Delayed Versus Immediate Reacquisition of the Rabbits' Nictitating Membrane Response Following Conditioned Inhibition Training

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This thesis is submitted in partial fulfillment of the requirements for the degree of Master of Arts in the Department of Psychology

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BY

MARCIA-LYNN POLLOCK

A thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

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Abstract

Current theories of classical conditioning (e.g., Rescorla & Wagner, 1972; Wagner, 1981) are congruent in the expectation that the training protocol of the conditioned inhibition paradigm should yield a conditioned stimulus with associative inhibitory properties. However, the empirical results from the conditioned inhibition paradigm are equivocal. While Marchant III and Moore (1974) and Pearce, Montgomery and Dickensen (1981) have found evidence of associative inhibition subsequent to conditioned inhibition training, other investigators have not (e.g., Rescorla, 1982, Experiment three; Williams, Pollock & Tait, Appendix). Studies indicating the presence of associative inhibition incorporated massed training procedures (e.g., Pearce et al., 1981) and immediate associative inhibition assessment tests (e.g., Marchant III et al., 1974). Conversely, studies failing to show the presence of associative inhibition incorporated distributed training and delayed associative inhibition assessment tests (e.g., Williams et al., Appendix). The present study attempted, in part, to reconcile the discrepant findings with respect to the effectiveness of the conditioned inhibition paradigm in yielding an associative inhibitor. It was hypothesized that response patterns inherent to the conditioned inhibition paradigm may decrease the likelihood of detecting associative inhibition with delayed, but not immediate, inhibitory assessment tests.

The nictitating membrane response was monitored in four groups of rabbits. Two experimental groups received conditioned inhibition training and the remaining two groups served as controls for the non-associative inhibitory effects of a stimulus. A reacquisition test served to assess whether or not associative inhibition developed during

conditioned inhibition training. One experimental group and its corresponding control group received reacquisition tests immediately after the last conditioned inhibition training session. The remaining two groups were tested on the day following the last conditioned inhibition session.

Although both conditioned inhibition groups displayed a decrease in responding on the inhibitory trials within sessions and recovery of responding between sessions, only the conditioned inhibition group which received the immediate reacquisition test showed evidence of associative inhibition. The results were discussed within the framework of current classical conditioning theories. Spontaneous recovery of the excitatory strength of the putative inhibitor and the presence or absence of motivational drives during reacquisition were forwarded as possible reasons for the obtained results.

Delayed Versus Immediate Reacquisition of the Rabbits' Nictitating Membrane Response Following Conditioned Inhibition Training

In classical conditioning an experimenter presents to the experimental subject two types of stimuli. One type, called the unconditioned stimuli (US), are characterized by their ability to produce specific, reliable responses from the subject (cf. Gormezano & Kehoe, 1975; Pavlov, 1927). The reliable responses to the US have been labelled unconditioned responses (URs). The second type of stimuli, labelled conditioned stimuli (CS), do not initially produce the responses of interest from the subject. By repeatedly presenting a CS and US in some constant temporal arrangement, a response, similar to the response produced by the US will develop to the CS. The CS controlled response is called the conditioned response (CR). If a CR develops to a CS, then the CS is said to have excitatory properties. The term excitatory is used to connote the associative processes that activate links between the CS and the effectors of the US (e.g., Pavlov, 1927; Rescorla & Wagner, 1972). However, not all temporal arrangements of CSs and USs lead to the development of CRs (cf. Pavlov, 1927) and putative excitatory processes are not sufficient to account for all classical conditioning effects (cf. Pavlov, 1927). To incorporate all classical conditioning phenomena, Pavlov (1927) introduced the concept of behavioral inhibition and asserted that inhibition is an active process that produces the suppression of behavior. Hence, the development and maintenance of CRs was thought to reflect the interaction between the activating (excitatory) and suppressing (inhibitory) processes.

The present paper elaborates on the basic concept of associative inhibition. Current classical conditioning theories addressing the acquisition of associative inhibition are discussed and an empirical assessment of a paradigm which theoretically maximizes the development of associative inhibition was performed.

Behavioral Inhibition

Pavlov (1927) delineated two categories of behavioral inhibition: external and internal inhibition. The distinction between these two categories was based on whether or not the subjects' past experience with a stimulus was causal to the inhibitory effects of that stimulus. External inhibition did not require any past experience with the stimulus and external inhibition was therefore referred to as unconditioned inhibition. Conversely, internal inhibition did require a subject's past experience with the stimulus eliciting the inhibition (Pavlov, 1927) and may therefore be thought of as learned or associative inhibition. Learning theorists have focused on the antecedent conditions necessary for a stimulus to become an associative inhibitor.

Pavlov (1927) outlined four procedures which should result in associative inhibition: extinction, differential conditioning, inhibition of delay, and conditioned inhibition. The extinction procedures requires at least two phases. In the first phase a CS is repeatedly paired with a US. As a result of this training, the CS elicits CRs and the CS is said to be excitatory. During the second phase, extinction proper, the excitatory CS is presented alone. After repeated presentations, the CS no longer elicits CRs. Pavlov (1927) inferred that the CRs were being inhibited during extinction. In the differential procedure, one CS is always paired with a US while a second

CS is never paired with the US. Subsequently, presentation of the paired CS produces a CR and presentation of the non-paired CS does not produce a CR. The non-paired CS is assumed to have acquired associative inhibition (Pavlov 1927). The procedure of inhibition of delay involves the presentation of a long duration CS which is paired with a US on every trial. After extended training, CRs are no longer elicited during the first portion of the CS. Pavlov (1927) inferred that the first portion of the CS was inhibiting CRs. And finally, the conditioned inhibition procedure involves the randomized presentation of two different types of trials. On some trials, a CS is always paired with a US while on other trials, the CS is paired with a second CS in the absence of the US. More recently, Rescorla and Wagner (1972) have labelled the US paired CS as 'A', the CS-US pairing as an 'A+' trial, the second CS as 'X', and the pairings of the two CSs as an 'AX-' trial. With this procedure, inhibition is thought to accrue to X (Pavlov, 1927).

Since 1927, other paradigms have been credited with creating associative inhibition. Backward conditioning is one such paradigm (Pavlov, 1932; Wagner & Rescorla, 1972). On a given trial in backward conditioning, the onset of a US precedes the onset of a CS. Inhibitory properties develop to the CS after such training (e.g., Seigel & Domjan, 1971). A variation of the backward procedure involves preceding the US with one CS and following the US with a different CS. Again, the CS following the US should become an inhibitor (Solomon & Corbit, 1974). And finally, Rescorla (1967) has proposed that any training procedure which involves a negative correlation between a CS and a US, such that the probability of a US following a CS presentation is low, should

result in the CS becoming inhibitory. This argument was extended to include the explicitly unpaired procedure whereby a CS and a US are randomly presented, alone, on a given trial (Wagner & Rescorla, 1972).

The preceding paragraphs state the antecedent conditioning histories assumed necessary for producing an associative inhibitory CS. However, the conditioning histories are not considered to be sufficient to confirm that a CS is an associative inhibitor. There appears to be a general consensus as to the other necessary criteria. First, the associative inhibitor must be a discrete stimulus or stimulus complex (Hearst, 1972; Rescorla, 1969). Second, the actions of the associative inhibitor must be specific with respect to the US employed (Hearst, 1972; Rescorla, 1969). Third, the effects of the associative inhibitor must be opposite to the excitatory effects of the US (Hearst, 1972; Rescorla, 1969). Fourth, the responding in the presence of the associative inhibitor must be at a level below that which would be observed had the associative inhibitor not been presented (Hearst, 1972). If a CS satisfies these criteria, then the CS is said to be an associative inhibitor and the conditioning history is assumed to result in associative inhibition.

Identification of Associative Inhibition

If a CS is an associative inhibitor then the subject would not be expected to respond when the CS is present. This expectation results in a methodological dilemma since the absence of responding could also result from an ineffective CS. In order to differentiate inhibitory effects from ineffective treatments and also to identify that responding in the presence of the associative inhibitor is less than responding in its absence, inhibition assessment procedures have been developed that

typically introduce the putative inhibitor at a time when excitatory responding would otherwise be expected (Hearst, 1972). The associative inhibition assessment techniques most applicable to classical conditioning procedures are: retardation of learning, combined cues, disinhibition, supernormal conditioning, and reaction of the reverse sign. Each assessment technique will be discussed in turn.

The retardation of learning procedure is also referred to as the retardation or reacquisition test. The latter will be adopted in this discussion as the term reacquisition most closely reflects the procedure. Prior to the reacquisition test, subjects are exposed to a training regime that is presumed to endow a CS with inhibitory properties. In the assessment phase, the putative inhibitory CS is paired with a US and the rate of acquisition of CRs to the putative inhibitory CS is compared to the rate of acquisition of CRs by a group of subjects that had received no prior experience with the putative inhibitory CS (control group). If the subjects that received inhibitory training exhibit a retarded rate of acquisition of CRs to the CS relative to the control group, then the CS is assumed to be an associative inhibitor (Hearst, 1972; Rescorla, 1969).

The second assessment procedure is the combined cues or summation test. As with the reacquisition test, the summation test follows a training phase in which inhibition is thought to develop to a CS. In the assessment phase, the putative inhibitory CS is presented in simultaneous compound with a preestablished excitatory CS. No USs are presented in the assessment phase. Inhibition is assumed to be identified if responding to the compound is less than responding in a control group that had previously been exposed only to the excitatory CS in the pre-assessment phase (Hearst, 1972; Rescorla, 1969).

The third assessment procedure was referred to by Pavlov (1927) as disinhibition. With this procedure, a novel stimulus that is known to be an external inhibitor is presented concurrently with the putative inhibitory CS. If the compound results in the reinstatement of a CR to the CS, then the CS was assumed to be inhibitory. The reinstated CR was thought to reflect the external inhibition of inhibition or disinhibition (Pavlov, 1927).

The fourth assessment procedure is the supernormal conditioning phenomena (Rescorla, 1971). After initially training the putative inhibitory CS, the CS is paired with a neutral CS and the compound is paired with a US. According to Wagner and Rescorla (1972), if inhibition accrued to the putative inhibitory CS, then reinforcing the compound should result in the neutral CS acquiring an inordinate amount of excitatory strength. If the excitatory conditioning to the neutral CS is greater than a control group that did not receive the inhibitory training, then inhibition may be inferred (Rescorla, 1971).

The last method of assessing inhibition is the reaction of the reverse sign. This test is applicable only when the inhibitory CS elicits a measurable response which is opposite to the response elicited by the excitatory CS (Gray, 1975). With the reaction of the reverse sign procedure the form and the direction of the CRs to an excitatory CS are determined (e.g., accelerated heart rate CRs). The next stage involves some form of training designed to endow a different CS with inhibitory properties. If, during inhibitory training the putative inhibitory CS produces a response which is opposite in form or direction to the response controlled by the excitatory CS (e.g., decelerated heart rate CRs), then associative inhibition is inferred (Gray, 1975).

Of the five assessment procedures, reaction of the reverse sign, disinhibition and supernormal conditioning may be of limited utility. The reaction of the reverse sign procedure is the most direct assessment procedure since the inhibitory CR is measured rather than inferred (Gray, 1975). The utility of the reaction of the reverse sign is limited, however, because not all response systems have an opposing measurable response (e.g., eyelid response). For such response systems, the reaction of the reverse sign could not be employed. The disinhibition test is based on the assumption that excitation must accrue to a CS before inhibition can develop to the CS (Rescorla, 1969). Rescorla (1967, 1969) has indicated that this assumption may not always hold true, particularly for the procedures of unpaired CS and US presentations. Therefore, the disinhibition test may not be appropriate for all inhibitory procedures. Although supernormal conditioning could be used to identify a putative inhibitory CS (cf. Wagner, 1971), application of supernormal conditioning has been restricted to only a few known inhibitory paradigms as the focus of research to date has been on the accelerated excitatory conditioning effect. Since not all inhibitory paradigms have been examined with the supernormal conditoning procedure, the generality of the phenomena is unknown. Because of these limitations, the use of the reaction of the reverse sign, disinhibition and supernormal conditioning procedures in the assessment of associative inhibition is restricted.

The most universal inhibition assessment procedures are the summation and reacquisition tests (Rescorla, 1969). Both tests measure inhibition through the suppression of excitatory CRs. However, the two testing procedures may not be trouble free. Both tests may be

differentially affected by attentional processes. Consider the summation test. Upon the presentation of the putative inhibitory CS compounded with an excitatory CS, the subjects' attention may be biased toward the inhibitory CS, thus less attention could be given to the excitatory CS. As a consequence, the excitatory CS would have less influence on behavior, with the result of reduced excitatory performance to the compound. Thus, an attentional bias could mimic the expected effect of associative inhibition on the summation test. However, if reduced responding on a summation test results from attentional rather than inhibitory properties of the putative inhibitory CS, then the same CS should facilitate performance on a reacquisition test in which the CS is paired with a US. On the other hand, if responding to the putative inhibitory CS is reduced during the reacquisition test because of too little attention to the putative inhibitory CS, then this same CS should not bias the summation test because the attention would then be biased toward the excitatory CS (Rescorla, 1969). Therefore, to conclusively identify the occurrence of associative inhibition and to eliminate the attentional interpretation, Rescorla (1969) advocated that both the summation and reacquisition tests be employed for the assessment of associative inhibition.

Theories of Associative Inhibition

The mechanism by which a CS becomes an associative inhibitor during classical conditioning procedures is not yet known. Currently, there are two major classes of classical conditioning theories which address the problem of the acquisition of associative inhibition. The first class of theories are based on a linear model of changes in associative

strength among the conditioned stimuli and the second class focuses on the dynamics of activity initiation and decay during memorial processes.

A linear model of classical conditioning was formally proposed by Rescorla and Wagner (1972) and Wagner and Rescorla (1972). The basic premise of the Rescorla-Wagner model is that the amount of associative strength that can accrue to a CS on a given conditioning trial is dependent upon the summed associative strength of all CSs presented on that trial. Stated mathematically, the theory takes the following form: $\Delta V_A = \alpha\beta(\lambda - \overline{V})$, where ΔV_A is the change in associative strength accrued to a CS A, on a given trial; α $(0 < \alpha < 1)$ and β $(0 < \beta < 1)$ are learning rate parameters for the CS and US, respectively, and reflect the relative intensity of the respective stimuli on that trial; λ is the level of conditioning a US will support, denoted by a positive value if a US is presented on that trial, zero otherwise; and $\overline{V}$ is the current summed associative strength of all the presented CSs (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). If $\lambda - \overline{V}$ is a positive value then the associative strength of the corresponding CSs are incremented. If the net associative strength of a CS is positive, then the CS becomes excitatory with respect to the US. On the other hand, if $\lambda - \overline{V}$ is a negative value, then the associative strength of the corresponding CSs are decremented. If the net associative strength of a CS is negative, then the stimulus becomes inhibitory (Wagner & Rescorla, 1972).

