its frequency or vigor), the more fear-arousing that event can be said to be. The studies of Mowrer and Solomon (1954) and Mowrer and Aiken (1954) are offered as examples of this type of procedure.

Test methods falling into the last category are characterized by the fact that the supposed fear-arousing event is presented noncontin-

gently while the organism is performing (or about to perform) some response. Depending upon the paradigm, increases or decreases in strength of responding may be used to define increases in level of fearfulness. Changes in the rates of two types of operant baselines are often used for this purpose--instrumental or consummatory responding for food reinforcement, and instrumental responding in unsignalled shock-avoidance paradigms. When an appetitive baseline is used, and a conditioned stimulus is presented, the method is typically referred to as the conditioned-emotional-response (CER) or conditioned-suppression paradigm. Decreases in rate of responding define increases in level of fearfulness (cf. Estes & Skinner, 1941). The aversively-motivated response baseline usually involves a Sidman avoidance task (Sidman, 1953). This method will be referred to here as the "transfer-to-Sidman" procedure. In this instance, increases above baseline response rate correspond to increases (excitation) in fearfulness, and decreases below baseline response rate correspond to decreases (inhibition) in fearfulness (cf. Rescorla & LoLordo, 1965).

When interest is specifically in acquired fearfulness, fear conditioning may take place either while the animal is performing a response such as barpressing ("on-the-baseline" conditioning) or under circumstances where responding is impossible ("off-the-baseline" conditioning). In the on-the-baseline procedure, conditioning and testing occur at the same time. However, in the off-the-baseline procedure, in order to test for fearfulness, the baseline must be re-established before the stimulus can be presented. There are advantages and disadvantages to each method. On-the-baseline conditioning permits a trial-by-trial record of the level

of fearfulness, whereas acquired fearfulness must always be measured in extinction when using the off-the-baseline method. In off-the-baseline conditioning, one can avoid the possible complications introduced by accidental punishment of the operant response by the US, or interactions with situational CRs related to the operant response during conditioning. However, because testing must always occur on-the-baseline, there is greater opportunity for stimulus-generalization-decrement effects in the off-the-baseline procedure. Although the data derived from both methods often permit the same conclusions to be made concerning the effects of variables purported to affect conditioned fear, in certain instances, different conclusions are reached (Wagner, Siegel, & Fein, 1967). There has not been a great deal of systematic comparison of these methods.

The transfer techniques involving appetitively- and aversively-motivated operant responses are instances in which fearfulness is defined in terms of the ability of a stimulus to affect the strength of a learned response. There are several techniques that define fearfulness in terms of a change in the strength of an unlearned response. An increase in the strength of the UR "normally" elicited by some US can be taken to define an increase in level of fearfulness. A decrease may indicate inhibition of fearfulness. The startle-probe method described by Brown, Kalish, and Farber (1951) is an example of this. They found that a stimulus that had been forwardly paired with shock enhanced the startle response elicited by a loud auditory stimulus. Changes in level of gross motor activity in the presence of fear-arousing stimuli might also be an example of a procedure falling into this category (e.g., open-field behavior; see also Klare, 1974). Another possibility has recently been proposed by

Rakover (1975). He measured the rat's response to electric shock when escape from shock required the rat to approach the alleged fear-arousing stimulus.

Several points must be made with respect to the test procedures described above. First, all may be used to define changes in level of fearfulness due to the conditioned or unconditioned effects of various stimuli. In each instance, to say that a change in fearfulness was due to learning would require comparison with some control condition. Second, to assess the effects of a "non-stimulus" factor (e.g., genetic background) on level of fearfulness, one must correlate variations in that factor with the level of fearfulness aroused by some standard stimulus (or in certain instances, the test conditions themselves provide the stimulus, e.g., the open field). Third, most of the tests were designed to measure fear excitation, and without modification, may not be appropriate for measuring fear inhibition. For example, in the escape-from-fear paradigm, an alleged inhibitory stimulus might not be expected appreciably to reduce the operant level of the target response if that response occurs infrequently in the absence of an explicit fear-arousing stimulus. This could be due simply to a measurement problem (e.g., a "floor effect"), or to the absence of any fear to inhibit. This point will be discussed again later.

Before continuing, two other test strategies must be added to the list. The first involves the use of physiological response measures (e.g., heart rate, respiration, blood pressure, muscle tension, skin conductance, etc.), and the second, verbal (written or spoken) responses. In these instances, level of fearfulness can be defined simply in terms

of the response to some standardized set of conditions in the presence of the supposed fear-arousing stimulus.

These are but a few of the tests that have been offered as candidates for the definition of fearfulness. McAllister and McAllister (1971) have discussed the merits and demerits of many of them at length. As has been suggested before, it may be necessary to use some combination of tests in arriving at the "best" (most useful) definition. For example, suppose that a stimulus has been found to depress response rate in a CER paradigm. Because stimuli that have been paired with food also depress response rate (Azrin & Hake, 1969), we may not wish to accept that behavior alone as defining fearfulness, especially if we want to restrict our notion of fearfulness to the effects of conditioned and unconditioned aversive stimulation. In that case, one might further demand that the stimulus enhance responding in a transfer-to-Sidman test or that it not affect the response in a startle-probe test. In both of these tests, conditioned appetitive stimuli do not appear to have the same effects as conditioned aversive stimuli (Grossen, Kostansek & Bolles, 1969; Trapold, 1962).

The tests for fearfulness described above have primarily been used to assess a level of fearfulness that was thought to be "excited." With the addition of a few conditions, however, all can be used to define inhibition of fearfulness as well. In the remainder of this section, attention will be focused upon techniques that might be used to define inhibition, and the logic behind their use. Because our interest is in conditioned inhibition of fearfulness, it will be assumed that control conditions are available to assess nonassociative factors (see next

section). In addition, although the discussion will be directed toward measuring conditioned inhibition, the techniques apply to the measurement of unconditioned inhibition as well.

Both Rescorla (1967b, 1969d) and Hearst (1972) have noted the bias among Western psychologists to view conditioning as either excitatory or absent. They have suggested that this bias is, in part, due to a measurement problem. When a stimulus that does not initially elicit a CR (the so-called "neutral" CS) acquires that ability through repeated pairings with the US, it seems reasonable to conclude that excitatory conditioning has occurred. However, consider the "neutral" CS that is imbedded in operations thought to make it inhibitory. According to the general definition proposed earlier, the conditioned inhibitor should produce a tendency opposite that produced by an excitor. But if the CS elicits no response prior to conditioning, it is difficult if not impossible to show a decrease in responding. Since no change in behavior is observed, one cannot say that learning has occurred. For this reason, a variety of special measurement techniques have been devised to show that an alleged inhibitor is no longer "neutral."

The first procedure, called the "summation" or "combined-cue" test, was first suggested by Pavlov. It involves the superimposition of the alleged inhibitory stimulus on a known excitatory stimulus. Given the assumptions that excitation and inhibition work in opposite directions and that their effects are algebraically additive, one might regard any loss in the strength of the response normally elicited by the excitor as evidence for inhibition. Implicit in this procedure is the notion that in order to demonstrate inhibition, there must be something

(excitation) there to inhibit. The types of excitors that have been used in combined-cue tests include: (1) an explicit excitatory conditioned stimulus (CS+), (2) backround cues and internal cues that have been paired with the US (Rescorla & LoLordo, 1965), and (c) the US itself. Experiments in which these techniques have been used are described in Appendix A.

With reference to the fearfulness tests already outlined, it is clear that each can be transformed into a combined-cue test for the inhibition of fearfulness. For example, a stimulus that has met the requirements of an excitatory stimulus in an escape-from-fear, CER, transfer-to-Sidman, startle-probe, or classical defense conditioning situation can be compounded (presented simultaneously) with the supposed inhibitor. In the case of the startle-probe and transfer-to-Sidman techniques, it may not even be necessary to provide an explicit excitor since the test procedures themselves may produce sufficient excitation. In each instance, there should be a decrease in the level of fearfulness if one is to say that the event was inhibitory. To assert that the change was due to conditioning, it must be shown to differ from that found in the control condition(s).

The results of a combined-cue test may not unambiguously support a conditioned-inhibition interpretation. Both the inhibitory-conditioning and control-condition procedures must be examined for their potential to produce differential stimulus generalization decrement and/or differential changes in attention. This will be discussed again shortly.

A second test procedure is the "retardation," "resistance-to-reinforcement," or "new-learning" procedure. Here, the potentially

inhibitory properties of a given stimulus are evaluated by examining the development of a new excitatory conditioned response to that stimulus. If the conditioned inhibitor elicits a negative tendency, it would be expected to retard the development of an excitatory one. Actual examples of these procedures are presented in Appendix A, but with respect to the fearfulness tests described previously, again, all can be used to assess the effect of prior inhibitory-conditioning procedures on the development of an excitatory tendency.

In this case, as before, however, retardation may not be due solely to conditioned inhibition. Thus, as Hearst (1972) has suggested, one might argue that through some attentional mechanism, the organism comes to "ignore" the stimulus that is used during inhibitory conditioning. Because ignoring the CS may retard the subsequent development of an excitatory tendency, and because this would not necessarily depend upon the prior CS - US experiences of the organism, this type of result could not be interpreted in terms of acquired inhibition. If the attentional deficit were purely a function of nonreinforced pre-exposure to the to-be-conditioned excitor, a control group that received an equal number of CS-US exposures, but in such a way as to preclude the development of an inhibitory tendency, might be an appropriate control for these types of deficits.

Rescorla (1969d) and Hearst (1972) have advocated the use of multiple tests to rule out interpretations contrary to conditioned inhibition. If the attentional mechanism described in the previous paragraph were responsible for retardation during a new-learning test, one would not expect to see evidence for inhibition during a combined-cue test. A

stimulus that was "ignored" should not affect responding to the excitor. Similarly, the attentional mechanism that might be invoked to explain positive evidence for inhibition on the combined-cue test could not explain retardation on a new-learning test. One might argue that the alleged inhibitor attracted the organism's attention during the combined-cue test, decreasing its attention to the excitor, and thereby produced a loss in response strength. However, if the supposed inhibitor were an "attention getter," it would be expected to facilitate rather than retard responding during a new-learning test.

Depending upon one's view of conditioned inhibition, there may be other ways to rule out nonassociative, attentional mechanisms. Rescorla's (1969d) definition of inhibition demands that the effects of the inhibitor be specific to excitation associated with the same US used to establish conditioned inhibition. Thus, whereas an attentional mechanism might predict retardation on all new-learning tests, Rescorla's conditioned inhibitor would only be expected to retard new learning involving the same US. Hearst's (1972) definition, however, does not restrict the action of the conditioned inhibitor. In contrast to Rescorla's approach, the question of specificity was left an empirical one, not a definitional one. Hearst suggested that the use of both combined-cue and new-learning tests may be sufficient to assess attentional hypotheses.

As can be seen in the review of the literature in Appendix A, combined-cue and new-learning tests have been used most widely to assess conditioned inhibition. However, there are other possibilities that have not yet been fully investigated. For instance, positive evidence for the response-reinforcing effects of an alleged inhibitory stimulus

might be used to define inhibition. As before, control conditions would be required to evaluate the associative nature of such a finding. Data in support of the putative reinforcing effects of a conditioned inhibitor will not be discussed here, but reference is made to a review by LoLordo (1969), and the studies of Braud (1968), Cunningham (1973, Exp. 3), Rescorla (1969c), and Weisman and Litner (1969b, 1972), for examples of this technique. There are several interesting issues posed by the secondary-reinforcement procedure. For instance, one might want to know whether a conditioned inhibitor can reinforce responding in the absence of an excited level of fearfulness. If conditioned inhibitors were to exert reinforcing effects in the absence of any excitation to be reduced might suggest that they actually have positive affective properties (cf. Solomon & Corbit, 1974).

Another alternative procedure for testing for inhibition might be based on the potential punishing effects of the response-contingent removal of a conditioned inhibitor. There appear to be no data available on the use of this type of procedure.

To summarize, a variety of techniques for defining level of fearfulness have been discussed in the preceding section. Although most of these procedures were developed in the context of measuring excitation of fearfulness, it has been suggested that through the use of combined-cue and/or new-learning test strategies, all can be used to assess inhibition of fearfulness as well. Whether defining excitation or inhibition, however, in order to assert that a change in level of fearfulness has been due to learning, one must consider the role of nonassociative factors. This topic will be discussed in the next section.

The Problem of Control Procedures in Pavlovian Conditioning

In the initial discussion of learning, it was suggested that if one's definition of learning required comparison of the experimental condition with a control condition(s), then the control condition(s), in effect, established the boundary conditions for "learning." The experimental group would only be said to have learned some response if the change in its behavior were different from that seen in the control condition(s). This approach is consistent with the general notion of control strategy--hold all operations constant except for the critical one--any differences that are observed can then be attributed to the critical operation. The problem of control procedures in Pavlovian conditioning has centered around two issues: (a) specifying the so-called "critical operation," and (b) determining whether the control condition has effects beyond the elimination of the critical operation.

Gormezano and Moore (1969, pp. 127-128) have pointed to three types of changes in the response to the CS that are generally considered <u>not</u> to be due to learning--sensitization, pseudoconditioning, and increases in the spontaneous rate of emission of the response. Sensitization refers to a reinstatement or augmentation of the original reflex response (alpha response) to the CS. This response may resemble the CR, but it is assumed to occur independently of the pairing of the CS and US. In some response systems, a latency criterion can be used to eliminate such responses, but in the case of most tests for fearfulness, a control condition must be employed. Differential- (within-subject control) and explicitly-unpaired- (between-subject control) conditioning procedures

have commonly been used to control for sensitization.

Pseudoconditioning refers to the emergence of a CR-like response to the CS as a result of prior US exposure. Because this effect occurs independently of CS-US pairings, it is also assumed not to be due to learning in the sense implied by our definition. Increases in spontaneous rate may also occur independently of CS-US pairing. US-only and CS-only control groups can be compared to a group that receives no CSs or USs to assess the contribution of each to spontaneous response rate, but a group that receives both may be better. In addition, a correction procedure can often be used to adjust responses observed during the interstimulus interval for responses occurring during non-CS intervals (e.g., difference scores, percentages). Gormezano and Moore conclude that a group that receives explicitly-unpaired presentations of the CS and US can be used to control for both pseudoconditioning and spontaneous rate changes. Since this type of control group receives the same number of CSs and USs as the experimental group, it is preferred to CS-alone, US-alone, or novel-CS controls.

The considerations in the preceding paragraphs reveal a potential bias in the approaches to control procedures in classical conditioning. All of the nonassociative effects that were mentioned were such as might be confused with conditioned excitatory changes in response strength. Moreover, all were evaluated with reference to an experimental condition in which the pairing of the CS and US was viewed as critical to learning. In a seminal review paper, Rescorla (1967b) noted this, and after a detailed consideration of a variety of control procedures, suggested that the traditional approach was biased against

inhibitory conditioning because its statement of the critical factor in conditioning was incomplete. Thus, all of the control procedures that had generally been used were said to be inappropriate. In his restatement of the critical aspect of conditioning, Rescorla proposed that the "contingency" between CS and US was the relevant relation. In contrast to the traditional "pairings" view, the contingency position emphasized not only what was paired with the CS, but also what was not paired with the CS. Specifically, contingency was said to be a function of the probability that a US occurred during or within a certain time interval following the CS (Pr[US/CS]). With precise specification of that time interval, this statement alone might be perfectly consistent with the pairings formulation. However, Rescorla insisted that the contingency formulation required consideration not only of this positive relation, but also of the negative relation, that is, the relation between the US and the absence of the CS. This "negative contingency" was assumed to be a function of the probability of the US in the absence of the CS (Pr[US/$\overline{CS}$]). Given this view, he argued that the only appropriate control group for conditioning was one in which there was no contingency between CS and US, that is, Pr (US/CS) = Pr (US/$\overline{CS}$). A group of this sort would receive CSs and USs, but these events would be programmed randomly and independently (the "truly-random" control).

Conventional control procedures were judged inappropriate either because the control did not receive the same number of CSs and USs (CS-alone, US-alone, and novel-CS groups), or because the control condition involved a negative CS-US contingency (Pr[US/CS]$<$Pr[US/$\overline{CS}$]) (explicitly-unpaired, backward, and differential-conditioning groups).

In Rescorla's view, negative contingencies may produce inhibitory effects. Therefore, even if an excitatory response were not "really" elicited in the experimental group, comparison with one of these controls might lead to the erroneous conclusion that excitatory conditioning had occurred. Presumably, the truly-random control provided the only "neutral" baseline against which to assess both excitatory and inhibitory effects.

Rescorla argued that one great advantage of the truly-random control was that it held all factors constant except CS-US contingency without demanding that the experimenter be able to specify in advance what factors might be operating. Moreover, regardless of whatever changes in behavior accompanied the truly-random treatment, conditioned changes (i.e., changes due to CS-US contingency) were to be evaluated as deviations from those shown by the random control.

Adoption of the truly-random control procedure as the control procedure in the definition of learning requires acceptance of Rescorla's contingency theory of learning--that positive contingencies produce excitatory conditioning and negative contingencies produce inhibitory conditioning. Much of the reluctance to accept the random control in recent years stems from disagreement with Rescorla's view of conditioning. From the traditional point of view, the chance pairings of CS and US that can occur during truly-random conditioning may be sufficient to establish an excitatory tendency. The difference between groups receiving forward conditioning and truly-random conditioning, then, is not the difference between "conditioning" and "no-conditioning," but between two levels of excitatory conditioning (the result of continuous vs partial reinforcement). Several studies involving the random control claim

to have shown this excitatory effect (Ayres, Benedict, & Witcher, 1975; Benedict & Ayres, 1972; Kremer, 1971; Kremer & Kamin, 1971; Quinsey, 1970). According to the traditional view, therefore, differences between a negative-contingency condition and a truly-random control might reflect excitation in the random group rather than inhibition in the negative-contingency group.

Even if one were to accept the contingency view, there may be reasons for rejecting the random control. For example, Seligman (1968) reported a CER study in which the truly-random control was used in an on-the-baseline conditioning procedure. The rats exposed to this condition soon stopped barpressing completely. In addition, they developed a mean of 9.1 ulcers each. Rats in a standard, forward-conditioning group gradually resumed barpressing between trials and developed no ulcers. Seligman argued that the unpredictability of shock had made the rats in the truly-random condition "chronically fearful," and that although the procedure had eliminated CS-US contingency, its side effects precluded meaningful comparison of the control condition with the experimental condition. In a subsequent paper, Seligman (1969) suggested that this experiment was but one example of the need to control not only for the presumed critical operation, but to consider carefully the empirical and theoretical implications of using any particular control condition. If the control condition produces side effects, the difference between the experimental and control groups may be due either to the elimination of the supposed critical operation or to the absence of the side effect in the experimental condition.

Because the control condition determines the empirical phenomena

that one's theory must account for, the theory cannot be said to be independent of the choice of the control condition. For example, choice of the truly-random control entails the assumption that the occurrence of the US during the CS is as effective in producing conditioned excitation as the occurrence of the US in the absence of the CS ($\overline{CS}$) is in reducing that excitation. The net associative value of the CS must be presumed to be zero, and any behavioral changes that occur (such as those shown by Ayres et al., 1975; etc) must be attributed to nonassociative factors. It is only with reference to some other control condition that one might argue that such changes were excitatory.

Interestingly, Rescorla and Wagner (1972) have subsequently attempted to account for excitatory-like effects in truly-random conditioning by proposing that the truly-random CS actually possesses excitatory associative value early in conditioning (although it presumably loses all associative value at asymptote). Thus, in terms of associative value, the truly-random control cannot always be considered an appropriate "zero" at every point during conditioning according to their theory. The associative value of the CS in a CS-only, US-only, or novel-CS group would more properly represent the neutral point within their theory, because the operations involved in those control procedures are not expected to affect the associative strength of the CS. Unfortunately, the theory contains no statements concerning the nonassociative consequences of using these types of control procedures (e.g., how does failure to equate for total number of CSs and USs affect the strength of the observed response), and it is not clear if they are to be preferred to the truly-random control early in conditioning.

Lacking theoretical structures that adequately take into account both excitatory and inhibitory associative factors and all possible nonassociative factors in conditioning, it is perhaps not surprising that the problem of control in Pavlovian conditioning has not yet been resolved to everyone's satisfaction. Any given control procedure can only be said to be "appropriate" to the extent that it maintains nonassociative factors constant, yet eliminates the relation(s) that, according to one's theory, produce excitatory and inhibitory changes in response strength.

Variables Affecting Strength of Conditioned Inhibition

It has already been stated that the present dissertation is concerned with factors that affect the strength of conditioned inhibition of fearfulness. Assuming that certain inhibitory changes in level of fearfulness are due to learning, it seems reasonable to expect that the strength of those associative tendencies will be a function of the same types of variables believed to affect excitatory associative tendencies (e.g., stimulus intensity and duration, temporal relations among stimuli, number of trials, etc.). Although extensive reviews of the effects of these variables on the conditioned excitation of fearfulness are readily available (e.g., McAllister & McAllister, 1971), there have been very few critical reviews of recent literature in the area of conditioned inhibition of fearfulness. It therefore seems appropriate to provide such a review here. This review is contained in Appendix A, and is briefly summarized in the following paragraphs.

In what was essentially a combined-cue procedure, Rescorla and LoLordo (1965) demonstrated that rate of responding by dogs on a Sidman avoidance schedule (hurdle jumping) was depressed by stimuli that had previously been (a) used as CS- in differential conditioning, (b) presented in close proximity to nonreinforced presentations of an established excitatory CS (before or after), or (c) "explicitly unpaired" with the US. Presumably, these stimuli reduced excitation associated with environmental, shock and internal cues, temporarily reducing motivation for the instrumental response. This finding has been replicated by several investigators, not only with dogs, but with rats in both wheel-turning and hurdle-jumping tasks (Bull & Overmier, 1968; Grossen

& Bolles, 1968; Herendeen & Anderson, 1968; Rescorla, 1966; Weisman & Litner, 1969a, 1971). Moreover, the effect appears following backward- and "cessation-" conditioning procedures (Moscovitch, 1972; Moscovitch & LoLordo, 1968). Control conditions have included presentation of a novel CS, prior CS-alone or US-alone exposures, as well as truly-random CS-US presentations.

Similar conditioning procedures have yielded outcomes consistent with a conditioned-inhibition interpretation in combined-cue and new-learning tests in both CER and eyelid (or nictitating membrane) conditioning (Cappell, Herring, & Webster, 1970; Hammond, 1966, 1967, 1968; Hammond & Daniel, 1970; Marchant, Mis, & Moore, 1972; Marchant & Moore, 1974; Plotkin & Oakley, 1975; Reberg & Black, 1969; Rescorla, 1969b; Rescorla & Wagner, 1972; Siegel & Domjan, 1971, 1974; Wagner, 1971; Wagner & Rescorla, 1972).

Although some of the following conclusions may be equivocal because of inappropriate control procedures (see Appendix A), generally, strength of conditioned inhibition has been shown to be: (a) an increasing function of number of conditioning trials (Hammond, 1968; Herendeen & Anderson, 1968; Rescorla, 1972 ; Siegel & Domjan, 1974; Weisman & Litner, 1969a), (b) positively related to the strength of the conditioned excitor with which the to-be-conditioned inhibitor is contrasted (Rescorla & Wagner, 1972), (c) inversely related to the amount of inhibition elicited by stimuli with which the stimulus is contrasted (Suiter & LoLordo, 1971), (d) positively related to the amount of shock-free time the stimulus precedes (Moscovitch & LoLordo, 1968; Weisman & Litner, 1971), and (e) positively related to the similarity

between place of conditioning and place of testing (Desiderato, 1970; Grossen, 1971).

In general, these findings are consistent with Pavlov's claim that the procedures that produce internal (conditioned) inhibition fall into the following categories: (a) extinction (nonreinforcement of an established conditioned excitor), (b) extended CS-US intervals (inhibition of delay), (c) differential conditioning (CS_1 is reinforced while CS_2 is not), and (d) "conditioned-inhibition" training (similar to differential conditioning except that nonreinforced presentations of CS_1 overlap [or occur closely to] CS_2 presentations). Rescorla (1969d) has suggested that all of these situations have one thing in common--a negative contingency between the US and CS.

The amount of research into the variables believed to determine strength of conditioned inhibition is clearly not as extensive as that devoted to the study of conditioned excitation. One reason for this might be that there have been few behavioral frameworks within which to view the inhibitory-conditioning process, particularly in a manner similar to that in which excitatory conditioning is handled. However, this trend may be reversing (cf. Boakes & Halliday, 1972). Recently, a model of conditioning that treats excitation and inhibition in a symmetrical fashion has been proposed by Rescorla and Wagner (1972). This model may offer a reasonable starting point for a thorough examination of the relations among the variables determining Pavlovian conditioning, and will be discussed in the next section.

THE RESCORLA-WAGNER MODEL OF PAVLOVIAN CONDITIONING

It can be argued that all of the procedures that Pavlov suggested would produce internal inhibition have one thing in common--nonreinforcement in the presence of stimuli that have been, or are similar to those that have been, associated with reinforcement. According to Pavlov (1927, p. 127), the strength of inhibition that develops under conditions of nonreinforcement is directly related to the strength of the excitatory process on the basis of which the inhibition is established. This is the hypothesis towards which the present dissertation is directed. Recently, Rescorla and Wagner have proposed a model of conditioning that incorporates these Pavlovian tenets in its treatment of conditioned inhibition. Because their theory provides a convenient reference point for the present series of experiments, a brief description of the theory will be given.

Basically, their model states that the effectiveness of reinforcement or nonreinforcement in changing the associative strength (V) of a stimulus depends on the existing associative strength of that stimulus as well as the associative strengths of all other stimuli present at the same time. In many respects, it is quite similar to Hullian theory, and in particular, proposes that changes in the associative strength of a particular stimulus, i, can be expressed in terms of a linear model:

$$\Delta V_i = \alpha_i \beta (\lambda - \overline{V})$$

where α represents stimulus salience (i.e., a learning-rate parameter associated with the CS), β a learning-rate parameter associated with the US, and λ, the asymptotic level of associative strength that the particular US is capable of supporting. Alpha and beta are confined to the

unit interval ($0 \leq \alpha, \beta \leq 1$), whereas λ is not formally bounded (although it is generally assumed to be equal to or greater than zero). Finally, $\overline{V}$ represents the combined strength of all stimuli present on any given trial. For purposes of simplicity, this has been assumed to be the sum of the strengths of the component stimuli (i.e., $\overline{V} = \sum_{i=1}^{n} V_i$)

Several things should be noted: First, the V values represent associative strengths (habits), and can be either positive (excitatory) or negative (inhibitory). Changes in associative strength on any given trial are excitatory if $\lambda > \overline{V}$ and inhibitory if $\overline{V} > \lambda$. Second, the changes in associative strength of elements (i) in stimulus compounds are dependent upon the discrepancy between λ and $\overline{V}$, not V_i. This represents one of the major changes from the linear model proposed by Hull (1943). With this assumption, the theory can account for the "blocking" effect (Kamin, 1969), as well as a variety of other outcomes involving compound stimulus conditioning. Finally, the theory as yet provides no performance rules, that is, no explicit rules for mapping V values into response magnitude or probability. The specification of such rules presumably will require investigation of a variety of "performance" or "motivational" variables. For the present, it has been suggested that this mapping procedure will preserve the ordering of the V values. Tests of the theory, therefore, usually involve comparison among the relative effects of different conditioning procedures.

Since the present experiments are concerned primarily with inhibitory conditioning, the remainder of this discussion will focus upon the manner in which the Rescorla-Wagner model might handle inhibitory-conditioning procedures. Specifically, we will consider the "A+,AX-"

inhibitory-conditioning procedure. This was the paradigm originally proposed by Pavlov for establishing "conditioned inhibition," and Wagner and Rescorla (1972) have suggested that perhaps all inhibitory-conditioning paradigms can be viewed as special cases of this procedure. In this procedure, reinforced conditioning trials to some stimulus, A, are contrasted with nonreinforced presentations of A in combination with a second stimulus, X. Eventually, A alone elicits a strong CR, whereas the AX compound elicits little or no response. To see how the Rescorla-Wagner model treats this, assume first that A has been paired sufficiently often with some US to bring its associative value (V_A) very close to asymptote (i.e., $V_A = \lambda_1$). Now, consider what happens when AX- trials are interspersed among the A+ trials. Assuming that the asymptote of conditioning that is supported by nonreinforcement is equal to zero (i.e., $\lambda_2 = 0$)(see Rescorla & Wagner, 1972, p. 80), the changes in V_A and V_X that occur on each AX- trial can be expressed as follows:

$$\Delta V_A = \alpha_A \beta_2 (\lambda_2 - [V_A + V_X])$$

$$\Delta V_X = \alpha_X \beta_2 (\lambda_2 - [V_A + V_X])$$

If X is "associatively neutral" to begin with, the initial value of $(V_A + V_X)$ will be equal to V_A. Since that value is positive, the quantity $(\lambda_2 - [V_A + V_X])$ will be negative, and changes in associative strength will be decremental. The positive V associated with A will be reduced on AX trials and V_X will actually become negative (inhibitory). A+ trials should restrengthen A, while not affecting X. As A+,AX- training continues, the quantity $(V_A + V_X = \overline{V})$ should eventually approach λ_2 (zero), and the changes occurring on any particular trial should be minimal. V_A will have a net positive value equal to the net negative

value of V_X.

It is clear that the amount of inhibition that accrues to X should be a function of the excitatory value of A. The stonger the A cue, the more inhibitory X should become as a result of being nonreinforced in the AX compound. Rescorla and Wagner (1972) reported three experiments that bear on this issue. In two of these studies, the strength of the response to A was manipulated by varying the number of conditioning trials, and in the third study, by varying US intensity.

In the first experiment, two groups of rabbits initially received forward eyelid conditioning to each of three stimuli, A, B, and X. A and X were each paired with shock 224 times, whereas B was paired only 28 times. Empirically, A and X elicited the CR more often than B, and presumably, their associative strengths differed. During the second phase of the experiment, one group received 32 nonreinforced AX trials and the other group received 32 nonreinforced BX trials. Because the strength of X was the same in each group, the groups differed only in terms of the excitatory value of the stimulus that was compounded with X. According to the theory, the decrement in the strength of X on each compound trial should have been greater in the AX group, because the discrepancy between $\overline{V}$ and λ was greater in that group. In the final phase of the experiment, X-alone was again paired with shock during a relearning test for the strength of X. As predicted by the model, the group that had received AX extinction trials was slower to recondition than the BX group. Presumably, the value of X had declined to a greater degree in the AX condition.

The eyelid preparation was also used in the second study. This

time, however, the X cue was associatively "neutral" when it was first compounded with the excitor during inhibitory training. During the first phase, A and B were each paired with shock (240 vs 8 trials, respectively) in two groups of rabbits. A third cue, C, was also paired with shock (548 trials). This cue was to be used as the conditioned excitor during a final combined-cue test. During the second phase, one group received A+, AX- training, and the second group received B+,BX- training (64 trials each). In both groups, X should have acquired a negative associative value. Moreover, because the conditioned excitors were expected to have different positive associative values (empirically, A elicited more CRs than B), X was expected to become more inhibitory when compounded with the stronger excitor. During the test phase, X was presented together with C in both groups. Although both groups responded at the same high level to C alone (about 80% CRs), Group AX responded reliably less often to the CX compound (about 40%) than did Group BX (about 65%). That X attenuated the response to C to a greater degree in the AX condition again supports the hypothesis that strength of conditioned inhibition depends upon the strength of the excitor with which the to-be-conditioned inhibitor is contrasted.

In the final experiment, the strength of the excitor was manipulated by varying US intensity during on-the-baseline CER conditioning. After VI food-rewarded barpress training, three groups of rats were given A+,AX- training. All AX trials were nonreinforced and the groups differed in terms of the intensity of the US paired with A (0, .5, or 1.0 mA). After 45 A+ and 75 AX- trials, a third cue, C, was established as a conditioned excitor on a 50% reinforcement schedule using a .5-mA

shock US. Testing involved a combined-cue procedure in which nonreinforced presentations of C and CX (two each per day) were given over a six-day period. Suppression ratios from the differential-conditioning phase were not reported, but presumably the associative strength of A was greatest in the 1-mA group, followed by the .5- and 0-mA groups, respectively (cf. Annau & Kamin, 1961). Suppression to the C cue was nearly complete in all groups (the median suppression ratios were about .03 or less). However, the stronger the US that had been paired with A, the greater the reduction in suppression when X was combined with C. Presumably, the stronger the A cue, the more inhibitory the X cue.

EXPERIMENT 1

In the present series of experiments, an attempt has been made to substantiate further the notion that the strength of conditioned inhibition is a function of the strength of the excitation on which the inhibition is based. Specifically, excitatory value was manipulated by varying US intensity in a CER situation. However, in contrast to the US-intensity experiment described above, a new-learning test was used to assess strength of inhibition. The more inhibitory the stimulus, the longer it should take for it to become excitatory when newly paired with shock.

There are several reasons for examining the US-intensity relation. First, there is a general lack of data concerning the dependence of strength of inhibition on strength of excitation. Of the variables believed to affect strength of excitation that have been manipulated (number of conditioning trials and US intensity), US intensity seems less firmly established as a determinant of strength of inhibition. Second, assuming that the combined-cue test showing the US-intensity effect was valid, the arguments presented by Rescorla (1969b) and Hearst (1972) seem to suggest that a new-learning test is required to rule out certain nonassociative attentional hypotheses. For example, one might propose that the differences among the groups in the US-intensity study described above were due to differences in attention to X. If for any reason, it could be argued that attentiveness to X was some increasing function of US intensity, then one might predict less suppression to CX, the greater the intensity of the US experienced during conditioning. This same argument, however, could not be used to explain positive

results for conditioned inhibition on a new-learning test.

Finally, there are several potential confounding factors in the US-intensity study. In all fairness, it should be noted that the unpublished report by Rescorla and Wagner was quite brief, and that a complete presentation of the procedure and results might clarify some of the problems discussed below.

A major problem may lie in the failure to equate the groups for the number and kind of USs each group received. Because of this, the outcome might have been due to: (a) differences in habituation, sensitization, or attentiveness to the alleged inhibitory stimulus; (b) differences in drive level during conditioning that might have affected the strength of the inhibitory tendency independently of its relation to a nonreinforced excitor of a particular associative value; and/or (c) differences in the amount of fearfulness conditioned to apparatus cues. A comparison among the response baselines across groups might have shed light on this last possibility (cf. Annau & Kamin, 1961).