To illustrate how inhibition would develop with the Rescorla-Wagner model, consider the conditioned inhibition paradigm. Recall that with this procedure, a CS, say A, is followed by a US on some trials (A+) and on other trials the same CS is presented in compound with another CS, say X, and the compound is never followed by a US (AX-). Prior to the

first US, the associative strength of the CSs, V_A and V_X, are zero thus their sum, $\overline{V}$, is also zero. On the first A+ trial, V_A becomes positive because λ is a positive value when a US is also presented and a positive value (λ) minus zero (V_A) results in a positive value. $\overline{V}$ is now also a positive value. On the first compound trial subsequent to an A+ trial, no US occurs, therefore $\lambda=0$. Since $\overline{V}$ has a positive value as a result of the previous A+ trial, $\lambda-\overline{V}$ will be negative and X accrues negative associative strength. With repeated trials, the A+ trials serve to maintain the positive associative strength to A and thus the positive value of $\overline{V}$ and the above process continues.

Thus for Wagner and Rescorla (1972) the acquisition of inhibition requires a source of excitation, otherwise $\overline{V}$ could not stay positive and thus result in a negative value when subtracted from λ on non-reinforced trials. The A+ trials in the A+/AX- paradigm very neatly supplies this source of excitation. However, backward, unpaired, differential and negative correlation procedures are also presumed to result in associative inhibition accruing to a CS (e.g., Pavlov, 1927, 1932; Rescorla, 1969), yet in these procedures there is not an excitatory CS serving to maintain a positive value of $\overline{V}$. To accommodate the apparent lack of excitation with these latter inhibitory training procedures, Wagner and Rescorla (1972) argued that contextual or background cues could become excitatory during classical conditioning procedures. The rationale for this assertion was that the context is always present, therefore, the context is always paired with the US and could, in this manner, acquire excitatory strength. Wagner and Rescorla (1972) argue that this contextual source of excitation is sufficient to produce inhibition in many circumstances but the context is not as effective as

an excitatory source as a discrete stimulus, such as A+, because the A+ is not prone to extinction between US presentations as are the contextual cues. Consequently, Wagner and Rescorla (1972) predict the conditioned inhibition paradigm (A+/AX-) to be superior in creating inhibitory properties to a CS, X in this case.

Another prediction of the Rescorla-Wagner model is that the presentation of an inhibitory CS alone should result in a positive associative strength accruing to the inhibitory CS, as λ would equal zero when a US is not also present and zero minus some negative value would result in a positive value (Wagner & Rescorla, 1972). Inhibition should therefore dissipate. Zimmer-Hart and Rescorla (1974) tested this prediction and found that, counter to the expectations of the Rescorla-Wagner model, inhibition did not extinguish if an inhibitory CS was repeatedly presented in the absence of a US. Subsequently, Rescorla (1979) modified his theory with regard to inhibition.

In the modified theory, Rescorla (1979) retained the mathematical form of the Rescorla-Wagner model but a certain asymmetry between excitation and inhibition was allowed under the special circumstance of presenting the inhibitory CS alone (non-reinforced). If an inhibitory CS was not reinforced, Rescorla (1979) predicted no change in the associative strength of that CS because the behavioral consequence of the CS presentation, i.e., no excitatory response, would not differ from the expectation had the CS not been presented. Rescorla's (1979) modification did not alter the previously stated predictions of the effectiveness of an inhibitory CS in the presence of excitation, i.e., the conditioned inhibition paradigm (A+/AX-) should result in the most

effective inhibitory CS relative to backward, differential, unpaired and negative correlation paradigm.

The essence of the Rescorla-Wagner model has been maintained in later theories such as Frey and Sears (1978), Pearce and Hall (1980) and Moore (1982). Each theory is mathematically based and uses the linear structure of the Rescorla-Wagner model: $\Delta V_A = \alpha\beta(\lambda - \overline{V})$. However, each theory places a greater emphasis on the attentional effects of the learning rate parameter of the CS (α) than did Rescorla and Wagner (1972) for whom α basically reflects CS intensity. The allowance for attentional processes were, in part, spurred by the Zimmer-Hart and Rescorla (1974) findings that an inhibitory CS does not extinguish in the absence of a US (Frey & Sears, 1978; Pearce & Hall, 1980; Moore, 1982). Frey and Sears (1978) argued that a conditioning model requires a dynamic attention rule. To incorporate the effects of attention into the Rescorla-Wagner model, Frey and Sears (1978) allowed the value of α to increase or decrease depending on the CS's current associative strength and whether or not the CS was followed by a US on a given trial. Pearce and Hall (1980) also allowed α to fluctuate in this manner but they also maintained the CS intensity factor of the Rescorla-Wagner model. The CS intensity factor was relabelled 'S'. The effects of the properties of the CS was then given the value resulting from the product of α and S (Pearce & Hall, 1980). Moore (1982), rather than altering the intended value of α in the Rescorla-Wagner model created a new parameter τ. τ is a function of the CS's associability, thus τ is analogous to the usage of α in the Frey and Sears (1978) and Pearce and Hall (1980) models. Despite the parameter changes related to the role of attention, the predictions pertaining to the acquisition and

strength of inhibition of the aforementioned theories are congruent with the Rescorla-Wagner model since the linear model was retained (Frey & Sears, 1978; Pearce & Hall, 1980; Moore, 1982).

While revisions of the Rescorla-Wagner model focus on the role of attention on conditioning, Wagner has revised his approach to classical conditioning by emphasizing dynamic memory processes (Wagner, 1981; Mazur & Wagner, 1984). Wagner refers to his new model of automatic memory processing by the abbreviation SOP, for standard operating procedures. In SOP, a memorial node for a US or a CS contains many elements. At any given point in time, the elements may be distributed among three states. The three states are: inactive (I), activity 1 (A1) and activity 2 (A2). At any given time, the number of elements across all nodes in the A1 or A2 states is a constant. An element may change from the I state to the A1 state only by the presentation of a stimulus. From the A1 state, the element activity decays to the A2 state and from A2 decays to the I state. The only exception to this activity change is through an associative link. If there is an associative link between two memorial nodes then A1 activation in one node will cause elements in the other node to change from the I state to the A2 state. In this instance, the A2 state is presumed to produce a CR.

Two types of associative links, excitatory and inhibitory, may be formed between two stimuli. An excitatory association between two nodes is formed when two nodes have simultaneous A1 activity. If one node has A1 activation while the other has A2 activity then an inhibitory association is formed (Mazur & Wagner, 1984). The strength of the association is a function of the number of overlapping elements across

nodes. As the number of overlapping elements increases, so does the strength of the association.

The SOP model's predictions for the development of inhibitory associations within the various inhibitory paradigms are as follows. Within the conditioned inhibition paradigm, two sets of excitatory associations are formed: one between stimulus A and the US, the second between stimulus A and stimulus X because the A1 states temporally overlap. In addition, on the AX- compound trials, A evokes a US A2 state (CR) which is paired with an X A1 state, therefore an inhibitory association forms between X and the US (Wagner, 1981; Mazur & Wagner, 1984).

In the backward paradigm, both excitatory and inhibitory associations may form. If CS onset coincides with US offset, a US A1 state would overlap with a CS A1 state and excitatory associations should form. As the US activity decays to A2, the US A2 activity may overlap with the CS A1 activity if the CS is present. In this latter instance, inhibitory associations would also form and the net associative strength would be determined by the difference between the strength of the excitatory and inhibitory associations (Wagner, 1981; Mazur & Wagner, 1984). Although it is beyond the scope of this paper to exhaust all of the possibilities of backward pairing procedures, it is possible for inhibitory associations to be formed with backward pairings but because of the coincident excitatory associations, the backward training procedure should be less effective than the conditioned inhibition procedure in creating inhibition.

The formation of associations should be more difficult with differential, unpaired and negative correlation procedures. The reason

for the increased difficulty is that the putative inhibitory CS and the US are presented on different trials with these procedures. The temporal discrepancy between the stimuli that results from the interpolated intertrial interval allows for decay of activity in the memory nodes. The activity decay results in a decrease in the number of elements in the A1 and A2 states of the CS and US nodes that are available coincidentally. Since the strength of association is directly related to the number of elements active during the formation of that association (Mazur & Wagner, 1984) the inhibitory associations formed during differential, unpaired and negative correlation procedures should be weaker than the inhibitory associations formed when there is no temporal delay between the CS and US. As there is no temporal delay between the stimuli in the conditioned inhibition procedure, the conditioned inhibition paradigm should yield stronger inhibitory associations relative to unpaired, differential and negative correlation paradigms.

Collectively, the current theories of classical conditioning are consistent in the expectation that the conditioned inhibition paradigm should yield the strongest associative inhibition relative to the backward, unpaired, differential and negative correlation procedures (Frey & Sears, 1978; Moore, 1982; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981). Furthermore, the theories based on linear models (Frey & Sears, 1978; Moore, 1982; Pearce & Hall, 1980; Rescorla & Wagner, 1972) lead to the expectation that the amount of excitatory conditioning of the contextual cues should be equal for the unpaired and backward paradigms and weakest for the differential paradigm. Consequently, the rank order of the paradigms for the development of

associative inhibition should be: conditioned inhibition, unpaired and backward, and lastly, differential. The associative inhibition resulting from negative correlation procedures is dependent upon the correlation between the putative inhibitory CS and the US, with the unpaired procedure having a zero correlation, thus the expected level of associative inhibition following negative correlation training is relative to the precise trial parameters employed (e.g., Wagner & Rescorla, 1972).

Similarly, with the exception of the conditioned inhibition paradigm, predictions of the strength of the associative inhibition based on the SOP model are specific to the stimulus intensities and trial parameters employed. For example, whether or not the activity between a CS and US node will overlap within the unpaired, negative correlation and differential procedures depends on the time between two trials (intertrial interval) and stimulus intensity. For the backward paradigm, the interstimulus interval between the US and CS within one trial and the intensity of the stimuli will affect the expected overlap between the A1 and A2 states of each node. However, within the constraints of the general procedures, the expectations based on the SOP model are as follows: (a) conditioned inhibition training should yield the strongest associative inhibition and, (b) backward training should yield stronger associative inhibition relative to unpaired, negative correlation and differential training (Wagner, 1981; Mazur & Wagner, 1984).

Although discrepant in the proposed mechanisms, current theories of classical conditioning are congruent with respect to the expectation that the conditioned inhibition paradigm should yield the strongest

associative inhibition. The major difference between the theories is the expectation of the strength of associative inhibition following the backward and unpaired training procedures. The backward procedure is expected to yield stronger associative inhibition in the SOP model (Mazur & Wagner, 1984) whereas unpaired training is thought to equal backward training in terms of associative inhibition in the linear based models (Frey & Sears, 1978; Moore, 1982; Pearce et al., 1980; Rescorla & Wagner, 1972).

Empirical Review of Associative Inhibition

The empirical evidence regarding the predictions based on current theories of classical conditioning will be reviewed in this section. The predictions pertaining to the rank ordering of the strength of associative inhibition with respect to the different inhibitory procedures will be discussed first. Next, the expectation that conditioned inhibition training will result in a stimulus acquiring associative inhibitory properties will be reviewed.

Recently, two experiments (Williams, Pollock & Tait, Appendix) were performed to test the prediction of the Rescorla-Wagner model (Rescorla & Wagner, 1972) that the strength of associative inhibition would be rank ordered from strongest to weakest, after conditioned inhibition, unpaired, backward and differential training procedures. Both experiments measured the nictitating membrane response in rabbits. All training sessions occurred on consecutive days, were 100 trials in length and the average intertrial interval was 60 sec. Experiment 1 employed six groups of rabbits. Each group experienced five phases: 1 day of adaptation, 3 days of acquisition training, 4 days of inhibition training, 3 days of reacquisition training, and 1 day of extinction.

During the acquisition phase, all groups received forward click-shock pairings in order to make the click an excitatory stimulus. During the inhibitory phase, the groups received one of the following treatments: (a) conditioned inhibition training (Group CI), (b) unpaired training (Group UP), (c) differential training (Group D), (d) backward training (Group B), (e) no stimuli (Group N), or (f) continued excitatory training to the click (Group E). For the groups receiving inhibitory training (Groups UP, B, D, and CI), a tone was the intended inhibitory stimulus. Groups N and E served as controls for the non-associative inhibitory effects of the tone (cf. Hendersen, 1973). Reacquisition and extinction phases for all groups consisted of forward tone-shock pairings and tone alone presentations, respectively.

The results of the first reacquisition session indicated that the strongest inhibition to the tone was acquired by Group UP, some inhibition was acquired by Groups B and D and no inhibition was evident in Group CI, relative to Groups E and N. As the performance of Group CI was in sharp contrast to theoretical predictions, Experiment 2 was performed.

Experiment 2 differed from Experiment 1 in the following ways: (a) the control group was restricted to Group E; (b) a 3 day summation assessment test was interposed between the inhibitory and reacquisition phases, (c) a tone and a click were separately trained in a 5 day acquisition phase, with the click subsequently to serve as the summation test excitor and the tone subsequently to serve as the excitor for inhibitory training of Groups CI and D, (d) during the inhibitory training phase the ratio of inhibitory versus excitatory trials was

changed from 1:1 to 2:1, (e) there were 3 more inhibitory training sessions and, (f) air was to be the inhibitory stimulus (Williams et al., Appendix).

During the summation test, associative inhibition was inferred if the percentage of CRs to the compound trials of the summation excitor and putative inhibitor was reliably lower than the percentage of CRs to the summation excitor alone trials, relative to control Group E. The percentage of CRs in Group E did not differ across these two trial types. Group UP demonstrated associative inhibition during each summation session while for Group D, associative inhibition was evident only during the first summation session. Interestingly, there was no evidence of associative inhibition in Group CI during the first and second summation sessions but during the third summation session, Group CI did elicit fewer CRs on the compound trials. It is unclear why Group CI started to discriminate between the two trial types during the third summation session as the reacquisition test initiated on the following day indicated that Group CI was not inhibitory; but Groups UP, B and D were in inhibitory. Since the summation excitor trials and the compound trials of the summation excitor and putative inhibitor are analogous to the A+ and AX- trials experienced during conditioned inhibition training, perhaps the summation test provided an extended form of inhibition training for Group CI (Rescorla, 1973).