In addition, there may be a problem in interpreting the results of the combined-cue test if it can be argued that the C cue was not equally excitatory across all groups. That the groups did not differ in amount of suppression to C does not necessarily contradict this, because suppression was virtually complete in all groups. There may have been differences in associative value that were obscured due to a "floor effect" (e.g., as in the study of Annau & Kamin, 1961). Moreover, there are reasons to believe that C might not have been equally excitatory across groups. Several studies suggest that prior exposure to the US retards subsequent development of the CR (Kamin, 1961; Mis & Moore, 1973; Siegel &

Domjan, 1971; Taylor, 1956), so it is not unreasonable to expect C to have been less excitatory in the two experimental (pre-shocked) groups than in the nonshocked control. In addition, there are data that show this effect to be dependent upon the intensity of the US during the pre-exposure phase (Mis & Moore, 1973; Rescorla, 1974).

Another problem might be that both C and A were in the same modality (250-Hz vs 1.2-kHz tone). If, as the theory demands, A were differentially excitatory across groups, it might be reasonable to expect differences in amount of generalized excitation to C. In any event, there appear to be a variety of ways in which C might have attained a different associative value in each of the groups. If that were the case, the assumptions of the combined-cue test were violated, and the outcome would have to be judged equivocal. Perhaps data on the rate of acquisition of suppression to C would illuminate these possibilities.

The use of different US intensities introduces one other potential problem. In the study described earlier, all groups were switched from the various training-level intensities to a common intensity during the final phase of the experiment. Thus, it might be possible to explain the obtained results in terms of between-group differences in the amount of stimulus generalization decrement between the training and test phases of the experiment. The conditions during the final test phase were probably most similar to those of the .5-mA group during conditioning.

As has already been mentioned, the Rescorla-Wagner model does not incorporate the types of nonassociative mechanisms described in the previous paragraphs. It would appear, then, that the only way to

manipulate US intensity and still provide a test of the model would be to equate the groups for the number of exposures to each US intensity in much the same way that Spence and his associates did in their investigations of the effects of US intensity on excitatory associative strength (e.g., Spence, Haggard, & Ross, 1958). Thus, in the present experiments, all groups were exposed to all US-intensity values during conditioning. However, in each group, the to-be-conditioned inhibitor was compounded only with nonreinforced presentations of a CS that had been paired with a particular intensity US. To minimize the possibility of differential conditioning of fear to the apparatus cues and to permit on-the-baseline assessment of the development of the excitatory tendencies, the "extra" USs were preceded by another stimulus (cf. Rescorla, 1972). To circumvent problems possibly introduced by generalization decrement between conditioning and the new-learning test, all the US-intensity values used during conditioning were also used during the new-learning test.

Specifically, in Experiment 1, two groups of rats were initially trained to barpress for food reward, and were then exposed to a conditioning procedure designed to endow two stimuli, A and B, with positive (excitatory) associative value. The groups differed in the intensity of the US paired with A (.65 vs 1.3 mA), the stimulus that was subsequently to be contrasted with the to-be-conditioned inhibitor, X. For each group, B was paired with whichever US had not been paired with A. A stimulus diagram of the conditioning trials given to each group is presented in Figure 1. This training began on the baseline to monitor the development of excitation and to see whether between- or within-group differences in amount of suppression to each CS would emerge as a function of US intensity.

Figure 1. Simulus diagrams of the three types of conditioning trials given to Groups Lo and Hi in Experiment 1. A flashing light or tone was used for the A stimulus and the X stimulus (counterbalanced), and a buzzer was used for the B stimulus.

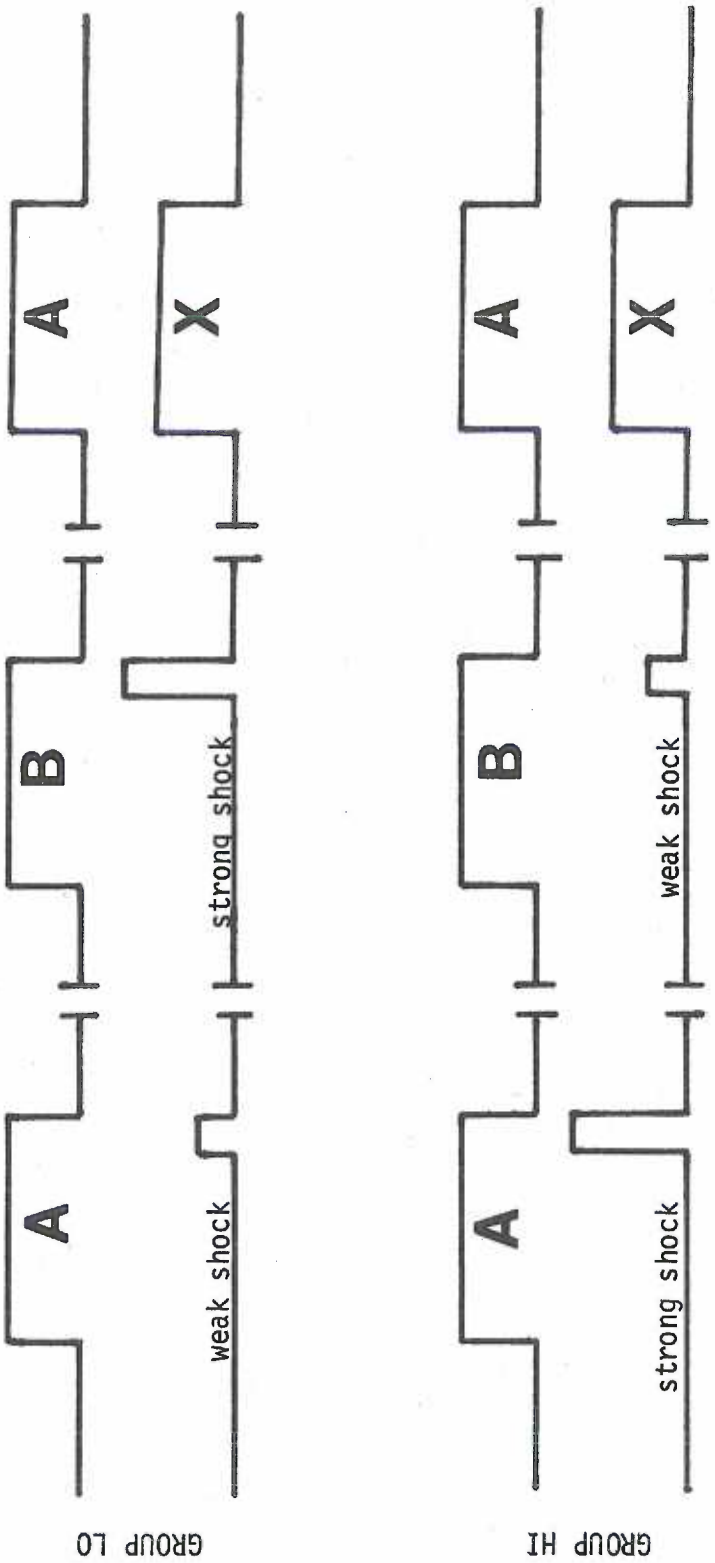
A
B
A
X
weak shock
strong shock
GROUP LO
A
B
A
X
strong shock
weak shock
GROUP HI

Figure 1

These intensities were chosen because a previous study involving US intensity in the CER paradigm had shown differences in suppression with similar values (Annau & Kamin, 1961). However, average US intensity had not been equated across groups in that study, and it was not clear that a within-group effect could be obtained. Once suppression to A and B had developed in the present study, excitatory conditioning was continued off-the-baseline. This was done in order to bring the associative value of each stimulus to asymptote without risking loss of the baseline level of barpressing unnecessarily. These tendencies were brought to asymptote before inhibitory training to assess certain predictions of the model regarding the development of the inhibitory tendency.

During the next phase of the experiment, reinforced A (A+) and reinforced B (B+) trials were still given, but in addition, nonreinforced presentations of the AX compound (AX-) were given. Presumably, these are the conditions that are appropriate for establishing an inhibitory tendency to X, a tendency that should be greater, the greater the associative value of A. This training began on-the-baseline, and when strong differential responding had appeared, conditioning was continued off-the-baseline in order to bring the strength of X to asymptote. The theory predicts that the group differences should be maximal at asymptote.

During the last phase, strength of inhibition to X was assessed by determining the rate at which X acquired suppressive properties when paired with shock on-the-baseline (a new-learning test). Specifically, X was paired with shock on a 50% reinforcement schedule. On half of the reinforced trials, X was paired with the weak US and on the other half, X was paired with the strong US. Finally, a series of 100%

nonreinforced X trials was given in order to assess the rate of extinction of the response conditioned to X in each group. It was expected that suppression would be less rapidly acquired and would dissipate more readily in the group for which A had been paired with the high-intensity US.

Method

Subjects

Sixteen naive, female albino rats, approximately 85-95 days old were used. These rats were Sprague-Dawley derived and were purchased from Carworth Farms, Portage, Michigan. All were caged individually and maintained in constant temperature conditions under a normal 12-hr light/dark cycle. Experimental sessions were conducted during the light part of the cycle at the same time each day.

The rats were gradually reduced to 80% of their initial body weight during the 15 days prior to the first day of barpress training. Throughout the experiment, each animal was weighed daily after its session and given lab chow in the home cage to maintain the 80% level. Water was available at all times in the home cage.

Apparatus

Four IOTEK (Iowa City, Iowa) operant-conditioning chambers (22.5 x 23 x 19 cm, inside) were used during all phases of the experiment. The end panels of each chamber were made of 1.5-mm aluminum and the side walls and ceiling were made of 6-mm clear Plexiglas. A Gerbrands feeder (Model D or D-1) delivered food pellets (45 mg, P. J. Noyes) to a Plexiglas foodcup attached in the center at the bottom of one of the end-panels. A Gerbrands rat lever was mounted to the left of the foodcup,

2.5 cm above the floor of the chamber. White pilot lights (6 W) were mounted on the panel to the left and to the right above the foodcup. These lights were 12 cm above the grid floor, and 11.5 cm apart, center to center. The grid floor of the chamber consisted of 2.3-mm stainless-steel rods mounted 1.27 cm apart. Each conditioning chamber was housed in a separate IOTEK ventilated, sound-attenuating chamber (36 x 71 x 34 cm, inside). A 6-W houselight was attached to the end wall of each chamber.

A Sonalert signalling device (4.5 kHz; Mallory SC628H) was mounted in the center of the ceiling in each operant chamber. The sound-pressure level of the tone was adjusted to 90 dB (re .0002 dyne/cm^2) as measured near the center of the chamber, 3 cm above the grid (H. H. Scott Sound-Level Meter, Type 450-B, A scale). A standard ac buzzer was attached to the ceiling of the sound-attenuating chamber, behind the wall opposite the food cup. Its intensity was 76 dB. In the absence of the tone and buzzer, the ventilating fans provided a masking noise of approximately 71 dB ($\pm$ 2 dB).

The US was the 350-V ac output of a step-up transformer wired in series with either 270 kΩ or 540 kΩ. These resistances provided short-circuit current values of 1.3 and .65 mA, respectively. Shock was delivered to the grid floor of each chamber via BRS grid scramblers (Model SC901).

A PDP8/F computer controlled all stimulus presentations and recorded all response measures.

Procedure

The experimental procedure is outlined in Table 1. On the first

Table 1

Procedure for Experiment 1

Days	Treatment
Pre-experiment	All rats reduced to 80% initial body weight; weighed and fed daily
	Barpress training
1	Shaping: minimum of 50 responses on CRF schedule
2 - 9	VI-2-min barpress training (90-min sessions)
10 - 11	On-the-baseline preconditioning exposures to A, B, X, and AX (once each per 100-min session)
	Excitatory conditioning (Phase 1)
	Group Hi: A + hi-US; B + lo-US Group Lo: A + lo-US; B + hi-US (totals: 42 A+ and 42 B+ trials)
12 - 18	On-the-baseline conditioning
19 - 24	Off-the-baseline conditioning
25 - 27	VI response baseline recovery sessions (no conditioning trials)
	Differential conditioning (Phase 2)
	Group Hi: A + hi-US; B + lo-US; AX alone Group Lo: A + lo-US; B + hi-US; AX alone (final totals: 84 A+; 84 B+; 100 AX-)
28 - 47	On-the-baseline conditioning
48 - 55	Off-the-baseline conditioning
56 - 57	VI response baseline recovery sessions
58 - 59	On-the-baseline conditioning
	New-learning test phase (On-the-baseline)
60	Pretest: Four nonreinforced X trials
61 - 66	CER acquisition to X: Four trials per 100-min session on 50% reinforcement schedule; half of reinforced trials with hi-US and half with lo-US
67 - 72	CER extinction: Four nonreinforced X trials per 100-min session

day of the experiment, all rats were magazine trained automatically with food pellets delivered on a variable-time 1-min schedule. In addition, the bar was smeared with wet food mash, and each barpress yielded a food pellet. The session continued until each animal had made 50 or more responses. Hand-shaping procedures were introduced if necessary. The next eight sessions (Days 2 - 9) were each 90 min long, and a variable-interval (VI) schedule of reinforcement was in effect. During the first 20 min of Day 2, the schedule was VI 1-min. During the remainder of that session, and during all subsequent sessions, the schedule was VI 2-min. The variable intervals were generated according to the constant probability formula proposed by Catania and Reynolds (1968) and their order was randomized.

Beginning on Day 10, all sessions were 100 min long. On each of Days 10 and 11, each of the following stimuli was presented while the rats were barpressing: flashing lights (houselight off/panel-lights on, twice per second), tone, flashing-lights-plus-tone compound, and buzzer. Each stimulus was presented for 2 min, and the order of presentation was randomized. Stimulus onsets occurred 17, 40, 65 and 80 min after the beginning of the session. Total responses were recorded during each stimulus presentation and during the 2 min preceding each CS. In order to compensate for individual differences in response rate, the relative-rate measure proposed by Annau and Kamin (1961) was used to index response strength. This suppression ratio was of the form CS/(CS + Pre-CS), where "CS" is the number of responses during the stimulus and "Pre-CS" is the number of responses during the 2 min before the stimulus. A ratio near .5 indicates little change in response

rate during the CS, whereas a ratio of 0.0 indicates complete cessation of responding during the CS.

At the conclusion of Day 11, four subgroups were formed (four rats each). Two of these groups were equated on the basis of their mean suppression ratio to the tone and the other two were equated on the basis of suppression to the flashing light. Tone or flashing light was the to-be-conditioned inhibitory stimulus (designated X) for each set of subgroups, respectively. One tone group and one light group were randomly assigned to each of the experimental conditions.

The first phase of Pavlovian conditioning began on Day 12. During this phase, each group received two types of conditioning trials. The buzzer (B) was presented on one of these trials for all groups. The flashing light or tone (whichever was not the X stimulus) was presented on the other trials. This latter stimulus, designated A, was the stimulus that was to be contrasted with X during subsequent differential conditioning. The groups differed in terms of the intensity of the US that was paired with A. In Group Hi, A overlapped and coterminated with a .5-sec, 1.3-mA shock, whereas B was paired with a .65-mA shock. These conditions were reversed in Group Lo.

An equal number of A+ and B+ trials was given on each conditioning day. The first eight trials (Days 12 - 18) to each stimulus were given while the animal was barpressing in order to monitor the development of suppression to each stimulus. Two trials to each stimulus were given on Day 12, and one trial to each stimulus was given on each of Days 13 - 18. The trials were distributed in this way in order to minimize the possibility of weakening the operant baseline. On Day 19, the foodcup

was removed and an aluminum cover was placed over the bar. Over the next six days (Days 19 - 24), conditioning was continued off-the-baseline. Six trials to each stimulus were given during each session at a mean intertrial interval of 6.9 min. Thus at the end of Day 24, each rat had been exposed to a total of 42 A+ and 42 B+ conditioning trials. The exact order of presentation of these trials is contained in the Appendix. The order of presentation was counterbalanced.

Days 25 - 27 were baseline recovery days. The foodcups were returned to the chambers and each animal was allowed to barpress for food on the VI-2 schedule. No stimuli were presented.

Differential conditioning was conducted over the next 28 days (Days 28 - 55). Each animal continued to receive A+ and B+ conditioning trials, but in addition, received nonreinforced AX compound trials. For the first 20 days, conditioning occurred on-the-baseline, with one A+, one B+, and two AX- trials each day. During the last eight days, conditioning was continued off-the-baseline. The intertrial interval was 6.9 min during off-the-baseline conditioning, and 12 trials were given during each session. By the end of this phase, a total of 82 A+, 82 B+, and 96 AX- trials had been given. The exact order of the trials is presented in Appendix B. Note that each AX- trial was preceded by an equal number of A+ and B+ trials. Also, the order of presentation of A+ and B+ trials was counterbalanced.

After two days of baseline recovery (Days 56 - 57), two more days of on-the-baseline differential conditioning were given (Days 58 - 59). On Day 60, X was presented alone on four trials, and no other stimuli were presented. The new-learning test was conducted over the next six

days (Days 61 - 66). On each of these days, X was paired with shock on a 50% reinforcement schedule--one trial terminated with the high-intensity US, one with the low-intensity US, and two trials were not reinforced. Extinction conditions were in effect over the final six days (Days 67 - 72). Four nonreinforced X trials were given during each session.

Results

The alpha level was set at .05 in all of the analyses described below. The data and major analysis of variance summary tables are contained in Appendix B.

Body Weights

There were no differences in the initial weights of the rats assigned to each group (F [1, 14] = 1.5). A mean weight was calculated for each rat over various phases of the experiment (see Appendix B) and a groups x phases analysis of variance was applied to these data. There was no groups effect or groups x phases interaction (Fs < 1.7), but there was a reliable overall change in weight across the various phases (F [7, 98] = 86.5). A followup comparison indicated that the effect was in part due to an overall 10-gm decrease in weight over the course of the experiment. Each group's mean weight weight over all phases was within .5% of 80% of its initial mean weight.

Response Baselines

Between-group differences in suppression ratios may be difficult to interpret if there are also between-group differences in the average rate of barpressing in the absence of the CS. To see whether baseline differences existed in the present experiment, the mean numbers of responses occurring during the 2-min pre-CS intervals were calculated for each rat during every on-the-baseline conditioning session. Baseline measures

were also obtained on the last VI-training day and on the baseline recovery days by recording the responses occurring in the intervals that would normally have preceded a conditioning trial. These scores were averaged over sessions across various phases of the experiment (see Appendix B), and between-groups (or groups x blocks) analyses were conducted. A reliable group difference appeared only once, during the first series of VI recovery days (Days 25 - 27)(F [1, 14] = 5.8). Group Lo responded less often than Group Hi on these days. However, since no group differences appeared during any phase in which stimuli were presented, it was concluded that interpretation of the suppression ratio data would not be obscured by differences in the baseline level of barpressing.

Two other effects emerged from these analyses, both due to increases in response rate over blocks of conditioning sessions. The first occurred during Phase-1 excitatory conditioning (Days 12 - 18)(F [3, 42] = 4.4), and the second during the initial Phase-2 differential conditioning (Days 28 - 47)(F [4, 56] = 6.2).

Suppression Ratios

Preconditioning exposures. A mean suppression ratio was calculated for each rat for each of the four types of stimulus pre-exposures. The overall mean suppression ratios were .48, .58, .47, and .51 for the stimuli that were to be used as A, B, X, and AX, respectively. The ratios for A and B are shown as the "P" points in Figures 2, 3, and 4. Group assignment (Hi or Lo) and stimulus designation (A, B, X, or AX) were used as "dummy" factors in an analysis of these data. There was no group difference, but there was an overall effect due to stimulus-type (F [3, 42] = 5.3). Simple followup comparisons indicated that the difference between the ratios to B and to every other stimulus was reliable. There were no differences among the ratios to A, X, and AX. These analyses suggested that there were no

Figure 2. Mean suppression ratios for Group Lo subgroups during Phase-1 and Phase-2 on-the-baseline conditioning. The "P" points represent the preconditioning suppression ratios obtained on Days 10 - 11. The "F" points represent the last two days of differential conditioning (Days 58 - 59).

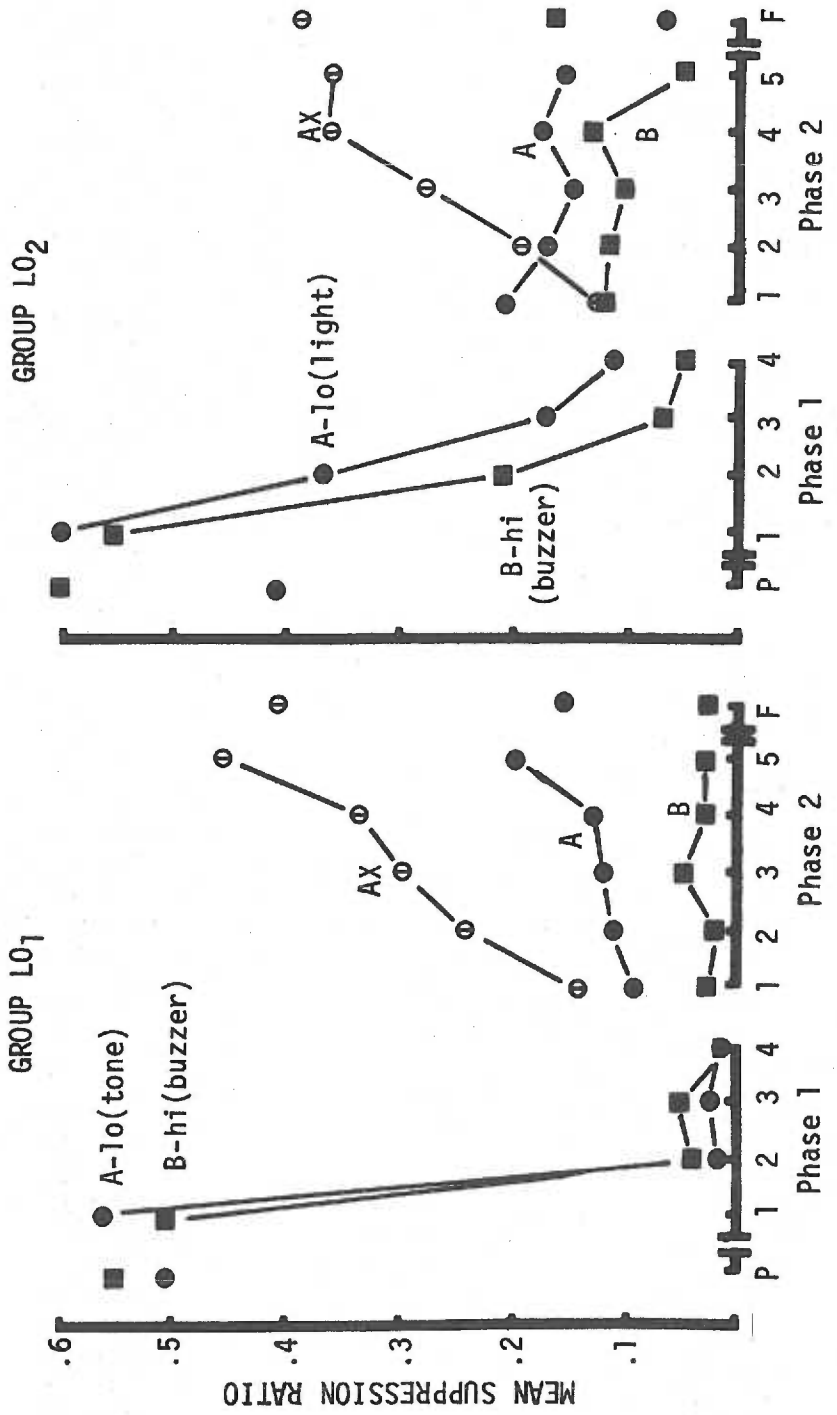
GROUP LO1
GROUP LO2
A-lo(tone)
B-hi(buzzer)
A-lo(light)
B-hi (buzzer)
AX
A
B
MEAN SUPPRESSION RATIO
.6
.5
.4
.3
.2
.1
P 1 2 3 4
1 2 3 4 5 F
Phase 1
Phase 2
TRIAL BLOCKS

Figure 2

Figure 3. Mean suppression ratios for Group Hi subgroups during Phase-1 and Phase-2 on-the-baseline conditioning. The "P" points represent the preconditioning suppression ratios obtained on Days 10 - 11. The "F" points represent the last two days of differential conditioning (Days 58 - 59).

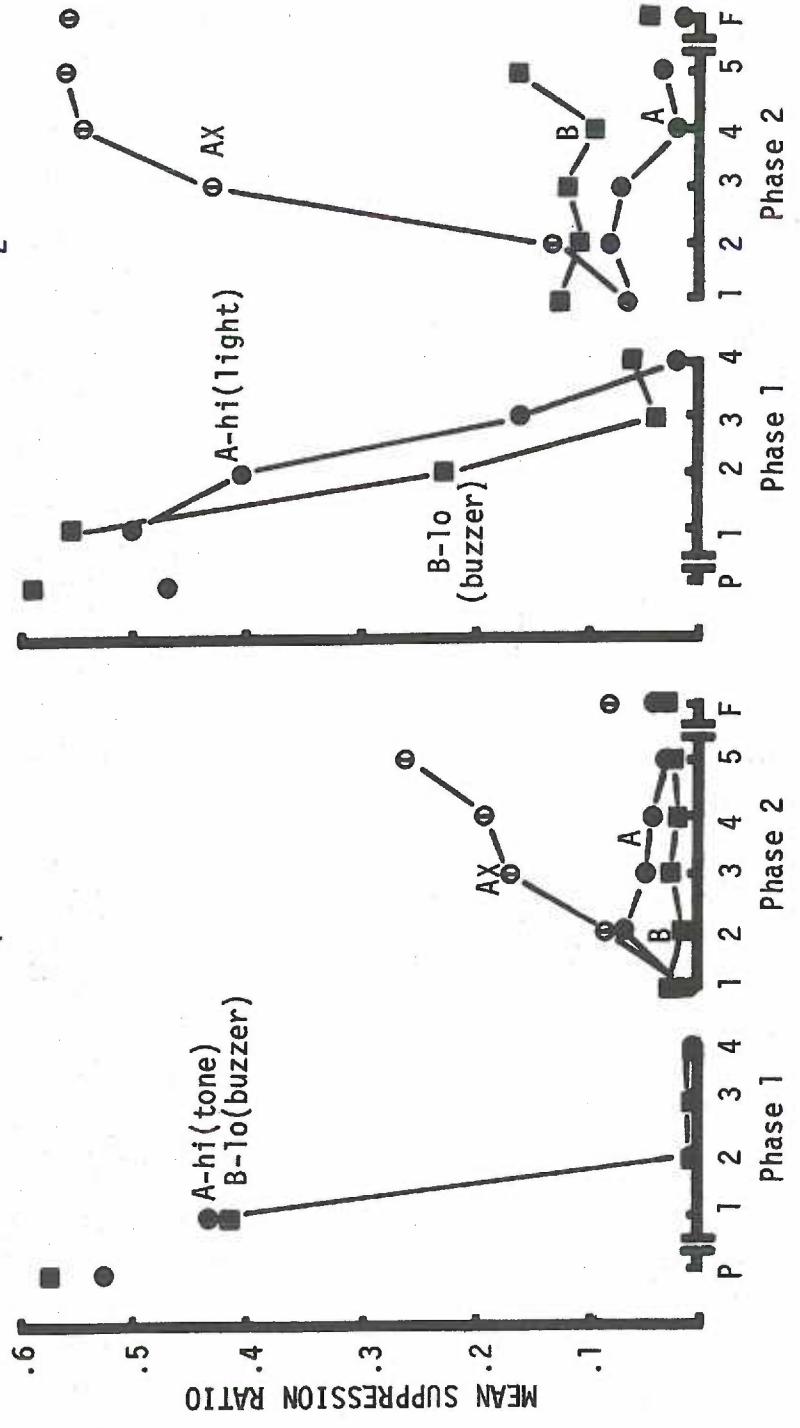
GROUP HI1
GROUP HI2
MEAN SUPPRESSION RATIO
.6
.5
.4
.3
.2
.1
A-hi(tone)
B-lo(buzzer)
A-hi(light)
B-lo
(buzzer)
AX
A
B
P 1 2 3 4
1 2 3 4 5 F
Phase 1
Phase 2
TRIAL BLOCKS

Figure 3

Figure 4. Mean suppression ratios for Groups Lo and Hi during Phase-1 and Phase-2 on-the-baseline conditioning. These data are the same as those presented in Figures 2 and 3, with the subgroup factor eliminated. The "P" points represent the preconditioning suppression ratios obtained on Days 10 - 11. The "F" points represent the last two days of differential conditioning (Days 58 - 59).

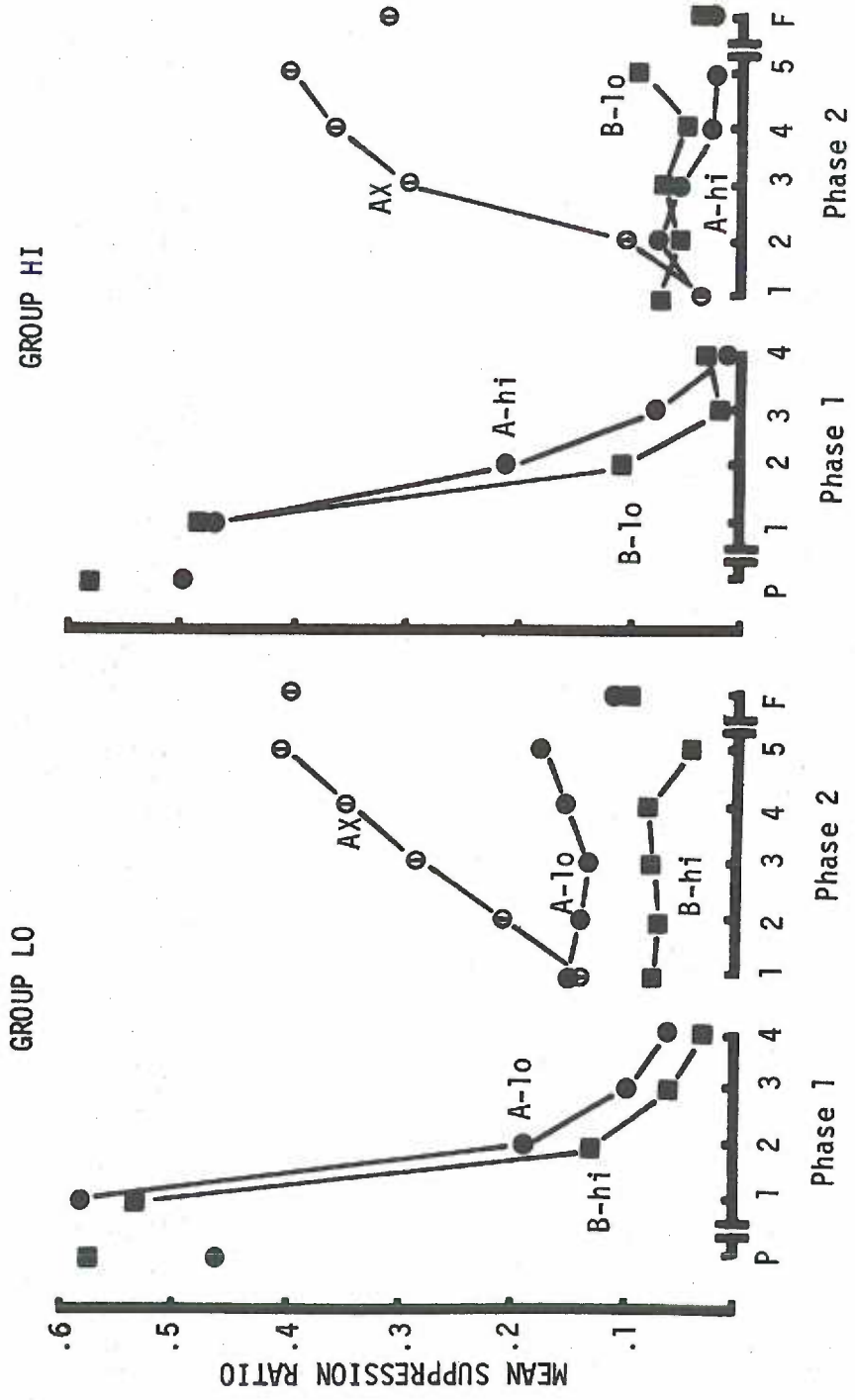
GROUP LO
GROUP HI
MEAN SUPPRESSION RATIO
.6
.5
.4
.3
.2
.1
A-lo
B-hi
AX
A-hi
B-lo
P 1 2 3 4
1 2 3 4 5 F
Phase 1
Phase 2
TRIAL BLOCKS

Figure 4

pre-treatment differences in the unconditioned responses to the stimuli of major importance in this experiment.

Because of the counterbalancing of A and X, the above analysis did not completely reflect the unconditioned reactions to the specific stimulus events used as CSs. In a second analysis, therefore, responses to the flashing light, tone, flashing-light-plus-tone, and buzzer were compared. The effect of stimulus type was reliable (F [3, 45] = 8.4), and followup comparisons suggested that the rats suppressed more to the light and light-tone compound (mean ratios of .45 and .47, respectively) than they did to the tone or buzzer (.53 and .58, respectively). In general, the auditory stimuli slightly facilitated barpressing whereas the visual stimulus had a mild suppressive effect. The unconditioned response to the compound appeared to be dominated by the visual component.

Acquisition: Phase 1. Data gathered during the initial excitatory-conditioning phase were analyzed in two-trial blocks according to stimulus type (A vs B). The mean suppression ratios during each block in Phase 1 are depicted for each of the four subgroups in Figures 2 (Group Lo) and 3 (Group Hi). Figure 4 shows these same data collapsed over subgroups. In general, the subgroups that received two auditory stimuli conditioned more readily than those receiving a visual and auditory stimulus. That the development of suppression to the buzzer was slower in the groups receiving buzzer and light than it was in the groups receiving buzzer and tone suggests the influence of stimulus generalization effects. The light appeared to acquire suppressive properties less rapidly regardless of the intensity of the US with which it was paired.

Overall, there seemed to be no within- or between-group effects of US intensity. A groups x stimulus-type x blocks analysis yielded significant main effects for stimulus-type (F [1, 14] = 6.2) and blocks (F [3, 42] = 81.5). Examination of the subgroup graphs suggests that the overall difference between suppression to A (mean = .21) and B (mean = .18) was due to the fact that when A was the flashing light, suppression developed less rapidly.

Acquisition: Phase 2. The mean suppression ratios to A, B, and AX during differential conditioning (Phase 2) are shown in the right-hand sides of the panels in Figures 2, 3, and 4. Mean ratios were computed in four-day blocks over the first 20 days of conditioning (Days 28 - 47). The final Phase-2 data point (marked "F" on the graphs) represents the mean suppression ratios for the two days following Phase-2 off-the-baseline conditioning (Days 58 - 59). From the figures it can be seen that in each subgroup, the compound (AX) came to elicit less suppression than either A or B over the first 20 days. Moreover, in three of the four subgroups, the mean suppression ratios to A, B, and AX were ordered according to the intensity of the US paired with each stimulus (Group HI_1 being the exception). In terms of individual mean suppression ratios over these 20 days, all eight rats in Group Lo were less suppressed to A (paired with the weak US) than they were to B (paired with the strong US). Five of the eight rats in Group Hi were more suppressed to A (paired with the strong US) than to B (paired with the weak US). All rats were less suppressed to the AX compound than they were to the stimulus paired with the high-intensity US, and 14 out of 16 (seven of eight in each group) were less suppressed to the compound than they were to the

stimulus paired with the low-intensity US.

A groups x type-of-stimulus x blocks analysis over the first 20 days of Phase 2 indicated reliable effects due to stimulus type (F [2, 28] = 24.2), blocks (F [4, 56] = 14.7), the stimulus-type x blocks interaction (F [8, 112] = 24.8) and the three-way interaction (F [8, 112] = 2.5). Examination of the data suggested that the triple-order interaction may have been due, in part, to the between-group difference in the divergence of suppression to A and to the AX compound over blocks. These data have been replotted in Figure 5. As can be seen, suppression to A and AX was the same within each group at Block 1, but overall, Group Hi was more suppressed than Group Lo. Over blocks, both groups showed a divergence in amount of suppression to these stimuli, but the absolute difference in suppression between stimuli was greater at Block 5 in Group Hi than it was in Group Lo. These observations were supported by a reliable group difference between the interaction contrasts based on responding to A and to AX in Blocks 1 and 5 (F [1, 112] = 4.7; see Table 2).

In an attempt to interpret further the higher-order interaction, groups x blocks analyses were applied separately for each stimulus type. The two-way interaction was nonsignificant in every case (Fs [4, 56] = 1.5, 1.9, and 1.7, for A, B, and AX, respectively), suggesting that the blocks effect for each stimulus did not differ as a function of groups. The only reliable main effects to emerge from these analyses were the group differences in responding to A (F [1, 14] = 5.5) and the blocks effect in responding to AX (F [4, 56] = 30.9). Responding to A was ordered in accord with the intensity of the US paired with A in each

Figure 5. Mean suppression ratios to A and to AX in Groups Lo and Hi during Phase-2 differential conditioning. Each trial-block mean is an average over four days of conditioning. The "F" points represent the last two days of differential conditioning (Days 58 - 59).

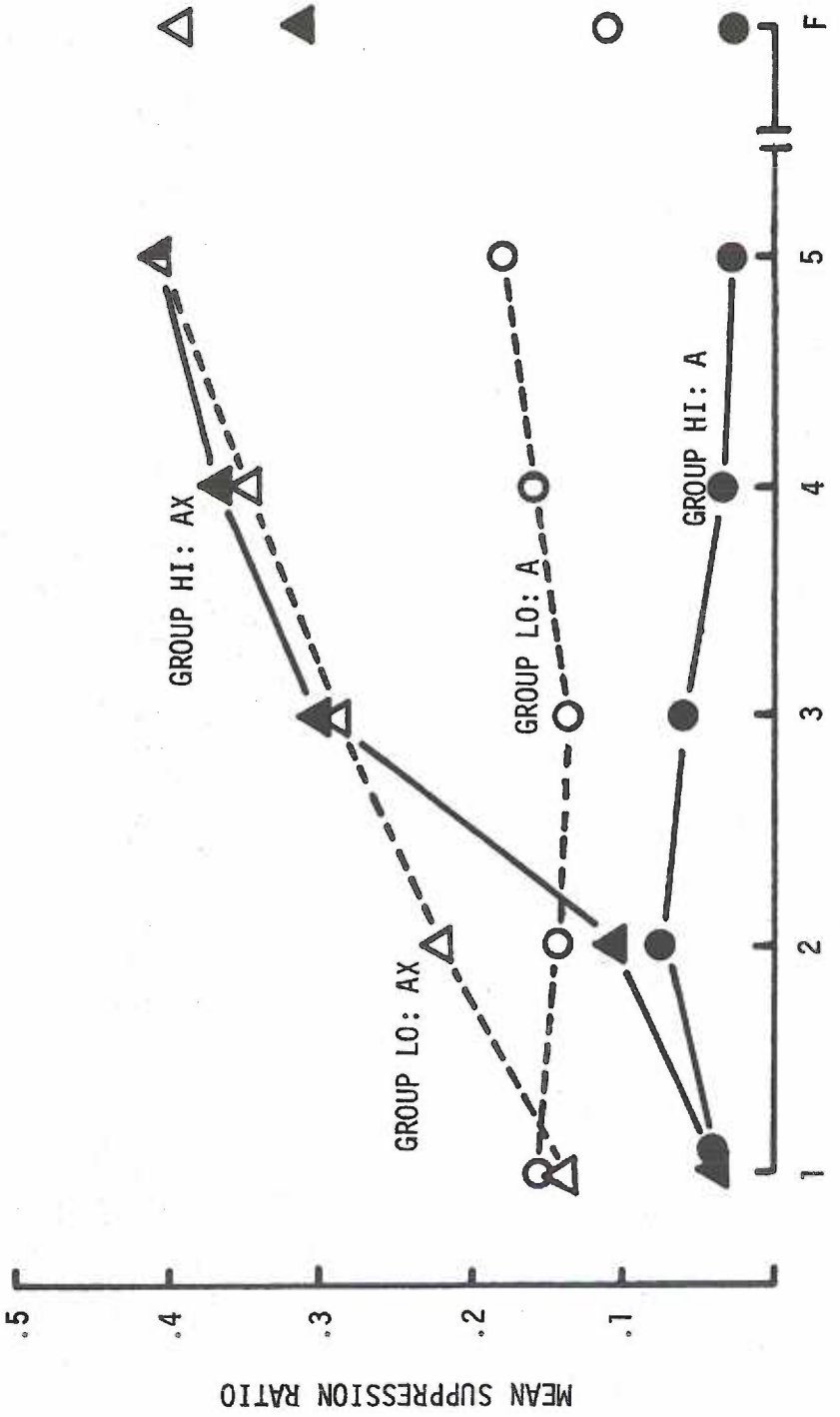
MEAN SUPPRESSION RATIO
.5
.4
.3
.2
.1
1
2
3
4
5
F
TRIAL BLOCKS
GROUP HI: AX
GROUP LO: AX
GROUP LO: A
GROUP HI: A

Figure 5

Table 2

Followup comparison between interaction contrasts based on responding to A and to AX during Blocks 1 and 5 of differential conditioning

Computational formula for numerator of F-ratio:

$$n/8\ [[\underbrace{(\overline{AX}_1 - \overline{AX}_5) - (\overline{A}_1 - \overline{A}_5)}_{\text{Group Lo}}] - [\underbrace{(\overline{AX}_1 - \overline{AX}_5) - (\overline{A}_1 - \overline{A}_5)}_{\text{Group Hi}}]]^2 =$$

$$[[(.140 - .412) - (.152 - .180)] - [(.039 - .411) - (.039 - .031)]]^2$$

$$= .018496$$

F-test:

$$F\ (1, 112) = .018496 / \text{MS error (stimuli x blocks)}$$

$$= .018496 / .003951$$

$$= 4.68 \quad (p < .05)$$

group. That is, Group Hi was more suppressed (mean = .05) than Group Lo (mean = .15). The group difference in responding to B was nonsignificant ($F < 1$; means of .072 and .070 for Groups Hi and Lo, respectively).

The stimulus-type x blocks interaction proved significant in separate two-way analyses for each group (Fs [8, 56] = 10.2 and 15.9, for Groups Lo and Hi, respectively). In each instance, these effects appeared primarily to be due to the divergence between the level of responding to AX (gradually increasing over blocks) and the levels of responding to A and B (relatively constant over blocks). Each of these analyses also indicated a reliable effect of stimulus type (Fs [2, 14] = 11.1 and 15.5, for Groups Lo and Hi, respectively). Because the contrast derived from the overall analysis had already examined the divergence between level of responding to A and to AX, only the difference between responding to A and B in each group was evaluated as a followup to these main effects. Essentially, these tests were directed towards determining whether within-group differences appeared as a function of US intensity. The difference between A and B was not reliable in either group (Fs [1, 14] = 3.3 and .4, for Groups Lo and Hi, respectively).

Although the subgroup condition was not formally considered as a factor in the above analyses, it would appear that the Group-Hi subgroup for which the flashing light had been the A stimulus (Group HI_2 in Figure 3) showed a much larger difference in responding to A and to AX than did the HI_1 subgroup. On the other hand, the Group-Lo subgroup that received the light as the A stimulus (Group LO_2 in Figure 2) showed about the same, if not less of a differential response than the LO_1 subgroup. However, because of the small n in each subgroup, and because there was

no reason to expect between-group differences in the direction of these effects, the counterbalancing factor was ignored in the present analysis.

A separate groups x type-of-stimulus analysis was applied to the mean suppression ratios recorded on the final two days of on-the-baseline differential conditioning (the points marked "F" in Figures 4 and 5). The main effect due to type-of-stimulus was the sole reliable outcome of this analysis (F [2, 28] = 22.5), and it appeared simply to reflect the greater suppression to A and B (means = .07 and .07, respectively) than to AX (mean = .36). The failure to find a reliable interaction suggested that the earlier between-group difference in the magnitude of the difference in responding to A and AX had disappeared. This seemed primarily to be due to an increase in suppression to A in Group Lo and an increase in suppression to AX in Group Hi. Visual inspection of the subgroup data (Figures 2 and 3) indicated that this latter increase was the result of a near complete loss of differential responding in the Group-Hi subgroup receiving light as the X stimulus (Group HI_1). To see whether the earlier between-group difference in responding to A had been maintained, a separate comparison was made between each group's responses to A on these two days. Group Lo showed a mean ratio of .11, whereas Group Hi showed a mean ratio of .03. This difference was of marginal significance (F [1, 14] = 4.42, $p < .054$).

New-learning test phase: Pre-exposures to X alone. A mean suppression ratio was computed for each rat based on responding to the nonreinforced presentations of X prior to the new-learning test. The data are represented by the "P" points in Figure 6. To see whether the groups

Figure 6. Mean suppression ratios to X in two-day blocks for Groups Lo and Hi during new-learning-phase acquisition and extinction. The "P" points represent the mean preconditioning suppression ratios obtained on Day 60.

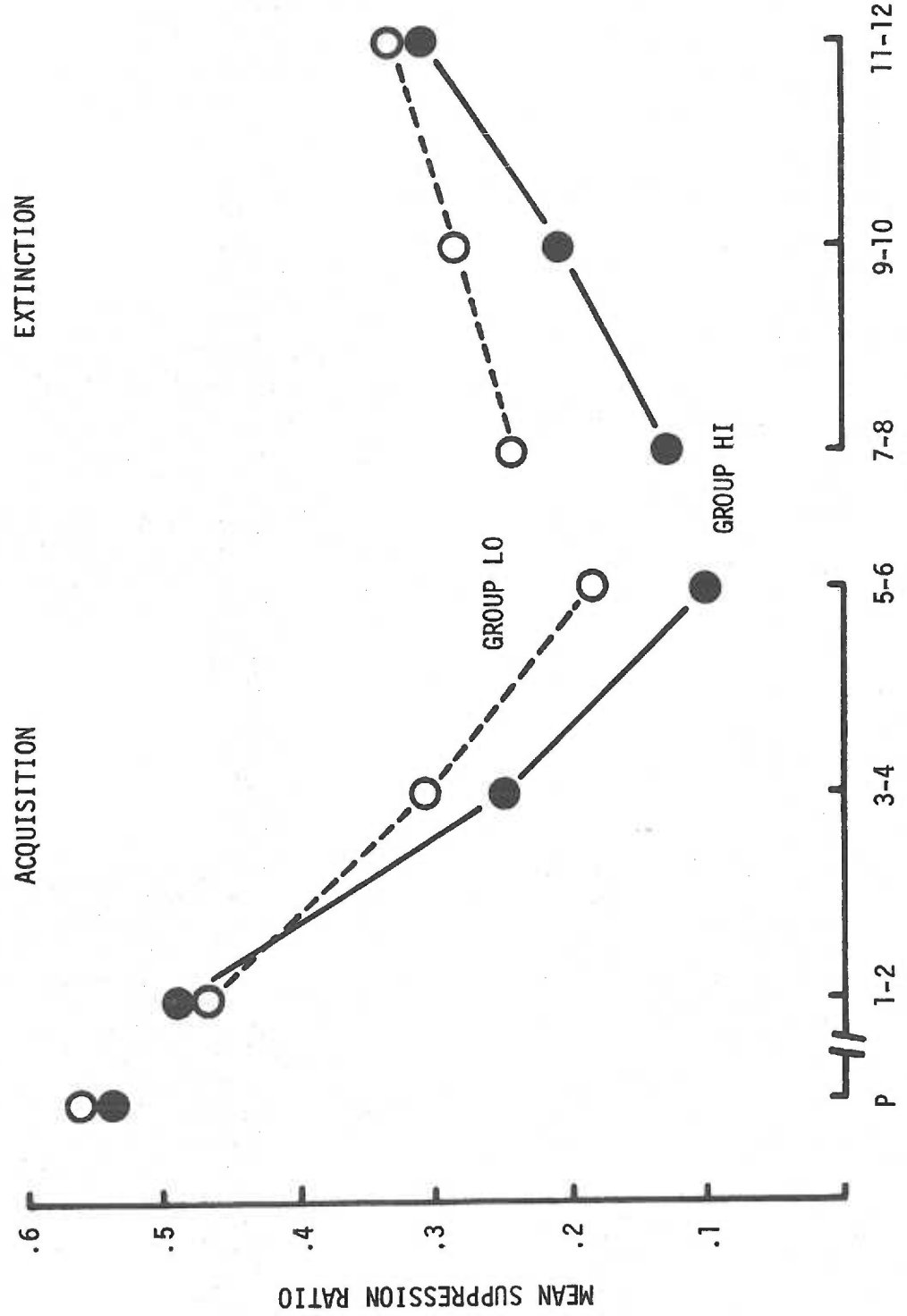
ACQUISITION
EXTINCTION
GROUP LO
GROUP HI
MEAN SUPPRESSION RATIO
.6
.5
.4
.3
.2
.1
P
1-2
3-4
5-6
7-8
9-10
11-12
DAYS

Figure 6

differed at this point, and to see whether their reactions to X alone were any different from their pre-conditioning reactions to X (as measured on Days 10 and 11 of the experiment), a groups x days analysis was applied to these data and to the mean pre-conditioning ratios to X. The mean pre-to-post conditioning ratios were .50 to .56 for Group Lo, and .51 to .54 for Group Hi. There was a slight but nonreliable increase from the pre- to post-conditioning session for both groups, and no differences between groups. These data were re-examined in terms of the actual stimulus event used as X (light or tone). The mean response ratio to the light was .49, and to the tone, .57. Once again, the mean post-conditioning ratios were slightly higher than the pre-conditioning ratios (.45 and .53 for light and tone, respectively). Moreover, the visual stimulus was slightly suppressive and the auditory stimulus was facilitative.

New-learning test phase: Acquisition and extinction. A mean suppression ratio was calculated for each rat in blocks of two days (eight trials) during both acquisition and extinction in the new-learning phase. These data are shown in Figure 6. Separate groups x days analyses were applied to each of these sets of data. In each case, the only reliable outcome of the analysis was the days effect (Acquisition: F [2, 28] = 73.6; Extinction: F [2, 28] = 20.4).

A "trials-to-criterion" measure was also derived from the acquisition suppression-ratio data. The scores were divided into two-trial blocks and the number of trials until each rat's mean suppression ratio was .2 or less was calculated. The median numbers of trials to criterion were 10.5 and 14.5 for Groups Hi and Lo, respectively. A Mann-Whitney

comparison indicated that this difference was not significant (U = 19.5).

Response Latencies

To obtain independent evidence for the effect of US intensity, the median latency to the first response after each type of conditioning trial (hi-US vs lo-US) was recorded during the initial portion of Phase-1 excitatory conditioning (Days 12 - 18). Overall, the rats waited an average of 10.4 sec after the weak US and 14.9 sec after the strong US. On the preconditioning exposure days (Days 10 - 11), the average median latency in the absence of shock had been 6.8 sec. A groups x US intensity analysis of the Phase-1 latencies indicated that the rats responded reliably slower after the strong US (F [1, 14] = 8.5).

The post-CS latencies were also recorded during the new-learning test phase. The median latency to the first response following non-reinforced, weak-US, and strong-US trials was calculated for each rat in two-day blocks during both the acquisition and extinction of suppression to X. A groups x trial-type x blocks analysis of the acquisition scores failed to show any reliable effects, although the interaction of trial-type x blocks approached significance (F [4, 56] = 2.45, $p < .1$). No reliable effects appeared in the analysis of extinction scores.

A median latency to the first response during the CS was also calculated in two-day blocks during the new-learning test phase. Separate groups x blocks analyses were performed on the acquisition and extinction scores. No group differences emerged, and the sole reliable outcome of these analyses was due to the change in response latency over blocks during acquisition (F [2, 28] = 5.8)(Means: Block 1 = 5.2; Block 2 = 20.2; Block 3 = 36.6 sec).

Discussion

The new-learning test phase of the preceding experiment failed to provide any support for the prediction that X would become more inhibitory the greater the excitatory value of the stimulus with which it had been compounded and nonreinforced. If anything, there was a tendency for the group that should have had the stronger inhibitory tendency (Group Hi) to become more suppressed during the test-phase CER acquisition and extinction. There are several ways to interpret this lack of a difference.

First, the theory may be wrong. Strength of conditioned inhibition might simply be a function of nonreinforcement, independent of the excitatory value of the stimulus compounded with the to-be-conditioned inhibitor. A theory that described the inhibitory process in terms of the relation between the to-be-conditioned inhibitor and the US, regardless of US intensity or the excitatory strength of any other stimuli in the conditioning situation, might more adequately account for the present results. The US-intensity effect reported by Rescorla and Wagner (1972) would then have to be attributed to the nonassociative factors discussed earlier. Rescorla's (1967b) contingency account of conditioning might provide an appropriate model because, as it was stated, only the relative probability of US occurrence was important, not US intensity. However, it might be that in using different US intensities, one might want to redefine the size of the time intervals used to bound "CS" and "non-CS" periods within the contingency framework.

Advocates of the hypothesized relation between strength of conditioned inhibition and strength of conditioned excitation might judge

abandonment of the Rescorla-Wagner model premature. There is always a danger in accepting the null hypothesis as evidence against a theory that predicts a difference in behavior. For example, it might be argued that the US-intensity values used in the preceding study were "not different enough." There have been no parametric studies of US intensity as a within-subject variable in the CER situation, so this argument cannot be countered by appealing to data other than that derived from the present experiment. One can only point to the reliable between-group difference in suppression to A during differential conditioning as support for the view that A had acquired a different associative value in each group. However, the failure to find within-group US-intensity effects, or a between-group difference in responding to B is not readily explained, and could be viewed as supporting the argument that the US-intensity values were not discrepant enough.

The only data in the preceding experiment that might be consistent with the Rescorla-Wagner model's treatment of conditioned inhibition were gathered during the first 20 days of differential conditioning. Assuming that the suppression ratios to A and AX reflected the associative values of A and AX, respectively, then the following aspects of the data were in agreement with predictions offered by the model: (1) Suppression to AX was initially greater in Group Hi than in Group Lo (cf. Figure 5) (Because X presumably had no associative value at that point, i.e., $V_X = 0$, the associative value of AX was determined entirely by the associative value of A in each group.). (2) Both groups reached the same final level of responding to AX, but the rate of loss of suppression to AX was greater in Group Hi (The asymptote, λ, associated with

nonreinforcement was the same in each group, but the discrepancy between λ and V_{AX} was initially greater in Group Hi.). (3) The absolute difference in amount of suppression to AX and to A was greater in Group Hi (The associative value of A was higher.). It is clear, however, that these outcomes do not necessarily depend upon the differential development of inhibition to X in each group. All the outcomes described above might simply be explained in terms of the extinction of a generalized excitatory response to the compound stimulus. It was because of ambiguity like this that a new-learning test (or combined-cue test) was required to assess strength of inhibition.

Unfortunately, even if the data from the first 20 days of differential conditioning were to be viewed as supporting a conditioned-inhibition interpretation, there is no easy way to explain the changes in differential responding to A and to AX after off-the-baseline conditioning in Phase 2 ("F" points in Figure 5). For whatever reason, the between-group difference in the magnitude of the difference in responding to A and to AX had disappeared. If the magnitude of that latter difference were related to the inhibitory strength of X (as the Rescorla-Wagner model suggests), then the fact that both groups showed the same final A - AX difference might be consistent with the failure to find differential acquisition of suppression to X during the new-learning test. What remains to be explained, however, is what happened to the group difference in the magnitude of the differential response.

There may be one other problem in interpreting the results of Experiment 1. It could be argued that the conditioning parameters (number of trials, temporal spacing, etc.) were somehow inappropriate for the

development of conditioned inhibition. As the experiment was designed, the ability to assert that different inhibitory tendencies had been established hinged upon the attainment of a group difference in the new-learning test. In the absence of such a difference, there was no control to assess whether X had become at all inhibitory in either group. The failure to find that difference might mean that in both groups, X was equally inhibitory, neutral, or even excitatory. The only comparison that might shed light on this would be between the new-learning test and Phase-1 excitatory conditioning. It is quite apparent that the acquisition of suppression to X was much slower than the original acquisition of suppression to A and B. However, this difference could be readily explained by appealing to the partial-reinforcement schedule during testing or to differences in prior experiences with the CSs and USs during each stage of conditioning. It would appear that the only argument in support of the view that X should have been inhibitory in Groups Lo and Hi must rely upon the similarity among the present conditioning procedures and the "successful" procedures described in the literature review (see Appendix A).

EXPERIMENT 2

Because of the problems posed by the outcome of Experiment 1, a second experiment was conducted in an attempt to resolve them. First in seeking to optimize the conditions for obtaining a US-intensity effect, the difference between the low and high intensity US was increased (.4 vs 1.6 mA). Presumably, this should have increased the differences between the associative strengths of A in each group.

Another major concern was the change in the amount of differential responding that had occurred after off-the-baseline conditioning in Phase 2 of Experiment 1. Shifting from an on-the-baseline procedure to an off-the-baseline procedure and then back on again apparently disrupted the pattern of responding that had been established during the initial stage of differential conditioning. It is not clear, however, why Group Hi seemed to be more affected by these changes than Group Lo (except, perhaps, simply because there was a greater initial difference in responding to A and to AX in Group Hi). Nonetheless, it might be argued that the procedure of shifting back and forth was undesirable because of potential stimulus-generalization-decrement effects. There were a number of potentially effective stimulus changes that occurred as a result of each shift. In going off-the-baseline, the foodcup and bar were removed, the internal cues associated with barpressing and eating were eliminated, and the average intertrial interval was reduced. Any one of these changes might have affected transfer of associative tendencies during the successive stages of differential conditioning.

In Experiment 2, all conditioning was conducted under the same condition--namely, off-the-baseline. Although this procedure did not

eliminate the possibility of generalization decrement upon transfer to the new-learning test (which had to be given on-the-baseline), it should have eliminated whatever detrimental effects might have been due to multiple changes in the "stimulus context" during successive stages of conditioning in the first experiment. Moreover, as has been mentioned earlier, the off-the-baseline procedure may have an advantage in that it minimizes interaction between Pavlovian fear conditioning and the variables associated with the maintenance of the operant response.

Another problem discussed above was whether X had acquired any inhibitory power in either group in Experiment 1. If the parameters used in Experiment 2 were also to fail to produce a US-intensity effect, knowing whether X was inhibitory might aid in interpreting such an outcome. Thus, a control group that should not have developed an inhibitory response to X was run. There appeared to be at least two ways to eliminate the development of inhibition to X, yet at the same time, to equate the control group for the number and kind of CSs and USs received by the experimental groups: (a) "unpairing" A and X on trials that would have been AX trials (i.e., randomly programming nonreinforced A and X presentations), or (b) "unpairing" A and the US on trials that would have been A+ trials. In both instances, X would be expected to acquire relatively little inhibitory value with respect to the experimental groups. However, the first method fails to equate all groups for prior exposure to X in compound with A. Mackintosh (1973) has shown that "latent-inhibition" effects are greater when X has been presented in compound with another stimulus. Thus, the second procedure was used in Experiment 2. It is described in greater detail in the

procedure section below.

Finally, a change was made in the counterbalancing of the stimuli. This was done to minimize the plausibility of an alternative explanation of a positive result in terms of generalization of excitation. Specifically, suppose that the predicted difference had been obtained on the new-learning test in Experiment 1. Although this result would have favored a conditioned-inhibition interpretation, it might have been explained as primarily due to generalized excitation. This is easiest to see in the subgroups that had received tone as the conditioned inhibitor. Because the tone and buzzer were in the same modality, the tone might have been expected to elicit a generalized excitatory response, a response that would have been greater in Group Lo than in Group Hi. The difference between groups on the new-learning test might then have been attributed to a facilitation of learning in Group Lo (because X elicited a stronger generalized excitatory response) rather than to retardation in Group Hi (because X elicited a stronger inhibitory response). Although this argument might not seem as reasonable in the case of the subgroups receiving light as the conditioned inhibitor, in principle, it can be made whenever the alleged inhibitor might be said to elicit a generalized response originally conditioned to the B stimulus. In Experiment 2, this problem was circumvented by holding the nature of the X stimulus constant across groups (flashing light), while counterbalancing the nature of the stimuli used as A and B (tone vs buzzer). Because the B stimulus was no longer the same across groups, it would be difficult to argue that test-phase results were due to a specific relation between the alleged inhibitor and a particular type

of B stimulus. Even though this procedure might limit the generality of a positive outcome to the effects of a visual conditioned inhibitor, the ability to eliminate alternative interpretations seemed worth the risk.

Method

Subjects

The subjects were 24 naive, female albino rats, 105 - 110 days old at the beginning of the experiment. They were obtained from the same supplier and maintained as in Experiment 1. All were reduced to 80% of their initial body weight before barpress training and were weighed and fed daily after each session.

Apparatus

The operant-conditioning chambers and stimuli described in Experiment 1 were also used in this experiment. The only changes were in the resistors wired in series with the 350-V ac shock sources. The low-intensity US was changed to .4 mA (890 kΩ) and the high-intensity US was changed to 1.6 mA (220 kΩ).

Procedure

The experimental procedure is outlined in Table 3. All rats were initially trained to barpress for food reward on a VI-2-min schedule as in Experiment 1. After the initial shaping day, each rat received seven more days of VI training (100-min sessions). On Day 9, each rat was exposed once to each of the following stimuli: tone, flashing light, and buzzer. Each stimulus was presented for 2 min. Stimulus onsets occurred 22, 50 and 75 min after the beginning of this 100-min pretest session. Order of presentation was randomized.

Table 3

Procedure for Experiment 2

Days	Treatment
Pre-experiment	All rats reduced to 80% initial body weight; weighed and fed daily
	Barpress training
1	Shaping: minimum of 50 responses on CRF schedule
2 - 8	VI-2-min barpress training (100-min sessions)
9	On-the-baseline preconditioning exposures to A, B, and X (once each in 100-min session)
	Conditioning (Off-the-baseline)
	Group Hi: A + hi-US; B + lo-US; AX alone Group Lo: A + lo-US; B + hi-US; AX alone Group Random: unpaired A and lo or hi US; B + hi or lo US; AX alone (totals: 40 A+; 40 B+; 70 AX-)
10 - 24	Off-the-baseline conditioning
25 - 27	VI response baseline recovery sessions (no conditioning trials)
	Post-conditioning test (On-the-baseline)
28 - 29	Nonreinforced test presentations of A, B, and AX (once each per 100-min session)
	New-learning test phase (On-the-baseline)
30 - 35	CER acquisition to X: Four trials per 100-min session on 50% reinforcement schedule. Half of reinforced trials with hi-US and half with lo-US
36 - 41	CER extinction: Four nonreinforced X trials per 100-min session
	Final test (On-the-baseline)
42	Nonreinforced test presentations of A, B, X, and AX (twice each during 100-min session)

On the following day, the foodcup was removed and the bar was covered. Off-the-baseline conditioning was conducted for the next 15 days (Days 10 - 24). Ten conditioning trials were given each day at an average intertrial interval of 8 min (range = 4 - 12 min). In general, the conditioning procedure resembled that described in Experiment 1. The two experimental groups, Groups Lo and Hi, received A+, B+, and AX- conditioning trials. The groups differed in the intensity of the US paired with A. For Group Lo, A was paired with the .4-mA US, and B was paired with the 1.6-mA US. The conditions were reversed in Group Hi. In contrast to the previous experiment, X was always the flashing light, whereas the stimuli that served as A and B were counterbalanced (tone or buzzer). Over the 15 conditioning days, a total of 40 A+, 40 B+, and 70 AX- trials were given. As before, each AX- trial was preceded by equal numbers of A+ and B+ trials. The exact order of presentation is listed in Appendix C.

The control group, Group Random, was exposed to conditions designed to equate the control group for the number and kind of stimulus presentations received by the experimental groups, but in such a way as to preclude the development of inhibition to X. This was done by attempting to endow A, the stimulus that was to be compounded with X, with minimal associative value by presenting it at random with respect to the shock with which it would have been paired in the experimental condition. Specifically, each of four subgroups of two rats received the stimulus conditions appropriate to one of the four experimental subgroups. However, during the unit of time normally reserved for each A+ trial (i.e., the 8-min interval), these control rats received random presentations of

A and shock. The only restriction was that the shock scheduled to occur on these "unpaired" trials could not begin sooner than 1 min after the preceding trial nor within 1 min of the next trial. All rats continued to receive B+ and AX- trials appropriate to the subgroup assignment. Although the AX compound was explicitly unpaired with shock during this procedure, and hence, might have been expected to become inhibitory (Rescorla, 1969b), this unpaired relation was also common to both of the experimental groups. What this procedure was intended to control for was the excitatory associative value of A. All groups remained equated for number and kind of stimulus exposures, level of shock-induced emotionality, the explicitly-unpaired relation of the AX compound to shock, and whatever generalization might have occurred as a result of excitatory conditioning to B.

After conditioning was complete, the foodcup and bar were reinserted into the chamber and each animal was allowed to barpress for food on the VI-2 schedule during each of three 100-min sessions (Days 25-27). No stimuli were presented during these sessions.

To determine the strengths of the responses to A, B, and AX prior to the new-learning test, one nonreinforced presentation of each of these stimuli was given on each of Days 28 and 29 during 100-min sessions while the rats were barpressing. Order of presentation was randomized. The new-learning test began on the next day (Day 30). The first two trials were nonreinforced presentations of X (17 and 40 min after the start of the session). On the last two trials, X was reinforced, once with the low-intensity US and once with the high-intensity US. The order of the last two trials was counter-

balanced within each subgroup (onsets at 65 and 80 min into the session). Conditioning continued over the next five days (Days 31-35) on a 50% reinforcement schedule with these same types of trials being given each day. The order of trial type was randomized each day, but the order of hi-US and lo-US trials on each day was counterbalanced within each subgroup. This was followed by six days of extinction to X (Days 36-41). Four nonreinforced trials were given during each session. The experiment concluded with a final test session (Day 42) during which X, AX, A and B were each presented twice without shock. The first trial began 15 min after the beginning of the session and subsequent trials occurred every 10 min. The order of presentation was counterbalanced within each subgroup.

Results

The alpha level was set at .05 in all of the analyses described below. The data and major analysis-of-variance summary tables are contained in Appendix C.

Body Weights

There were no differences in the initial weights of the rats assigned to each group (F [2, 21] = 1.2). Mean weights over all phases of the experiment were calculated for each rat (see Appendix C) and a groups x phases analysis of variance was applied to these data. There was no groups effect nor interaction (both Fs $<$ 1.2), but there was a reliable phases effect (F [5, 105] = 3.5). The endpoints comparison was not reliable, and the effect appeared to be due to small fluctuations in weight across the various phases. However, there was no difference between group means greater than 6 gm across

any phase of the experiment. Each group's mean weight over all phases was within .52% of 80% of its initial mean weight.

Response Baselines

The mean number of responses occurring during the 2-min pre-CS intervals were analyzed in a manner similar to that described in Experiment 1 (see Appendix C). In no instance was there a between-groups difference in the baseline level of barpress responding.

Suppression Ratios

Preconditioning exposures. The overall mean suppression ratios obtained during the preconditioning session (Day 9) were .42, .52, and .39, for the stimuli that were to be used as A, B, and X, respectively. Group assignment (Hi, Lo, Random) and stimulus-designation (A, B, X) were used as "dummy" factors in an analysis of these scores. There were no group differences, however, as in Experiment 1, there was an overall effect due to stimulus-type (F [2, 42] = 10.3). Followup comparisons showed that the difference between the ratios to B and X were reliably different, but the ratios to A did not differ from those to X or B. It was concluded that interpretation of subsequent analyses would not be obscured by group differences in the pretreatment reactions to these stimuli.

As in Experiment 1, these data were reanalyzed in terms of the actual stimulus events that were presented. The mean suppression ratios to the flashing light, tone, and buzzer were .39, .46, and .48, respectively. The effect of stimulus-type was reliable (F [2, 42] = 4.5), and the means were ordered in the same way that they had been in the first experiment.

Post-conditioning test (Days 28-29). The mean suppression ratios to the nonreinforced presentations of A, B, and AX on the post-conditioning test days are depicted in Figure 7. A groups x stimulus-type x days analysis of variance indicated that each of the factors had a significant effect (Groups: F [2, 21] = 35; Stimulus-type: F [2, 42] = 56.8; Days: F [1, 21] = 4.6) as did the interaction of groups x stimulus-type (F [4, 42] = 31.1). Followup analyses showed a reliable effect of stimulus-type within each group and reliable group differences in the response to each stimulus. Simple comparisons supported the following conclusions: (a) each group's response to the A stimulus differed from its response to the B stimulus (all Fs > 10), but not from its response to the AX compound; (b) Group Hi's responses to each of A, B, and AX differed from those of Groups Lo and Random (all Fs > 23), however, the latter groups did not differ in their responses to any of the stimuli. The reliability of the within-group effects of US intensity was supported by the observation that all but one rat (in Group Hi) showed greater suppression to the stimulus that had been paired with the more intense US during conditioning. Although the difference in responding to A and to AX was not statistically significant, all eight rats in Group Hi, and five out of eight rats in each of Groups Lo and Random were less suppressed in the presence of AX than in the presence of A.