Collectively, the results of the two experiments (Williams et al., Appendix) indicate that the unpaired training produces the strongest associative inhibition, differential is more effective than backward training in establishing associative inhibition, and conditioned inhibition training was ineffective as an associative inhibition

training procedure. These findings are in sharp contrast to the expectations of the Rescorla-Wagner model (Wagner & Rescorla, 1972) and its derivatives (Frey & Sears, 1978; Moore, 1982; Pearce & Hall, 1980) as well as Wagner's SOP model (Wagner, 1981). This conclusion follows for three reasons. First, the unpaired group, not the conditioned inhibition group, demonstrated the strongest associative inhibition. Second, the strength of associative inhibition was greater in the differential group relative to the backward group. Third, there was no evidence that conditioned inhibition training resulted in associative inhibition.

Other experiments investigating the rank ordering of inhibition amongst various treatment groups have also failed to support theoretical predictions. Mahoney, Kwaterski and Moore (1975) investigated the effect of interstimulus interval on the acquisition of associative inhibition and found that the response patterns of the conditioned inhibition and differential groups did not reliably differ. Similarly, Gaffan and Hart (1981), using autoshaping and pigeons did not obtain reliable differences in responding between a conditioned inhibition and unpaired group during a summation test. Finally, Hoffman and Fitzgerald (1982), in a heart rate study, found no differences between a conditioned inhibition, an unpaired and a differential group during a summation test, although in a subsequent reacquisition test it appeared that the differential group was less inhibitory than either the conditioned inhibition or unpaired groups, which did not differ. It should be noted that the unpaired training in the Hoffman and Fitzgerald (1982) study was atypical. Recall that the unpaired procedure typically involves alternating a CS (the to be inhibitor) and a US across trials.

In the Hoffman and Fitzgerald (1982) study, the unpaired group received alternating presentations of a tone and light (the to be inhibitor) compound and a US, therefore the generalizability of their findings regarding the unpaired group is restricted.

From the results of Mahoney et al. (1975), Gaffan and Hart (1981) and Hoffman and Fitzgerald (1982) studies, it would appear that the associative inhibitory strength does not differ appreciably between conditioned inhibition, differential and unpaired groups. However, such a conclusion is not warranted for two reasons. First, when the terminal levels of inhibition are the same across the different inhibitory groups, the rate of acquisition of inhibition could be used as an index of the effectiveness of the different training procedures. Data relevant to the rate of acquisition of inhibition was reported by Gaffan and Hart (1981) who found that the unpaired group seemed to show stronger acquisition of inhibition relative to the conditioned inhibition group. Also, Hoffman and Fitzgerald (1982) reported that the differential group acquired inhibitory CRs faster than the conditioned inhibition group during the inhibitory phase. Second, Hendersen (1973) has shown that a stimulus may possess non-associative (external) inhibitory properties. Specifically, using rats as subjects, Hendersen (1973, Experiment 1) showed that subsequent to differential training, the response suppression observed upon presentation of the putative inhibitory CS, relative to the excitatory CS, was not different than the response rate when the putative inhibitory CS was presented to a group of rats that had no prior exposure to any CSs. In this instance, the external inhibitory effects of the stimulus were sufficient to account for the response decrements to the putative inhibitor observed after

differential training. Therefore, in order to assess the associative effects of a stimulus, a control group not receiving exposure to the putative inhibitory CS prior to the assessment test should be included in the study. Since the Mahoney et al. (1975), Gaffan and Hart (1981) and Hoffman and Fitzgerald (1982) studies did not employ a non-associative inhibitory control group, conclusions concerning associative inhibition are premature.

Given the inconclusive nature of the Gaffan and Hart (1981), Hoffman and Fitzgerald (1982) and Mahoney et al. (1975) studies, the studies neither support nor contradict the theoretical positions regarding the rank ordering of inhibition across treatment groups. It seems then that the only available evidence regarding this prediction is from the experiments performed by Williams et al. (Appendix) and as earlier reported, expectations were not upheld. The major exception to the predicted rank ordering was the lack of inhibition detected in the conditioned inhibition group.

The results from other studies investigating the inhibitory properties of the conditioned inhibition paradigm are equivocal. Interpretation of the relevant data is clouded, in part, by the previously mentioned omission of a control group assessing the non-associative inhibitory effects of a stimulus. Also, ambiguity arises from the specific procedures employed during the inhibition assessment tests and the reporting of results. These latter points will be elaborated in the following review of studies investigating the inhibitory properties of the conditioned inhibition paradigm.

A recurring problem in some assessment tests following conditioned inhibition training is that a true summation test is not applied. There

are three major abuses of the summation test. First, if A+ and AX- stimulus presentations are intermixed during the summation test, then the summation test is essentially extended conditioned inhibition training and therefore, is not an appropriate assessment technique. This testing procedure has been employed by Rescorla (1982, Experiments one and two) and Zimmer-Hart and Rescorla (1974, Experiments two and three). Second, in a typical summation test, no USs are presented. If USs are incorporated into the summation test (e.g., Gaffan & Hart, 1981), then the USs would serve to maintain the excitatory strength of the summation excitor. Maintenance of excitatory strength to the summation excitor may create conditions analogous to conditioned inhibition training. Third, a more subtle abuse of the summation procedure is to test X in compound with the excitor with which it was compounded in the conditioned inhibition phase, rather than to test X in compound with an alternative excitor (e.g., Marchant, Mis & Moore, 1972; Zimmer-Hart & Rescorla, 1974). Under these conditions, reduced responding to the training compound might be due to within compound associations formed between A and X during the conditioned inhibitory phase (e.g., Cunningham, 1981) and not necessarily to an inhibitory association between X and the US.

Other difficulties in interpretation arise when the data of a three day summation test are presented as an overall average (e.g., Marchant et al., 1972). This is problematic in a within subjects design because the summation test may advantageously mimic continued inhibitory training for the conditioned inhibition group (Rescorla, 1973 and Williams et al., Appendix). Specifically, the summation excitor alone trials and the simultaneous compound trials of the summation excitor and

putative inhibitor are analogous to the A+ and AX- trials of the conditioned inhibition procedure, respectively. Recall that this possibility was observed by Williams et al. (Appendix) who found that the effects of the conditioned inhibition group were not evident until the third day of the summation test. Therefore, the results of the first day of the summation test tend to be the most informative; if this data is not reported it can not be determined if inhibition was present prior to or was an artifact of the summation test.

Experimenters who provide some evidence of associative inhibition after conditioned inhibition training include Pavlov (1927), Marchant III and Moore (1974) and Pearce, Montgomery and Dickensen (1981); whereas both Williams et al. (Appendix) and Rescorla (1982, Experiment three) do not provide evidence of associative inhibition following conditioned inhibition training. Interestingly, both Marchant III and Moore (1974) and Pearce et al. (1981) used massed conditioned inhibition training procedures. Specifically, Marchant III and Moore (1974) used 700 daily training trials with a constant intertrial interval of 40 sec, while Pearce et al. (1981) employed 600 daily training trials with a 60 sec intertrial interval. Even with the large number of daily trials, Pearce et al. (1981) observed that responding on the A+ and AX- trials during conditioned inhibition sessions did not differ. Therefore, they altered the ratio of the number of stimulus presentations from 1:1 to 1:2 for A+ and AX- trials, respectively. This change resulted in lower responding to AX-. In both the Marchant III and Moore (1974) and Pearce et al. (1981) studies, reacquisition to the putative inhibitor (X) was retarded relative to control groups. For Marchant III and Moore (1974), the reacquisition test was initiated half-way through the last

inhibition training session and for Pearce et al. (1981), the reacquisition test was initiated on the day following the last inhibitory training session.

It would appear that massed conditioned inhibition training is a sufficient condition for the development of associative inhibition, but it is not clear that it is a necessary condition. The studies that failed to identify associative inhibition with the conditioned inhibition paradigm differed from the successful experiments in two ways. First, trials were distributed over days. Second, the assessment test was applied on the day following the last inhibitory training session. Recall that when Marchant III and Moore (1974) initiated the reacquisition test mid-way through the last conditioned inhibition session, associative inhibition was detected but a control group receiving the reacquisition test on the day following the last inhibition session was not included so it can not be determined if the inhibition was a function of the massed training or the timing of the reacquisition test.

The success of the immediate reacquisition test should not be ignored given the observations of Williams et al. (Appendix). In both experiments, Williams et al. (see figure 13, Appendix) observed that during conditioned inhibition training responding to the AX- compound decreased over each daily session yet responding recovered to high levels at the start of each new session. These within session decrements to AX- were statistically reliable, more notably in Experiment two which had a more extended inhibitory training phase than Experiment one. Although within session decrements were also observed on the trials when a US was not presented in the differential and

unpaired groups, the recovery across these sessions did not approach the level of recovery observed for the conditioned inhibition group. Specifically, at the start of each new session, the conditioned inhibition group responded to 85 percent of the no US trials whereas the differential and unpaired groups responded to less than 40 percent.

It is possible that the observed between sessions recovery in the conditioned inhibition group places the conditioned inhibition group at a disadvantage relative the the differential, unpaired and backward groups when the reacquisition test is given on the day following the last inhibitory training session. There are two possible reasons why the conditioned inhibition groups might have demonstrated recovery to the AX- trials across conditioned inhibition sessions. First, the between session recovery to the AX- trials across conditioned inhibition sessions may be due to the excitatory strength of A. If the response recovery across the conditioned inhibition sessions is due to the strength of A, then a delayed reacquisition test should not bias against the detection of associative inhibition because A is not presented during the reacquisition test; therefore, responding to X during reacquisition should not be artifactually accelerated by the presence of an excitatory stimulus. Second, Wagner and Rescorla (1972) have indicated that prior to X becoming inhibitory during conditioned inhibition training, X first acquires some excitatory strength. If the response decrements within conditioned inhibition sessions are due to extinctive inhibition accruing to X, then the between sessions recovery to the AX- trials may be due to the spontaneous recovery to X. If spontaneous recovery of responding occurs to X, then responding to X during the first few trials of a delayed reacquisition test will be

observed. Since X is followed by a US during reacquisition, responding to X should continue at a high rate for the remaining course of the reacquisition test. In this instance, a delayed reacquisition test would bias the test against the detection of associative inhibition to X.

In summary, the lack of support for the theoretical expectation that the conditioned inhibition paradigm should result in strong associative inhibition to a CS (Frey, & Sears, 1978; Moore, 1982; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) may be due to either the use of distributed training procedures or a delayed reacquisition test (Williams et al., Appendix). Since current classical conditioning theories (Frey & Sears, 1974; Moore, 1982; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) predict that the conditioned inhibition paradigm should produce the strongest indices of associative inhibition, and since demonstrable associative inhibition is obtained with the backward, differential and unpaired paradigms with distributed training (Williams et al., Appendix), demonstrating associative inhibition with distributed conditioned inhibition training is a critical question. Accordingly, the present focus will be on the possible effects of a delayed reacquisition test in the detection of associative inhibition following distributed conditioned inhibition training.

Experimental Hypothesis

The experiment was designed to: (a) reassess the associative inhibitory properties of the conditioned inhibition procedure and, (b) determine if the reacquisition test on the day following conditioned inhibition training underestimates the putative inhibition.

Four groups of animals were employed. Two experimental groups received conditioned inhibition training (A+/AX-) during the inhibitory training phase. One of the experimental groups received a reacquisition test immediately following the last conditioned inhibition session; the other experimental group had the reacquisition test on the day following the last conditioned inhibition session. Each experimental group was matched to a control group that received A+ trials but not AX- trials during the conditioned inhibition sessions.

Reacquisition was chosen as the associative assessment procedure for four reasons. First, the response system used in this study was the extension of the nictitating membrane of the rabbit which does not have a known measurable response that is opposite in form or direction to the excitatory response. Accordingly, the most direct assessment procedure, the reaction of the reverse sign, could not be used. Second, Marchant III and Moore (1974), Pearce et al. (1981) and Williams et al. (Appendix) all employed the reacquisition procedure. In order to compare the results to these studies, methodological consistency was desired. Third, although both summation and reacquisition procedures are relatively direct associative inhibition assessment procedures (Rescorla, 1969) the summation procedure very closely resembles conditioned inhibition training (Williams et al., Appendix). As the conditioned inhibition paradigm was under study, a summation test may confound the obtained results. Fourth, Rescorla's (1969) caution that the reacquisition test might be affected by attentional processes was considered. The basic concern was that retarded acquisition to the putative inhibitor during reacquisition may be due to the putative inhibitor receiving too little attention during the inhibitory training

phase. But Williams et al. (Appendix) found that during conditioned inhibition training, there were significant within session decrements in responding on the AX- trials relative to the A+ trials. If X was not attended to then responding to the AX- trials should not have differed from the A+ trials. It seems then that X is attended to in the conditioned inhibition paradigm so the reacquisition test should not be affected by attentional processes.

Method

Subjects

Thirty-two male and female New Zealand albino rabbits, approximately ninety days of age and weighing about 2.5 kg, were obtained from the Kleefeld Rabbitry in Tourond, Manitoba. The rabbits were housed in 60 cm by 45 cm wire-mesh cages located in a continuously lighted colony room maintained at 23°C ± 1°C. The rabbits had ad lib access to Landmark rabbit chow and water.

Apparatus

During all experimental procedures, each rabbit was individually restrained in a Plexiglas holder equipped with adjustable backplates, foam padded headstock and ear clamps in order to minimize the rabbits' head and body movements. Also, movement around the rabbits' eyes was minimized by adjustable Velcro eyestraps which were tipped, on each end, with Newey tailor hooks. The rabbits' upper eyelid was inserted in one hook, the eyestrap wound around the rabbits' head and then the other hook was used to grip the lower eyelid. Once the hooks were in place the eyestraps were adjusted so that eyelids were retracted to a comfortable position.

Extension of the rabbits' third eyelid, i.e., the nictitating membrane (NM), was detected and recorded in the following manner. A suture embedded in the free edge of the NM permitted attachment of the NM to a 10K ohm microtorque potentiometer. The potentiometer was mounted on a frame which was securely fitted on top of the rabbit's head. A piano wire armature was mechanically fixed to the potentiometer. A thread leading from the armature was tipped with a staple which was hooked through the suture in the NM. The tautness of the thread was then adjusted relative to the retracted NM such that there was no slack in the thread (Gormezano, 1966). A minimum .5 mm extension of the NM caused a clockwise motion of the armature which produced a change of .2V in the potentiometer and transformed the signals to a digital code such that the .5 mm NM movement corresponded to a hexidecimal output of 20.

All conditioning sessions took place in one of eight legal-size drawers of two, fire-proofed, sound attenuating file cabinets. A ventilation fan was situated at the back of and external to each cabinet drawer (chamber). A white panel formed the false front wall of each chamber and concealed electrical wires, speakers, air tubing and the chamber light. Holes were cut through the front panel to allow the presentation of experimental stimuli. An 8 ohm auditory speaker was mounted behind one hole (dia = 6.5 cm) which was centered relative to the vertical sides of the panel and 12.7 cm from the floor of the chamber. Two holes (each dia = 6 cm) were cut on either side of the auditory speaker. The opening on the left was covered with translucent plastic in order to diffuse the light from a 6 watt 30 volt light bulb. The other opening accommodated a second 8 ohm speaker which lay flush

with the panel wall. Centered at the top of the panel was a three-pin female stereo socket (dia = 1.5 cm) that served as a receptacle for the potentiometer. Female banana plugs were mounted in the lower right corner of the panel to permit the delivery of shock. A plastic tube passed through a 0.5 cm opening in the panel and could be inserted in a side hole (dia = 0.25 cm) of the Plexiglas restrainer. The tube allowed the delivery of a puff of air to the rabbits' rib cage.

Delivery of the stimuli to each chamber was controlled and time by the Raytheon computer. The stimuli consisted of two CSs, a 500 msec 80 db 1000 Hz tone and a 500 msec, 20 p.s.i. airpuff. The US was a 50 msec 2.5 mA, 60 Hz constant current shock delivered through two 9 mm stainless steel Autoclips implanted 9 mm caudal to a 10 mm above and below the horizontal plane of the rabbits' right eyes. Finally, 72 db of white noise provided a masking background hum in the experimental room during all conditioning sessions.

Procedure

One day after their arrival the rabbits were brought to the laboratory and individually restrained. The area surrounding their right eye was shaved and then depilitated with Nair. Subsequently, the rabbits' eyelids were retracted with the eyestraps and the NM extended from the inner canthus of the eye by a Q-tip (Life). The epithelial layer of the extended NM was sutured with 00 Ethicon monofilament nylon and a 2 mm loop was made. In order to prevent infection, Vetropolycin antibacterial cream was then applied to the inner sides of the eyelids immediately, and after each conditioning session. Following the suturing, the rabbits were randomly assigned to one of four groups (n = 8) and numbered from one to eight within each group. The numbering

procedure ensured that each rabbit was suited with the same equipment, restrainer, chamber and home cage for the duration of the experiment.

On the second day after suturing, each group of rabbits were restrained and fitted with a headset. Each restrained rabbit was then placed in its conditioning chamber and remained there for a 96 min adaptation session. No stimuli were presented. On the day following adaptation, conditioning sessions commenced. There were four conditioning phases for all groups: tone acquisition, inhibition training, reacquisition, and extinction. During tone acquisition, each group received a daily conditioning session for three consecutive days. Each session consisted of 96 trials of forward tone-shock pairings with the offset of the tone coinciding with shock onset. The average intertrial interval was 60 sec.

On the fourth day, the inhibitory phase commenced and extended for eight consecutive days. During the inhibitory training phase, two groups received conditioned inhibition training (CI) and two groups continued to receive only excitatory (E) training. During each inhibitory session, the CI groups received 32 trials of forward tone-shock pairings and 64 trials of non-reinforced simultaneous compound pairings of the tone and airpuff. The two types of trials were randomly intermixed under the constraint that no more than 3 consecutive presentations of the same trial type could occur. Again the intertrial interval averaged 60 sec. During this phase, the E groups received only 32 forward tone-shock pairings over the 96 min session. Trial onset for the E groups were temporally matched to the tone-shock trials of the CI groups.

Following inhibitory training, each group received a daily reacquisition session on 2 consecutive days. Each reacquisition session consisted of 96 forward air-shock pairings with the air offset coinciding with shock onset. The average intertrial interval was 60 sec. One CI group (Group CI-I) and one E group (Group E-I) commenced the first reacquisition session immediately following the last inhibitory training session. The remaining two groups, Groups CI-N and E-N, received the first reacquisition session on the next day following the last inhibitory training session.

On the day following the last reacquisition session, all groups received one session of extinction. Extinction consisted of 96 air alone trials. The intertrial interval averaged 60 sec.

During the experiment three rabbits developed pneumonia and therefore, the rabbits were killed by a Nembutal overdose. The loss of the subjects resulted in an n of 7 for Group E-I and an n of 6 for Group E-N. Groups CI-I and CI-N maintained an n of 8.

Response Definition

During each session, a 0.5 mm extension of the NM was recorded as a CR if it occurred during the 500 msec presentation of a CS. Although no stimuli were presented in adaptation, baseline CRs were recorded if the response occurred in a time interval that a CS would have been presented in the acquisition phase. Similarly, a CR base rate was recorded in the E groups during the inhibitory training phase if the response occurred in the time interval coincident with the tone-air pairings of the CI groups. The percent CRs during each 24 trial block of the adaptation, acquisition, reacquisition and extinction sessions was calculated for each group. In a similar way, the percent CRs for trial types A+ and AX- were calculated for the inhibitory phase.

Results

In each phase of the experiment the mean percentage conditioned responses (CRs) was analyzed by a repeated measures ANOVA with two between factors. The between factors were the type of training during the inhibition phase (Training) and the timing of the reacquisition phase (Test Time). Training had two levels: conditioned inhibition (CI) or excitatory only (E). Similarly, Test Time had two levels: immediate (I) or next day (N). The combination of the two Factors resulted in four groups: conditioned inhibition-immediate (CI-I), excitatory-immediate (E-I), conditioned inhibition-next day (CI-N) and excitatory-next day (E-N). Although the two factors served only as dummy variables in the ANOVA prior to the inhibitory phase, when the Training factor became relevant, and during the reacquisition phase, when the Test Time factor operated, the factors were included in the analyses prior to the inhibitory and reacquisition phases. This was done to ensure that responding was similar across the factors prior to the differential treatments.

Adaptation Phase

During the adaptation session the mean percentage of responses on the observation trials was less than 3 for all groups. The repeated measures ANOVA yielded no significant differences between the sets of means for Test Time ($F(1,25) = .45$), Training ($F(1,25) = .25$) and Test Time X Training ($F(1,25) = 2.17$).

Tone Acquisition Phase

The mean percent CRs over the acquisition phase for the I and N Test Time conditions was 57.9 and 54.4, respectively, while the mean percent CRs for the CI and E Training conditions was 53.3 and 58.9,

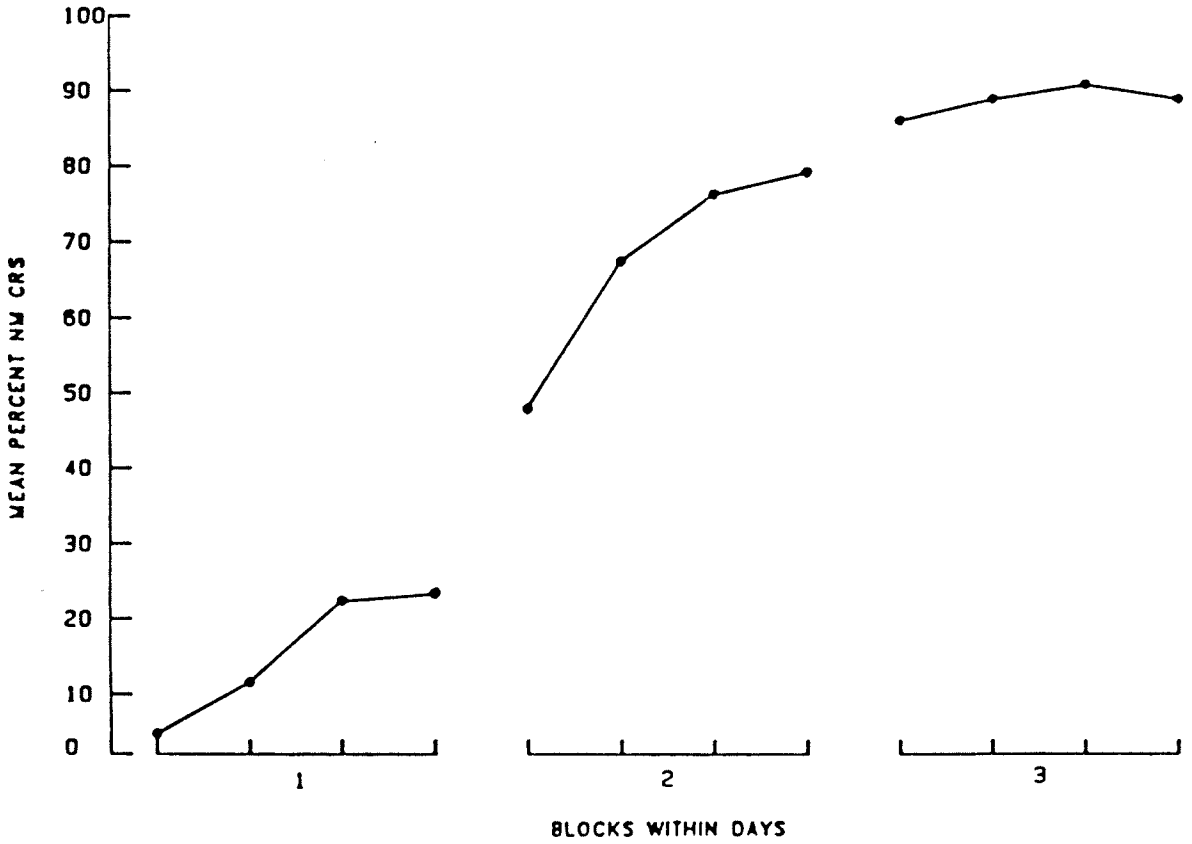
respectively. The similarity of these means resulted in non-significant effects for Test Time ($F(1,25) = .22$), Training ($F(1,25) = .55$) and Test Time X Training ($F(1,25) = 2.07$).

Figure 1 displays the mean percentage of CRs, collapsed across groups, as a function of the four, 24 trial blocks within each daily session of tone acquisition training. Percentage CRs increased over days and over the four blocks within each daily session. The within session increase was largest on Day 2. The graphical interpretation was confirmed by the repeated measures ANOVA which contained significant Days ($F(2,50) = 120.91$, $p < .05$), Blocks ($F(3,75) = 32.28$, $p < .05$) and Blocks X Days ($F(6,150) = 3.66$, $p < .05$) effects. Orthogonal components for trend revealed that percent CRs showed a negatively accelerating increase over Days ($F(1,25) = 54.37$, $p < .05$ and $F(1,25) = 13.11$, $p < .05$ for the linear and quadratic components, respectively) and Blocks ($F(1,25) = 53.37$, $p < .05$ and $F(1,25) = 13.11$, $p < .05$, for the linear and quadratic trends, respectively). Trend analysis applied to the Block X Day interaction yielded a significant linear component ($F(1,25) = 5.07$, $p < .05$) which identified the greater increase in percentage CRs over Day 2, relative to the increases observed over Day 1 and 3. Neither Test Time nor Training interacted with Days or Blocks.

Insert Figure 1 about here

The ANOVA did contain a significant Training X Block X Day interaction ($F(6,150) = 2.43$, $p < .05$). This interaction is plotted in Figure 2 which displays the mean percentage CRs for Condition CI and E as a function of each block within each daily session of tone

Figure 1 Caption: Mean percentage CRs as a function of the four blocks within each of the three days of tone acquisition training.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BLOCKS WITHIN DAYS

acquisition training. The within session increment in percentage CRs for Condition E is substantially larger than Condition CI on Day 1 but much smaller on Day 2 and about the same on Day 3. The reversal in response increments rates on Day 1 and Day 2, for Conditions E and CI, produced the significant interaction.

Insert Figure 2 about here

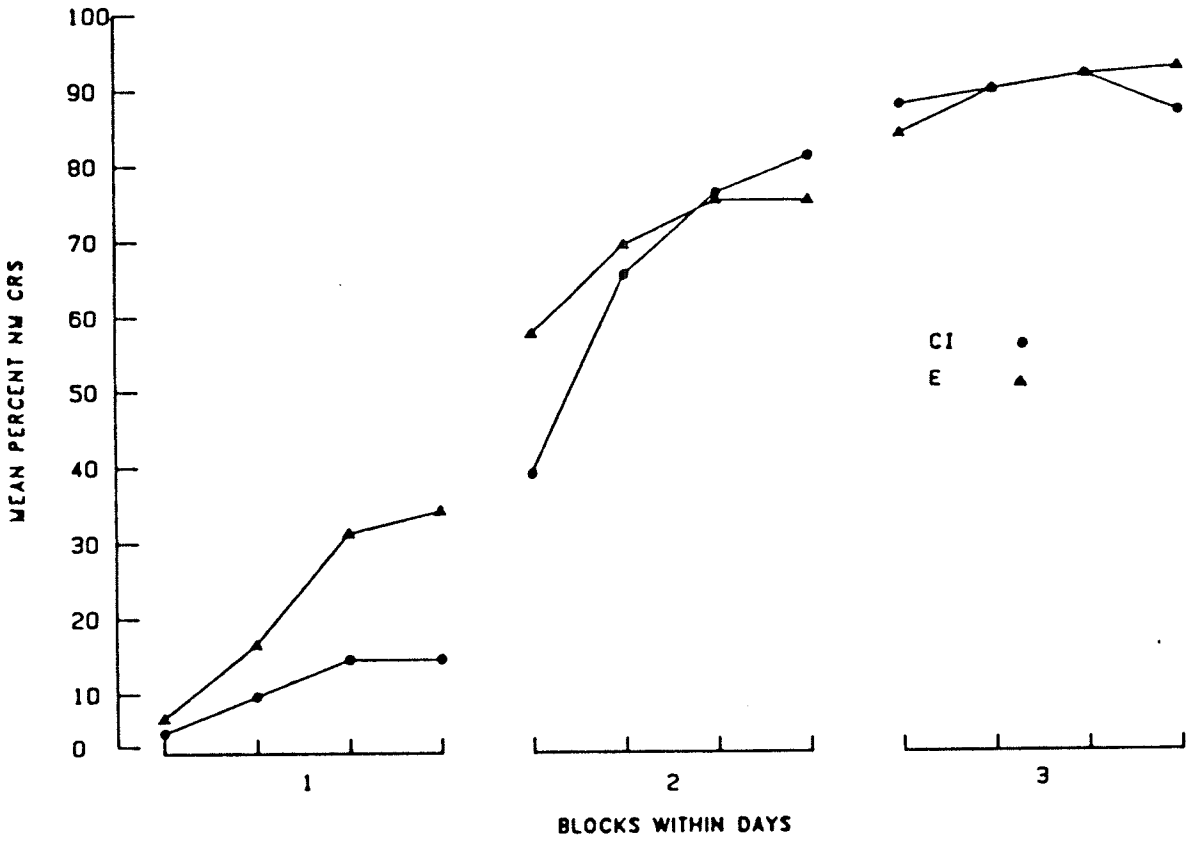
Although CR acquisition rates to the tone differed early in training for Conditions E and CI, convergence in acquisition rates was obtained on Day 3. No other significant effects involving the between-subject factors were obtained. The lack of significant between-subjects effects indicates that the tone was equally excitatory for all groups by the end of the tone acquisition phase.