A visual comparison of the responses in the experimental groups suggested that the suppression which each group showed to its A stimulus was less than that shown by the other group to the stimulus that had been paired with the same intensity US (i.e., the other

Figure 7. Mean suppression ratios to A, B and AX for Groups Hi, Lo and Random during the Post-Conditioning test sessions (Days 28 - 29).

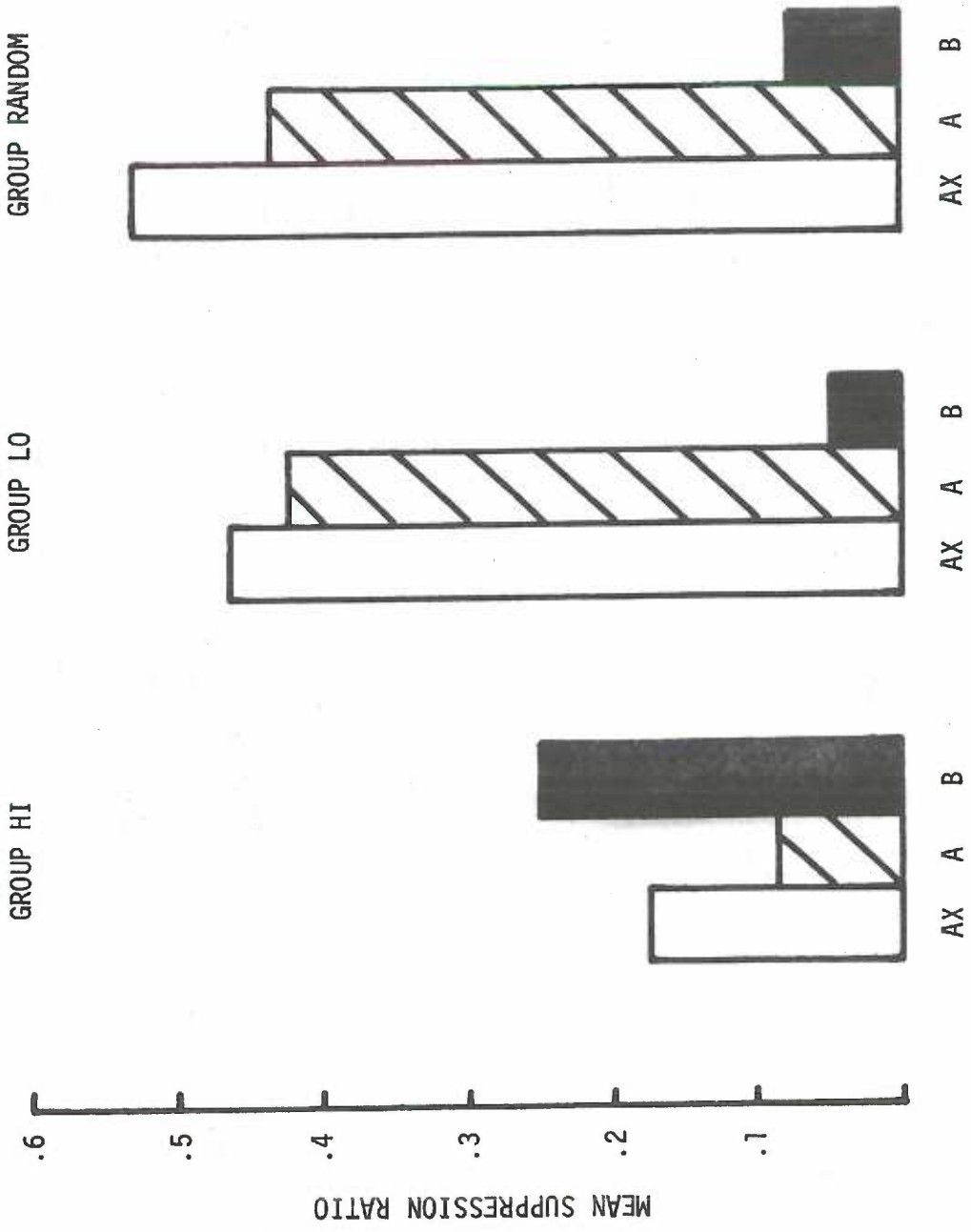
GROUP RANDOM
GROUP LO
GROUP HI
AX A B
AX A B
AX A B
STIMULUS
MEAN SUPPRESSION RATIO
.6
.5
.4
.3
.2
.1

Figure 7

group's B stimulus). Thus, subsequent to the analyses described above, a direct comparison was made between the response to A in each group and the response to B in the other group. The groups differed only in their responses to the stimuli paired with the low-intensity US (Group Hi's B vs Group Lo's A) (F [1, 14] = 12.1).

The days effect was attributable to a slight overall loss in suppression across the two test days (from .25 on Day 28 to .30 on Day 29), suggesting that extinction had begun to occur.

New-learning phase: Acquisition and extinction. Figure 8 shows the mean suppression ratios recorded for each group during consecutive two-day blocks of acquisition and extinction. As can be seen, Groups Hi and Lo were ordered as they had been in Experiment 1 (cf. Figure 6), with Group Hi showing greater suppression throughout. Group Random appeared generally to be less suppressed than Group Hi, and not substantially different from Group Lo.

To see whether the groups differed in their reactions to X prior to the introduction of shock during this phase, a one-way analysis of variance was applied to the means of the suppression ratios obtained on the first three trials of Day 30 (the first day of new-learning acquisition). The groups did not differ ($F < 1$). The means were .45, .47, and .48 for Groups Hi, Lo and Random, respectively.

Since primary interest was in the comparison between Groups Hi and Lo during this phase, their scores were initially subjected to separate groups x blocks analyses during both acquisition and extinction. In each analysis, the overall change in suppression over blocks proved reliable (Acquisition: F [2, 28] = 49.8; Extinction:

Figure 8. Mean suppression ratios to X in two-day blocks for Groups Hi, Lo and Random during new-learning-phase acquisition and extinction.

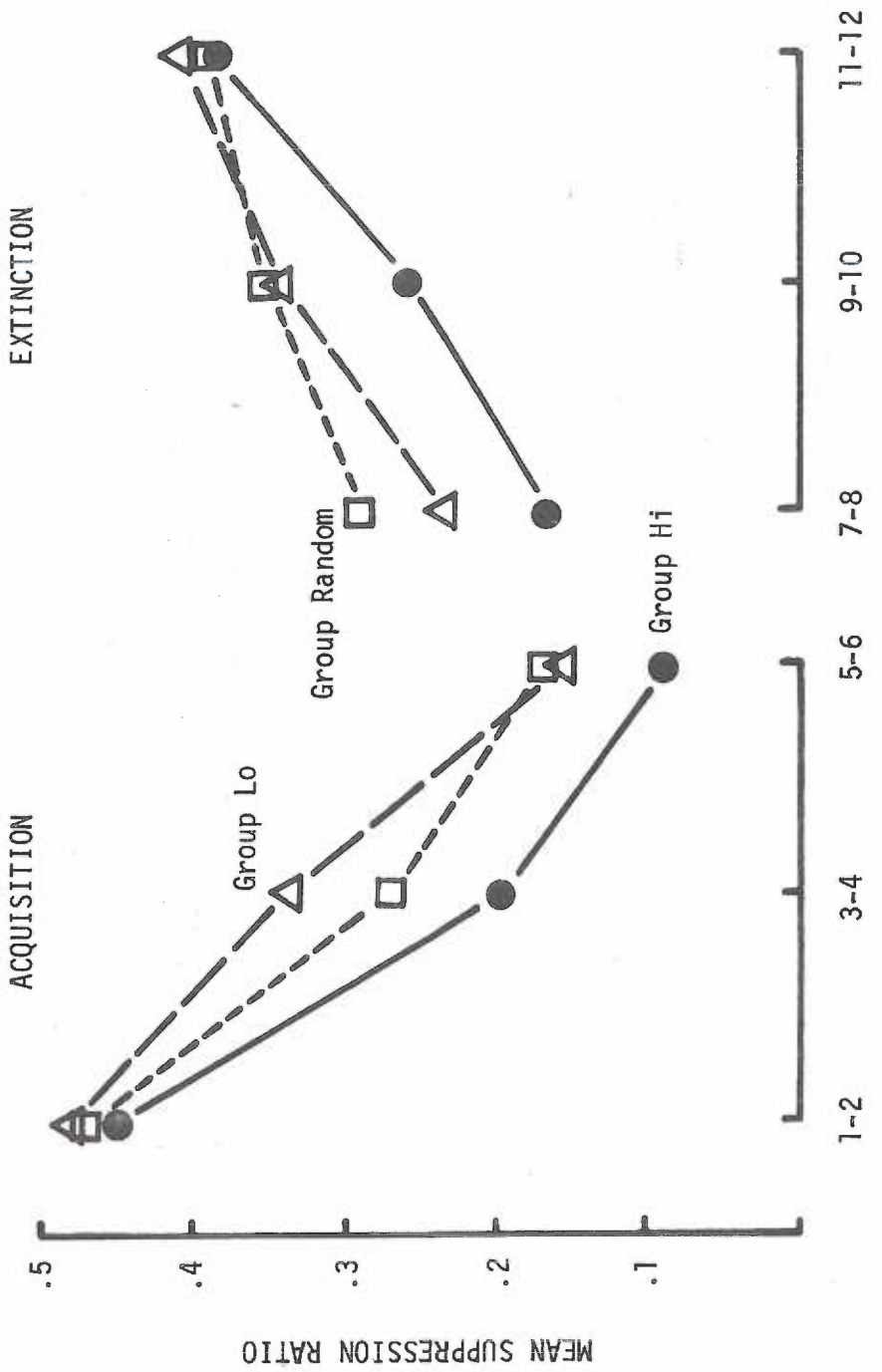
ACQUISITION
EXTINCTION
Group Lo
Group Random
Group Hi
MEAN SUPPRESSION RATIO
.5
.4
.3
.2
.1
1-2
3-4
5-6
7-8
9-10
11-12
DAYS

Figure 8

F [2, 28] = 45.8). In addition, the overall difference between Groups Hi and Lo during acquisition was significant (F [1, 14] = 5.6). Comparisons between each experimental group and the random control revealed only one difference. During extinction, the groups x blocks interaction involving Groups Hi and Random was reliable (F [2, 28] = 5.6). This effect appeared simply to be due to the convergence of these groups over blocks of extinction trials.

The "trials-to-criterion" measure reported in Experiment 1 was also analyzed in this experiment. The median numbers of trials to reach a two-trial mean suppression ratio less than .2 were 10, 13.5, and 15.5 for Groups Hi, Random and Lo, respectively. A two-tailed Mann-Whitney comparison indicated that the difference between Groups Hi and Lo was reliable (U = 12). The difference between Groups Hi and Random approached significance (U = 15, p = .08), but Groups Lo and Random did not differ (U = 25.5).

Final test day. The group mean suppression ratios to the non-reinforced presentations of A, B, X and AX on Day 42 are depicted in Figure 9. With the exception of the responses to X, unless the new-learning-phase treatment affected these responses, these data should have been similar to those gathered on Days 28-29 (cf. Figure 7). A visual comparison suggests that the mean ratios on Day 42 were ordered the same way that they had been during the earlier test, but that the differences between Groups Lo and Random were larger. An overall analysis of these data yielded significant effects of groups (F [2, 21] = 6.0), stimulus-type (F [3, 63] = 36.8) and their interaction (F [6, 63] = 10.1). Followup tests indicated reliable

Figure 9. Mean suppression ratios to A, B, AX, and X for Groups Hi, Lo, and Random during the final test session (Day 42).

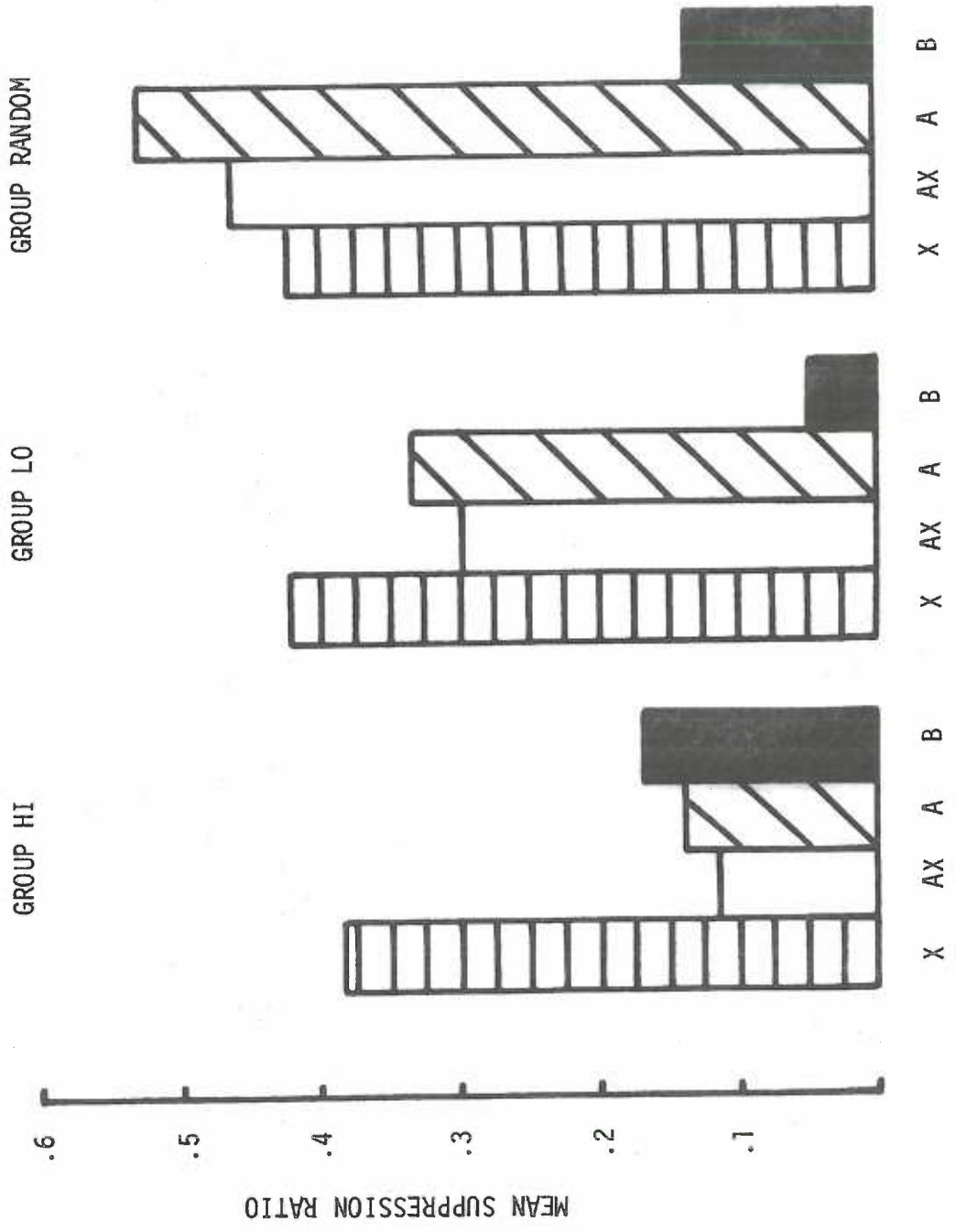
GROUP HI
GROUP LO
GROUP RANDOM
X
AX
A
B
STIMULUS
.6
.5
.4
.3
.2
.1
MEAN SUPPRESSION RATIO

Figure 9

stimulus-type effects in each group (all Fs > 8.8). The groups effect was reliable for A and AX (Fs > 11.8) but not for B or X (Fs < 1.9). Subsequent comparisons supported the following conclusions: (1) each group differed from every other group in its response to A and to AX (all Fs > 5.4), (2) Groups Hi and Lo were reliably more suppressed to the AX compound than to X (Fs > 12.5), (3) Groups Lo and Random were reliably more suppressed to B than to A (Fs > 53.8), and (4) no group showed a difference between its response to A and its response to AX (all Fs < 1.5). In general terms, there were within-groups effects of US intensity in Groups Lo and Random, but not in Group Hi, and there was a between-groups effect of US intensity for the A stimulus, but not for the B stimulus. In addition, at least in Groups Hi and Lo, the response to the compound appeared to be governed primarily by the A component.

As was done earlier, a direct comparison was made between each group's response to the A stimulus and the response made by the other group to the stimulus that had been paired with the same intensity US (i.e., B). This time, the groups differed in their response to the stimuli paired with the high-intensity US (Group Hi's A vs Group Lo's B) (F [1, 14] = 5.2). The difference between the responses to the stimuli paired with the low-intensity US approached significance (F [1, 14] = 3.6, $p < .1$).

Response Latencies

Latency to first CS response. Median latencies to the first response during the CS were calculated for each rat in two-day blocks throughout the new-learning test phase. As can be seen in Figure 10,

Figure 10. Mean latency to the first response after the onset of X in two-day blocks for Groups Hi, Lo and Random during new-learning-phase acquisition and extinction.

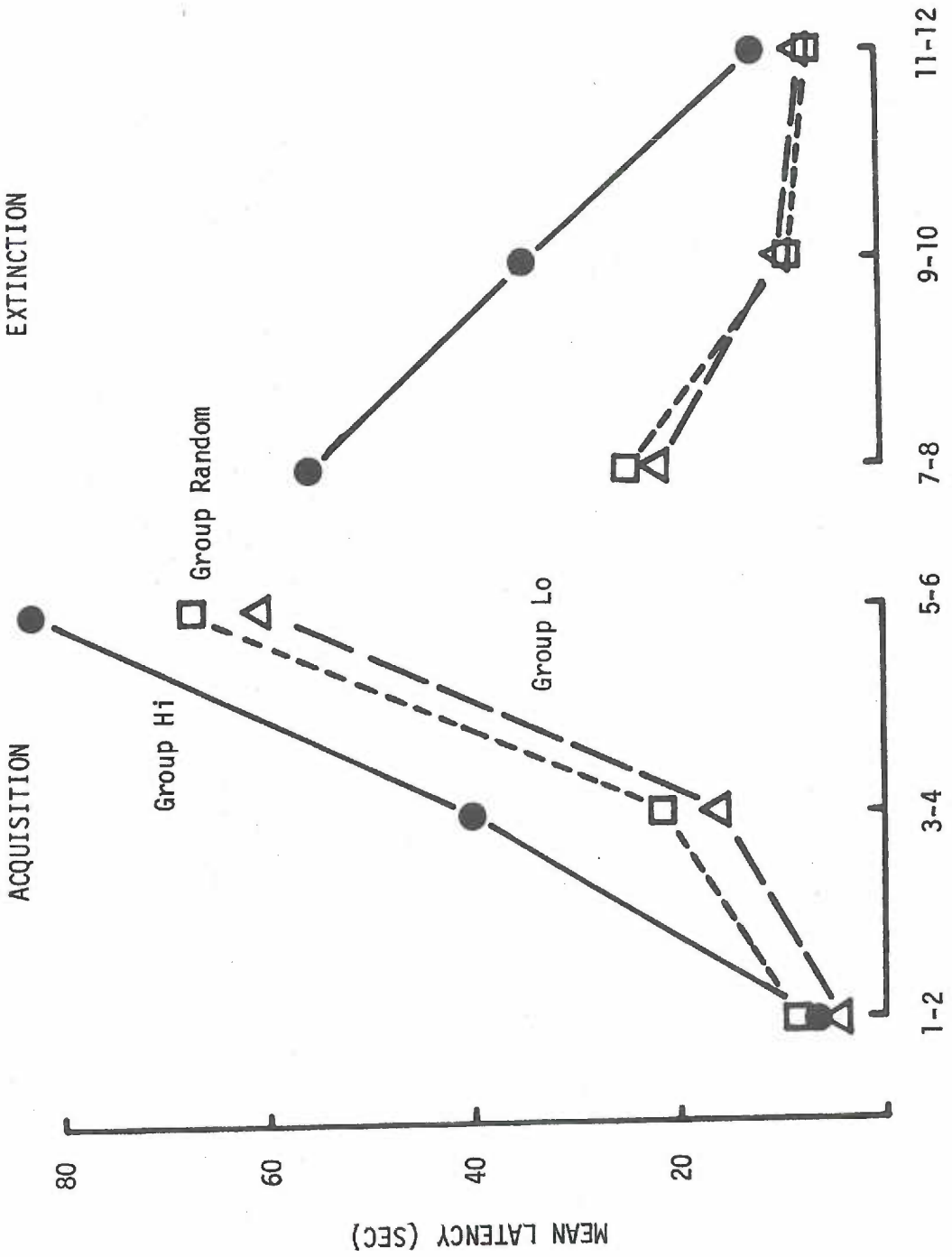
EXTINCTION
ACQUISITION
Group Random
Group Hi
Group Lo
MEAN LATENCY (SEC)
80
60
40
20
1-2
3-4
5-6
7-8
9-10
11-12
DAYS

Figure 10

the latency data were ordered in the same way as the suppression-ratio data (cf. Figure 8), with Group Hi showing the longest latencies. Separate groups x blocks analyses during acquisition and extinction failed to produce a reliable effect of groups or a groups x blocks interaction. Nonetheless, the change in response latency over blocks within each phase was significant (*Fs* [2, 42] = 34.4 and 9.2 for acquisition and extinction, respectively).

Latency to first post-CS response. As in the previous experiment, median latencies to the first response following nonreinforced, weak-US, and strong-US trials were computed in two-day blocks during the new-learning acquisition phase. These scores have been collapsed across groups and are depicted in Figure 11 as a function of trial-type over the three blocks of training. As the figure suggests, on the average, the rats initially responded quickly (within 5 sec) after a nonreinforced conditioning trial, but much more slowly following shock trials (Block 1). As training continued, however, this pattern reversed and by the final block, the rats were showing the longest latencies following nonreinforced trials. These observations were supported by a reliable trial-type x blocks interaction (*F* [4, 84] = 13.8) that emerged as a result of a groups x trial-type x blocks analysis of variance. The change in suppression over blocks was significant in separate analyses for each stimulus (all *Fs* > 4.9). The differences between the latencies following no-shock trials and the combined shock trials were significant in each block (all *Fs* > 6.3). The difference between the latencies following weak and strong shock was marginally reliable in Block 1 (*F* [1, 42] = 4.02).

Figure 11. Mean latency to the first response after the offset of X in two-day blocks as a function of the intensity of the US paired with X (0, .4, and 1.6 mA) (collapsed over Groups).

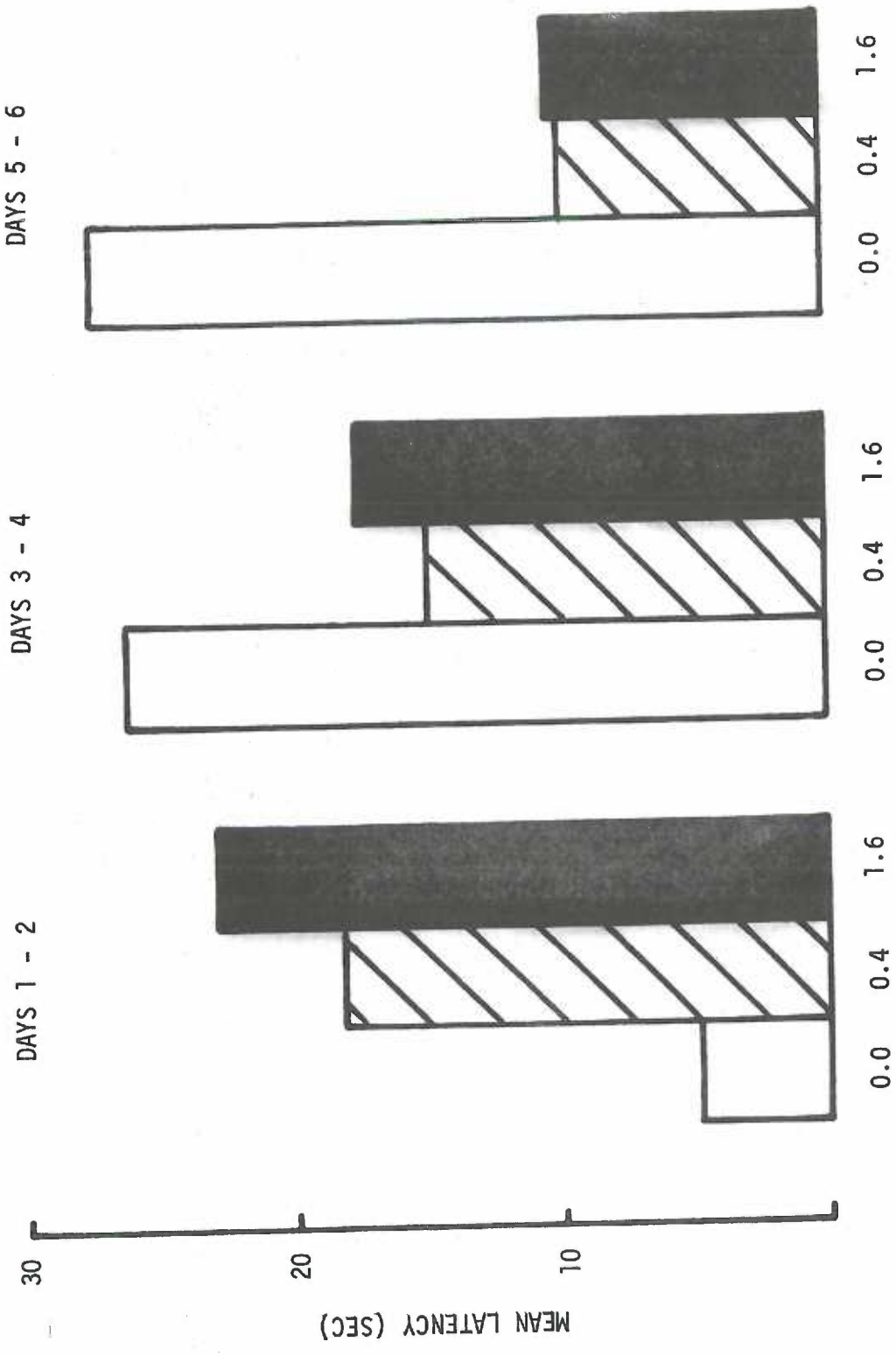
DAYS 1 - 2
DAYS 3 - 4
DAYS 5 - 6
0.0
0.4
1.6
US INTENSITY (mA)
MEAN LATENCY (SEC)
30
20
10

Figure 11

Analysis of latencies to the first post-CS response across two-trial blocks in extinction revealed a significant blocks effect (F [2, 42] = 5.0), but no group differences. The mean latencies were 5.9, 4.3, and 4.0 sec for Blocks 1, 2, and 3, respectively.

Discussion

The suppression data from the new-learning test phase clearly contradict the prediction that Group Hi should have been slower to acquire suppression to X than Group Lo. Moreover, not only was Group Hi consistently more suppressed than Group Lo, but it tended also to show greater suppression than the random control group. These findings suggest that, at least under the conditions of Experiment 2, the proposed relation between the strength of conditioned inhibition and strength of conditioned excitation does not hold. These data seem more compatible with the supposition that procedures designed to increase the associative value of A make X less inhibitory (or, depending upon one's interpretation of the random control, more excitatory) when conditioning involves the A+, AX- ("conditioned inhibition") paradigm. Current theories of conditioned inhibition do not appear to predict this outcome. This point will be discussed in greater detail later.

To evaluate fully the significance of this outcome, however, consideration must first be given to the data gathered during the post-conditioning and final test sessions (cf. Figures 7 and 9). Presumably, these data reflect the associative strengths of the various stimuli that were involved in the "conditioning" of X prior to the new-learning phase. The existence of certain between- and within-group differences may be critical to the assertion that the present experiment constituted a valid test of the hypothesis under consideration. First, the between-group differences in suppression to A suggest that the US-intensity manipulation was successful in endowing A with different associative values in each group. Group Hi differed from both Group Lo and Group

Random, and during the final test, Group Lo showed less suppression to A than Group Random did. This latter finding is consistent with the suggestion that forward pairings of A with weak shock were more effective in suppressing barpressing than were random presentations of A and shock. The between-group differences in suppression to B (post-conditioning test) and the various within-group US-intensity effects further support the contention that the shock values used in Experiment 2 were effective in producing different degrees of associative value.

One other finding related to the US-intensity variable is the difference between each experimental group's response to the A stimulus and the other experimental group's response to the stimulus that had been paired with the same intensity US (i.e., the other group's B stimulus). There was a tendency for each group to show less suppression to its A stimulus than the other group did to its B stimulus. This suggests that generalization of extinction (inhibition) from nonreinforced presentations of AX may have reduced the amount of suppression elicited by A in each group.

The failure to find within-group differences in suppression to A and AX in the experimental groups may pose a problem for a conditioned-inhibition analysis. Despite 70 AX- conditioning trials, there was no indication of a differential response to A and the AX compound. Although the existence of a differential response would have clearly been consistent with an inhibition analysis, the failure to see such a difference does not necessarily mean that inhibition had not developed to X. It might be argued that the associative value of A was very high in Group Hi and very low in Group Lo, and hence, that

"floor" and "ceiling" effects prohibited the observance of A - AX differences. However, without modification of existing theories of conditioned inhibition, this would not explain the results obtained on the new-learning test.

One other possible reason for the failure to see A - AX differences is that the shift from off- to on-the-baseline conditioning (for the new-learning test) produced stimulus generalization decrement, resulting in a reduction in the inhibitory strength of X. That the excitatory tendencies appeared not to have suffered this loss would be consistent with the beliefs expressed by Pavlov (1927) and Hull (1943) that inhibitory tendencies are less stable in this regard than excitatory tendencies. However, the generalization-decrement explanation seems unlikely, especially given the several studies that show successful transfer of inhibition after a similar shift in conditions (e.g., Rescorla, 1969b). Even if generalization decrement were responsible for the lack of an A - AX difference, there is still no reason for a reversal in the predicted outcome. At best, the generalization-decrement notion would predict no differences between groups during new-learning.

Before considering possible interpretations of the test-phase suppression-ratio data, the latency data obtained in this experiment should be mentioned. In general, the suppression-ratio measure seems to be preferred in the analysis of CER experiments, although occasionally, response latency measures are thought better to reflect the effects of certain independent variables (e.g., Crowell & Anderson, 1972). In both of the present experiments, however, the latency to

the first CS response failed to differentiate among groups, although it did increase with increases in suppression during CER acquisition. These data were quite variable and it may be that latency measures based on different criteria could have proved more stable (e.g., latency to first 5 or 10 responses after CS onset).

Although they did not indicate between-group differences, the post-CS response latency data were quite interesting. It will be recalled that during the new-learning phase, the tendencies to resume responding after CS offset reversed over trial blocks in a manner that differed depending upon the presence or absence of the US. Initially, rats resumed responding faster after nonreinforced trials than after reinforced trials. By the end of training, however, the latencies following nonreinforced trials had become very long and those following shock trials had become much shorter. This finding suggests that the unconditioned suppressive effects of shock decreased over trials and/or that some sort of differential conditioning was occurring in the post-CS interval. Apparently, the presence or absence of shock at the end of the CS became an important determinant of the suppression elicited by post-CS cues.

If Experiment 2 represents a valid test of the relation between strength of conditioned inhibition and strength of conditioned excitation, it appears that the suppression-ratio data from the new-learning test phase demand a revision of existing theories of conditioned inhibition. It has already been noted that according to the Rescorla-Wagner model, Group Hi should have been less rather than more suppressed than Group Lo. Although there were ways to handle

the failure to find an effect in Experiment 1, the model simply does not predict that the associative value of A in Group Hi would ever become more positive than that of A in Group Lo. That the groups were equated for nonassociative variables at all times eliminates a number of alternative explanations.

Hull's (1943) inhibition theory is similarly unable to explain this finding in any simple manner. According to Hull (1943, p 277 ff), whenever an excitatory response is evoked, there is a negative after-reaction that tends to produce a cessation of the original response. This after-reaction was referred to as reactive inhibition (I_R) and was assumed to have "primary negative motivational" properties (although it did not appear to interact with other sources of drive in determining the strength of Hull's general D construct). Within the context of Hull's drive-reduction theory of learning, the diminution of this post-response, negative-motivational condition (which occurred as a function of time following the response) was thought to be sufficient for the conditioning of inhibitory potential. Conditioned inhibitory potential ($_SI_R$) was loosely described as a "negative habit," a habit of <u>not</u> doing something. Hull proposed that inhibition developed whenever a response was evoked, even under conditions of reinforcement. Of greater importance to the present considerations, however, he suggested that inhibition would accrue not only to an established excitatory stimulus during its extinction, but also to any neutral stimulus that was nonreinforced in conjunction with an excitatory stimulus (e.g., X in the A+, AX- paradigm). The primary inhibitory reaction was said to be a function of the "work" or "energy consumption"

involved in the performance of the response. Although Hull appears not to have specified the determinants of "work" in the case of classically conditioned responses, if it can be assumed that the greater the strength of a given habit, the greater the energy consumption involved in executing the response, then, the greater the strength of the habit, the greater the inhibitory reaction following the response, and hence, the greater the strength of conditioned inhibition. Thus, the theories of Hull (1943) and Rescorla and Wagner (1972) offer the same predictions for the present experiments, and both seem equally at odds with the obtained result.

Spence's (1960, Chapter 6) treatment of inhibition differed considerably from Hull's, although both theorists advocated counterconditioning views of extinction. In the instrumental reward situation, Spence assumed that it was not response evocation per se that produced inhibition, but rather nonreinforcement under conditions where the organism had learned to "expect" or "anticipate" reinforcement (i.e., where a fractional anticipatory goal response, r_g, had been conditioned to stimulus cues in the instrumental chain). Inhibition in such situations was viewed primarily as a frustration phenomenon. The absence of reinforcement after evocation of r_g was presumed sufficient to produce an emotional (anger, frustration) response, designated as r_f. The frustration response was allegedly capable of becoming conditioned to stimulus cues in much the same way as r_g, and the stimulus consequences (s_f) of r_f were thought to elicit learned or unlearned responses, some of which would compete with the instrumental response.

For a variety of reasons, Spence believed that the factors governing habit strength and inhibition were different in the case of classical defense conditioning. Habit strength was governed not only by the number of occurrences of the response (which alone determined habit strength in the instrumental reward situation), but also by the intensity of the US. Inhibition was assumed simply to be a function of the number of nonreinforced trials. Although Spence did not elaborate the mechanism underlying inhibition in the classical defense conditioning situation, he did indicate that a direct application of the frustration analysis appeared implausible. Strictly speaking, since inhibition was said to be a function only of the number of nonreinforced trials, Spence's 1960 analysis would lead to the prediction of no differences among groups in the present experiments. However, a more detailed consideration of the aversive situation might have led him to the conclusion that the strength of the inhibitory response was a function of the strength of the anticipatory response (hence, a function of US intensity), in much the same way that the strength of r_f was believed to depend upon the strength of r_g.