Inhibitory Phase

During the inhibitory phase, Condition CI received two types of trials in a random sequence. On one-third of the trials, forward tone-shock pairings (A+) were presented, while on the remaining two-thirds of the trials, a simultaneous tone-air compound (AX-) was presented. Since the excitatory control groups received only the A+ trials, a comparison of the performance of all groups on the A+ trials will be presented prior to a description of performance on the inhibitory conditioning trials.

A+ Responding. The mean percentage of CRs during the A+ trials over the inhibitory phase was high (95.8%) and fairly constant. The low variability in percentage CRs resulted in significant effects with small differences between means. The mean percentage CRs during the A+ trials

Figure 2 Caption. Mean percentage CRs as a function of the four blocks within each day of tone acquisition training for Conditions CI and E.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for the I and N Test Time Conditions was 95.5 and 96.2, respectively, while the mean percentage CRs was 94.0 and 97.6 for the CI and E Training Conditions, respectively. The repeated measures ANOVA identified a significant Training main effect ($F(1,25) = 5.07$, $p < .05$) but neither a Test Time main effect ($F(1,25) = 0.20$) nor a Test Time X Training interaction ($F(1,25) = .01$).

The mean percentage CRs across blocks of A+ trials (i.e. collapsed across groups and days) was 96.7, 96.2, 95.6 and 94.1 for Blocks 1 through 4, respectively. The decrease in responding over blocks resulted in a Block main effect ($F(3,75) = 3.15$, $p < .05$). Trend analysis applied to the Block effect confirmed that the decrease was linear ($F(1,25) = 5.55$, $p < .05$). Also, the Training X Block interaction approached significance ($F(3,75) = 2.41$, $p < .08$). Trend analysis applied to the Training X Block interaction yielded a significant difference between the linear components to the interaction ($F(1,25) = 4.07$, $p < .05$). The difference in linear trend was caused by a greater decrease in responding over blocks for condition CI ($F(1,25) = 7.98$, $p < .05$) compared to the fairly constant rate of responding over blocks for condition E ($F(1,25) = 0.18$, $p > .05$).

There were no other significant effects with respect to blocks nor were there any effects with respect to days. Thus, the only deviation from stable asymptotic responding on the A+ trials was the within session decrease in percent CRs for condition CI.

AX- Responding. Responding during the inhibitory conditioning trials was analyzed for Groups CI-I and CI-N by a repeated measures ANOVA with one between factor (Test Time). The mean percent of CRs to

the AX- compound, over the entire inhibitory training phase, was 83.8 and 78.8 for Groups CI-I and CI-N, respectively. The ANOVA did not yield a significant difference between the group means ($F(1,14) = 0.18$).

Figure 3 displays the mean percent of CRs to the AX- compound, collapsed across Groups CI-I and CI-N, as a function of days of inhibitory training. The figure shows that responding was maintained at a fairly constant level, around 85 percent, and then gradually decreased in level after Day 5. The decrease in responding to the AX- trials was reflected in the ANOVA as a significant Day main effect ($F(7,98) = 3.75$, $p < .05$). Trend analysis applied to the Day effect indicated that the decrement in responding had only a significant linear component ($F(1,14) = 5.98$, $p < .05$).

Insert Figure 3 about here

The mean percentage CRs to the AX- compound for each block of inhibitory training, collapsed across Groups and Days, was 89.1, 83.6, 78.6 and 73.9 for Blocks 1 through 4, respectively. The steady decrease in responding over blocks was reflected in the ANOVA by a significant Block main effect ($F(3,42)$, $p < .05$). Orthogonal trend analysis applied to the block effect revealed that the decrease was linear ($F(1,14) = 4.41$, $p < .05$). And finally, there was a significant Block X Day interaction ($F(21,294) = 1.63$, $p < .05$) which resulted from larger decrements occurring within a session as the number of days increased. The Test Time factor did not interact with either Blocks ($F(1,14) = 1.75$) or Blocks X Days ($F(21,294) = 1.01$).

Figure 3 Caption. Mean percentage CRs to the AX- trials for each of the eight days of inhibitory training.

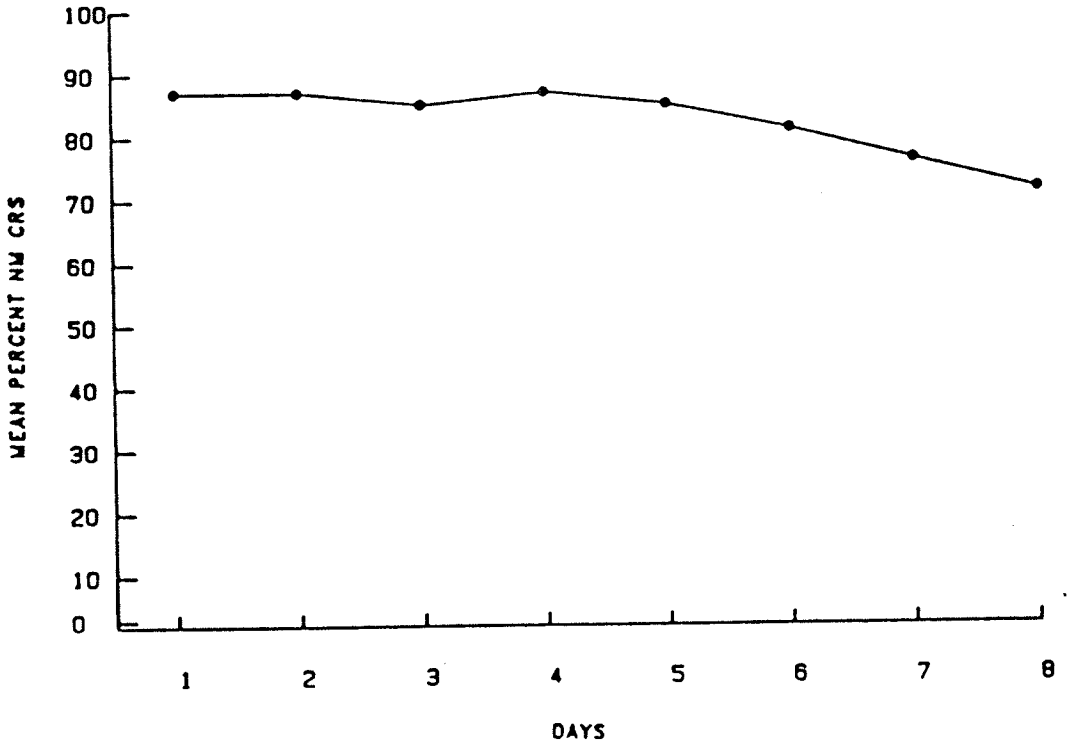
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Figure 4 displays the mean percentage of CRs during the A+ and AX- trials, collapsed across Groups CI-I and CI-N, as a function of each block within each day of inhibitory training. Responding to the A+ trials was higher than responding to the AX- trials. This discrepancy was fairly constant over blocks for the first three days. However, during Day 4 the difference in percentage CRs between the A+ and AX- trials increased over blocks within a session. This pattern persisted over the remaining days of training, thus the discrepancy between the two trial types increased. A repeated measures ANOVA confirmed the graphical interpretation by yielding significant effects for Trial Type ($F(1,14) = 5.68$, $p < .05$), Day X Trial Type ($F(7,98) = 2.89$, $p < .05$) and Block X Trial Type ($F(3,42) = 7.40$, $p < .05$).

Figure 4 also suggests that responding during the AX- trial during Block 1 was higher than responding during Block 4 of the previous session. To determine whether the between session recovery was a reliable effect, a separate ANOVA with Block 4 versus Block 1, and the number of times the Block 4 - Block 1 transitions occurred (transitions) as within factors was conducted. The ANOVA indicated that the percentage CRs during Block 1 was significantly higher than the percentage CRs during Block 4 of the previous day ($F(1,14) = 9.58$, $p < .05$). In addition, percentage CRs decreased over Transition ($F(6,84) = 2.60$, $p < .05$) which confirmed the lower levels of AX- responding that developed over inhibitory conditioning. No other significant effects were identified. Thus, there was a similar

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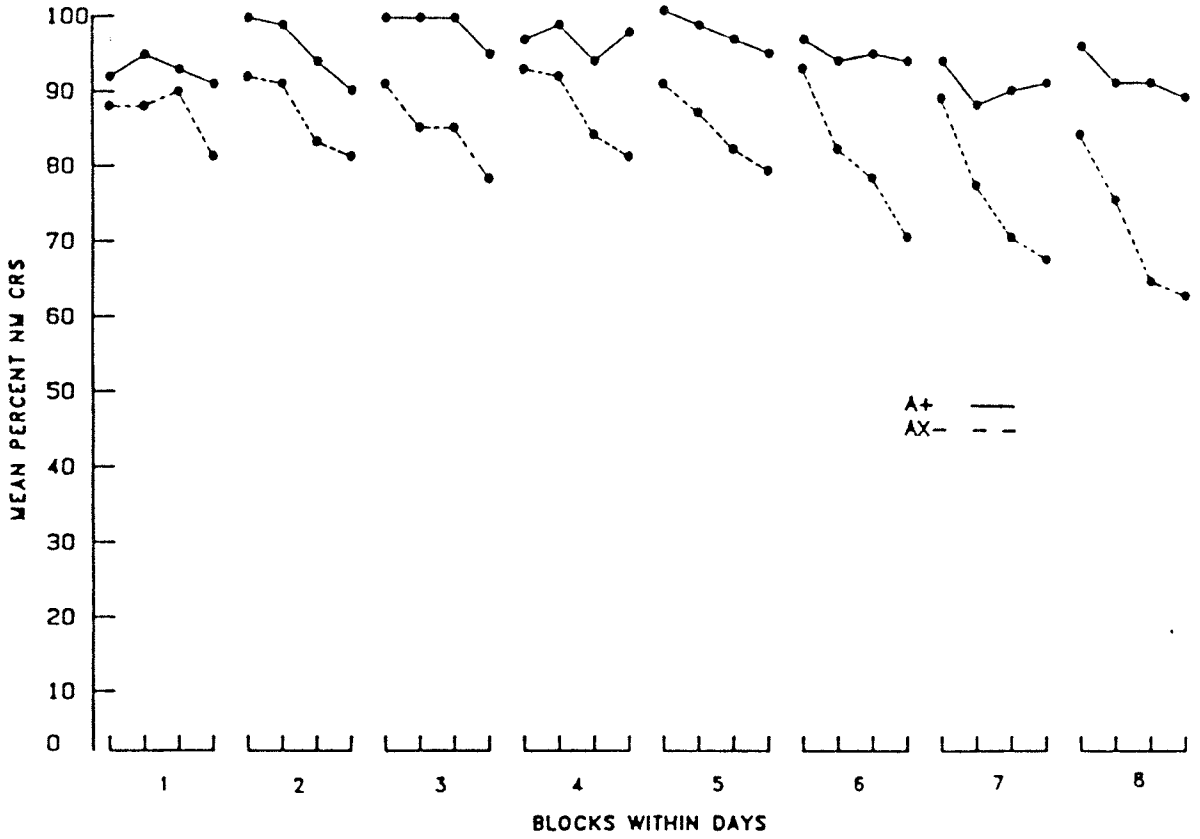
Insert Figure 4 about here

Figure 4 Caption. Mean percentage CRs during both the A+ and AX- trials as a function of each block within each day of inhibitory training, collapsed across Groups CI-I and CI-N.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30
20
10
0
MEAN PERCENT NW CRS
A+
AX–
1
2
3
4
5
6
7
8
BLOCKS WITHIN DAYS

significant recovery of percentage CRs between sessions for Groups CI-I and CI-N.

Air Reacquisition Phase

A repeated measures ANOVA with two between factors, Test Time and Training, was used to analyze the data arising from the reacquisition phase. Subsequently, the hypotheses central to the thesis were assessed by a priori tests which included contrasts between the experimental Groups, CI-I and CI-N, and their respective controls, Groups E-I and E-N.

The mean percentage CRs during the air-shock trials, over the reacquisition phase, for the I and N Test Time conditions was 88.8 and 89.7, respectively, while the mean percentage CRs for the CI and E Training Conditions was 88.1 and 90.0, respectively. The similarity of the means was reflected in the ANOVA which did not yield significant effects for Test Time ($F(1,25) = .14$), Training ($F(1,25) = .31$) and Test Time X Training ($F(1,25) = .58$).

The mean percentage CRs for Sessions 1 and 2 was 83.4 and 94.4, respectively. The ANOVA confirmed the higher response levels during Session 2 by yielding a significant Session effect ($F(1,25) = 27.22$, $p < .05$). The ANOVA also revealed significant Session X Test Time ($F(1,25) = 11.92$, $p < .05$) and Session X Training ($F(1,25) = 5.13$, $p < .05$) interactions. The Session X Test Time interaction is plotted in Figure 5 which displays the mean percentage CRs for the I and N Test Time Conditions as a function of sessions. Figure 5 shows that during the first session, responding was lower in Condition I relative to Condition N. However, during Session 2, Condition I was slightly higher

than Condition N. A Student Neuman-Keuls (SNK) test applied to the Session X Test Time interaction confirmed the graphical interpretation by indicating that the percentage CRs for Condition I was significantly lower than both the percentage CRs for Condition N during Session 1 and the percentage CRs for Conditions I and N during Session 2. Thus, the analysis suggests that an immediate test for inhibition retarded reacquisition performance.

Insert Figure 5 about here

The Session X Training interaction is plotted in Figure 6 which displays the mean percentage CRs for Conditions CI and E as a function of the two reacquisition sessions. Figure 6 indicates that the percentage CRs was lower for Condition CI relative to Condition E during Session 1, but the difference between the conditions disappeared as the Conditions converged on a higher value in Session 2. A SNK test applied to the Session X Training interaction confirmed the graphical interpretation by indicating that percentage CRs for Condition CI was significantly lower than the percentage CRs for Condition E in Session 1 and the percentage CRs for Conditions CI and E in Session 2. In addition, the SNK test confirmed that the Session 1 percentage CRs for Condition E was significantly lower than the percentage CRs for Conditions CI and E during Session 2. Thus, the analysis suggests that prior inhibition training retarded reacquisition.

Insert Figure 6 about here

Figure 5 Caption. Mean percentage CRs as a function of each reacquisition session for Conditions I and N.

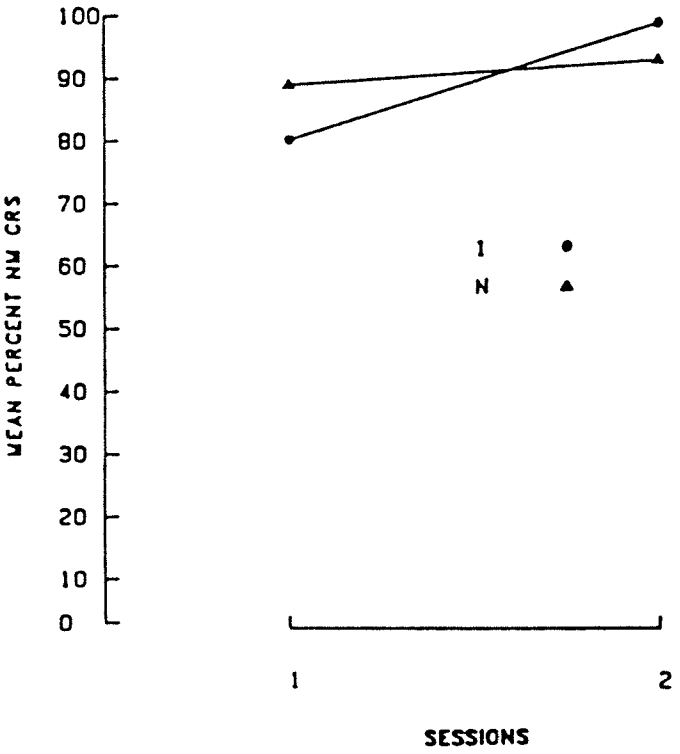
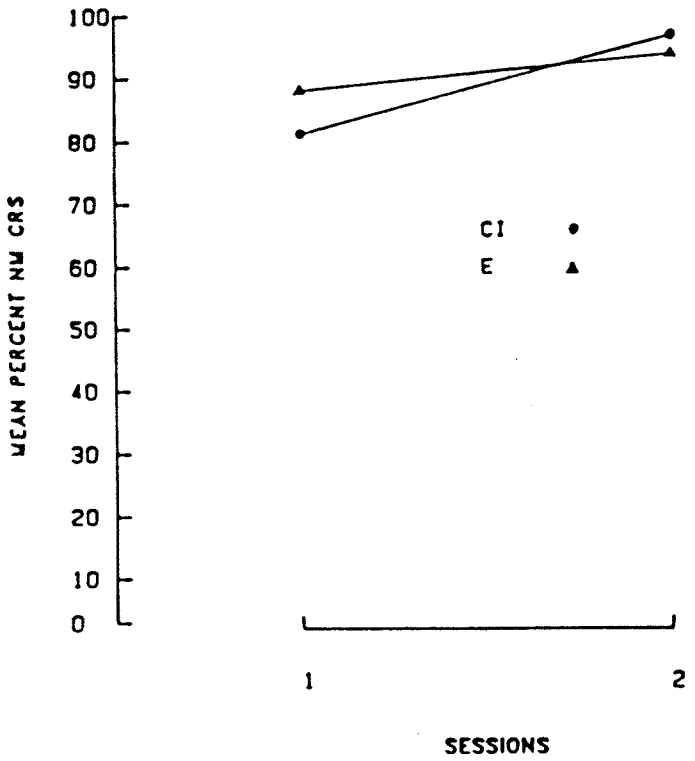

100
90
80
70
60
50
40
30
20
10
0
MEAN PERCENT NW CRS
I
N
1
2
SESSIONS

Figure 6 Caption. Mean percentage CRs as a function of each reacquisition session for Conditions CI and E.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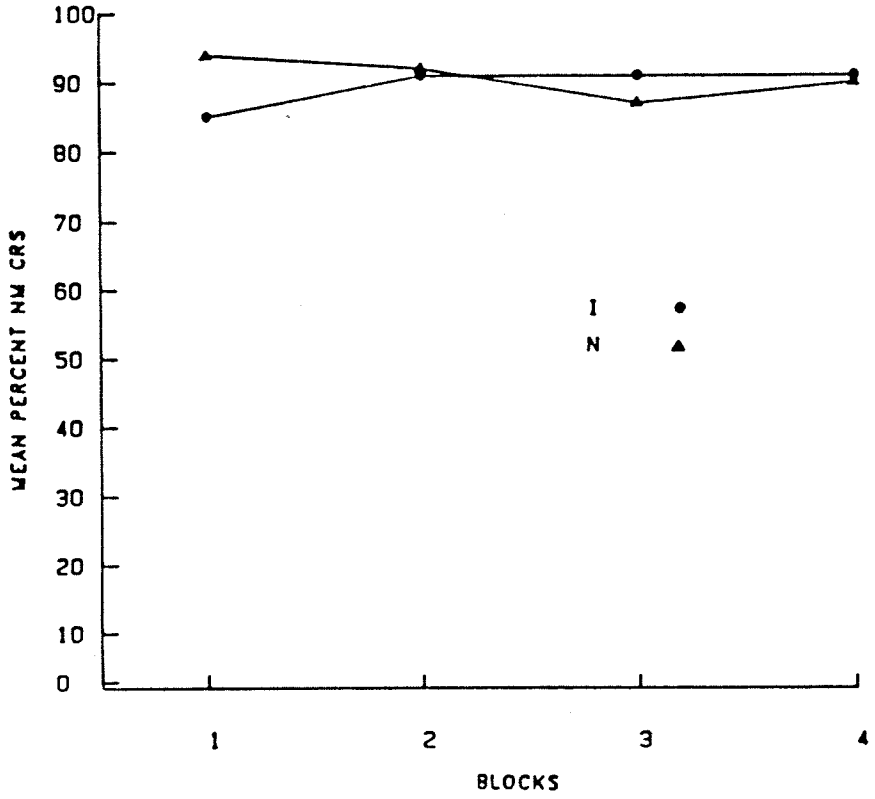
0
MEAN PERCENT NW CRS
CI
E
1
2
SESSIONS

The different rates of reacquisition that produced the significant Session X Test Time interaction were also apparent over the four, 24 trial blocks within each session. The repeated measures ANOVA yielded a significant effect for Block X Test Time ($F(3,75) = 4.34$, $p < .05$). This interaction is plotted in Figure 7 which displays the mean percentage CRs for Conditions I and N as a function of the four blocks of reacquisition, collapsed across sessions. Figure 7 shows that Condition I exhibited a negative accelerating increase in percentage CRs over blocks, whereas Condition N exhibited a negatively accelerating decrease. Orthogonal components for trend applied to the Block X Test Time interaction confirmed the graphical interpretation by yielding significant linear ($F(1,25) = 4.73$, $p < .05$) and quadratic ($F(1,25) = 5.87$, $p < .05$) components to the interaction.

Insert Figure 7 about here

The ANOVA also yielded significant effects for Session X Block ($F(3,75) = 4.11$, $p < .05$), and Session X Block X Test Time ($F(3,75) = 4.30$, $p < .05$). The Session X Block interaction is plotted in the upper frame of Figure 8 which displays the mean percentage CRs during the air-shock trials as a function of the four blocks within each session of reacquisition training. Percentage CRs increased over blocks during Session 1, but decreased over blocks during Session 2. This graphical interpretation was confirmed by orthogonal trend analysis which revealed significant linear ($F(1,25) = 6.58$), $p < .05$) and quadratic ($F(1,25) = 4.25$, $p < .05$) components to the interaction. The linear and quadratic components of the Session X Block X Test Time interaction were also

Figure 7 Caption. Mean percentage CRs as a function of each block of reacquisition training, collapsed across Sessions, for Conditions I and N.


100
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MEAN PERCENT NW CRS
I
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1
2
3
4
BLOCKS

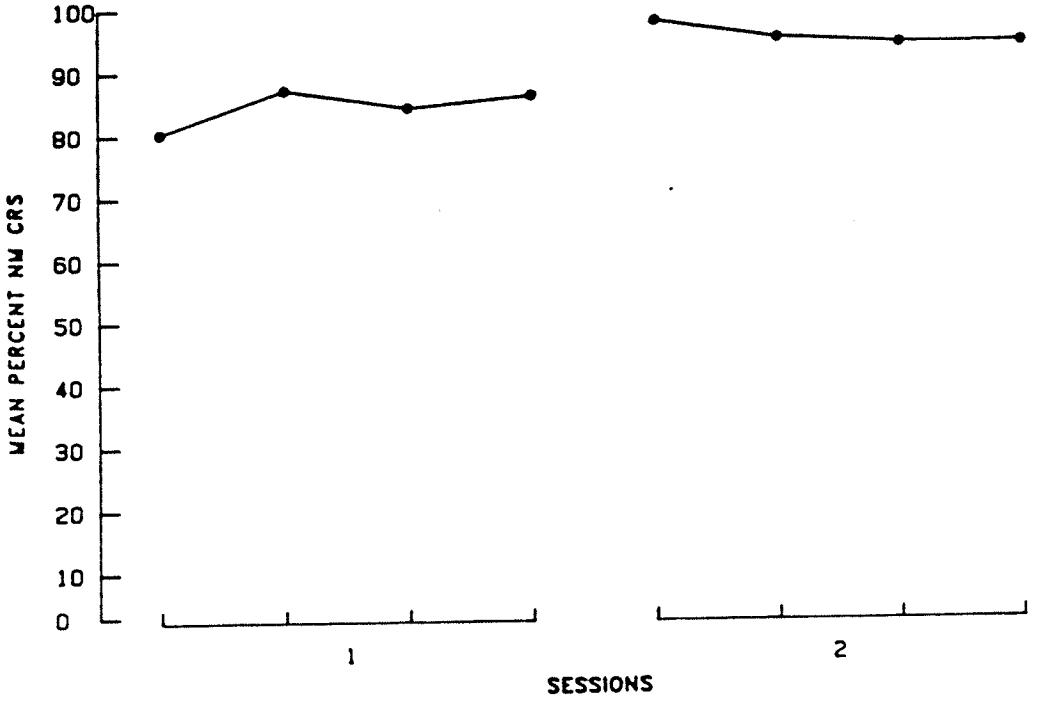
significant ($F(1,25) = 7.03$, $p < .05$) and $F(1,25) = 4.36$, $p < .05$, respectively). The trend components for the I and N Test Time conditions are plotted in the lower frame of Figure 8 which displays the mean percentage CRs as a function of blocks within each session. The figure shows that during the first session Condition I exhibited a negatively accelerated increase in percentage CRs whereas Condition N exhibited a negatively accelerated decrease. During Session 2 the linear decrease in responding was more pronounced for Condition N relative to Condition I.

Insert Figure 8 about here

The contrasts central to the present study are between Groups CI-I and E-I; Groups CI-N and E-N; and Groups CI-I and CI-N. Since the air-shock acquisition occurred rapidly, with asymptotic convergence of all groups completed by Session 2, it is possible that the overall analysis of reacquisition masked the observation of differences during reacquisition. Therefore, a separate analysis which included the *a priori* tests was conducted on just Session 1 results.

Figure 9 displays the mean percentage CRs for each group as a function of the 24 trial blocks of the first reacquisition session. During Block 1, responding was low for Group CI-I, moderate for Group E-I and high for Groups CI-N and E-N. Responding for all groups converged over the remaining blocks. As a consequence, the repeated measures ANOVA did not identify either significant Group ($F(3,25) = 1.93$) or Block ($F(3,75) = 1.94$) effects. However, the Group X Block interaction was significant ($F(9,75) = 2.65$, $p < .05$). A SNK

Figure 8 Caption. Upper frame: Mean percentage CRs as a function of blocks for each of the 2 reacquisition sessions. Lower frame: Mean percentage CRs as a function of each block within each reacquisition session for Conditions I and N.