Attempts to interpret the present outcome may hinge in part upon one's view of the random control group. This consideration is related to remarks made earlier about establishing a reference condition against which to evaluate the effects of alleged inhibitory stimuli (pp 12 - 17). Given that Hi was more suppressed to X during the new-learning test than Groups Lo and Random, and that the latter groups did not differ, in the absence of a reference point, the outcome might be attributable to: (a) retardation of CER learning in Groups Lo and

Random (with respect to Group Hi), (b) facilitation of CER learning in Group Hi (with respect to the other groups), or (c) some combination of retardation and facilitation (with respect to some unknown condition). Acceptance of the performance of the random control group as an appropriate reference point would support the second conclusion. Acceptance of this control may not be unreasonable since the final test data indicated that the random control procedure was effective in producing minimal associative strength to A. It might also be possible empirically to determine whether retardation or facilitation was involved (e.g., with a combined-cue test), but once again, the ability to make a final decision would rest upon the choice of a control for nonassociative factors. If the random control is judged inappropriate, there may be little reason to favor any one of the above alternatives over the others.

There are two theories that may help to explain the results of Experiment 2. The first involves attentional mechanisms in conditioning, and the second involves the role of higher-order conditioning during conditioned-inhibition training. The ways in which attentional theories might handle the conditioned-inhibition paradigm have been outlined by Wagner (1969), Rescorla and Wagner (1972) and Hearst (1972). One attentional version of the outcome of Experiment 2 might be as follows (see Wagner, 1969, p 38). First, assume that the likelihood of attending to A is an increasing function of the intensity of the US paired with A on A+ trials. Suppose also that the likelihood of attending to one component of a compound is inversely related to the likelihood of attending to other components. Given these

assumptions, one might predict that rats in Group Lo would be more likely to attend to X than would rats in Group Hi because of differences in the likelihood of attending to A. If nonreinforcement of the AX compound were sufficient to produce conditioned inhibition, then inhibition to X would presumably develop to a greater degree in Group Lo. However, this theory might also predict that inhibition should have been greatest in the Random group since attention to A and to X would not be expected to differ initially. This latter prediction becomes even stronger when one considers the evidence suggesting that random conditioning may act directly to decrease attention to A (Mackintosh, 1973).

As Rescorla and Wagner (1972) have suggested, attentional accounts of changes in responding often do little more than redescribe the data. The utility of such accounts may be minimal due to their failure to specify the trial-by-trial events that control conditioning. It is interesting to note that the Rescorla-Wagner model does provide a means of incorporating certain types of attentional changes through the CS-saliency parameter, α. If rules could be given to describe the way in which changes in α occur, differences in rates of acquisition of inhibition might be attributed to differences in α rather than to differences in the initial values of $\overline{V}$. Without added assumptions, however, the model would still be unable to handle the present results.

A second hypothesis that may explain the obtained result must appeal to the development of a second-order conditioned fear response during "conditioned-inhibition" training. Although this may initially

seem an outrageous proposal, close examination of the conditions alleged to support conditioned inhibition and higher-order conditioned excitation reveals certain similarities. In both instances, excitatory conditioning to some stimulus, S_1, must precede nonreinforced presentations of a second stimulus, S_2, in close temporal proximity to S_1. For second-order conditioning, it is generally required that the onset of S_2 precede that of S_1, although as will be mentioned shortly, this need not always be true. The relative arrangement of the two stimuli does not appear to be as important to the development of conditioned inhibition (Rescorla & LoLordo, 1965). In addition to the apparent procedural commonalities, there are empirical and theoretical reasons for supposing that conditioned inhibition and higher-order conditioning are interrelated. It has been shown, for example, that after a few pairings of S_2 and S_1 in a standard second-order conditioning arrangement (S_2 preceding and overlapping S_1), S_2 shows secondary excitatory properties, but that after many trials, S_2 shows conditioned inhibitory effects (Herendeen & Anderson, 1968; for a related result, see Rescorla, 1973).

McAllister and McAllister (1971) and Rescorla (1973) have suggested that inhibitory conditioning and second-order conditioning are interrelated, although each has done so for different reasons. The McAllisters were attempting to examine Rescorla's (1967b) contingency formulation of conditioning within the framework of a "more traditional view of conditioning." According to that traditional view, in order for a CS to become inhibitory, it had to become excitatory first, either through direct conditioning or through

stimulus generalization. Inhibition could then develop through the nonreinforcement of that stimulus. It is not clear whether the McAllisters are the source of the "traditional view" of conditioning or whether that view was intended to be representative of earlier theories such as Hull's (1943). If the latter is true, it should be mentioned again that Hull did not demand that a to-be-conditioned inhibitory stimulus ever possess excitatory properties. Under certain circumstances (e.g., A+, AX - conditioning), a neutral stimulus could acquire inhibitory potential (Hull, 1943, p 282).

Nevertheless, according to the McAllisters' view of conditioning, a stimulus that is explicitly unpaired with shock must first become excitatory before it can become inhibitory. In describing how this might occur for the CS in an "explicitly-unpaired" conditioning procedure, they proposed that the to-be-conditioned inhibitory stimulus acquires its initial excitatory potential by being paired with fear-arousing apparatus cues, that is, through a process of higher-order conditioning. Since the CS is never paired with shock, extinction is alleged to take place, making the CS inhibitory. Presumably, this analysis could be extended to the A+, AX- paradigm by assigning the role played by fear-arousing apparatus cues to the explicit conditioned excitor, A. Because the second-order conditioned response would be expected to be greater, the greater the associative value of A, one might predict that, under conditions such as those in Experiment 2, X would initially possess a more positive associative value in Group Hi (when second-order conditioning was at its peak), but that with continued training, it would possess a more negative

associative value in Group Hi (when inhibitory conditioning reached asymptote). This analysis would suggest that the test for conditioned inhibition in Experiment 2 was conducted too soon, that is, before the second-order conditioned response had extinguished.

There are several problems with the formulation described in the preceding paragraphs. First of all, there is a certain amount of slippage in the description of the conditions thought to be responsible for the development, maintenance, and extinction of a second-order conditioned response. In the treatment of the explicitly-unpaired procedure, for example, the first-order conditioned response is presumed to be elicited by apparatus cues, that is, by cues that precede, occur together with, and follow the explicitly unpaired stimulus. Despite the apparent irregular relation between the "onset(s)" of the apparatus cues and the onset of the CS, it is argued that the CS acquires conditioned excitatory properties. Even if this process were to occur, it is not clear why the second-order conditioned response would ever extinguish given that the apparatus cues continued to be paired with shock.

The major distinction between the McAllisters' treatment of inhibition of that of Hull (1943) or Rescorla and Wagner (1972) appears to be the McAllisters' insistence that the to-be-conditioned inhibitory stimulus initially elicit an excitatory response. It seems that a "traditional view" such as Hull's could handle the explicitly-unpaired (or A+ , AX-) procedure without appealing either to generalization of fear from apparatus cues to the explicit CS or to higher-order conditioning. For example, one might argue

that fear directly conditioned to apparatus cues (A) initially generalizes to the compound stimulus consisting of the apparatus cues plus the explicit CS (AX). Repeated nonreinforcement would be expected to lead to the extinction of the response elicited by this generalized CS, thereby leading to the development of inhibition to the compound (and hence, to each component). However, it is clear that this analysis does not require that X itself ever possess excitatory properties before becoming inhibitory. If fear conditioned to apparatus cues (A) does not generalize to the explicit CS (X), the McAllisters' demand that X be excitatory before it can become inhibitory necessitates invocation of the concept of higher-order conditioning and a consideration of the problems that such an interpretation entails.

One last consideration related to the McAllisters' analysis is whether the inhibition that accrues to an extinguished excitatory stimulus is capable of affecting excitation elicited by any other positive CS. Within Hull's theory, for example, the inhibitory potential developed under such conditions was thought to be sufficient only to cancel the habit strength originally associated with the extinguished CS. Moreover, there are data that suggest that an extinguished CS does not possess the inhibitory power acquired by stimuli in standard conditioned-inhibition paradigms (Rescorla, 1969d). If the McAllisters' view of inhibition reduces all such conditioning to the extinction of an excitatory tendency, it may not be able to integrate these conflicting data.

As was indicated earlier, Rescorla (1973) has also noted the procedural similarities and empirical outcomes that suggest a relationship between second-order fear conditioning and (first-order) conditioned

inhibition of fear. He has proposed that under certain circumstances, the development of conditioned inhibition becomes superimposed upon second-order conditioning. Assuming that the rate at which the second-order response develops is less than that at which conditioned inhibition develops, one might predict that over conditioning trials, a given stimulus would initially arouse fear, but later inhibit fear (e.g., Herendeen & Anderson, 1968). Rescorla (1973, p 143) summarizes the process as follows:

> Initially, S_2 develops second-order conditioning because it is followed by the fear-producing S_1. Because S_2 is presented with an adequate fear elicitor and nonreinforced, however, it gradually develops conditioned inhibition. That inhibition attenuates the response to S_1, which in turn makes it a less potent reinforcer and thus allows extinction of the second-order conditioning to S_2.

In attempting to extend this analysis to the present data, one might question whether the simultaneous compounds were such as might be expected to promote the development of second-order conditioning. Although simultaneous conditioning does not appear in the traditional classical-conditioning literature (e.g., Fitzgerald & Martin, 1971; Gormezano, 1972), there is some evidence that simultaneous conditioning is effective for both first- and second-order fear conditioning (see Rescorla, 1973). It might also be argued that the intermittency of the light component of the compound promoted the development of higher-order conditioning despite the nominal simultaneity of onsets of the components. If the preceding analysis correctly reflects the processes

underlying the development of excitation and inhibition in Experiment 2, then the outcome might be consistent with the suggestion that the new-learning test was given prior to the complete attenuation of the second-order conditioned response.

Since Rescorla's (1973) analysis appears generally compatible with the Rescorla-Wagner model, an attempt had been made here to incorporate into that model modifications that reflect the potential contribution of second-order conditioning in the A+, AX- paradigm. To do this, the associative value of X (V_X) at any given instant is assumed to be jointly determined by the associative value of the first-order conditioned inhibitory tendency (V_{X1}) and the associative value of the second-order excitatory tendency (V_{X2}). These tendencies summate algebraically to determine V_X (i.e., $V_X = V_{X1} + V_{X2}$). Inhibitory value, V_{X1}, develops in the manner specified by the Rescorla-Wagner model. Its growth is assumed to be independent of the current strength of V_{X2}. The change in associative value on each trial is expressed in this way:

$$\Delta V_{X1} = \alpha_X \beta_2 (\lambda - V_{AX1})$$

Increments in the second-order tendency, however, depend upon the current strengths of both A and X_1. In other words, the growth of second-order fear conditioning depends primarily upon the strength of the first-order conditioned excitor. However, the ability of that stimulus secondarily to reinforce X depends upon the strength of the first-order conditioned inhibition. In terms of the model, the asymptote of second-order conditioning is determined by the joint strengths of the first-order inhibitory and excitatory tendencies:

$$\Delta V_{X2} = \alpha_X \beta' (V_{AX1} - V_{X2})$$

It should be noted that the quantity that corresponds to the current associative value of all the stimuli present on any given trial differs depending upon whether the change in associative value is first-order or second-order. Changes in V_A and V_{X1} occur with respect to the current value of V_{AX1}, and changes in V_{X2} occur with respect to the current value of V_{X2}. While there may be no apriori reason for this assumption, it might be consistent with the suggestion that first- and second-order conditioning involve fundamentally different underlying processes (Rescorla, 1973).

To illustrate the changes that these assumptions make in predictions offered by the model, a sample learning run is depicted in Figure 12. For purposes of this example, it was assumed that: (1) A and X were equally salient ($\alpha_A = \alpha_X = 1$); (2) the asymptotic associative value (λ) was 40 on A+ trials and 0 on AX- trials; (3) the learning-rate parameter (β) for first-order conditioning was .4 on reinforced trials and .05 on nonreinforced trials; (4) the learning-rate parameter for second-order conditioning equalled that for first-order excitatory conditioning (i.e., $\beta' = .4$). The initial values of V_A, V_{X1}, and V_{X2} were set at 0, and conditioning alternated between A+ and AX- trials.

The curve labeled X represents the algebraic sum of the values of V_{X1} and V_{X2} at each point in training. As can be seen, under the above assumptions, the net value of X is initially excitatory and then becomes inhibitory. The degree to which this initial excitation occurs depends upon the relation between the learning-rate parameters for second-order conditioning (β') and nonreinforcement (β_2). Second-order conditioning will always "retard" the development of inhibition,

Figure 12. Sample learning run that illustrates the changes introduced by the proposed modification of the Rescorla-Wagner model. Conditioning trials alternated between A+ and AX-. The curve labeled X_1 shows the hypothetical strength of the first-order inhibitory tendency, whereas the X_2 curve shows the strength of the second-order excitatory tendency. The X curve represents the algebraic sum of the values of X_1 and X_2 at each point in training (see text for parameter values). The curves representing the strength of the first-order excitatory tendency (A) and that of the compound (AX) are identical to those generated by the original model.

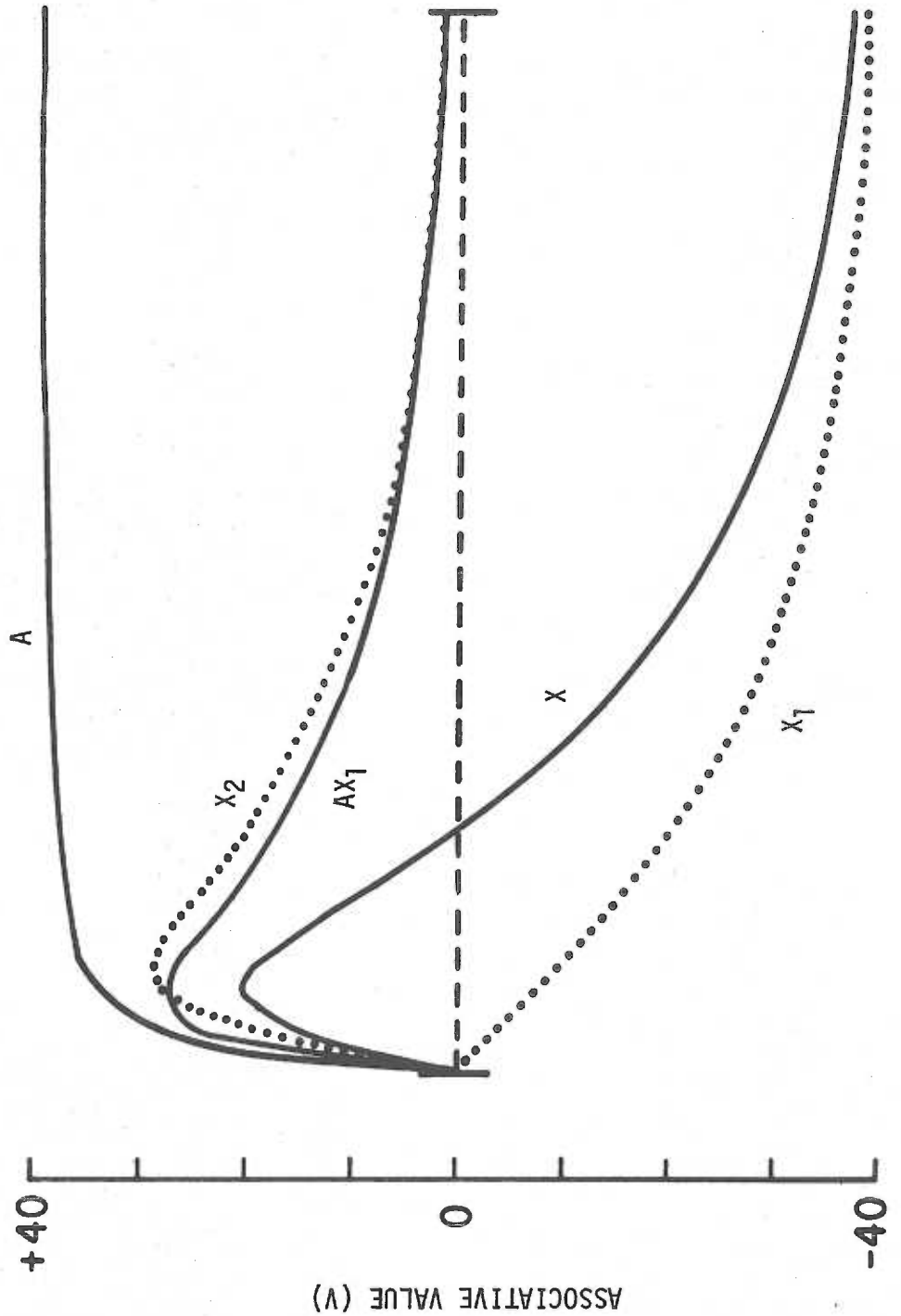

Figure 12

but the initial excitation will appear only if $\beta' > \beta_2$.

The curves in Figure 12 labeled A, X_1 and AX_1 are identical to those generated using the original Rescorla-Wagner model. The modification suggested here poses an interesting question concerning the net associative value of the AX compound. Namely, do the excitatory strengths of the first- and second-order conditioned responses summate in determining V_{AX}? If so, under conditions such as those depicted in Figure 12, the compound will initially possess greater associative value than the A component. Whether this seems reasonable awaits empirical determination.

Figure 13 shows the curves generated for V_X when USs of different intensity are used to establish V_A. The parametersused to generate the curve for the hi-US condition were those used in Figure 12. For the lo-US condition, λ was fixed at 10 on reinforced trials and the learning-rate parameters for first- and second-order excitatory conditioning were both set at .3. As can be seen in the figure, the modified model makes the same predictions as the original model concerning the relative strengths of V_X in each group at asymptote. The novel feature, of course, is that this final reaction represents a reversal in the relation earlier in conditioning when both groups showed net excitatory values for X.

The worth of the modified model in "explaining" the results of Experiment 2 is extremely questionable given the large number of assumptions involved. Not only must all the general relations be similar to those used in making Figure 13, it must be assumed that the new-learning test began <u>just before</u> the alleged reversal of the curves. It will

Figure 13. Sample learning runs generated by the modified Rescorla-Wagner model showing the net values of X when different intensity USs are paired with A in the A+, AX- (conditioned inhibition) paradigm. In both conditions, X is initially excitatory and then becomes inhibitory. The conditions are ordered differently depending upon the level of training (see text for parameter values).

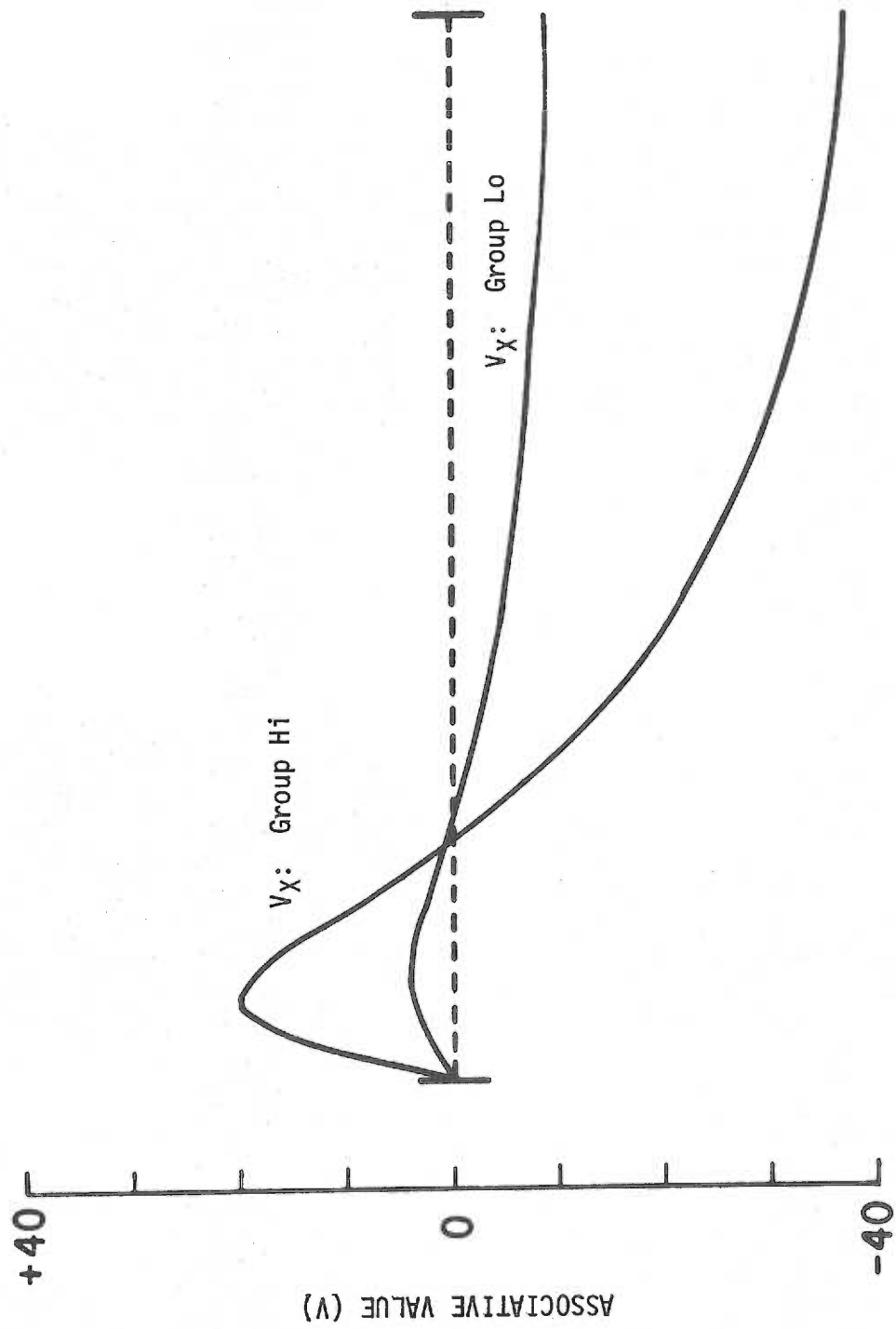

Figure 13

be recalled that the groups showed no differences in their reactions to X on the first few trials of new learning. Whatever second-order conditioning was there would have to have been below some (unspecified) threshold for excitation. It is also not known that continued training under the A+, AX- procedure would have produced a crossover in the associative strength functions. Comparison of the number of AX- trials given in Experiment 2 with the numbers of trials typically used in conditioned inhibition experiments suggests that enough trials had already been given. At the very least, however, the model does seem consistent with the data reported by Herendeen and Anderson (1968) and Rescorla (1973).

Regardless of the viability of any of the theories described above, the present experiments would seem to represent valid tests of the hypothesis that strength of conditioned inhibition is directly related to strength of conditioned excitation. That the obtained relation was opposite this, and appears to contradict existing views of the inhibitory process demands a serious re-evaluation of the empirical and theoretical bases of that process.

SUMMARY AND CONCLUSIONS

Two experiments were conducted to test the hypothesis that the strength of conditioned inhibition of fear is positively related to the strength of conditioned fear during inhibitory training. Strength of conditioned fear was manipulated by varying unconditioned-stimulus (US) intensity in a conditioned-emotional-response (CER) procedure using the Pavlovian "conditioned-inhibition" paradigm. In this paradigm, the to-be-conditioned inhibitory stimulus (X) is nonreinforced (i.e., presented without the US) in compound with a stimulus (A) that is normally reinforced when it is presented alone. Presumably, the greater the intensity of the US paired with the A stimulus, the greater the inhibition that should develop to the X stimulus.

In both experiments, food-deprived rats were initially trained to barpress for food reward on a variable-interval (VI) schedule. After conditioned-inhibition training, a "new-learning" test was used to assess strength of inhibition. This involved pairing the alleged inhibitor with shock in an on-the-baseline procedure. It was assumed that the slower the development of response suppression to X, the greater its inhibitory power. All groups were equated for the number of exposures to each US intensity during both conditioning and testing.

In Experiment 1, after a series of on- and off-the-baseline excitatory conditioning trials, conditioned-inhibition training was given first on- and then off-the-baseline. In Group Hi, X was always compounded and nonreinforced with a stimulus that was normally paired with a 1.3-mA shock, whereas in Group Lo, X was compounded and

nonreinforced with a stimulus that was normally paired with a .65-mA shock. By the end of inhibitory training, both groups showed less suppression in the presence of the compound than in the presence of the excitatory stimulus. Moreover, response suppression in the presence of the excitatory stimulus differed between groups and was ordered in accord with the intensity of the US paired with that stimulus. However, no group differences emerged during the new-learning test when X was paired with shock (24 acquisition trials on a 50% reinforcement schedule followed by 24 extinction trials). There appeared to be several possible reasons for failing to observe the predicted outcome.

Experiment 2 was conducted to resolve problems posed in attempting to interpret the previous finding. Three major changes were introduced: (1) the discrepancy between the US-intensity values was increased (.4 vs 1.6 mA); (2) all conditioning prior to the new-learning test was given off-the-baseline; and (3) a "random" control group was included. During the inhibitory-conditioning phase, this group received random presentations of shock and the stimulus that was normally paired with shock in the experimental groups. All other conditioning trials were identical to those received by the experimental groups. During post-conditioning on-the-baseline test sessions, none of the groups showed a difference between its response to the compound and its response to the excitatory stimulus, although suppression to the excitatory stimulus differed among groups and was ordered in accord with the intensity of the US paired with that stimulus. Group Hi showed the most suppression to X during the new-learning test phase, while Groups Lo and Random did not differ. Thus, contrary to

expectation, the conditioned-inhibition training with the stronger US made X less inhibitory (more excitatory).

This finding appears to conflict with predictions offered by most theories of conditioned inhibition and is not readily explained. The data may be consistent with either: (a) an attentional interpretation of US-intensity effects in the conditioned-inhibition paradigm, or (b) the suggestion that higher-order excitatory conditioning precedes inhibitory conditioning in the conditioned-inhibition paradigm. The second possibility was illustrated by a proposed modification in the Rescorla-Wagner model of Pavlovian conditioning.

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APPENDIX A

EMPIRICAL STUDIES OF CONDITIONED INHIBITION: A REVIEW OF THE LITERATURE

Empirical Studies of Conditioned Inhibition: A Review of the Literature

It has already been stated that the present dissertation is concerned with factors that affect the strength of conditioned inhibition of fearfulness. Assuming that certain inhibitory changes in level of fearfulness are due to learning, it seems reasonable to expect that the strength of those associative tendencies will be a function of the same types of variables believed to affect excitatory associative tendencies (e.g., stimulus intensity and duration, temporal relations, number of trials, etc.). Although extensive reviews of the effects of these variables on the conditioned excitation of fearfulness are readily available (e.g., McAllister & McAllister, 1971), there have been very few critical reviews of recent literature in the area of conditioned inhibition of fearfulness. It therefore seems appropriate to provide such a review before discussing the particular problem to be considered in this investigation.

Although all of the experiments that might be relevant to the topic of conditioned inhibition cannot be presented, an attempt has been made to review the major studies of the last 10 years (1965-1975). This is the literature that contemporary theories of learning must address, and, to varying degrees, the studies conducted since 1965 reveal a sensitivity to the problem of control not found in earlier studies. The following criteria were used to restrict studies that were to be included in the review: (a) The experimenter maintained control over the number, duration and distribution of CSs and USs during conditioning, (b) the US used during conditioning was one generally considered to be "aversive,"

and (c) the US used during a new-learning test or during a combined-cue test was also considered aversive. The choice of these criteria does not imply that studies that fall outside their boundaries are irrelevant to the notion of conditioned inhibition of fearfulness. Rather, it was based on the belief that, given the tenuity of our current knowledge in this area, it might be easier to consider only those studies that had these things in common.

Before continuing, a terminological point must be made. As it has been used in this paper, "conditioned inhibition" has referred simply to an inhibitory tendency that was learned. Pavlov, on the other hand, used "conditioned inhibition" to refer only to the outcome of a special type of differential-conditioning procedure, and used the term "internal inhibition" in the more general sense of learned inhibition. Pavlov suggested that there were four types of internal inhibition, resulting from four general types of conditioning procedures: (a) extinction (repeated nonreinforcement of an established conditioned excitor), (b) extended CS-US intervals (inhibition of delay), (c) differential conditioning (CS_1 is reinforced whereas CS_2 is not reinforced), and (d) the "conditioned-inhibition" procedure (similar to differential conditioning except that nonreinforced presentations of CS_1 overlap CS_2 presentations). The procedures described in the studies that follow generally fall into these categories. Rescorla (1969d) has suggested that a simpler way to characterize the situations believed to produce learned inhibition is in terms of a negative contingency between the US and CS.

Combined-Cue Tests

The transfer-to-Sidman and CER procedures have been most often used as combined-cue tests for inhibition. Although a conditioned excitatory stimulus is typically used in the CER procedure, an explicit excitatory stimulus is generally not used in the Sidman procedure. It is assumed that the cues provided by the avoidance schedule constitute an excitatory stimulus complex (e.g., backround cues, shock, response-produced cues, etc.).

Transfer-to-Sidman procedure. Rescorla and LoLordo (1965) have reported three experiments in which the effects of several conditioning procedures were examined by noncontingently superimposing the conditioned stimulus during avoidance responding. In each experiment, dogs were initially trained to jump a hurdle in a two-way shuttlebox to avoid brief shocks on a Sidman schedule. After the first three training sessions, avoidance sessions alternated with Pavlovian conditioning sessions during which the dog was confined to one side of the shuttlebox. In the first experiment, each dog was exposed to a Pavlovian "conditioned-inhibition" procedure. On half of the trials, one tone (CS_1) was paired with shock under trace conditions with a variable ISI. On the remaining trials, a second tone (CS_2) followed CS_1 instead of shock. In the second experiment, one group received differential conditioning wherein CS_1 coterminated with US onset, and CS_2 was presented alone. A second group received a variation of the "conditioned-inhibition" procedure. This treatment was identical to that given the differential-conditioning group save that CS_2 offset coincided with the onset of a nonreinforced presentation of CS_1. A third group was not exposed to tone or shock

during the conditioning phase. Two other groups were run during the third experiment. The first group received CS_2 and shock in an explicitly-unpaired manner. Essentially, the procedure was that used for the differential-conditioning group, except that CS_1 was omitted. The other group was simply exposed to both CSs in the absence of shock. In all of the experiments, the CSs and US were 5 sec in duration. Animals receiving both CSs were given a total of 90 trials of each type over the five conditioning days, at an average intertrial interval of 1.5 min.

Each experiment concluded with a single 1-hr test session. The dogs performed the Sidman response under extinction conditions (i.e., no shocks were delivered), and were given 60 5-sec presentations of each tone in a random order (average intertrial interval of 25 sec). Responses were recorded during 5-sec intervals before, during and after each stimulus presentation. The effect of each tone was measured in terms of the change in response rate from the pre-CS interval to the CS and post-CS intervals. In both conditioned-inhibition groups, and in the differential-conditioning group, response rate increased during CS_1, the stimulus that had been positively correlated with the US. Responding was also elevated following CS_1 offset in the first experiment, but was actually lower than the pre-CS response level in the second experiment. In all of the experimental groups, response rate declined in the presence of CS_2 and remained below the pre-CS level during the 5 - 10 sec following its offset. In each of the control conditions (no conditioning control in Experiment 2; CS-only control in Experiment 3), presentation of the CS had little effect on the avoidance response, and

in each case, the differences between the experimental and control conditions were reliable.

Rescorla and LoLordo interpreted their findings in terms of the excitation and inhibition of Pavlovian conditioned fear. They reasoned that if the avoidance response was motivated, in part, by a conditioned fear reaction, then a stimulus that elicits fear should enhance responding, whereas a stimulus that inhibits fear should depress responding. Their control conditions eliminated some, but not all alternative interpretations. With respect to the notion of control in general, the authors suggested that, because CS_2 had shown inhibitory properties following differential and explicitly-unpaired conditioning, these procedures may not be appropriate conditions against which to gauge excitatory conditioning. It was suggested that "truly random" exposures to CS and US might provide a better control for nonassociative factors.

Subsequently, Rescorla (1966) compared the performance of dogs exposed to a truly-random sequence of CSs and USs with that of dogs exposed to conditions in which the CS and US were either positively or negatively correlated. In general, the training procedure paralleled that of Rescorla and LoLordo (1965). During each of five conditioning sessions, every group received 24 5-sec tone presentations. In the random group, 24 5-sec shocks were also presented on an independently-programmed schedule. The "positive-prediction" group received only the shocks that were scheduled to occur within 30 sec following CS onset, whereas the "negative-prediction" group received all ahocks except those scheduled during that 30-sec interval. The intertrial interval averaged 2.5 min.

The Sidman schedule remained in effect during the test session and each dog received 24 noncontingent 5-sec test trials. Comparison of response rates before, during and after the CS revealed little change in the responding of the random group. However, reliable increases and decreases were seen in the positive- and negative-predictions groups, respectively. These effects persisted following CS offset and responding only gradually returned to pre-CS levels.

Rescorla suggested that these findings substantiated those of Rescorla and LoLordo (1965), and supported a view of conditioning in which the degree to which the CS allows the subject to predict US occurrence is seen as the critical factor. Despite the fact that the random group received the same number of "pairings" (CS followed by US within 30 sec) as the positive-prediciton group, it did not show enhanced responding to the CS. The nature of the contingency between CS and US was emphasized as being more important than pairings in the establishment of excitatory and inhibitory response tendencies.

It should be noted that Rescorla's procedures resulted in each group's receiving a different number of USs. The random group received the most shocks, followed by the negative and positive-prediction groups, respectively. Although this factor was uncontrolled across groups, because the final ordering of the groups did not vary systematically as a function of number of shocks, there seems to be no simple way to provide an alternative explanation based solely on this difference.