100
90
80
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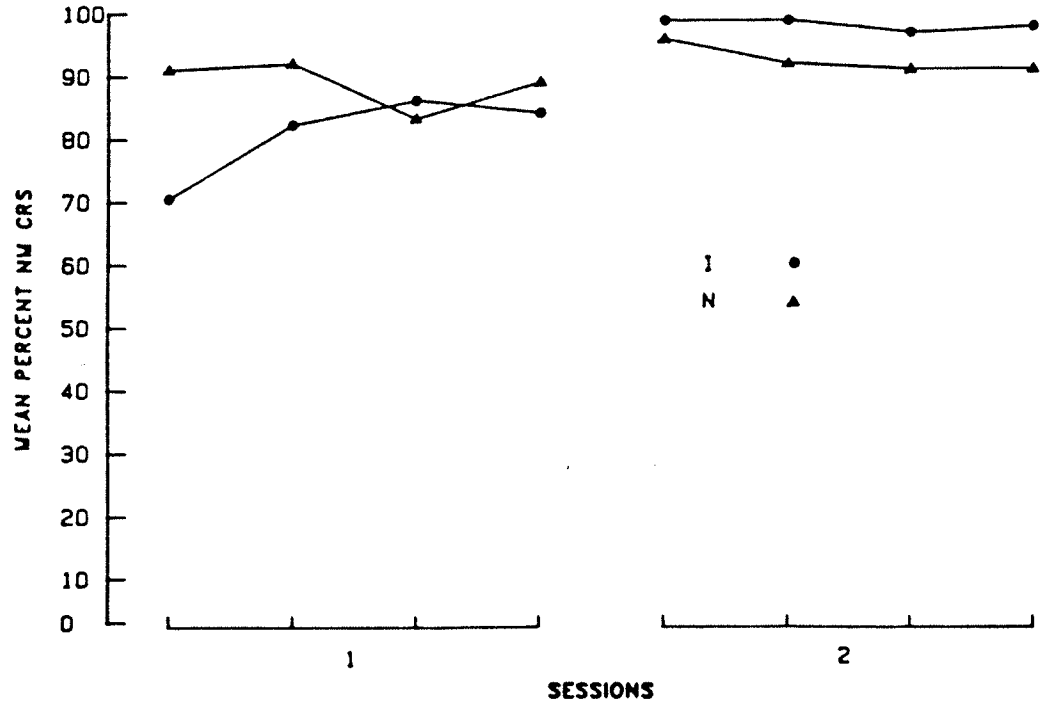
30
20
10
0
MEAN PERCENT NW CRS
1
2
SESSIONS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MEAN PERCENT NW CRS
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2
SESSIONS

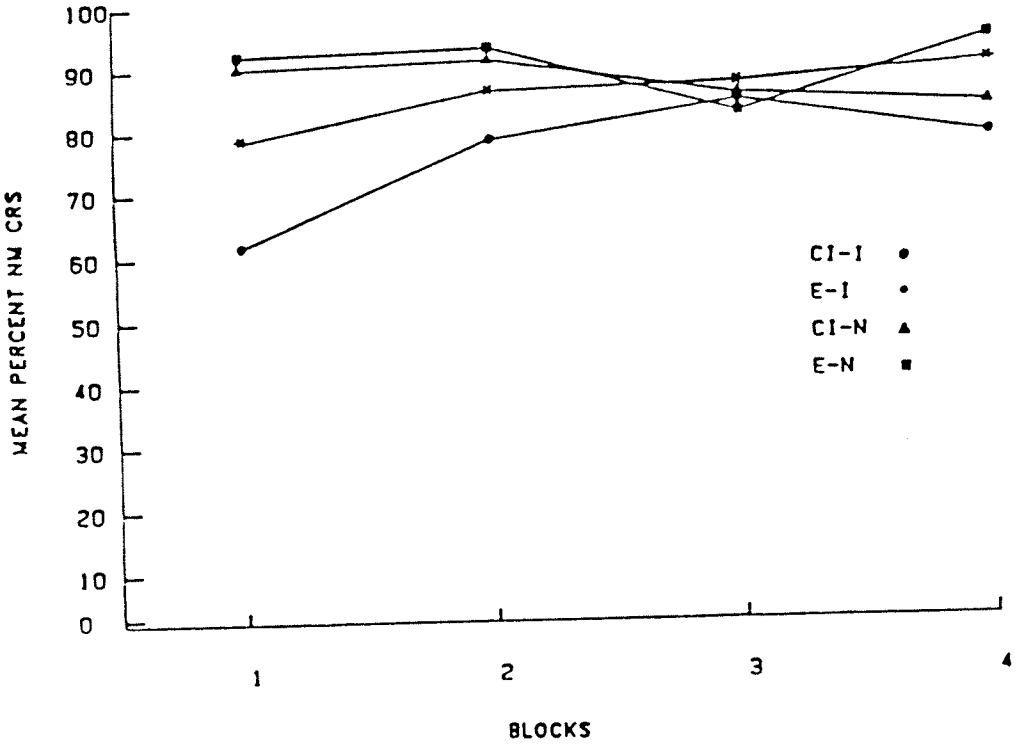
test applied to the Group X Block interaction indicated that during Block 1, conditioned responding was significantly lower for Group CI-I relative to Group E-I, CI-N and E-N. Furthermore, percentage CRs for Group CI-I, during Block 1, was significantly lower relative to all the possible remaining Block X Group combinations. No other contrasts were significant. Orthogonal trend analysis applied to the Block X Group interaction yielded significant linear ($F(3,25) = 3.25$, $p < .05$) and quadratic ($F(3,25) = 3.41$, $p < .05$) components to the interaction. Individual trend analysis revealed that only Group CI-I had reliable linear and quadratic components over blocks of Session 1 ($F(1,7) = 6.60$, $p < .05$) and $F(1,7) = 14.36$, $p < .05$, respectively). Thus the increase in responding over Session 1 in Group CI-I was the primary effect that produced the significant Group X Block interaction. Also, it seems that the initial deficit in responding for Group CI-I caused the significant Session X Test Time interaction because responding for Group E-I did not differ from Groups CI-N and E-N during the first reacquisition session.

Insert Figure 9 about here

Air Extinction Phase

The mean percentage CRs during the air trials of the extinction phase for the I and N Test Time Conditions was 60.4 and 69.7, respectively, while the mean percent CRs for the CI and E Training Conditions was 64.3 and 65.7, respectively. The repeated measures ANOVA indicated that the differences within the sets of means were not significant ($F(1,25) = .80$ and $F(1,25) = .02$, for Test Time and Training, respectively). However, the Test Time X Training interaction

Figure 9 Caption. Mean percentage CRs as a function of each block of the first reacquisition Session for Groups CI-I, E-I, CI-N, E-N.


100
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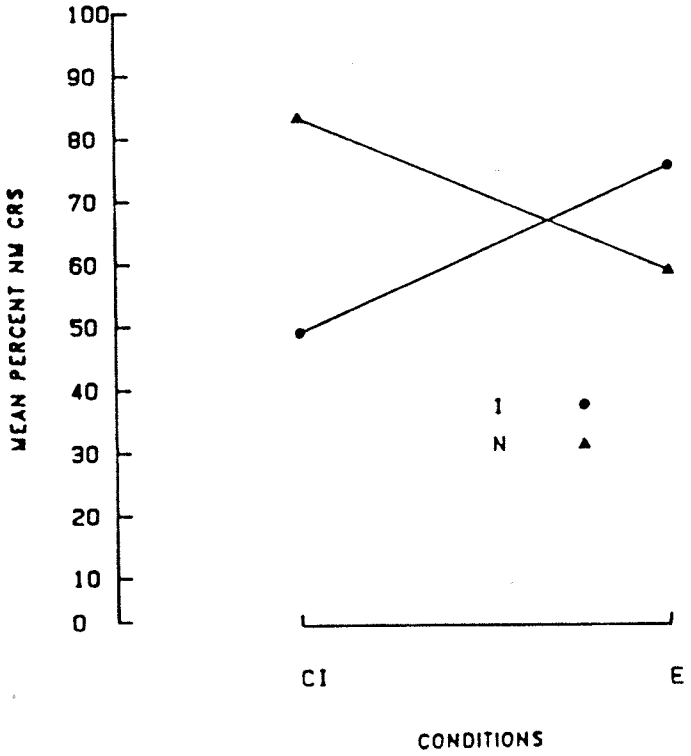
0
MEAN PERCENT NW CRS
CI-I
E-I
CI-N
E-N
1
2
3
4
BLOCKS

was significant ($F(1,25) = 6.36$, $p < .05$). This interaction is plotted in Figure 10 which displays the mean percent CRs in extinction for Conditions CI and E as a function of Conditions I and N. The percentage CRs was lower for Group CI-I relative to Group CI-N, but slightly higher for Group E-I relative to Group E-N. A SNK test applied to the interaction revealed that the difference between Groups CI-I and both Groups CI-N and E-I approached significance ($p < .10$). Groups CI-N, E-I and E-N did not differ ($p > .10$).

Insert Figure 10 about here

The mean percentage of CRs over the four, twenty-four trial blocks of extinction was 88.6, 69.2, 53.0 and 47.8 for Blocks 1 through 4, respectively. This decrease over blocks resulted in a significant Block main effect ($F(3,75) = 27.71$, $p < .05$). Orthogonal components for trend applied to the Block effect identified a negative decelerating extinction function ($F(1,25) = 43.50$, $p < .05$) and $F(1,25) = 5.37$, $p < .05$, for the linear and quadratic trends, respectively). The quadratic component over blocks was affected by the interaction between Test Time and Training ($F(1,25) = 7.56$, $p < .05$). This interaction is plotted in Figure 11 which displays the mean percentage of CRs to the air CS, for Groups CI-I, CI-N, E-I and E-N, as a function of the four blocks of extinction. Both Groups CI-I and E-N showed faster rates of extinction over Blocks 1 and 2 relative to Groups CI-N and E-I. Over Blocks 3 and 4, Groups CI-N and E-I continued to show a steady decrease in percentage CRs while Groups CI-I and E-N exhibited more or less constant rates of

Figure 10 Caption. Mean percentage CRs during extinction, for Condition I and N as a function of Conditions CI and E.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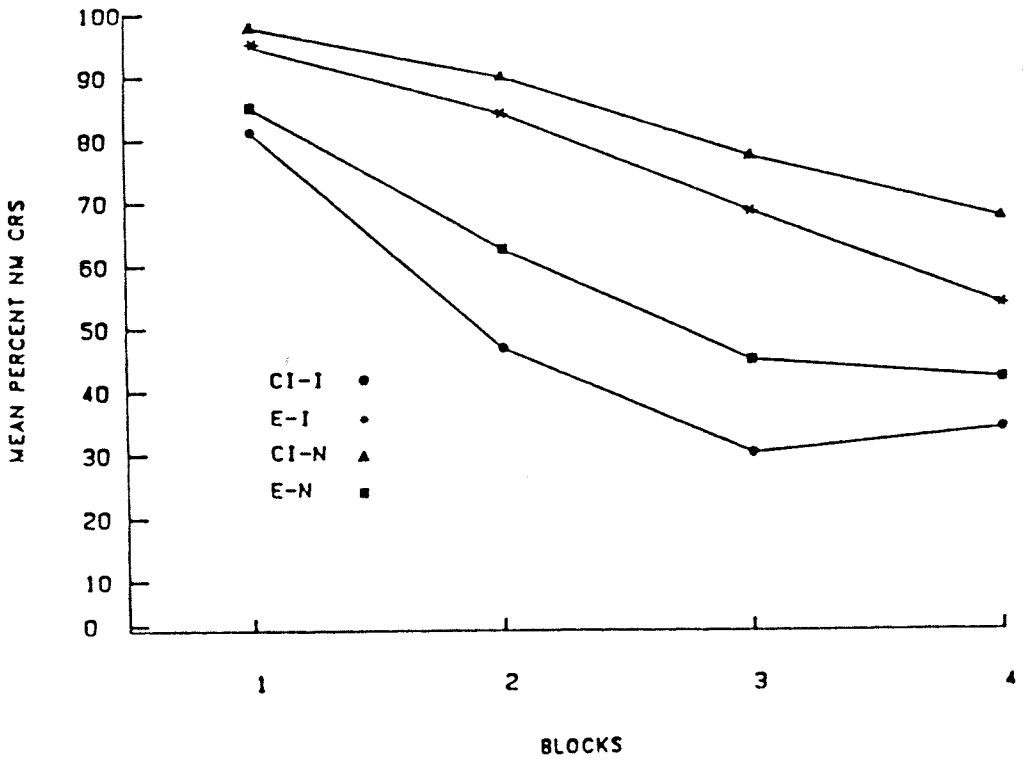
MEAN PERCENT NM CRS
I
N
CI
E
CONDITIONS

responding. Individual trend analysis revealed that this pattern of responding resulted in quadratic trends for both Groups CI-I ($F(1,7) = 8.08$, $p < .05$) and E-N ($F(1,6) = 7.06$), $p < .05$), but not Groups CI-N and ($F(1,7) = .03$) and E-I ($F(1,5) = .06$).

Insert Figure 11 about here

Figure 11 Caption. Mean percentage CRs as a function of each block of extinction for Groups CI-I, E-I, CI-N and E-N.


MEAN PERCENT NW CRS
100
90
80
70
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50
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30
20
10
0
CI-I
E-I
CI-N
E-N
1
2
3
4
BLOCKS

Discussion

The major findings of the present study are: a) Condition CI showed small within session decrements on the A+ trials during the inhibitory phase whereas Condition E did not; b) Condition CI showed large within session decrements on the AX- trials during the inhibitory phase; c) Condition CI showed a recovery in responding on the AX- trials between inhibitory sessions; d) Condition CI responded less on the AX- trials relative to the A+ trials during the inhibitory phase; e) Group CI-I but not Group CI-N showed retarded acquisition to X during reacquisition and f) Group CI-I seemed to show a faster rate of extinction to X relative to Group CI-N. Each finding will be discussed in turn.

During the A+ trials of the inhibitory training phase fewer CRs were observed for Condition CI relative to Condition E. Although the magnitude of this difference was small, it was significant and is consistent with previous studies that have examined the conditioned inhibition paradigm with the rabbits' nictitating membrane response (e.g. Marchant & Moore, 1974). The poorer performance on the A+ trials for Condition CI can be accounted for within the framework of both the Rescorla-Wagner theory (1972) and Wagner's SOP model (1981). According to Rescorla and Wagner (1972) a stimulus gains associative strength when it is followed by an unconditioned stimulus (US) and loses associative strength when it is not followed by a US. During the inhibition phase A was reinforced (followed by a US) during the A+ trials but A was not reinforced during the AX- trials. Therefore, the response decrements during the A+ trials for Condition CI may have resulted from a loss of associative strength to A incurred during the AX- trials.

A different account of the response decrements on the A+ trials arises from the SOP model (Wagner, 1981). According to SOP, an excitatory association between A and the US is formed during the A+ trials because of temporally overlapping A1 states produced by the presence of each stimulus. As a consequence, stimulus A will elicit a US A2 state during the AX- trials. On the AX- trials the US A2 state is concomitantly paired with the A1 state of both A and X. The simultaneous US A2 and A A1 states results in an inhibitory association which algebraically subtracts from the existing excitatory association between A and the US and reduces the net associative strength of A. The weakening of the excitatory association would result in response decrements on the A+ trials (Wagner, 1981).

During the inhibitory phase, the magnitude of the within session decrements on the AX- trials increased over training and resulted in significantly fewer CRs during the AX- trials relative to the A+ trials. The pattern of within session decrements to the AX- compound was also observed in the study by Williams et al. (Appendix). Both Rescorla and Wagner (1972) and Wagner (1981) can account for the response decrements to the AX- compound over inhibitory training. Both accounts are based on the assumption that associative inhibition with respect to the US is acquired by X, although the explanatory mechanisms central to the two theories differ. Rescorla and Wagner (1972) believe that negative associative strength is acquired by X because X is presented simultaneously with an excitatory stimulus in the absence of reinforcement. On the other hand, Wagner (1981) contends that X acquires an inhibitory association with the US during the AX- trials because the X A1 state becomes paired with the US A2 state produced by the presence of the excitatory A stimulus.