Rescorla (1967a) also used the transfer-to-Sidman technique to show inhibition of delay in Pavlovian fear conditioning. Pavlov reported inhibition of delay in salivary conditioning when the CS-US interval

was progressively extended over conditioning trials. He found a gradual loss in the strength of the response elicited by CS onset, with a greater part of the CR occurring near the end of the ISI. Moreover, not only did the CR disappear from the early part of the CS, but that portion of the CS acquired the ability to inhibit CRs elicited by other stimuli. In his fear-conditioning analogue, Rescorla initially trained two groups of dogs to jump a hurdle on a Sidman schedule, and then alternated conditioning sessions and avoidance sessions. Two test sessions were given, one after three conditioning days (72 trials) and a second after five conditioning days (120 trials). The experimental group received delayed-conditioning trials on which a 30-sec tone coterminated with a 5-sec shock. The control group received an equal number of CSs and USs in a random and independent manner. On the first test day, both groups responded identically on the Sidman schedule during the pre-CS period and first 5-sec of the CS. The experimental group, however, showed a gradual increase in response rate over the 30-sec interval, and was responding reliably more often than the control group by the end of the CS. The pattern of responding was generally the same on the second test day, except that the experimental dogs responded less often than controls (and below the pre-CS baseline) right after CS onset, although they continued to respond more often during the later part of the interval. Thus, inhibition of delay was shown both in the sense of a confinement of the CR to the end of the CS-US interval, and in the ability of the early part of the CS to inhibit situationally-aroused fear. In a later study, Rescorla (1968) replicated this finding using a trace-conditioning procedure in which the CS was presented only during the first

5 sec of the 25-sec CS-US interval.

Rescorla (1967a) also reported inhibition of delay in an experiment in which Pavlovian conditioning was actually superimposed on a Sidman-avoidance task (on-the-baseline conditioning). This was done in order to examine more closely the development of excitation and inhibition under the delay-conditioning procedure. The dogs were restrained in a Pavlovian harness throughout the experiment, and were able to avoid shocks on the Sidman schedule by pressing either of the panels mounted on each side of the head. After initial avoidance training, each of eight dogs received 168 30-sec presentations of the to-be-conditioned CS (tone) without shock while responding on the avoidance schedule. Although CS onset disrupted responding at first, little change was observed after 168 exposures. During the next phase, each dog received 300 pairings of the CS with a brief shock. The CS-US pairings were independent of panel pressing, and shocks programmed under the avoidance schedule were still given. Over conditioning trials, the CS acquired the ability to increase response rate over the pre-CS baseline. On early conditioning trials, responding was uniform throughout the CS-US interval, but as more trials were given, fewer responses occurred during the early part of the interval, and more occurred later in the interval. Eventually, CS onset produced a reliable depression in response rate. As the interval progressed, however, response rate increased, reaching a maximum during the last 5 sec.

At the end of the conditioning phase, an attempt was made to show disinhibition and external inhibition by presenting a 5-sec flashing light during the periods of minimum and maximum responding, respectively.

Disinhibition was observed in the form of a uniform rise in response rate during the CS. However, the effect disappeared within six trials. Evidence for external inhibition of excitation (i.e., a decline in late-interval responding) was not observed.

During the final phase of the experiment, 300 nonreinforced CS presentations were given while the dogs continued to respond on the avoidance schedule. Response rates during the CS slowly returned to the pre-CS baseline level. Although a formal analysis of these data was not reported, it appeared that the later part of the CS lost its excitatory effect more rapidly than the early part lost its inhibitory effect.

The inhibition-producing effects of backward- and "cessation-" conditioning procedures were studied in two experiments by Moscovitch and LoLordo (1968). In both studies, dogs were first given three days of hurdle-jump training in a shuttlebox on a Sidman shock-avoidance schedule. Pavlovian conditioning sessions were administered on the next five even-numbered days, while Sidman training was continued on odd-numbered days. The dogs were confined to one side of the apparatus on each conditioning day and exposed to a sequence of 24 tones and variable duration shocks (4, 5, or 6 sec). In Experiment 1, CS onset occurred 1 sec after shock offset in the backward group, and 1 sec before shock offset in the cessation group. The intertrial interval averaged 2.5 min (range = 2 - 3 min). Testing was accomplished during a single test session in which the tone was presented 24 times. No shock was delivered during this session. Both groups showed a reliable decline in response rate during the 5-sec CS presentation, with the backward group displaying

reliably greater suppression than the cessation group. During the 30-sec following CS offset, both groups continued to respond at a low rate. In fact, during the first 5-sec post-CS block, the cessation group responded even less than it had during the CS itself, and did not differ from the backward group throughout the 30-sec post-CS interval. The performance of both groups was compared to that of a random-control group that had been tested in the same apparatus by Rescorla (1966). This group showed virtually no change in response rate during the CS, and Moscovitch and LoLordo concluded that the response-inhibiting properties of the CS in their two groups were associative in nature. They also suggested that the cessation group may have shown less interference to the CS because (a) it was a "redundant predictor" of shock offset (since shock duration was not too variable, its onset may have provided sufficient "information" concerning its offset), and/or (b) it may have acquired some excitatory properties as a result of its overlap with shock, and/or (c) stimulus generalization decrement occurred in the test session due to the absence of shock.

In their second experiment, an attempt was made to determine whether the backward-conditioning procedure produced inhibition because of its backward relation to shock termination or its forward relation to a long shock-free interval ("safety"). The general procedure was identical to that described above except for the US-CS relation during conditioning. One group received CS onset 15 sec after US offset, followed by an intertrial interval that ranged from 2 - 3 min (mean of 2.5 min). For the other group, CS onset occurred 1 sec after US offset, but the intertrial interval ranged from 0 sec to 15 min

(mean of 2.5 min). The test performance of both groups was compared to that of the backward group in the first experiment. The 15-sec backward group suppressed responding during both the CS and the 30 sec following its offset, and was indistinguishable from the 1-sec backward group in Experiment 1. However, despite the fact that the backward US-CS relation was the same, the 1-sec group with the highly variable intertrial interval showed little inhibition to the CS and responded reliably more often during the CS than the first 1-sec backward group. Taken together, these findings suggested that the interval between the CS and the preceding shock was less important to the development of inhibition than the interval between the CS and succeeding shock. When this latter interval was consistently 2 min or longer, inhibition developed. But when shock was occasionally permitted to occur more closely following the CS, inhibition did not appear. Moscovitch and LoLordo concluded that the backward relationship of the CS to shock termination was unimportant, and that the forward safety-signal properties of the CS were responsible for inhibition. This conclusion, however, does not seem to be entirely warranted. It is possible to argue, for example, that the backward US-CS relation did promote the acquisition in the 1-sec, variable-intertrial-interval group, but that this was counteracted by occasional forward (trace) pairings of the CS with the next US.

Bull and Overmier (1968) reported excitatory and inhibitory effects following differential conditioning using a special discrete-trial discriminated-avoidance test paradigm. Dogs were first trained to jump a barrier in a two-way shuttlebox to avoid shock under a standard dis-

criminated-avoidance procedure (light offset as S^D; 10-sec ISI; 80-sec mean ITI). After a criterion of 10 successive avoidances in a single session had been met (2 - 7 days), the schedule was changed so that responses were reinforced (S^D offset and no shock) on a VI schedule following S^D onset. Each trial began with a variable interval "hold" period during which responses were ineffective. The first response within 5 sec of the end of the hold period terminated the trial. Otherwise, a .5-sec shock was presented. The average duration of the hold period was gradually increased over days (to an average of 8 sec), and by the end of training all dogs were responding at a stable rate during the S^D (12 - 15 responses per minute).

Pavlovian conditioning alternated with avoidance training until six conditioning sessions had been given. During conditioning, the dogs were confined to one side of the shuttlebox. The experimental group received differential conditioning to two tone stimuli: CS+ was followed by a brief shock after a variable ISI (5-sec mean), and CS- occurred an average of 80 sec (the mean ITI) before the next shock. A total of 126 CS+ and 126 CS- trials were given. The control group was exposed to a completely random sequence of tones and shocks.

Testing was done during extinction of the avoidance response and involved trials that began with S^D alone, S^D plus CS+, and S^D plus CS-. A 10-sec hold period was in effect on each trial, and the first response after 10 sec ended the trial. Otherwise, the trial terminated after 30 sec. The major dependent variable was the response rate during the 10-sec hold period on each trial. Both groups responded at approximately the same rate on S^D trials. However, the differential-condition-

ing group showed a reliable increase in rate on CS+ trials and a reliable decrease on CS- trials. The controls responded identically on tone trials and on S^D trials.

Thus, Bull and Overmier confirmed the excitatory and inhibitory effects reported by Rescorla and LoLordo (1965), and in addition, provided a control for CS-US contingency, eliminating several alternative explanations of their findings. Their special discriminated-avoidance procedure provided an explicit conditioned excitor against which the summative properties of both excitation and inhibition could be judged. It would have been interesting to see the results of test trials to CS+ and CS- in the absence of S^D. Presumably, the experimental dogs would have responded to CS+, but not to CS-. The controls should not have responded to either stimulus.

In another variation of the transfer-to-Sidman procedure, Grossen and Bolles (1968) extended the findings of Rescorla and associates to the population of rats. In addition, they compared the performance of a US-only control to excitatory, inhibitory, and random conditioning groups. The rats initially learned to jump a hurdle in a two-way shuttlebox on a Sidman schedule. They were then placed into a separate conditioning chamber, and over a period of three days, were exposed to 90 CSs and 90 USs (except for the US-only control). In Group CS+, shock occurred only within 30-sec following the onset of the 5-sec tone, whereas in Group CS-, shock never occurred within 30 sec. For the random group, tone and shock occurred randomly in time. The intertrial interval averaged 90 sec.

Testing was conducted while the rats were performing the avoidance

response, and 10 test presentations of the CS were given during each of four sessions. In contrast to earlier studies, presentation of the CS was response dependent, beginning 1 sec after a hurdle jump. The dependent variable was the latency to the next response following CS onset. Each groups's mean latency on CS trials was compared to its latency on non-CS trials and to every other groups's mean latency. Analyses showed that the CS+ and CS- groups responded differently on CS trials than on non-CS trials. Group CS+ responded faster, whereas Group CS- responded more slowly. Both of these groups differed from the controls, which in turn did not differ between themselves or from the no-CS baseline level.

Herendeen and Anderson (1968) noted that one of the conditioning paradigms used by Rescorla and LoLordo (1965) to establish inhibitory conditioned responses was exactly that used by other investigators to establish higher-order excitatory conditioned responses. Specifically, they referred to the procedure wherein the to-be-conditioned inhibitor immediately preceded nonreinforced presentation of an established excitatory CS. It was suggested that the differences in the outcomes of these experiments might be related to the number of conditioning trials that have usually been given in each instance. Rescorla and LoLordo administered 90 differential-conditioning trials, whereas most demonstrations of second-order conditioning had involved fewer than 40 trials. Herendeen and Anderson proposed that perhaps excitatory conditioning did occur following a small number of trials, but that with extended training, the second-order CS became a signal for shock omission, and eventually acquired inhibitory properties. In order to test this,

they examined the properties of a second-order CS after either 20 or 200 nonreinforced pairings with an excitatory CS. Second-order trials were interspersed among first-order trials during off-the-baseline conditioning sessions. The 10-sec second-order CS overlapped and coterminated with the 5-sec first-order CS. Control groups received either 20 or 200 random presentation of CS_1 and CS_2 instead of second-order trials. Testing involved the noncontingent presentation of each CS to rats during shuttlebox Sidman-avoidance responding. As expected, all groups showed an increase in responding to the first-order CS. The group receiving 20 second-order trials tended to respond at a higher rate during the second-order CS than its random control, whereas the 200-trial group showed a depression relative to its control. It was concluded that a second-order CS becomes excitatory after a few trials, but inhibitory after many trials. Rescorla (1972) has reported a similar finding in the CER situation.

Weisman and Litner (1969a) have replicated and extended the finding that CS- suppresses avoidance responding by examining the behavior of rats in a wheel-turning Sidman task. After several days of avoidance training, off-the-baseline conditioning sessions were interspersed among on-the-baseline test sessions. Different groups of rats were exposed to one of four conditioning paradigms: (a) differential conditioning (CS+ [light or tone] preceded shock; CS- was explicitly unpaired), (b) conditioned inhibition (same as differential conditioning except that CS- was always preceded by nonreinforced presentations of CS+), (c) CS+ only (same as differential conditioning, but without CS- trials), or (d) CS- only (an explicitly-unpaired procedure). Test

sessions occurred after 20, 40, 60, and 80 conditioning trials. During each test session, 20 nonreinforced presentations of CS+ and CS- were given while the rat was performing the Sidman response. Response rates before, during, and after each stimulus presentation were the major dependent variables. By the end of training, rats were showing reliable increases in response rate during CS+ (Groups a, b, c) and reliable decreases to CS- (Groups a, b, d). Enhanced responding to CS+ appeared after the first conditioning session (after 20 trials), but three to four sessions were required before reliable interference was produced by CS-. Weisman and Litner suggested that this might indicate that conditioned inhibition of fear could not develop until conditioned fear itself had been established. Alternatively, it may have been that whatever was learned on CS- trials required more repetitions to produce a given level of performance. Although direct comparison of the magnitude of the response change to CS+ and CS- was not made among the groups, the conditioned-inhibition procedure appeared to produce the most dramatic effects, followed by the differential-conditioning group and the two contrast groups, respectively. Formal between-group comparisons, however, may not have been made because of what appeared to be group differences in the baseline response rates.

Extinction of Pavlovian conditioned excitation and inhibition was examined in a second experiment by Weisman and Litner (1969a). Following four days of conditioning identical to that described above, different groups of rats were given 15 consecutive test sessions during which neither stimulus was explicitly reinforced. In general, response enhancement to CS+ disappeared more rapidly (within six sessions) than

did response suppression to CS- (9 - 11 sessions). That CS- appeared to inhibit responding after CS+ no longer enhanced responding suggests that the inhibitory properties of CS- were not necessarily dependent upon the strength of fear elicited by the excitatory stimulus with which it was originally contrasted. Depression of response rate must then be interpreted in terms of inhibition of situationally-aroused fear.

As an aside, it should be noted that this last study does not unambiguously support the view that nonreinforced presentations of CS- in the absence of an explicit relation to shock leads to extinction of inhibition. Since shocks programmed on the Sidman schedule were still delivered throughout the test sessions, it is reasonable to suggest that if CS- had its expected effect (i.e., lowered response rate), then the frequency of shocks occurring shortly after its offset would increase, thus providing conditions sufficient for trace excitatory conditioning (see Zimmer-Hart & Rescorla, 1974).

Using the test procedure described above, Weisman and Litner (1971, Exp. 1) have also found that the inhibitory effects of the CS- in differential conditioning increase as the intertrial interval (interval between successive CSs, regardless of type) is increased from .5 to 5 min. As before, enhanced responding to CS+ occurred after the first 20 conditioning trials and remained relatively constant over the remaining trials. In addition, this effect was independent of the length of the intertrial interval. The strength of inhibition to CS-, however, showed only a gradual increase over conditioning days, and the course of its development depended upon the intertrial interval. The longer the

interval, the sooner the inhibitory effect appeared.

In a second experiment (previously reported by Litner & Weisman, 1970), the amount of inhibition elicited by a backwardly-paired CS (5-sec US-CS interval) was shown to be, in part, a function of the amount of conditioned inhibition elicited by a second stimulus. Different groups of rats received differential inhibitory conditioning in which one backwardly-paired CS always preceded a 200-sec shock-free interval, while a second backwardly-paired CS preceded shock-free intervals of 10, 110, 200, 290, or 390 sec. After 160 conditioning trials to each stimulus, test presentations were given during the Sidman wheel-turning task. The amount of response suppression that occurred during presentation of the second stimulus was an increasing function of the length of the intertrial interval that it had preceded during conditioning. Interference during the CS that had preceded the 200-sec interval in all groups, however, was an inverse function of the interval associated with the second stimulus. The shorter that interval, the greater the inhibitory power of the first CS. Statistical analyses suggested that a linear function best described the relation between the relative amount of suppression to the first CS and the proportion of the total nonshock time with which it had been correlated.

The authors suggested that this finding was an instance of what Pavlov had described as "negative induction." Pavlov used that term to refer to the intensification of inhibition that occurred as a result of prior excitation (Pavlov, 1927, p. 196). As an example, he cited the experiments of Krjishkovsky in which attempts had been made to destroy the inhibitory properties of a compound CS by pairing it with the

US. Krjishkovsky discovered that inhibition could be reduced in this manner quite readily, but only if no conditioned excitatory stimuli were presented during the same session. If reinforcement of the conditioned inhibitor alternated with reinforcement of a previously established conditioned excitor, the inhibitor proved to be exceedingly resistant to the effects of forward pairings with the US. Presumably, excitation aroused by the positive CS enhanced the inhibition elicited by the succeeding negative CS. In Weisman and Litner's experiment, the less inhibitory (more excitatory) of the two stimuli supposedly produced a similar effect. It may be reasonable to suggest that the reciprocal process, positive induction, was also at work. Presentation of the more inhibitory stimulus might be expected to intensify whatever excitation was associated with the other stimulus.

Weisman and Litner have noted the similarity between the CS- in differential conditioning and response-produced feedback stimuli in avoidance conditioning. Both events precede long shock-free intervals. These authors have reported that a conditioned inhibitor can be used differentially to reinforce high and low rates of responding in the wheel-turning Sidman-avoidance task (Weisman & Litner, 1969b), and have recently suggested a revised two-factor theory of avoidance in which the reinforcing effects of Pavlovian conditioned inhibitors play a major role (Weisman & Litner, 1972)(For a general discussion of the role of feedback stimuli in avoidance learning, see Cunningham, 1973).

CER procedure. Hammond (1966) examined differential conditioning in an on-the-baseline CER procedure. For the experimental group, a 3-min tone (CS+) consistently preceded a .5-sec, .72 mA shock while a

flashing light (CS-) was not reinforced. Three CS+ and three CS- trials were given during each of 15 2-hr sessions. A control group received the same sequence of tones and lights, but in the absence of shock. CS+ reliably suppressed responding below the level shown by controls. Response rate during CS- showed an increase relative to the 3 min preceding its onset, and in fact, over Days 3 to 9, the response ratios of the experimental group actually exceeded those of the controls. Examination of baseline response levels (responses/min during the 3-min, pre-CS interval) in the experimental group showed an initial decline in overall rate of responding (to about 50% of the preconditioning level) that gradually returned to preconditioning levels. Enhanced response ratios in the presence of CS- reflected momentary returns to this preconditioning level, rather than increases in absolute level of responding. As the baseline returned to its earlier level, the difference in response rate between the pre-CS interval and CS- interval diminished. Hammond suggested that the lowered baserate resulted from fear conditioned to apparatus cues. CS- temporarily inhibited this fear, permitting the response rate to approach preconditioning levels. However, with continued conditioning, apparatus cues in the absence of CS+ themselves presumably became inhibitory, leading to a recovery of the baseline. In the absence of situationally-aroused fear, CS- no longer enhanced response rate in its presence.

Subsequently, Hammond used an explicit conditioned excitor in a combined-cue test to demonstrate the inhibitory properties of CS- in the CER paradigm. In this study (Hammond, 1967), the inhibitory-conditioning group received 30 CS+ and 30 CS- trials over a 10-day

period. This differential-conditioning procedure was identical to that described in the previous experiment. During the second phase of the study, CS+ and CS- were presented simultaneously in the absence of further shocks. Presumably, if CS- had acquired inhibitory properties, suppression in the presence of the compound should have been less than that in the presence of CS+ alone. In order to show that this loss in suppression was above and beyond that attributable to stimulus generalization decrement, Hammond compared responding in the experimental group with a control that had received the light stimulus at random with respect to tone-shock pairings during the initial conditioning phase. As predicted, the differential-conditioning group suppressed less in the presence of the compound than did the random control. During the final test to CS+ alone, both groups showed the same amount of suppression, suggesting that extinction of the excitatory CS was not facilitated by compounding it with a conditioned inhibitor.

Hendry (1967) used a "conditioned-inhibition" paradigm in an on-the-baseline CER procedure. A 2-min intermittent noise was paired with a .5-sec, 1-mA shock, except when presented in compound with a dim light (light onset preceded noise onset by 5 sec). Four reinforced trials (noise plus shock) were interspersed among 12 nonreinforced trials (noise plus light) during each of 12 96-min sessions. Examination of response rates during this phase showed that the suppression ratios to the noise alone averaged between .1 and .2, whereas suppression to the compound averaged between .4 and .5. This difference alone, however, does not unambiguously support the view that the light (or compound) had become inhibitory. No controls were included to assess non-

associative effects of the conditioning procedure. For example, lack of suppression to the compound may have resulted from generalization decrement or loss of attention to the noise stimulus. Moreover, the failure to counterbalance the stimuli used as excitor and inhibitor did not eliminate the possibility that the visual stimulus "overshadowed" the auditory stimulus in compound, producing the obtained result independent of any conditioning mechanism.

Hendry attempted to provide further data in support of the view that light had become inhibitory. In a second phase of the experiment, pressing the bar that had originally produced food reward was still effective, but in addition, pressing a second bar produced 5-sec presentations of either the noise stimulus (3 rats) or light stimulus (3 rats). During this phase and a subsequent reversal, rats tended to press more often when the light was presented. Hendry interpreted this as supporting the view that the conditioned inhibitor functioned as a conditioned reinforcer. These findings are equivocal for a variety of reasons. First, the comparison was made between the effects of an alleged inhibitor and a known excitor instead of some "neutral" stimulus. The difference that appeared could have been due entirely to the secondary-punishing effects of the noise. Hendry argued against this by pointing out that with repeated presentations, the light appeared gradually to lose its apparent reinforcing effect, suggesting that conditioned inhibition was undergoing extinction. However, the loss in reinforcing value might also be consistent with the suggestion that the light initially exerted an unconditioned reinforcing effect that gradually habituated. A more meaningful comparison might have been

made with a group that received response-contingent light presentations, but for which light had not been contrasted with a conditioned excitor during conditioning. A second problem with this alleged demonstration of secondary reinforcement is that there was no control for the response-eliciting effects of the light. Since the light had been presented during the conditioning phase while the animals were pressing the first bar, it is possible to argue that during Phase 2, the first few barpresses served merely to reinstate conditions that had been associated with a comparatively high rate of responding on the first bar. Such reasoning might lead to the prediction that responding to the first bar would be greater in the presence of the light than in its absence. However, these data were not reported.

Reberg and Black (1969) used a combined-cue procedure to detect both excitation and inhibition in a CER situation. In their first experiment, hungry rats that had been trained to barpress for food reward were given forward, on-the-baseline conditioning trials in which either a white-noise or the offset of the houselights was paired with a brief shock (89.5-sec ISI). After four reinforced trials with each stimulus, different groups were given a single nonreinforced trial with either noise, darkness, or the noise-darkness compound as the stimulus. Rats receiving the compound suppressed more than they did to the noise or darkness alone, and more than rats that received single stimulus test trials. During extinction, individual tests with each stimulus and with the compound showed that 11 of 13 rats were more suppressed during the compound than during either of the single stimuli.

In a second experiment, after two days of forward conditioning to

one of the stimuli (six trials total), one group of rats received two additional days of differential conditioning in which the second stimulus was also presented three times per day, but was not reinforced. A control group simply received three CS+ trials on each of these days. In extinction, all rats were tested with each individual stimulus and the compound. The differentially-conditioned rats were reliably less suppressed to the compound than to CS+. The control group showed no such difference. The authors suggested that these results strongly supported the notion that excitation and inhibition can summate algebraically to determine amount of suppression in the CER paradigm. They argued that the control condition in the second experiment ruled out novelty or stimulus generalization decrement as alternative explanations of the inhibitory effect of CS-. However, they assumed that generalization decrement and novelty were unaffected by preexposure to the alleged inhibitory component of the compound. For example, one might argue that the compound was "more novel" for the control group than for the experimental group because the controls had never been exposed to one of the components. If "novelty" leads to greater suppression, their results need not be taken as support for conditioned inhibition.

Inhibitory stimulus control that could be "turned on and off" was reputedly demonstrated by Cappell, Herring, and Webster (1970) in a differential CER conditioning study. During initial on-the-baseline differential conditioning, CS+ and CS- were light and tone (or tone and light), respectively, for two groups of two rats each. After 30 trials of each type, all rats showed differential suppression, responding less

during CS+ than during CS-. Suppression developed more rapidly when the tone was CS+, and there was a tendency toward increased responding during the light when it was CS-. During the test phase of the experiment, three CS+ and three CS- trials were still given on each conditioning day. However, CS- was also present during the first or second minute of the 3-min CS+. The data were presented in terms of the percentage of the total responses during CS+ that occurred in each minute. For all rats, the greatest percentage of responses were emitted during CS- when it was presented during the first minute. When CS- was presented during the second minute, only the light CS- rats showed a greater percentage of responses during that minute. The authors concluded that CS- had exerted an inhibitory effect, and that this effect could be turned on and off during consecutive minutes of an excitatory conditioning trial. Unfortunately, there were no controls to determine whether this was a conditioned inhibitory effect or whether it was due simply to stimulus generalization decrement.

Startle-probe technique. Using a startle-probe technique similar to that originally described by Brown et al. (1951), Galvani (1970) attempted to assess the inhibitory properties of CS- following differential conditioning. The experimental group received 25 CS+ and 25 CS- trials on each of four successive conditioning days. On CS+ trials, a 5.5-sec tone coterminated with a .5-sec shock, while CS- (a different tone) occurred in the absence of shock and at least 60 sec before the next shock. The control group received an equal number of exposures to each CS and shock, but these were programmed independently in a random manner. At the end of every session, a single test trial to each CS

was administered. Five sec following CS onset, a brief air puff was delivered through the end panels of the chamber. The stabilimeter device was attached to a recording system that produced a graphic measure of the startle response to the air puff. Comparisons of the responses elicited during CS+ between the experimental group and control group indicated a reliable enhancement of startle magnitude. However, no depression of startle magnitude was seen in the presence of CS-. Since startle responses were obtained at levels well below the mean value shown by the random control, the failure to see response depression did not appear to be attributable to a physical floor effect. Galvani raised the possibility that there may not have been a conditioned excitatory baseline against which to assess inhibition, but suggested that fear conditioned to apparatus cues may have been sufficient. However, the fact that there was no overall increase in the responding of the control animals over conditioning days does not support this suggestion. If fear were being conditioned to apparatus cues in this experiment, one might reasonably expect such an increase (see Meryman, 1952).

New-Learning Tests

CER and Classical-defense-conditioning procedures. Hammond (1968) reported two studies in which new-learning test procedures were used. The first experiment was a factorial in which type of conditioning (differential vs random) was combined with number of conditioning trials (15 vs 30). The general procedure was similar to that of Hammond's previous experiments, save that the test phase consisted of a reversal in the roles of tone (previously CS+) and flashing light

(previously CS-). Attention was focused on response-rate changes in the presence of the light, particularly during excitatory conditioning. The random controls consistently showed greater suppression to the light during the conditioning phase than either of the inhibitory groups. In addition, the more training trials, the greater the retardation, as indicated by the number of conditioning days required to show a mean suppression ratio of .1 or less to the light. This latter finding, however, was independent of the type of conditioning procedure (there was no interaction between number of trials and type of conditioning), and suggested that simply increasing the number of stimulus pre-exposures, regardless of the stimulus-shock relation, had produced greater interference.

In the second study, Hammond compared differential- and random-conditioning groups to "habituation" and "novel-CS" control groups in a new-learning test. The habituation group received the same number and kind of stimuli as the differential- and random-conditioning groups, but did not receive shock until the test phase. The novel-CS group received an equal number of tone-shock pairings (45), but was not exposed to the flashing light until it was paired with shock during the test phase. Analysis of the test suppression ratios showed that the differential group conditioned more slowly to the former CS-, but was reliably different only from the habituation group. The failure to find any difference between the experimental group and either the random or novel-CS group does not support the view that CS- had acquired inhibitory properties. In general, the results were more consistent with the notion that prior US exposure retards subsequent

conditioning as a result of US habituation (Kamin, 1961). Hammond suggested that the failure to see inhibition following 45 differential-conditioning trials possibly reflected nonmonotonicity of the acquisition curve for Pavlovian inhibition.

A "negative-contingency-discrimination" paradigm was used by Hammond and Daniel (1970) to show conditioned inhibition as a within-subject effect. Rats that had been trained to barpress for water reinforcement were exposed to a special differential-conditioning procedure over a period of 30 days. During every 2-hr session, each rat received three shocks, three "safe" stimuli, and three "random" stimuli while barpressing. The safe stimulus (tone or light) never occurred during or within 6 min after a shock. The random stimulus was presented independently with respect to the occurrences of shock or the safe stimulus. In order to maximize the possibility of observing response facilitation to the safe stimulus (cf. Hammond, 1966), shock intensity was adjusted daily for each rat in an attempt to depress the operant baseline.

Analyses of the baseline indicated that this manipulation was successful in reducing the baseline over conditioning days. During this phase, both stimuli produced response facilitation (suppression ratios greater than .5), but this facilitation was greater in the presence of the safe stimulus (NB: trials that overlapped with shock or the other stimulus were not included in this analysis). Thus, reliable differential responding occurred as a result of the conditioning procedure.

In the second phase of the experiment, both stimuli were paired with shock in a forward manner on a 50% partial-reinforcement schedule. All rats (n = 8) were slower to suppress to the formerly safe stimulus than they were to the formerly random stimulus. The overall difference in suppression for the 12 conditioning days was quite reliable. Assuming that the random stimulus was associatively neutral (as Rescorla's contingency analysis suggests), this experiment would appear to provide strong evidence for conditioned inhibition. If one were to argue from a "pairings" viewpoint, however, it might be reasonable to suggest that the performance during differential conditioning reflected different degrees of conditioned excitation, with background cues being most fear arousing (evidenced by depressed baseline), followed by the random CS (occasionally paired with shock), and the safe stimulus (never paired with shock). The difference in the final test would then be attributed to differences in amount of fear aroused by each of the stimuli at the end of differential conditioning, not to differences in amount of conditioned inhibition.

Siegel and Domjan (1971) demonstrated the conditioned inhibitory properties of a stimulus that had been backwardly paired with a shock US in both CER (rats) and eyelid (rabbits) conditioning situations. In each case, a new-learning test was used to assess inhibition. Backward-paired groups were compared to groups receiving CS-alone, US-alone, random CS and US, or no stimulus presentations. In the CER experiment, US and CS onsets were simultaneous (0.5-sec US, 2-min CS) in backward condition (actually simultaneous conditioning). In the eyelid study, however, US offset coincided with CS onset

(0.1-sec US, 0.5-sec CS). Fifty off-the-baseline exposures preceded forward on-the-baseline CER conditioning, and 550 exposures preceded eyelid conditioning. In both experiments, backward pairings produced the most interference during subsequent excitatory conditioning. Groups receiving no stimulus pre-exposures conditioned most readily, while the three other groups fell between the no-pre-exposure group and the backward group. These three groups did not differ among themselves, except in the eyelid study where the random group conditioned reliably slower than either the CS or US group (though all were still better than the backward group). It was suggested that this difference between experiments may have been due, in part, to the fact that the procedure used to generate intervals for the random group in the CER experiment restricted inter-US intervals to multiples of 30 sec. No such restriction was in force for the eyelid study. Siegel and Domjan concluded that these studies showed a conditioned inhibitory effect due to backward pairings that was above and beyond non-associative effects attributable to prior CS exposure (e.g., latent inhibition; cf. Lubow, 1973; Siegel, 1972), US-exposure (e.g., US habituation; cf. Kamin, 1961; Taylor, 1956), or exposure to both CS and US in the absence of pairings. Moreover, the findings appeared to support Rescorla's (1967b) contingency formulation.

In a later set of experiments, Siegel and Domjan (1974) attempted to show that the inhibitory effect produced by backward pairings of the CS and shock was a function of the number of pairings. Once again, both CER and eyelid conditioning procedures were used, and in each case, different groups were exposed to 0, 5, 10, 25, or 50 US-CS

presentations prior to excitatory conditioning. In both experiments, there was an inverse relation between the number of pre-exposures and strength of conditioning. However, only the differences between each 50-trial group and its no-exposure control were significant. The authors argued that these results did not support the contention that inhibitory effects in backward conditioning were dependent upon extensive training, as had been suggested by Heth and Rescorla (1973). Unfortunately, the experiment lacked the controls necessary to support this assertion. In fact, in an earlier CER experiment by these same investigators (Domjan & Siegel, 1971), different numbers of pre-exposures to the CS-alone produced effects that were nearly identical to those reported above, suggesting that "latent inhibition," rather than conditioned inhibition, may have been responsible for reduced conditioning.

The retarding effect of prior backward conditioning on subsequent forward conditioning was also examined by Plotkin and Oakley (1975) using the nictitating-membrane preparation. During the initial phase, two groups of rabbits received 125 backward pairings of a 200-msec CS and a 200-msec US at ISIs of either 200 or 500 msec. The intertrial interval was 55 sec. Several other groups were also run: a no-conditioning, restraint group; a latent-inhibition (CS-only) group; and a forward, trace-conditioning group (200-msec ISI). In addition, a "safety-signal" group was run in an attempt to control for the fact that the CS in the backward groups always preceded a long shock-free interval in addition to being backwardly paired with the US. The backward US-CS interval was extended to 55 sec, while the forward CS-US interval remained at 55 sec. All groups then received forward delay conditioning

in which the CS (now 500-msec long) overlapped and coterminated with the 200-msec US.

Examination of the response to the CS in the backward groups revealed no indication of an excitatory response during backward conditioning. During the test phase, the backward groups did not differ, but both were reliably slower to condition than the no-conditioning, latent-inhibition, or safety-signal controls. These latter groups did not differ among themselves. The forward-trace group transferred to the delay procedure with no loss in response strength.

Plotkin and Oakley concluded that the retardation that followed backward conditioning was independent of the forward safety-signal function of the CS and the effects of latent inhibition. The failure to find a difference between the CS-only group and the no-conditioning group (failure to show latent inhibition) was attributed to the relatively small number of pre-exposures (e.g., Siegel & Domjan, 1971, used 550), and to possible differences between the nictitating membrane and the other response systems that have shown latent-inhibition effects.

The authors argued that the safety-signal group controlled for the relation between the CS and the shock-free interval, but suggested that that relation might still be important if a signal that precedes the entire shock-free interval (independent of its duration) becomes a stronger inhibitor than one that precedes only half of the shock-free interval. Whether or not that is the case, one might still question whether this group represents an appropriate control for the effects of prior US exposure. Since the intershock interval was twice as long in this group as it was for the backward groups, the rate of US

habituation (or even CS habituation) might not have been comparable among these groups. The retardation in the backward groups might have been due completely to the effects of this differential prior exposure to the US (cf. Mis & Moore, 1973).

Multiple-tests

There have been two major series of experiments in which both combined-cue and new-learning tests have been used to assess the inhibitory effects of certain types of conditioning procedures. One series employed the CER technique and the second used a classical defense-conditioning paradigm.

Rescorla (1967b, 1969a, 1969d) has suggested that the contingency between CS and US is an important dimension in establishing conditioned inhibition (and excitation). In fact, he has noted that all of the procedures that produce conditioned inhibition appear to have one thing in common--namely, a negative CS-US contingency (i.e., $Pr[US/CS] < Pr[US/\overline{CS}]$). Studies involving the transfer-to-Sidman procedure support this notion, and in an attempt to extend the generality of those findings, Rescorla (1969b) manipulated degree of negative CS-US contingency in two CER experiments with rats. In the first experiment, a new-learning test was used to assess inhibition, and in the second experiment, a combined-cue test. In both experiments, conditioning was conducted off-the-baseline after initial barpress training. The to-be-conditioned inhibitor was a 2-min tone and it was presented 12 times per day over five conditioning days at a mean intertrial interval of 10 min. Shock frequency in the presence of the tone and in its absence was dependent upon group assignment.

In the first experiment, there were two levels of negative contingency, and for each of those experimental conditions, there were two control groups. In the experimental groups, shock never occurred during the CS or during the 2 min following its offset. However, during all other 2-min intervals, shock occurred with a probability equal to .1 (Group 0-1) or .4 (Group 0-4). In addition, shock onset was equiprobable in each 1-sec interval during those portions of the session in which it was schedule to occur. There were two random control groups (one for each experimental group) for which the probability of the US in the absence of the CS equaled its probability in the presence of the CS (Groups 1-1 and 1-4). It should be noted that this does not mean that an equal number of shocks occurred in the presence and in the absence of the CS. Rather, these figures refer to the probability that shock would occur in any given 2-min interval. Because there were more non-CS intervals than CS intervals, presumably more shocks occurred in the absence of the CS than in its presence. Two additional groups received negative-contingency conditioning, but to a different stimulus (Groups 0-1 Light, and 0-4 Light). All groups were then exposed to forward, on-the-baseline pairings of the tone and shock (new-learning test).

Analysis of suppression ratios during the test phase revealed that the negative-contingency tone groups suppressed less to the tone than their respective controls. Moreover, the degree of retardation was greater in the group pre-exposed to the larger negative contingency, Group 0-4. The controls did not differ among themselves. Rescorla suggested that these findings were consistent with the view that negative contingencies produce conditioned inhibition, but admitted

that the comparisons between each experimental group and its controls might not be the most appropriate ones for substantiating this position. For example, one might argue that the random-conditioning procedure endowed the CS with excitatory properties as a result of chance CS-US pairings. In that instance, the differences between each of the experimental groups and its random control might be viewed in terms of facilitation under the random condition rather than retardation under the negative-contingency condition. Rescorla noted that this view would predict that the random groups should have conditioned more rapidly than the light groups. However, because the control groups differed among themselves in terms of the total number of US exposures during the intermediate conditioning phase (the random groups received more shocks on the average than the light groups), the failure to see that difference does not necessarily vitiate the argument.

Similarly, the differences between each experimental group and its light control did not unambiguously support a conditioned-inhibition interpretation. It might be argued that mere pre-exposure to the CS was responsible for retardation in the experimental group ("latent inhibition"). In conclusion, Rescorla suggested that the difference between the two experimental groups was critical to the contingency hypothesis. The group with the greater negative CS-US contingency conditioned more slowly. Unfortunately, the experimental groups also differed in terms of the number of pre-exposures to the US, with Group 0-4 receiving more shocks. Because degree of retardation may be a direct function of number of US pre-exposures (Mis & Moore, 1973), Rescorla's critical finding may still be ambiguous.

In Rescorla's (1969b) second experiment, three groups of rats were initially exposed to different levels of negative contingency (0-1, 0-4, 0-8) between tone and shock. A fourth group received an equal number of tones but no shock (0-0). At that point, a flashing light was established as a conditioned excitor in an on-the-baseline procedure (total of 12 trials on a 50% reinforcement schedule). Testing involved nonreinforced presentations of the flashing light alone and flashing-light-plus-tone compound (combined-cue test).

Rescorla reported that suppression to the light alone was strong and "about equal" in all groups. No formal analysis of these scores was reported. The bar graph that he presented showed the mean suppression ratio to the light in each of the experimental groups to be less than that shown by the CS-alone control. If those differences were reliable, differences obtained on compound test trials might be open to interpretation. Siegel and Domjan's (1971) finding that as few as 50 US pre-exposures reliably retarded CER acquisition might lead one to suspect a similar effect here (Rescorla's Groups 0-1, 0-4, and 0-8, should have received an average of 18, 72, and 144 US pre-exposures, respectively).

Even if the apparent differences in suppression to light were not significant (or if they are simply ignored), it is not clear that the data obtained on the compound test trials support the contingency interpretation. In general, all groups (including the control) were less suppressed during the compound than during light alone. Moreover, the greater the negative contingency, the less the suppression to the compound. Rescorla reported that a one-way analysis of variance involving all four groups indicated that the tone produced a reliable

"disruption" in suppression to the light. It is not clear whether the dependent variable in this analysis was the absolute level of suppression to the compound, or the difference in suppression between the compound and light alone. Use of a difference score might have partially corrected for the apparent between-group differences in suppression to the light alone. In any event, no matter which dependent variable was used, one cannot be sure that the effect was not entirely due to the difference between the control and combined experimental groups, as opposed to a systematic difference among levels of negative contingency (no followup comparisons were reported). Although a difference between the CS-only group and the experimental groups is consistent with the contingency analysis, because the control group did not receive the same numberof prior US exposures, this effect might be attributable to nonassociative factors. As Rescorla (1967b) himself has suggested, rate of habituation to the CS might be interfered with by US presentations. The experimental groups in his second study may simply have been showing greater amounts of external inhibition (or stimulus generalization decrement) in the presence of the tone because habituation was not as complete as it had been for the controls.

Rescorla concluded that his demonstration of inhibitory effects both in a retardation test and in a summation test clearly supported the view that stimuli predicting greater reductions in US probability become greater inhibitors. The present analysis would suggest a more cautious conclusion.

Combined-cue and new-learning tests were used to assess conditioned inhibition of the rabbit's nictitating-membrane response by Marchant,

Mis, and Moore (1972). Two groups first received forward delay conditioning to each of three stimuli--light (CS_1), white noise (CS_2), and tone (CS_3). The ISI was 500 msec and each stimulus co-terminated with a 50-msec infraorbital shock. Training during this phase continued until each animal reached an 80% criterion level of responding (approximately 140 trials to each CS). Conditioned-inhibition training was given to one group over the next seven sessions. During each session, 50 reinforced CS_1 trials were interspersed among 50 nonreinforced CS_1-CS_3 compound trials. The control group also received a random sequence of 50 CS_1 trials and 50 CS_1-CS_3 compound trials, but both were reinforced on a 50% schedule. This training was designed to make CS_3 more inhibitory in the experimental condition than in the control.

Combined-cue testing was given over the next three days, and it consisted of nonreinforced presentations of each stimulus alone and of all possible two-stimulus compounds. This was followed by a new-learning test during which all three stimuli were again forwardly paired with shock. Training continued until an 80% criterion was attained.

Analysis of percentage CRs during the final days of the differential-conditioning phase indicated that the experimental group responded reliably less often to the CS_1-CS_3 compound than to CS_1 alone. The controls showed no such difference, responding to both CS_1 and CS_1-CS_3 at a high level. Excitatory summation was observed during testing as a positive difference between responding to the CS_1-CS_2 compound and CS_2 alone. It was also seen as a difference between

responding to CS_1-CS_2 and CS_1 alone in the control group. It was suggested that the failure to see this latter difference in the experimental group was possibly due to the relatively high level of responding to CS_1 in the experimental condition. No attempt was made to explain the discrepancy in response strength to CS_1 between the groups. Although both groups received the same number of pairings of CS_1 and shock, since the control group received half of those pairings in Phase 2 in compound with an established excitor (CS_3), "blocking" may have occurred (cf. Kamin, 1969; Rescorla & Wagner, 1972).

Because the response to CS_1 was not the same across groups, the critical evidence for inhibitory summation was the comparison between level of responding to CS_2 (which was the same in each group) and level of responding to CS_2-CS_3. The control group showed no difference in its response to the compound, whereas the experimental group responded reliably less often to the compound, suggesting that CS_3 was indeed inhibitory in the latter group. When responding to CS_1-CS_3 was compared to responding to CS_1, the experimental group again showed a decline consistent with an inhibitory interpretation. The control group, however, showed an increase in responding to the compound. If this finding means that CS_3 had actually acquired excitatory properties in the "pseudo-conditioned-inhibition" control, then the differences that were presented as evidence for inhibitory summation might more parsimoniously be interpreted simply in terms of a loss of excitation in the experimental group.

During the final reacquisition, the experimental group recon-ditioned to CS_3 reliably slower than it did to CS_2. Moreover, they

differed reliably from the control group. Reacquisition to CS_3 was also slower than to CS_1 in the experimental group. However, unlike CS_2, CS_1 elicited different levels of responding across the groups at the beginning of the retardation test.

Marchant et al. concluded that conditioned inhibition had been shown as a tendency opposite that produced by conditioned excitation, and that their control condition, and the use of both combined-cue and new-learning tests eliminated a number of alternative explanations. In a subsequent study, however, Marchant and Moore (1974) addressed the possibility of the "loss of excitation" interpretation mentioned above. They conducted another membrane-conditioning experiment and compared their inhibitory-conditioning procedure to a variety of additional controls in a new-learning test. In contrast to their previous study, they wanted to provide a control group that represented a "zero level," below which inhibitory effects might be observed. They chose a group that was simply restrained in the conditioning apparatus during the initial phase of the experiment (no CSs or USs). The experimental group received reinforced light trials (L +) and nonreinforced light-tone compound trials (LT-) during this phase. Another group received L+ trials and nonreinforced light-noise trials (LN-) to assess the effect of inhibitory training per se, and a fourth group received N+, LT- training to control for the number of LT- exposures, but under conditions not expected to produce inhibition. Finally, a "naive" group received no treatment prior to the retardation test. During that test, the tone was paired with shock for all groups.

Analysis of trials to criterion (80% CRs over three consecutive blocks) and percentage CRs yielded essentially identical results. The experimental group was reliably slower to condition than either the no-CS or naive control. The two other conditioning groups did not differ, but each conditioned reliably faster than either the no-CS or naive control group.

Marchant and Moore viewed the difference between the experimental and no-CS controls as critical to the assertion that the inhibitory-conditioning procedure had produced a CS with "negative value." They argued that the effect was not readily interpreted as due to "attentional decrement" because (a) their criterion during differential conditioning (better than 80% CRs to L+ and less than 30% to LT-) demanded the animal's attention to the tone throughout training, and (b) Group N+, LT- should have shown an attentional decrement also (since it had received an equal number of LT- exposures), but did not. This second point may not be as strong as it seems if it could be argued that the tone elicited a generalized excitatory tendency in Group N+, LT- by virtue of the fact that the tone was more similar to the conditioned excitor (same modality) in the experimental condition. That Group N+, LT- was facilitated in its acquisition to the tone is consistent with this suggestion. The failure to see generalization of inhibition in Group L+,LN- from noise to tone might further indicate that inhibition had not been established under the differential-conditioning procedure. Thus, the only argument in favor of a conditioned-inhibition interpretation would appear to be the logical one that appeals to the differential responding in the conditioning

phase as evidence against a nonassociative, attentional-decrement hypothesis.

Special Issues

Unconditioned inhibitory effects. That the unconditioned inhibitory properties of a stimulus need to be considered in evaluating conditioned inhibition has been emphasized by Hendersen (1971). In his first experiment, he found that rats that had never been exposed to the tone used in a transfer-to-Sidman procedure showed a depression in response rate during the tone comparable to that shown by rats for which the tone had been CS- during prior differential conditioning. The inhibitory effect of CS- could not clearly be attributed to an acquired inhibition of fear. The change in response rate shown by the controls suggested that the effect was due to unconditioned fear-inhibiting properties or simply to external inhibition of the Sidman avoidance response.

Hendersen reasoned that if the tones were "true" unconditioned inhibitors of fear, then they should facilitate rather than reduce a baseline that had already been depressed by a conditioned excitor (e.g., barpressing in CER). However, if the tones acted as external inhibitors of the operant response, they should only further reduce the baseline. To test this, he compared the unconditioned effects of the tone on several different baselines--barpress Sidman avoidance, barpress VI-2-min food reward and barpress DRL-30-sec food reward. In addition, he compared the unconditioned and conditioned effects of the tone on the VI-2 baseline in groups whose responding was depressed due to the presence of CS+ during on-the-baseline CER conditioning.

The Sidman barpress response, like the shuttle response, was depressed when the unconditioned tone stimuli were presented. On the other hand, in the absence of CER training, no effect was observed when the unconditioned tones were superimposed on the VI or DRL baselines. However, whether conditioned or not, the tones facilitated responding when presented during CS+ in the CER procedure. In addition, CS- was more effective in attenuating suppression than the novel tone. Hendersen concluded that the unconditioned effects of the tone were such as to actively inhibit fear. If the tones had merely produced external inhibition of the operant response, one might have expected barpressing to be more suppressed rather than less suppressed during CS+. That the tone exerted no unconditioned suppressive effect in the VI-2 condition supports this conclusion.

Although Hendersen's conclusion seems appropriate on a descriptive level, one might still question whether the "unconditioned inhibitory effect" of the tone could be more simply interpreted as due to stimulus generalization decrement or external inhibition of the conditioned fear response. In this way, the increase in barpressing in the CER and the decrease in barpressing in the Sidman task could both be viewed as due to a loss in excitation resulting from a change in the fear-arousing stimulus complex.

In any event, Hendersen's data emphasize the importance of determining the unconditioned effects of to-be-conditioned inhibitory stimuli in evaluating the effectiveness of conditioning procedures alleged to produce conditioned inhibition. In addition, Hendersen suggested that it might be useful to consider whether conditioning

changes those initial properties or whether they are merely "masked" during conditioning. As an example of a situation in which this distinction might be useful, he discussed the problem of whether an extinguished excitor can become inhibitory ("extinction below zero"). If the stimulus had inhibitory properties prior to any conditioning, "extinction below zero" might simply reflect a return to that unconditioned state rather than the acquisition of an inhibitory tendency. In his final experiment, Hendersen demonstrated that nonreinforcement of an established tone CS+ produced inhibitory effects in both the Sidman and CER situations. However, in accord with his contention, these effects appeared to be the same as the initial unconditioned effects of the tone, suggesting that things had simply been returned to their original condition. It seems quite possible that Kamano's (1968) demonstration of the inhibitory properties of an extinguished CS+ can be interpreted similarly.

Situational effects. There are two studies that suggest that the effects of a Pavlovian conditioned inhibitor of fear may be restricted to the situation in which conditioning occurs. Desiderato (1970) trained dogs to jump a hurdle in a shuttlebox on a Sidman schedule and then transferred them to a Pavlovian harness in a different room for fear conditioning. Avoidance sessions were alternated with conditioning sessions. The experimental group received differential conditioning to two tone stimuli while restrained in the harness. The 5-sec CS+ was followed at a variable ISI (mean of 5 sec) by a 5-sec shock, and CS- was never followed by shock. The control group was simply exposed to the tones without shock. Each stimulus was presented a total of 90 times, at an average ITI of 60 sec.

Testing occurred while the dogs were performing the avoidance response in the shuttlebox under extinction conditions. Each dog received 50 noncontingent, nonreinforced exposures to each tone. The CS+ enhanced responding relative to the pre-CS baseline in the experimental group, but the effect disappeared within the first 20 trials. At no point did the experimental dogs show a reliably different response to CS-. Nor did the controls respond differentially in the presence of either tone. Moreover, relative to baseline, the experimental group responded no differently to CS- than the control group did to the tones.

Thus, in an aversive-to-aversive transfer situation, the effects of CS-, but not of CS+, appeared to depend upon the similarity between place of conditioning and place of testing. However, this assumes that the conditioning parameters were such that had differential conditioning been given in the shuttlebox, CS- would have proved inhibitory. Unfortunately, Desiderato did not run groups that were conditioned and tested in the same place. That the earlier studies in the Pennsylvania laboratory used a somewhat longer ITI (e.g., Rescorla & LoLordo, 1965, used a 90-sec ITI; Rescorla, 1966, and Moscovitch & LoLordo, 1968, used a 150-sec ITI) suggests that Desiderato's parameters may have been less than optimal for replicating previous results (cf. Weisman & Litner, 1971). Moreover, Grossen and Bolles (1968) and Herendeen and Anderson (1968) have reported conditioned inhibitory effects in rats when fear conditioning occurs someplace other than the shuttlebox used for testing.

A second study that is purported to show a situation-specific effect of a conditioned-inhibitory CS was done by Grossen (1971), and involved transfer to an appetitively-motivated instrumental task. In contrast to the previous study, however, a comparison was made between rats conditioned and tested in the same place and rats conditioned and tested in different places. Initially, food-deprived rats were trained to barpress for food reward on a discriminated DRL 30-sec schedule. This schedule was designed to produce a low overall response rate and to restrict responding to the interval during which the S^D was present. At the end of the DRL interval, the houselight (S^D) was turned on. The next barpress operated the pellet dispenser and ended the trial. If the rat failed to respond during the S^D, the trial was ended after 30 sec. Responses that occurred during the DRL interval reset the DRL timer. Training continued until the rat made 110 or fewer responses to obtain 100 pellets.

Conditioning was done either in the operant chamber (with bar removed) or in a 10-in. cube box with a grid floor. Three groups were conditioned in each place, a forward-conditioning group (5-sec ISI), a "safety-signal" group (CS followed by at least 30 sec of no shock), and a random-conditioning group. Ninety trials were given to each group over a three-day period at an average ITI of 1 min. All rats were then returned to the operant chamber to make sure that DRL performance was still at criterion. Test sessions were identical to DRL training sessions except that on 10 trials (randomly determined), the tone came on instead of S^D. Latency to respond following tone onset was the major dependent variable.

In general, response latencies were longer on test trials than on S^D trials. Moreover, they were longer when both conditioning and testing occurred in the operant chamber. Under that condition, the performance of the forward-conditioning group was depressed relative to the random control, whereas that of the safety-signal group was facilitated. However, when conditioning and testing occurred in different places, these three conditions were not different. Thus, both excitatory and inhibitory effects appeared to depend upon the similarity between place of conditioning and place of testing.

Grossen suggested that the effect of the safety signal depended upon fear conditioned to apparatus cues since the safety-signal group that had never been shocked in the test apparatus showed no facilitation. He also suggested that the danger signal (forward-conditioning group) depended upon a "relevant motivational state," because it too had no effect when the conditioning and testing environments differed. He said that excitatory cues had to be relevant to the animal's motivational state in order to have an excitatory effect. Apparently, the rat must expect to be fearful in order to be fearful when the CS comes on.

Grossen argued that differences in place of conditioning and testing are unimportant when the motivational states relevant to conditioning and testing are the same (i.e., aversive-aversive transfer and appetitive-appetitive transfer). When these motivational states differ, conditioning and testing must be done in the same environment in order to obtain excitatory and inhibitory effects. Presumably, in so doing, the test environment acquires the ability to arouse the motivational state necessary to mediate the conditioning effects.

Effects of prior inhibitory conditioning on excitatory and inhibitory conditioning with compound CSs. When a "neutral" stimulus is compounded with a previously established conditioned excitatory stimulus, and the compound is reinforced, the new stimulus often does not become as excitatory as it would if compounded with another neutral stimulus. Kamin (1969) termed this the "blocking" effect, and the model described by Rescorla and Wagner (1972) accounts for it in terms of the discrepancy between the excitatory value of the compound and the maximum associative strength that the US can support. In the case where one of the component stimuli already has excitatory value, this discrepancy is smaller than in the "neutral" condition, hence, subsequent reinforcements are less effective in increasing the associative strength of each component. The model also predicts that if one of the components is an established inhibitor, reinforcement of the compound will result in "superconditioning" to a previously neutral component.

Rescorla (1971) tested this prediction in a CER situation with rats. Following barpress training, several off-the-baseline conditioning sessions were given. In one group, a 1.8-kHz tone was established as a conditioned excitor by being paired with shock on an 80% partial-reinforcement schedule (Group E). A second group (Group I) received the same number of tones and shocks, but in a manner designed to make the tone inhibitory, that is, no shocks occurred during the 2-min CS or during the 2 min after its offset. A third group received an equal number of shocks and tones, but in a completely random fashion (Group R). A fourth group received only US presentations (Group US). All groups

received 60 trials over a five-day period at an average ITI of 10 min.

Following three days of baseline recovery, the tone was presented in compound with a flashing light on a 50% reinforcement schedule. Suppression to the compound developed most rapidly in Group E, which had received prior excitatory training to the tone. The two control groups did not differ, however, both were slower to condition than Group E and faster than Group I. In addition to being part of the "treatment" in this experiment, the second conditioning phase constituted a resistance-to-reinforcement test for the effects of prior conditioning. Relative to random CS-US or US-only pre-exposures, forward conditioning enhanced subsequent conditioning to the compound, whereas explicitly-unpaired CS-US presentations retarded conditioning.

The final test involved examination of the amount of suppression evoked by the light alone in extinction. With respect to the controls (which did not differ), Group E was reliably less suppressed, whereas Group I was reliably more suppressed. Respectively, these differences replicated Kamin's blocking effect, and confirmed the prediction that prior inhibitory training to a stimulus enhances the effectiveness of a reinforcer in establishing excitation to a concurrent stimulus. Wagner (1971) has reported a similar "superconditioning" effect in eyelid conditioning with rabbits.

Rescorla (1971) reported a second experiment in which he attempted to provide evidence for another prediction of the model that is related to the effects of prior inhibitory training. Specifically, he was concerned with the effects of prior inhibitory conditioning on subsequent inhibitory

training. One might suppose that, because prior excitatory conditioning to a component stimulus "blocks" excitatory conditioning to the other element of the compound, prior inhibitory conditioning to a component would similarly block inhibitory training to the other element of the compound. The Rescorla-Wagner model not only suggests that the "neutral" component will become less inhibitory, it predicts that nonreinforcement of the compound will actually make the second component excitatory.

The CER procedure was used in this second experiment. After initial barpress training, conditioning was conducted in two phases and was followed by a "savings" test for excitation to the previously neutral component of the Phase-2 inhibitory compound. Several additional groups were run in an attempt to control for nonassociative effects during each of the conditioning phases. The following table will aid in the description of these conditions:

Group	Phase 1	Phase 2	Test
1	T_1+, T_1L-	T_2L	T_2+,T_2-
2	T_1+, T_1L-	T_2,L	T_2+,T_2-
3	T_1+, T_1-	T_2L	T_2+,T_2-
4	T_1,T_1L	T_2L	T_2+,T_2-

Group 1 was the experimental condition. During the first phase, a 1.8-kHz tone (T_1) was consistently paired with shock, except when presented in compound with a flashing light (TL-). Presumably, this training established L as a conditioned inhibitor. During the second phase, L was presented in compound with an intermittent 250-Hz tone and no shocks were given. If the model is correct, the net associative

value of this compound was negative. Nonreinforcement should have had the effect of positively incrementing the associative value of T_2. During the test phase, suppression would be expected to develop rather rapidly to T_2. Group 2 received identical inhibitory training during Phase 1, but during Phase 2, L and T_2 were presented in an explicitly-unpaired manner. Presumably, this would produce no change in the associative value of T_2. For Group 3, T_1 was presented on a partial reinforcement schedule (50%) during Phase 1. Ignoring the presence of L, this group received the same number of reinforced and nonreinforced T_1 trials as Groups 1 and 2. However, L would not be expected to acquire inhibitory properties, and hence, would have no effect when compounded with T_2 in the second phase. The final group received no shock until the test phase. As in Group 3, L would not be expected to exert an inhibitory effect in Phase 2. The test phase was identical for all groups, with T_2-alone being reinforced on a 50% schedule.

The experimental group tended to suppress slightly more than the other groups during the test phase. The effect was quitesmall, however, and only the difference in the second two-trial block (out of four) was reported to be reliable. No difference appeared in the first block as might have been expected. Rescorla suggested that the small magnitude of the effect may have been due to generalization decrement resulting from the change from off-the-baseline conditioning (Phases 1 and 2) to on-the-baseline (test phase). He also pointed out that, according to the theory, if L and T_2 were equally salient, T_2 could only be expected to become half as excitatory as L was inhibitory. If Phase-1 training were to make L more salient, the excitatory value that T_2 could be expected to attain would be even less.

There are several reasons for questioning the worth of this finding, aside from its small magnitude. There are at least two alternative explanations of these results that do not depend upon the alleged excitatory effect of nonreinforcement. First, the control for inhibitory conditioning during Phase 2 (Group 2) is not appropriate. Mackintosh (1973) has found that latent-inhibition effects are greater when a stimulus is presented alone during pre-exposure than when it has been presented in compound with some other stimulus. One could argue that Rescorla's Phase-1 conditioning had absolutely no effect, and still account for test differences in terms of differential exposure to the to-be-conditioned stimulus in Phase 2. Although Groups 3 and 4 were exposed to the compound during Phase 2, their test performance cannot be compared to Group 1 because they received a different number of Phase-1 exposures to T_1L (Group 3) or shock (Group 4). A better control condition might have been attained if Group 4 had received an equal number of randomly-distributed shocks during Phase 1.

The second alternative interpretation of these results relies upon stimulus generalization. Recall that the conditioned excitor in Phase 1 (T_1) was in the same modality as the stimulus alleged to become excitatory as a result of nonreinforcement in Phase 2 (T_2). If T_1 was differentially excitatory across groups, differences in the excitatory strength of T_2 might be attributable to differences in generalized excitation. The excitatory strength of T_1 should have been the smallest in Group 4, since T_1 was never paired with shock. Although Group 3 received the same number of T_1-US pairings as Groups 1 and 2, one might argue that T_1-alone trials (Group 3) detracted more

from T_1's excitatory strength than did T_1L trials (Groups 1 and 2). The difference between Groups 1 and 2 could have been due to a similar effect involving differential extinction of generalized excitation to T_2 during Phase 2. Thus, Rescorla's "savings" effect could be attributed to differences in generalized excitation.

Baker (1974) attempted to substantiate and extend the findings of Rescorla's (1971) second experiment by maintaining the strength of the original inhibitory component during Phase-2 conditioning. During Rescorla's second phase, the alleged inhibitor (L) was compounded with the neutral stimulus (T_2) in the absence of further T_1+,T_1L- training. According to the Rescorla-Wagner model, nonreinforcement of an established inhibitor should produce a loss in inhibition. Baker suggested that Rescorla's effect might have been stronger had he continued the Phase-1 training during Phase 2. In this way, the established inhibitor would be expected to maintain its strength and the new stimulus would be expected to become even more excitatory.

Baker's procedure was similar to that used for Rescorla's Group 1, with the following major exceptions: (a) a noise stimulus (N) replaced T_1, (b) Phase-1 conditioning was continued during Phase 2, (c) T_2-alone was pre-tested before Phase 2, (d) there were two levels of Phase-2 training (12 days vs 24 days), (3) no control groups were run, (f) all conditioning was done while the rat was barpressing, and (g) the final test consisted simply of nonreinforced tone trials (i.e., there was no "savings" test).

No evidence was found to support the contention that the tone had become excitatory as a result of Phase-2 conditioning. The mean

suppression ratios to the tone were between .47 and .53. There was no difference in suppression between the last pretest day and the final test, nor was there any difference between the group tested after 12 days and the group tested after 24 days.

In an attempt to resolve the discrepancy between his and Rescorla's data, Baker tried to replicate Rescorla's finding. Noting the possibility that Rescorla's control group (Group 2) may actually have been retarded due to latent inhibition, Baker ran a control that received identical Phase-1 conditioning, but no Phase-2 conditioning. During a "savings test, he found that the experimental group differed from a group that had received tone-alone presentations during Phase 2 (similar to Rescorla's Group 2). In fact, this difference was reliable in the exact same block as Rescorla's (Block 2). However, there was virtually no difference between the experimental group and the control that had not received the tone during Phase 2, suggesting that the difference between Rescorla's Groups 1 and 2 did not reflect enhanced acquisition in Group 1, but retardation in Group 2. Latent inhibition would seem to account entirely for these results. Baker concluded that the evidence in support of the Rescorla-Wagner model's prediction was equivocal at best, and suggested that the model's treatment of excitation and inhibition as symmetrical processes might require modification.

One other series of experiments has been concerned with the role of inhibitory training to a single stimulus on subsequent inhibitory training to a compound containing that stimulus. In these experiments, however, attention was focused on the weaker prediction offered by the Rescorla-Wagner model, namely, that inhibition to the new element of the

compound would be blocked. Using the CER technique, Suiter and LoLordo (1971) gave a new-learning test for inhibition to the second component following a two-stage conditioning procedure. During the first stage, the experimental group was exposed to explicitly-unpaired presentations of light (L) and shock. The control group received only shock. Next, both groups received explicitly-unpaired presentations of shock and a light-tone (LT) compound. Finally, both groups received forward tone-shock pairings (new-learning test). The experimental group conditioned much faster than the control group, suggesting that prior inhibitory training to L had blocked inhibitory training to T in Phase 2.

Although their first experiment indicated some blocking, there was no control to assess whether the tone had acquired any inhibitory properties (i.e., whether blocking was complete). In fact, if Rescorla's (1971) analysis is correct, the experimental group may have been facilitated in its acquisition of suppression because T was excitatory. In their second experiment, Suiter and LoLordo compared the experimental condition to one which involved an equal number of L trials, but in the absence of any T trials before the test phase. They argued that if the tone were inhibitory for the experimental group, the acquisition of suppression should be retarded with respect to the novel-stimulus control group. They found no difference, and concluded that blocking had been complete. However, even if they had observed a difference in the right direction, it would have been possible to explain it in terms of latent inhibition.

In a final experiment, they attempted to determine whether the results of Experiment 1 had depended upon the order in which the

conditioning treatments were administered. In other words, would inhibitory training to L alone affect the strength of inhibition to T even if the order of L- and LT- training were reversed? Two groups were treated like the two groups in Experiment 1 except that LT- training preceded L- training (experimental group) or US-alone presentations (control group). No differences emerged during subsequent acquisition of suppression to T, supporting the view that L- training had to precede LT- training in order to block inhibition to T. Although no control was provided in Experiment 3, comparison with the test data of the experimental groups in the previous experiments suggests that the tone was inhibitory (i.e., acquisition was retarded) in both groups. Suiter and LoLordo concluded that the outcomes of their experiments were in agreement with the general predictions offered by the Rescorla-Wagner model.

APPENDIX B

STIMULUS SEQUENCES, DATA, AND ANALYSIS OF VARIANCE SUMMARY TABLES FOR EXPERIMENT 1

Table B1

Stimulus Sequences used during Phase-1 and Phase-2 Conditioning*

Day(s)												
	Phase 1: On-the-baseline											
12 - 14	A	B	B	A		B	A		A	B		
15 - 18	A	B		B	A		B	A		A	B	
	Phase 1: Off-the-baseline											
19	B	A	A	B	A	A	B	B	B	A	A	B
20	A	B	B	A	B	B	A	A	A	B	B	A
21	A	B	B	A	B	B	A	A	A	B	B	A
22	B	A	A	B	A	A	B	B	B	A	A	B
23	B	A	A	B	A	A	B	B	B	A	A	B
24	A	B	B	A	B	B	A	A	A	B	B	A
	Phase 2: On-the-baseline											
28 - 29	A	B	AX	AX		AX	B	A	AX			
30 - 31	AX	AX	B	A		AX	A	B	AX			
32 - 33	B	A	AX	AX		AX	AX	A	B			
34 - 35	AX	A	B	AX		AX	B	A	AX			
36 - 37	AX	AX	A	B		B	A	AX	AX			
38 - 39	AX	B	A	AX		AX	A	B	AX			
40 - 41	A	B	AX	AX		AX	B	A	AX			
42 - 43	AX	AX	B	A		A	B	AX	AX			
44 - 45	AX	A	B	AX		B	A	AX	AX			
46 - 47	AX	AX	A	B		AX	B	A	AX			
	Phase 2: Off-the-baseline											
48	AX	AX	A	B	AX	B	A	AX	AX	AX	B	A
49	A	B	AX	AX	AX	AX	AX	B	A	AX	AX	AX
50	AX	AX	AX	A	B	AX	AX	B	A	AX	AX	AX
51	AX	AX	B	A	AX	A	B	AX	AX	AX	A	B
52	A	B	AX	AX	AX	A	B	AX	AX	B	A	AX
53	AX	AX	AX	B	A	AX	AX	A	B	AX	AX	AX
54	B	A	AX	AX	AX	AX	AX	A	B	AX	AX	AX
55	B	A	AX	AX	AX	B	A	AX	AX	A	B	AX
	Phase 2: On-the-baseline											
58 - 59	AX	AX	B	A		AX	A	B	AX			

* A flashing light or tone was used for A and X (counterbalanced); A buzzer was always used for B. For half of the rats in each subgroup, the order of A and B in the above sequence was reversed.

Table B2

Mean body weights (gm)

Group	Subject	80%	Phase							
			A*	B	C	D	E	F	G	H
Hi	1	174	178	176	170	171	171	169	173	169
	2	179	181	186	174	181	176	176	175	172
	3	166	170	171	164	163	165	167	168	161
	4	170	173	175	170	169	169	169	168	167
	5	171	177	177	170	170	169	169	170	166
	6	166	174	173	166	167	166	165	167	161
	7	178	182	181	174	172	175	174	173	171
	8	165	173	170	164	167	163	165	164	160
Lo	1	178	182	184	174	177	175	177	174	172
	2	170	177	175	171	168	169	169	168	165
	3	178	182	183	176	175	176	174	174	173
	4	187	190	189	183	187	185	187	182	181
	5	189	193	194	184	189	186	185	184	183
	6	162	167	169	163	163	161	162	162	157
	7	177	181	183	174	171	174	175	174	171
	8	166	178	174	165	164	164	161	163	160

*Key: A = Shaping and VI training (Days 1 - 9)
B = Preconditioning exposures (Days 10 - 11)
C = Phase-1 conditioning (Days 12 - 24)
D = VI recovery sessions (Days 25 - 27)
E = Phase-2 conditioning (Days 28 - 55)
F = VI recovery sessions (Days 56 - 57)
G = Phase -2 conditioning (Days 58 - 59)
H = New- learning test phase (Days 60 - 72)

Analyses:

Initial Body Weights

Source	df	ms	F
Groups	1	90.25	1.5
error(b)	14	59.98	

Groups x Phases

Groups	1	634.57	1.7
error(b)	1	367.20	
Phases	7	229.20	86.5
Groups x Phases	7	2.27	0.9
error(w)	98	2.65	

Table B3

Response Baselines: Mean number of responses per 2-min pre-CS interval

Group	Subject	Phase												
		A*	B	C1	C2	C3	C4	D	E1	E2	E3	E4	E5	F	G
Hi	1	16	20	28	38	39	33	30	44	44	55	63	71	37	51
	2	24	23	12	1	20	28	21	24	33	42	48	42	23	22
	3	30	29	36	36	41	43	37	66	70	77	75	77	46	65
	4	24	26	20	33	35	22	27	35	34	36	35	36	35	46
	5	17	13	14	18	23	15	15	17	18	24	22	26	21	32
	6	16	15	15	26	29	19	23	22	22	20	22	22	23	19
	7	36	46	38	56	23	28	39	44	57	60	60	77	50	86
	8	17	24	15	22	33	29	37	31	25	22	35	35	25	21
Lo	1	27	22	24	35	40	33	23	27	26	34	39	38	34	35
	2	27	25	18	2	34	45	35	40	44	43	43	66	45	59
	3	15	19	19	21	26	26	20	31	31	31	25	22	19	31
	4	14	13	6	16	16	19	11	13	12	13	14	13	12	13
	5	13	9	17	23	21	19	10	10	13	14	13	16	16	17
	6	6	10	11	15	23	21	18	19	24	45	33	42	27	29
	7	12	14	19	16	19	16	13	15	18	16	13	15	11	16
	8	20	18	13	26	37	22	18	30	19	21	21	19	16	22

Group	Subject	H	I1	I2	I3	J1	J2	J3
Hi	1	47	73	80	45	53	78	65
	2	22	33	27	21	26	29	22
	3	44	58	49	42	32	42	46
	4	42	46	49	41	37	39	46
	5	36	30	29	29	25	21	21
	6	22	27	29	24	24	19	16
	7	84	95	99	88	99	70	86
	8	16	21	20	23	31	29	21
Lo	1	31	23	32	30	34	39	31
	2	55	67	41	44	49	33	32
	3	22	22	17	26	25	23	28
	4	9	11	10	12	11	10	11
	5	13	16	24	25	7	22	27
	6	14	27	20	26	29	42	47
	7	16	21	24	15	13	14	13
	8	23	25	25	20	20	22	24

*Key: A = Last VI day (Day 9)
B = Preconditioning exposures (Days 10 - 11)
C = Phase-1 conditioning (two-trial blocks)(Days 12 - 18)
D = VI recovery sessions (Days 25 - 27)

Table B3 (cont.)

E = Phase-2 differential conditioning (four-day blocks) (Days 28 - 47)
F = VI recovery sessions (Days 56 - 57)
G = Phase-2 differential conditioning (Days 58 - 59)
H = Exposures to X (Day 60)
I = New-learning acquisition (two-day blocks)(Days 61 - 66)
J = New-learning extinction (two-day blocks)(Days 67 - 72)

Analyses:	Source	df	ms	F
A.	Groups (G)	1	132.2	2.4
	error (b)	14	55.1	
B.	G	1	272.3	4.0
	err(b)	14	68.4	
C.	G	1	449.4	2.0
	err(b)	14	224.8	
	Blocks (B)	3	267.1	4.4
	G x B	3	44.0	0.7
	err (w)	42	60.6	
D.	G	1	410.1	5.8
	err(b)	14	70.1	
E.	G	1	5232.6	4.2
	err(b)	14	1253.7	
	B	4	221.9	6.2
	G x B	4	43.8	1.2
	err(w)	56	35.6	
F.	G	1	400.0	3.0
	err(b)	14	134.4	
G.	G	1	900.0	2.3
	err(b)	14	398.1	
H.	G	1	1056.3	3.1
	err(b)	14	339.6	
I.	G	1	4820.0	4.1
	err(b)	14	1166.9	
	B	2	126.6	2.8
	G x B	2	105.3	2.4
	err(w)	28	44.6	
J.	G	1	2867.5	2.9
	err(b)	14	990.3	
	B	2	7.8	0.1
	G x B	2	13.3	0.2
	err(w)	28	56.8	

Table B4

Mean suppression ratios (x 1000) on Preconditioning exposure Days 10 - 11

Group	Subject	Stimulus			
		A	X	AX	B
Hi	1	492	553	365	549
	2	500	474	474	704
	3	507	482	427	446
	4	599	350	470	592
	5	500	629	412	573
	6	474	497	397	644
	7	441	565	478	541
	8	460	550	386	593
Lo	1	470	470	493	483
	2	560	361	456	580
	3	415	415	500	490
	4	569	568	536	638
	5	515	419	562	505
	6	389	554	690	842
	7	409	500	487	471
	8	367	694	397	580

Groups x Stimulus-type analysis:

Source	df	ms	F
Groups (G)	1	1056.3	0.1
err(b)	14	7507.9	
Stimulus-type (S)	3	37244.7	5.3
G x S	3	12154.8	1.7
err(w)	42	6997.1	

Table B5

Mean suppression ratios (x 1000) in two-trial blocks during Phase-1 conditioning (Days 12 - 18)

Group	Subject	Stimulus:	A				B			
		Block:	1	2	3	4	1	2	3	4
Hi	1		525	000	000	000	380	000	016	000
	2		315	000	000	000	593	000	000	000
	3		526	042	000	011	462	025	000	010
	4		358	000	000	000	220	000	000	000
	5		515	189	191	025	376	034	000	050
	6		423	508	218	000	613	341	044	198
	7		565	287	000	064	633	015	084	015
	8		499	630	234	000	604	518	040	000
Lo	1		528	000	000	017	411	028	000	000
	2		543	000	022	000	581	074	012	024
	3		581	024	077	031	503	044	203	000
	4		588	027	000	000	514	023	000	000
	5		502	530	270	324	617	476	147	124
	6		629	326	107	000	542	366	000	042
	7		451	446	286	137	472	022	120	048
	8		813	173	032	000	586	021	013	000

Groups x Stimulus-type x Blocks analysis:

Source	df	ms	F
Groups (G)	1	33832.5	0.6
err (b)	14	57006.9	
Stimulus-type (S)	1	41508.0	6.2
G x S	1	2784.4	0.4
err (S)	14	6743.3	
Blocks (B)	3	1551863.6	81.5
G x B	3	8784.1	0.5
err (B)	42	19044.8	
B x S	3	7796.4	1.2
G x S x B	3	5361.8	0.8
err (SB)	42	6718.5	

Table B6

Mean suppression ratios (x 1000) in four-day blocks during Phase-2 conditioning (Days 28 - 47)

Stimulus:		A					B					AX				
Block:		1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Group	Subject															
Hi	1	026	163	083	022	016	010	012	014	004	003	036	210	460	398	530
	2	029	079	096	044	045	070	031	050	022	064	003	085	061	085	108
	3	008	024	011	014	025	022	023	052	043	032	000	016	025	003	058
	4	007	008	007	105	017	018	000	000	008	010	006	031	146	284	363
	5	000	015	080	000	009	184	140	262	128	188	030	138	481	529	491
	6	025	122	117	033	012	146	155	071	085	293	041	187	453	556	585
	7	110	028	073	033	008	099	065	083	098	081	047	129	403	618	618
	8	108	151	020	016	115	068	063	054	059	086	152	069	398	475	532
Lo	1	130	028	071	072	173	091	000	043	014	025	034	114	125	191	410
	2	067	162	214	190	247	022	009	029	013	025	254	396	474	489	425
	3	133	221	165	213	326	000	058	083	096	054	288	463	556	577	633
	4	035	042	030	047	050	000	020	039	000	014	008	000	045	094	371
	5	484	467	408	469	322	397	443	414	463	160	294	484	412	489	481
	6	071	040	012	144	103	000	017	011	019	030	173	166	465	651	637
	7	231	034	050	094	034	050	000	000	000	000	016	004	118	068	074
	8	061	142	130	013	181	051	020	000	060	021	050	145	132	263	262

Groups x Stimulus-type x Blocks analysis:

Source	df	ms	F
Groups (G)	1	130107.3	0.9
err (b)	14	142100.7	
Stimulus-type (S)	2	871588.6	24.2
G x S	2	59264.6	1.6
err (S)	28	36017.1	
Blocks (B)	4	94480.6	14.7
G x B	4	5095.6	0.8
err (B)	56	6424.1	
S x B	8	97963.4	24.8
G x S x B	8	9747.8	2.5
err (SB)	112	3951.5	

Table B7

Mean suppression ratios (x 1000) on final Phase-2 conditioning days (Days 58 - 59)

Group	Subject	Stimulus		
		A	B	AX
Hi	1	000	004	044
	2	138	114	258
	3	018	006	004
	4	000	000	008
	5	000	108	491
	6	039	022	617
	7	022	030	595
	8	000	036	536
Lo	1	091	013	406
	2	222	000	309
	3	215	101	530
	4	088	000	404
	5	250	520	497
	6	000	068	591
	7	000	000	007
	8	027	091	467

Groups x Stimulus-type analysis:

Source	df	ms	F
Groups (G)	1	68026.0	1.90
err (b)	14	36145.9	
Stimulus-type (S)	2	450953.4	22.50
G x S	2	789.1	0.04
err (w)	28	20042.1	

Table B8

Mean suppression ratios (x 1000) to X during New-learning test phase (Days 60 - 72)

Group	Subject	Blocks:	P*	A1	A2	A3	E1	E2	E3
Hi	1		598	545	512	408	172	229	233
	2		480	439	216	015	132	289	392
	3		463	455	225	014	112	056	185
	4		518	530	380	138	171	239	354
	5		462	417	052	022	031	059	233
	6		561	474	108	137	137	360	424
	7		605	563	173	036	101	094	189
	8		613	524	335	058	184	353	467
Lo	1		516	434	417	219	187	160	172
	2		504	458	212	195	321	481	508
	3		485	466	531	291	390	424	480
	4		565	437	093	028	101	100	130
	5		646	585	498	444	630	499	582
	6		743	472	193	070	078	149	303
	7		509	396	196	036	021	065	118
	8		523	501	326	189	233	417	392

*Key: P = Pretest exposures (Day 60)
A = New-learning CER acquisition (two-day blocks)(Days 61 - 66)
E = New-learning CER extinction (two-day blocks) (Days 67 - 72)

Analyses:	Source	df	ms	F
P vs Initial response to X (see Table B4)	Groups (G)	1	162.0	0.01
	err(b)	14	8940.9	
	Test Session (T)	1	15753.1	2.52
	G x T	1	3003.1	0.48
	err (w)	14	6240.5	
A.	G	1	16688.0	0.49
	err(b)	14	33576.9	
	Blocks (B)	2	459678.1	73.60
	G x B	2	12345.4	1.97
	err(w)	28	6243.5	
E.	G	1	63438.0	1.02
	err(b)	14	61608.2	
	B	2	73208.3	20.37
	G x B	2	7998.5	2.22
	err(w)	28	3592.3	

Table B9

Trials to criterion during New-learning acquisition

Group	Subject:	1	2	3	4	5	6	7	8
Hi		24	9	11	14	9	10	10	14
Lo		21	14	23	11	25	12	8	15

Table B10

Median latency to first post-CS response during Phase-1 conditioning (Days 12 - 18)

Subject	Group Hi		Group Lo	
	US intensity .65 mA	1.3 mA	US intensity .65 mA	1.3 mA
1	9.5	23.9	11.6	23.6
2	28.1	28.7	12.3	27.5
3	7.0	7.6	5.0	4.1
4	12.6	16.8	18.4	16.1
5	12.0	13.0	11.5	20.2
6	4.3	9.0	15.4	27.1
7	4.5	6.5	4.1	2.6
8	7.2	6.0	3.6	5.1

Groups x US-intensity analysis:

Source	df	ms	F
Groups (G)	1	3.95	0.03
err(b)	14	117.56	
US-Intensity (I)	1	157.75	8.47
G x I	1	10.07	0.54
err(w)	14	18.62	

Table B11

Median latency (sec) to first post-CS response in two-day blocks as a function of US intensity during new-learning acquisition (Days 61 - 66)

US intensity (mA):		0.0			.65			1.3	
Blocks:	1	2	3	1	2	3	1	2	3
Group	Subject								
Hi	1	1.9	1.2	7.3	16.1	10.4	3.3	13.8	11.6	3.3
	2	1.9	18.0	120.0	2.4	2.6	3.5	4.1	2.1	120.0
	3	4.2	6.8	5.6	15.0	3.5	1.7	8.1	2.7	11.8
	4	2.3	2.2	3.3	5.4	5.8	1.8	11.3	4.4	21.0
	5	7.6	10.3	10.2	6.1	2.1	1.2	3.8	2.2	2.2
	6	2.6	1.5	3.8	5.0	1.2	1.7	10.1	1.9	1.2
	7	2.3	11.6	3.0	6.5	4.3	1.9	2.7	6.0	16.9
	8	3.3	3.3	3.9	6.9	5.5	5.9	5.2	10.2	4.0
Lo	1	5.8	11.9	5.6	10.5	2.4	15.4	7.4	2.5	2.6
	2	3.3	7.0	2.3	15.1	7.7	2.2	12.6	5.1	0.1
	3	6.4	9.0	5.9	4.9	0.3	5.3	4.3	0.2	29.4
	4	50.9	120.0	104.9	32.8	35.1	7.0	31.5	65.9	4.6
	5	6.7	4.0	3.5	4.2	3.9	1.2	4.2	3.1	5.9
	6	4.9	21.5	10.8	12.6	2.5	3.8	11.5	7.0	3.8
	7	2.7	2.4	3.9	2.3	1.4	1.6	1.9	2.1	0.7
	8	7.6	3.8	6.4	3.6	5.4	5.9	5.1	4.2	5.1

Groups x US-intensity x Blocks analysis:

Source	df	ms	F
Groups (G)	1	227.1	0.2
err (b)	14	1484.8	
US-Intensity (I)	2	616.2	1.7
G x I	2	284.0	0.8
err (I)	28	365.1	
Blocks (B)	2	209.8	0.5
G x B	2	556.8	1.2
err (B)	28	465.6	
I x B	4	334.2	2.5
G x I x B	4	154.4	1.1
err (IB)	56	136.4	

Table B12

Median latency (sec) to first post-CS response in two-day blocks during new-learning extinction (Days 67 - 72)

Group	Subject	Block		
		1	2	3
Hi	1	3.7	1.6	2.9
	2	23.4	2.2	2.4
	3	5.3	3.4	3.7
	4	1.7	3.2	1.5
	5	3.4	2.1	2.9
	6	7.8	6.2	5.1
	7	2.4	2.0	1.7
	8	9.1	6.6	5.0
Lo	1	3.9	3.9	1.9
	2	2.7	1.9	2.0
	3	2.4	3.4	3.2
	4	15.2	8.6	14.3
	5	2.3	1.8	3.4
	6	8.9	3.0	2.2
	7	2.4	2.4	3.9
	8	4.6	6.4	7.5

Groups x Blocks analysis:

Source	df	ms	F
Groups (G)	1	0.2	0.01
err(b)	14	29.2	
Blocks (B)	2	30.4	2.91
G x B	2	12.6	1.20
err(w)	28	10.4	

Table B13

Median latency (sec) to first response during CS in two-day blocks during new-learning acquisition and extinction (Days 61 - 72)

Group	Subject	Block					
		A1*	A2	A3	E1	E2	E3
Hi	1	4.1	2.2	1.4	3.8	5.6	6.9
	2	7.3	34.5	120.0	82.2	8.0	10.1
	3	3.1	7.2	80.1	102.6	5.5	26.2
	4	3.5	1.9	34.0	64.5	37.1	6.6
	5	2.1	49.1	1.9	4.4	2.6	2.4
	6	2.5	3.7	4.4	4.5	9.4	7.9
	7	2.0	3.2	3.4	1.4	3.8	1.9
	8	5.9	1.7	2.7	3.8	3.9	10.5
Lo	1	13.7	12.9	27.1	26.6	12.0	55.9
	2	0.8	5.0	19.8	6.0	3.7	2.8
	3	3.6	7.6	24.2	8.2	11.8	7.2
	4	17.5	120.0	120.0	10.7	52.2	63.9
	5	4.4	2.6	2.7	5.1	3.6	3.0
	6	2.4	2.1	8.7	6.7	1.8	2.9
	7	5.3	68.5	120.0	120.0	94.8	77.4
	8	5.8	2.1	14.1	36.7	6.2	8.2

*A = Acquisition; E = Extinction

Analyses	Source	df	ms	F
A.	Groups (G)	1	1095.4	0.6
	err(b)	14	1990.3	
	Blocks (B)	2	3927.2	5.8
	G x B	2	146.0	0.2
	err(w)	28	677.3	
E.	Groups (G)	1	933.7	0.5
	err(b)	14	2009.2	
	Blocks (B)	2	929.4	2.3
	G x B	2	674.9	1.7
	err(w)	28	408.4	

APPENDIX C

STIMULUS SEQUENCES, DATA, AND ANALYSIS OF VARIANCE SUMMARY TABLES FOR EXPERIMENT 2

Table C1

Stimulus Sequences used in Experiment 2*

Day										
10	A	B	B	A	AX	B	A	A	B	AX
11	AX	A	B	AX	AX	B	A	AX	A	B
12	B	A	AX	AX	AX	A	B	AX	AX	AX
13	AX	B	A	A	B	AX	AX	B	A	AX
14	AX	AX	B	A	A	B	B	A	AX	AX
15	A	B	AX	AX	AX	AX	AX	AX	B	A
16	AX	B	A	AX	A	B	AX	AX	A	B
17	AX	AX	AX	B	A	AX	A	B	AX	AX
18	B	A	AX	AX	AX	AX	AX	A	B	AX
19	AX	B	A	A	B	AX	AX	AX	A	B
20	A	B	B	A	AX	AX	A	B	AX	AX
21	AX	A	B	AX	B	A	AX	AX	A	B
22	A	B	AX	AX	AX	AX	B	A	AX	AX
23	AX	AX	B	A	AX	A	B	B	A	AX
24	AX	A	B	AX	AX	B	A	AX	AX	AX

* A flashing light was always used for X; a tone or buzzer was used for A and B (counterbalanced).

Table C2

Mean body weights (gm)

Group	Subject	80%	Phase					
			A*	B	C	D	E	F
Hi	1	182	181	180	182	182	180	182
	2	183	182	177	182	182	178	181
	3	198	196	194	199	196	196	197
	4	199	197	195	200	196	198	200
	5	188	188	182	189	188	186	187
	6	196	193	195	196	197	196	196
	7	182	180	177	183	183	182	182
	8	185	181	185	185	187	185	184
Lo	1	186	187	184	188	187	188	186
	2	190	190	194	191	189	188	190
	3	188	186	188	187	186	187	189
	4	190	187	193	190	189	190	190
	5	181	183	172	182	182	182	182
	6	188	189	189	189	186	185	188
	7	184	182	190	185	185	183	184
	8	182	180	182	183	180	182	182
Random	1	183	182	182	184	184	179	184
	2	170	173	169	173	171	170	171
	3	182	181	183	183	181	179	182
	4	194	192	198	193	193	190	195
	5	180	181	176	182	181	184	181
	6	190	188	190	190	189	192	190
	7	185	185	182	186	182	185	185
	8	190	191	184	191	191	192	191

*Key: A = Shaping and VI training (Days 1 - 8)
B = Preconditioning exposures (Day 9)
C = Conditioning (Days 10 - 24)
D = VI recovery sessions (Days 25 - 27)
E = Post-conditioning test (Days 28 - 29)
F = New-learning phase and final test (Days 30 - 42)

Analyses	Source	df	ms	F
Initial weights	Groups	2	48.4	1.2
	err(b)	21	40.7	
Groups (G) x Phases (P)	G	2	166.9	0.7
	err(b)	21	233.8	
	P	5	14.4	3.5
	G x P	10	4.5	1.1
	err(w)	105	4.1	

Table C3

Response baselines: Mean number of responses per 2-min pre-CS interval

Group	Subject	Phase										
		A*	B	C	D	E1	E2	E3	F1	F2	F3	G
Hi	1	13	10	11	12	15	14	19	17	15	14	15
	2	19	24	12	16	19	13	19	17	17	18	13
	3	38	26	22	33	23	24	30	31	31	28	27
	4	41	35	26	35	41	44	56	59	40	30	27
	5	28	32	17	35	49	44	45	48	43	37	33
	6	34	30	30	42	40	32	29	40	52	62	60
	7	48	49	49	55	82	89	75	70	61	48	33
	8	22	15	20	27	22	25	32	23	30	39	37
Lo	1	30	32	21	27	30	28	24	27	24	25	24
	2	11	6	10	12	8	6	5	7	8	7	6
	3	23	16	16	18	19	22	25	24	22	24	25
	4	25	26	23	42	32	40	38	51	52	37	43
	5	36	52	60	71	85	78	87	62	54	72	99
	6	50	47	39	25	29	20	17	38	21	22	20
	7	17	16	11	21	16	13	18	18	21	19	20
	8	70	61	51	71	55	61	46	57	68	65	79
Random	1	9	17	10	20	22	19	16	23	19	15	14
	2	25	41	12	22	17	20	16	28	35	25	21
	3	13	11	12	12	9	9	8	11	10	13	11
	4	5	5	7	9	6	6	7	7	8	7	6
	5	24	33	29	50	26	37	42	39	24	31	36
	6	25	32	21	33	31	29	26	27	22	27	22
	7	25	28	19	23	25	30	23	34	24	27	26
	8	19	25	26	50	42	37	51	58	61	67	67

*Key: A = Last two VI training days (Days 7 - 8)
B = Preconditioning exposures (Day 9)
C = VI recovery sessions (Days 25 - 27)
D = Post-conditioning test (Days 28 - 29)
E = New-learning acquisition (two-day blocks)(Days 30 - 35)
F = New-learning extinction (two-day blocks)(Days 36 - 41)
G = Final test (Day 42)

Analyses:	Source	df	ms	F
A.	Groups (G)	2	492.8	2.6
	err(b)	21	192.4	
B.	G	2	128.4	0.6
	err(b)	21	225.8	

Table C3 (cont.)

Analysis:	Source	df	ms	F
C.	Groups (G)	2	282.5	1.5
	err(b)	21	189.8	
D.	G	2	144.7	0.4
	err(b)	21	327.3	
E.	G	2	1213.0	1.0
	err(b)	21	1242.6	
	Blocks (B)	2	2.3	0.1
	G x B	4	10.7	0.4
	err(w)	42	23.8	
F.	G	2	607.6	0.6
	err(b)	21	954.8	
	B	2	42.9	1.0
	G x B	4	4.8	0.1
	err(w)	42	45.1	
G.	G	2	407.8	0.7
	err(b)	21	549.0	

Table C4

Mean suppression ratios (x 1000) on Preconditioning exposure Day 9

Group	Subject	Stimulus		
		A	B	X
Hi	1	077	667	280
	2	150	500	327
	3	551	743	520
	4	500	788	470
	5	323	426	333
	6	476	522	490
	7	548	474	240
	8	610	684	500
Lo	1	302	500	339
	2	722	467	625
	3	551	643	286
	4	435	421	485
	5	433	505	395
	6	273	430	384
	7	400	333	344
	8	173	529	174
Random	1	500	483	250
	2	281	274	279
	3	545	500	556
	4	533	727	500
	5	379	537	238
	6	439	438	458
	7	455	404	447
	8	400	524	405

Groups x Stimulus-type analysis:

Source	df	ms	F
Groups (G)	2	11691.1	0.4
err(b)	21	32365.6	
Stimulus-type (S)	2	116683.1	10.3
G x S	4	14721.5	1.3
err(w)	42	11323.4	

Table C5

Mean suppression ratios (x 1000) to A, B, and AX on Post-conditioning Test Days 28 - 29

	Stimulus:	A		B		AX	
	Day:	1	2	1	2	1	2
Group	Subject						
Hi	1	000	077	500	316	000	125
	2	000	267	000	290	143	500
	3	194	111	114	421	293	400
	4	222	060	013	130	061	270
	5	033	000	034	571	000	065
	6	073	017	064	588	041	148
	7	017	120	507	090	308	120
	8	032	167	182	217	227	100
Lo	1	121	553	040	028	509	500
	2	516	412	000	211	500	368
	3	680	361	091	167	485	500
	4	542	438	026	000	487	547
	5	500	277	000	045	136	523
	6	725	214	000	071	468	606
	7	400	475	000	000	488	500
	8	049	484	013	015	425	347
Random	1	640	475	192	038	452	471
	2	030	514	233	032	528	806
	3	481	647	077	000	414	474
	4	450	438	143	111	611	500
	5	356	467	161	016	479	573
	6	231	470	038	065	429	422
	7	523	536	071	000	478	508
	8	227	485	000	016	578	753

Groups x Stimulus-type x Days analysis:

Source	df	ms	F
Groups (G)	2	408780.4	35.0
err(b)	21	11681.7	
Stimulus-type (S)	2	898551.5	56.8
G x S	4	492140.8	31.1
err(S)	42	15811.4	
Days (D)	1	87418.8	4.6
G x D	2	15093.2	0.8
err(D)	21	19020.3	
S x D	2	2578.6	0.1
G x S x D	4	35483.7	1.7
err(SD)	42	21097.5	

Table C6

Mean suppression ratios (x 1000) to X during New-learning test phase (Days 30 - 41)

Group	Subject	Blocks: P*	A1	A2	A3	E1	E2	E3
Hi	1	458	442	395	091	242	485	605
	2	459	380	307	341	400	383	459
	3	528	512	099	092	231	328	487
	4	421	444	126	005	002	037	140
	5	347	368	082	045	139	329	446
	6	463	493	091	058	010	125	306
	7	443	419	365	095	219	325	454
	8	510	539	117	008	102	085	231
Lo	1	520	511	431	327	451	450	500
	2	514	503	502	171	391	526	445
	3	475	490	178	150	225	374	392
	4	420	420	439	070	103	269	380
	5	472	474	118	030	064	111	285
	6	402	514	260	190	160	343	388
	7	488	492	301	106	156	330	457
	8	472	426	459	209	340	361	449
Random	1	428	413	368	235	351	388	432
	2	407	504	394	550	488	449	488
	3	560	566	325	091	310	446	447
	4	549	460	425	081	418	401	457
	5	476	473	188	100	228	325	333
	6	481	454	219	199	339	412	361
	7	477	401	033	014	020	133	257
	8	472	523	209	108	185	237	357

*Key: P = Mean of first three trials on Day 30
A = New-learning CER acquisition (two-day blocks)(Days 30 - 35)
E = New-learning CER extinction (two-day blocks)(Days 36 - 41)

Analysis:	Source	df	ms	F
P.	Groups (G)	2	1549.3	0.6
	err(b)	21	2503.8	
Planned comparison: Hi vs Lo (Acquisition)	G	1	71842.7	5.6
	err(b)	14	12851.6	
	Blocks (B)	2	466217.6	49.8
	G x B	2	12386.4	1.3
	err(w)	28	9352.8	

Table C6 (cont.)

Analysis:	Source	df	ms	F
Planned comparison: Hi vs Lo (Extinction)	G	1	39675.0	0.9
	err(b)	14	45959.2	
	B	2	158922.6	45.8
	G x B	2	4229.3	1.2
	err(w)	28	3469.8	

Table C7

Trials to criterion during New-learning acquisition

Group	Subject:	1	2	3	4	5	6	7	8
Hi		13	8	9	11	10	10	17	10
Lo		19	19	10	18	10	13	13	19
Random		18	14	15	15	13	13	8	13

Table C8

Mean suppression ratios (x 1000) to A, B, AX, and X on Final Test Day 42

Group	Subject	Stimulus			
		A	B	AX	X
Hi	1	121	519	318	542
	2	215	178	125	553
	3	308	396	361	467
	4	250	063	009	000
	5	150	100	054	333
	6	044	008	0 8	407
	7	034	048	027	508
	8	000	047	000	252
Lo	1	446	050	393	579
	2	558	143	443	542
	3	433	053	290	454
	4	220	024	222	324
	5	155	004	036	189
	6	111	034	209	302
	7	410	000	395	482
	8	340	040	390	507
Random	1	610	220	417	584
	2	428	216	361	599
	3	491	000	465	350
	4	634	225	643	515
	5	486	076	167	175
	6	518	349	519	409
	7	521	000	553	338
	8	548	006	588	406

Groups x Stimulus-type analysis:

Source	df	ms	F
Groups (G)	2	281346.8	6.0
err(b)	21	47142.3	
Stimulus-type (S)	3	369696.1	36.8
G x S	6	101241.6	10.1
err(w)	63	10045.3	

Table C9

Median latency (sec) to first response during the CS in two-day blocks during new-learning acquisition and extinction (Days 30 - 41)

Group	Subject	Block					
		A1*	A2	A3	E1	E2	E3
Hi	1	13.8	27.1	119.9	14.1	15.0	4.5
	2	12.2	17.8	21.2	8.6	10.6	11.7
	3	3.5	9.2	95.7	21.0	6.4	2.1
	4	5.7	68.2	120.0	120.0	77.9	65.6
	5	12.5	113.8	62.1	83.9	21.0	5.2
	6	2.1	79.1	120.0	120.0	39.1	3.5
	7	2.2	3.5	9.3	3.0	3.7	2.4
	8	2.6	4.2	120.0	76.0	105.7	4.0
Lo	1	4.6	2.7	17.5	6.7	3.1	3.3
	2	6.4	16.3	94.4	47.4	18.0	13.5
	3	5.9	21.2	60.9	41.9	3.6	3.8
	4	6.9	5.0	67.5	45.7	27.1	12.3
	5	0.8	57.9	112.4	4.9	2.7	4.7
	6	3.0	20.3	36.3	5.2	9.0	8.1
	7	4.4	5.7	96.6	18.1	5.7	10.5
	8	5.5	2.2	2.2	5.6	6.2	4.6
Random	1	5.7	4.4	28.4	8.8	2.9	13.0
	2	8.4	15.9	4.9	0.9	6.0	7.1
	3	12.6	26.3	120.0	34.1	15.7	6.8
	4	8.6	16.2	105.2	8.3	5.4	6.2
	5	16.3	17.9	50.2	2.8	5.0	4.5
	6	6.1	2.7	46.8	8.8	8.3	10.9
	7	7.6	63.5	120.0	114.0	25.9	5.2
	8	5.1	28.4	64.7	15.1	5.4	1.7

*Key: A = Acquisition; E = Extinction

Analyses:	Source	df	ms	F
A.	Groups (G)	2	1636.8	1.4
	err(b)	21	1204.8	
	Blocks (B)	2	25753.8	34.4
	G x B	4	365.5	0.5
	err(w)	42	748.1	
E.	G	2	3584.4	2.8
	err(b)	21	1275.2	
	B	2	3848.3	9.2
	G x B	4	555.6	1.3
	err(w)	42	419.5	

Table C10

Median latency (sec) to first post-CS response in two-day blocks as a function of US intensity during new-learning acquisition (Days 30 - 35)

US intensity (mA):		0.0			0.4			1.6		
Blocks:		1	2	3	1	2	3	1	2	3
Group	Subject									
Hi	1	7.4	8.5	8.9	20.1	6.9	4.1	33.7	9.4	6.8
	2	5.0	20.8	9.1	9.5	4.0	11.5	9.7	8.7	8.9
	3	3.0	36.8	34.3	11.2	6.5	5.5	11.1	9.7	3.9
	4	2.3	10.7	25.5	3.8	2.1	1.2	21.9	5.8	8.8
	5	5.0	34.8	51.2	10.6	5.9	4.1	9.3	4.4	3.2
	6	3.4	51.2	28.5	19.0	19.6	2.2	28.1	14.8	7.1
	7	2.2	3.2	79.4	14.7	3.3	3.8	5.5	9.9	8.5
	8	3.9	45.9	14.6	3.9	6.3	5.9	4.9	8.7	3.8
Lo	1	4.8	2.8	8.8	15.6	9.2	6.0	12.6	8.3	2.6
	2	13.0	9.7	11.9	13.3	18.2	9.1	21.1	23.4	39.2
	3	3.3	40.1	8.0	17.8	26.5	11.9	22.1	6.0	9.1
	4	2.0	6.1	11.4	22.0	7.0	7.3	32.2	7.3	33.1
	5	3.4	66.8	55.2	11.6	60.6	25.8	32.4	77.9	17.9
	6	13.3	27.3	46.2	15.1	6.5	3.4	18.3	8.4	3.7
	7	5.7	68.9	30.4	14.9	24.0	52.7	24.2	18.0	11.4
	8	1.7	1.8	6.1	27.6	32.1	24.8	0.2	33.3	11.2
Random	1	4.2	7.0	9.6	23.6	31.0	13.8	7.4	67.6	19.0
	2	4.9	6.2	6.4	36.3	25.3	4.2	42.8	4.0	3.4
	3	6.7	26.9	73.8	44.7	29.7	9.8	77.1	48.2	18.8
	4	7.6	17.5	20.7	13.1	5.4	10.7	30.9	5.0	3.3
	5	3.7	29.5	19.8	16.6	10.2	1.5	2.4	12.7	4.4
	6	1.9	11.8	6.1	19.4	10.7	7.4	32.9	21.9	12.8
	7	4.2	78.3	81.0	36.0	3.5	3.7	37.9	2.6	1.5
	8	4.3	20.7	16.7	17.4	6.6	5.5	31.3	6.6	3.4

Groups x US-intensity x Blocks analysis:

Source	df	ms	F
Groups (G)	2	902.0	1.6
err(b)	21	571.5	
US-Intensity (I)	2	497.4	2.0
G x I	4	384.8	1.5
err(I)	42	250.0	
Blocks (B)	2	399.2	1.8
G x B	4	303.0	1.4
err(B)	42	221.6	
I x B	4	2470.8	13.8
G x I x B	8	136.1	0.8
err(IB)	84	179.1	

Table C11

Median latency (sec) to first post-CS response in two-day blocks during new-learning extinction (Days 36 - 41)

Group	Subject	Block		
		1	2	3
Hi	1	5.3	9.2	3.4
	2	6.6	8.9	4.0
	3	3.5	3.5	3.1
	4	7.6	3.4	1.5
	5	8.0	1.7	6.6
	6	6.0	5.1	3.2
	7	5.2	1.8	1.4
	8	5.6	5.0	2.8
Lo	1	3.8	2.2	1.6
	2	14.6	5.7	10.0
	3	7.6	3.4	3.6
	4	5.0	2.4	3.4
	5	11.7	5.2	3.0
	6	5.0	4.8	7.7
	7	6.1	2.2	3.2
	8	1.7	2.8	2.7
Random	1	5.8	2.1	6.9
	2	1.9	2.1	2.6
	3	2.1	9.7	8.0
	4	10.3	2.4	6.6
	5	4.0	7.2	2.6
	6	3.5	2.5	1.4
	7	10.4	8.6	7.1
	8	1.8	1.1	1.4

Groups x Blocks analysis:

Source	df	ms	F
Groups (G)	2	0.7	0.04
err(b)	21	14.5	
Blocks (B)	2	25.8	5.02
G x B	4	7.3	1.41
err(w)	42	5.1	