The pattern of response recovery to the AX- compound between consecutive sessions of conditioned inhibition training was observed in the Williams et al. study (Appendix) and was replicated in the present study. There are two possible reasons for the recovery between sessions. First, it is possible that the strength of the excitatory association between A and the US supercedes the putative inhibitory association between X and the US at the start of each daily inhibitory session. Implicit in the assertion that A may dominate responding, is the notion of differential forgetting rates for excitatory and inhibitory associations, such that over the 22 hour interval between sessions the inhibitory associations are more readily forgotten. Some support for the labile nature of inhibitory associations arose from a study by Hendersen (1978) who found that excitatory associations were better remembered than inhibitory associations over long intervals. The intervals used in the Hendersen (1978) study were in the order of 25 to 35 days and thus not directly generalizable to the 22 hour interval inherent in the present study. However, Hendersen's (1978) findings lend credence to the possibility that inhibitory associations may be more readily forgotten than excitatory associations.

Neither the Rescorla and Wagner (1972) nor Wagner (1981) theories, in the present form, have accounted for the possibility of differential forgetting rates between excitatory and inhibitory associations. Rescorla and Wagner (1972) are less able to accommodate differential forgetting rates as a cause of response recovery to the AX- trials because the presence of an excitatory source should enhance, not override, the behavioral indices of associative inhibition accrued by X. Therefore, the Rescorla-Wagner (1972) model would predict greater

inhibition during the first block of an inhibitory session, a time period prior to the loss of excitatory associative strength to both A and the context, through non-reinforcement. In the present study, however, inhibition was least pronounced during the first block of an inhibitory training session.

An alternative account of the observed response recovery to the AX- trials between inhibitory training sessions could stress the spontaneous recovery of the excitatory strength to X. Rescorla and Wagner (1972) acknowledge that X acquires excitatory strength early in conditioned inhibition training. Within the SOP model (Wagner, 1981) X can acquire excitatory associative strength through its excitatory association with A which has an excitatory association with the US. If, over conditioned inhibition training, X accrues extinctive inhibition, then spontaneous recovery of X's excitatory strength between sessions would be expected. Thus, both theoretical positions contain mechanisms that would allow a between session recovery in response levels to result from spontaneous recovery.

During the reacquisition phase, Group CI-I showed evidence of associative inhibition to X but Group CI-N did not. It appears that the timing of the reacquisition test affected the detection of associative inhibition. The finding that X accrued inhibitory properties in Group CI-I indicates that the observed within session decrements to the AX- compound were due to associative inhibition accruing to X. Detection of associative inhibition after conditioned inhibition training is congruent with both the Rescorla and Wagner (1972) theory and its derivatives (Frey & Sears, 1978; Moore, 1982 and Pearce & Hall, 1980), as well as Wagner's SOP model (1981).

The lack of associative inhibition for Group CI-N, during reacquisition, although consistent with the Williams et al. study (Appendix) is more difficult to reconcile theoretically. Rescorla and Wagner (1972) predict that X should be inhibitory after conditioned inhibition training regardless of any time parameters or associations with other stimuli. On the other hand, Wagner (1981) allows for associations between stimuli to affect behavior. Specifically, an excitatory association between A and X should form during the AX- trials (Wagner, 1981). As a consequence of the reacquisition test, the presentation of X arouses an A A2 state which in turn produces a US A2 state. The US A2 state results in a conditioned response. Thus early in reacquisition, CRs could result from the presentation of X. As X is followed by a US during reacquisition, the subsequent excitatory association between X and the US would maintain high levels of CRs. However, high levels of responding would be expected for both conditioned inhibition groups. Yet, Group CI-I demonstrated a lower level of percent CRs during reacquisition. It appears that at the time of testing, the inhibitory association was stronger than the excitatory association between X and the US for Group CI-I relative to Group CI-N. Thus, for Wagner to account for the present results, he would need to add the hypothesis that inhibitory associations are forgotten or disrupted more easily than excitatory associations.

Hendersen (1978) provided some support for the labile nature of inhibitory associations over 25 day intervals. Williams et al. (Appendix), however, found little evidence of forgetting of inhibition over a 22 hour interval when associative inhibition was produced with differential and explicitly unpaired paradigms. The

persistence of inhibitory associations over the shorter intervals suggests that inhibition can be remembered over the intervals imposed in the present study. Therefore, invoking differential forgetting rates of excitatory and inhibitory associations, to account for the lack of association inhibition for Group CI-N, is tenuous.

One suggestion which accounts for the differences between Groups CI-I and CI-N that does not rely on differential forgetting rates is that of spontaneous recovery of the excitatory strength of X occurring over the interval preceding reacquisition for Group CI-N. Implicit in the argument for spontaneous recovery is that during conditioned inhibition training, X acquires an excitatory association with the US as well as an inhibitory association. Both Konorski (1967) and Wagner (1981) predict that during conditioned inhibition training, X acquires excitatory strength from A on the AX- trials. Evidence of X's excitatory strength is inferred from the high levels of responding to the AX- compound. The inhibitory associations are inferred from the within session decrements in responding to the AX- trials. If the reacquisition test is given when the inhibitory strength of X is greatest, i.e. at the end of the inhibitory training session, then inhibition should be detected. The finding of associative inhibition for Group CI-I is consistent with the notion that the within session decrements were due to inhibition accruing to X. The interval imposed between the last inhibitory training session and reacquisition for Group CI-N, however, allows for the spontaneous recovery of any excitatory strength accrued by X. Thus, the excitatory strength of X could obfuscate the measurement of inhibition for Group CI-N.

During the second reacquisition session, responding converged for all groups. Thus, at the end of reacquisition, the excitatory associative strength to X should be the same for all groups (Rescorla & Wagner, 1972). Yet, during X extinction, Group CI-I extinguished more rapidly than Groups CI-N and E-I. Because of the variability inherent in the extinction phase (Konorski, 1967), this finding was only significant at the .10 level. However, it is consistent with Williams et al. (Appendix) who found that groups demonstrating inhibition during reacquisition extinguished more rapidly than groups not showing evidence of inhibition. If the current associative strength of X is the only determinant of the rate of extinction (Rescorla & Wagner, 1972), then all groups should have extinguished at the same rate because X was equally excitatory for all groups by the end of the reacquisition phase. Similarly, if the current strength of associations between stimuli determines performance (Wagner, 1981), then at the commencement of extinction training X should elicit a US A2 state of equal strength for all groups. Therefore, the formation of inhibitory associations between X and the US during extinction should not differ among the groups. The finding that Group CI-I extinguished more rapidly than Groups CI-N and E-I contradicts the predicted no difference of both Rescorla and Wagner (1972) and Wagner (1981).

Contrary to the positions of Rescorla and Wagner (1972) and Wagner (1981), Konorski (1967) asserted that the past training history of a stimulus is crucial to the understanding of current behavior to that stimulus. For Konorski (1967) all stimuli, whether CSs or USs, are represented in the central nervous system by systems of neurons which he called gnostic units. A gnostic unit contains information regarding the

physical characteristics of the stimulus. USs have a second representation called a drive unit, which represents the motivational consequences of a US. Thus an aversive US would evoke a gnostic unit associated with somatic pain information and drive units which reflect fear information. In addition, each drive unit is reciprocally related to an antidrive unit which, when activated, inhibits the drive unit.

The development of excitatory CRs in defensive conditioning is equivalent to the establishment of linkages between the CS gnostic units and both the US gnostic units and drive units (Konorski, 1967). The development of inhibitory CRs in defensive conditioning is equivalent to the establishment of linkages between the CS gnostic units and the antidrive units (Konorski, 1967). Thus, within the conditioned inhibition paradigm, A would develop linkages to the fear drive units, whereas X would develop linkages with the antidrive units. Konorski (1967) viewed the development of the two linkages as independent of one another with the strength of each linkage determined by the past training history of the respective stimuli. If A and X are presented together, the presence or absence of a CR will be determined by the stronger of the two linkages.

In addition to the linkages involved with discrete CSs, such as tones, Konorski (1967) asserted that the experimental context, a relatively constant set of complex stimuli, would act as a CS. Thus, linkages between the gnostic units for the context and the fear drive units are established when the US occurs. The consequence of establishing a linkage between the context and fear drive units is the observation of fear CRs to the conditioning context while the subject is in the context (Konorski, 1967). The contextually controlled fear CRs

were labelled tonic fear CRs by Konorski (1967) and were thought to be necessary for the performance of CRs to discrete stimuli (CSs).

If Konorski's (1967) principles are applied to the phases in the present study the following linkages should develop. During the tone acquisition phase (A+) the A gnostic units should form a linkage with the US gnostic units and the fear drive units. The linkages should be of equal strength across all Conditions. In addition, tonic fear CRs to the context should develop in all Conditions. During the inhibitory phase the linkages between the A gnostic units and the fear drive units should be further strengthened on the A+ trials for all Conditions. In addition, for Condition CI linkages between the A gnostic units and the fear antidrive units and between the X gnostic units and fear antidrive units develop on the AX- trails. Subsequent reacquisition training (X+) should serve to develop linkages between X gnostic units and the fear drive units across all Conditions. Conversely, during extinction (X-), linkages between the X gnostic units and the fear antidrive units should develop.

Since Konorski's (1967) position advocated an interaction of the substrate of discrete and tonic CRs, the theory can be used to account for the within session effects observed during the inhibitory conditioning phase. During the first block of an inhibitory session, tonic fear CRs are elicited to the context. Since performance of discrete CRs requires the presence of the fear drive units, the tonic fear CRs would activate the fear drive units thereby allowing high levels of responding to occur. As the session proceeds, the inhibitory AX- trials produce two major consequences. First, X establishes a linkage to the fear antidrive units, which through reciprocal inhibition

reduces the action of the fear drive units. Second, the reduced fear drive reduces the effectiveness of the tonic CRs to activate discrete trial performance. Responding on the AX- trials would be affected by both consequences and, therefore, responding should decrease across an experimental session as X becomes increasingly more inhibitory. Responding on the A+ trials would be affected only by the second consequence. Therefore, A+ response levels should decrease across an experimental session, but the magnitude of the decrease would be smaller than the decrease observed on the AX- trials. If the linkage between X and the fear antidrive is not stable and weakens over the 22 hour inter-session interval, then at the beginning of the next session, the tonic CRs would again be at full strength and high levels of responding would be observed. The instability of the linkage between X and the fear antidrive may be due to second-order excitatory conditioning of X by presenting X in compound with the pre-established excitatory stimulus, A, on the AX- trials (Konorski, 1967).

The presence of tonic fear CRs upon returning a subject to an experimental setting where shock has been previously given could also explain the differences in reacquisition performance between Groups CI-I and CI-N. Although both Groups CI-I and CI-N demonstrated within session decrements on the AX- trials during inhibitory training, only Group CI-I showed evidence of inhibition to X during reacquisition. Group CI-I, however, received the reacquisition test immediately after the last inhibitory training session, at a time when the fear antidrive should be strong and therefore, when the tonic fear CRs were weak. Conversely, Group CI-N was removed from the experimental setting after the last inhibitory training session and placed back the next day for

reacquisition. Thus, for Group CI-N reacquisition would occur in the presence of tonic fear CRs produced by the linkage between the context and fear drive units. The presence of the fear drive and tonic fear CRs may have caused the higher initial level of responding to X for Group CI-N during reacquisition.

Given that shock followed X during reacquisition, the tonic CRs would be strengthened for Groups CI-I and CI-N. However, a greater strengthening of the linkage between X and the fear drive units would occur in Group CI-N since performance in Group CI-I would be lower because of the actions of the active fear antidrive units. The finding that X extinction occurred more slowly for Group CI-N relative to Group CI-I is congruent with the expectation that the presence of a strong fear drive during reacquisition caused a stronger linkage between the X gnostic units and the fear drive units for Group CI-N. Otherwise, Groups CI-I and CI-N would have extinguished to X at the same rate because of the equal amount of exposure to the reinforced and non-reinforced trials.

Thus, the results from each phase of the present experiment are consistent with a post hoc application of Konorski's (1967) theory. The greater concordance with Konorski's (1967) position relative to the theories of Rescorla and Wagner (1972) and Wagner (1981) suggests that a renewed focus on theories that allow the past associative strength of a stimulus to influence present conditioning to that stimulus should be undertaken.

In summary, the main findings of the present study were a) the within session decrements to the AX- compound during inhibition training were the result of associative inhibition accruing to X, and b) a

delayed reacquisition test failed to identify that associative inhibition was accrued by X. The finding of associative inhibition following conditioned inhibition training is consistent with current classical conditioning theories, but the labile nature of the inhibition is not (Frey & Sears, 1978; Moore, 1982; Pearce & Hall, 1980; Rescorla & Wagner, 1972 and Wagner, 1981). Two suggestions for the disruptive effects of the delayed reacquisition test were offered: a) between session spontaneous recovery of the excitatory strength of X, and b) whether reacquisition occurred in the presence of a strong fear antidrive versus a strong fear drive. The present experiment does not allow for a choice between the spontaneous recovery and drive accounts but does provide these accounts as viable directions for future research.

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Appendix

Differential Development of Associative Inhibition with the Rabbit's Nictitating Membrane Response

In Rescorla and Wagner's (1972; Wagner & Rescorla, 1972) general model of Pavlovian conditioning, the associative strength of a predictive cue is represented by a point on an associative strength dimension which is symmetrically distributed about a null value. If the value of a cue's associative strength is positive, then excitatory conditioning to the cue is observed; whereas, if the cue's associative strength is negative, then inhibitory conditioning to the cue results. Change in the associative strength of a cue was hypothesized to occur gradually and incrementally with repeated cue presentations. The direction and magnitude of change was a function of the discrepancy between the level of conditioning that a US will support and the current value of the summed associative strengths of the aggregate of available predictive cues. Increments to the associative strength of a predictive cue accrue when the discrepancy is positive; whereas, decrements in associative strength result from negative discrepancies. Thus, the direction and magnitude of change in associative strength for a predictive cue will depend on that cue's current associative strength, the summed associative strength of all predictive cues acting and the maximum associative strength that is supported by a US, and not the associative history of the predictive cue of interest.

Stated in these terms, associative inhibition is simply a negative level of associative strength, and has no unique status in terms of its mechanisms. For conditioning paradigms that are commonly employed to create an inhibitory CS (e.g., unpaired presentations, backward,

conditioned inhibition and differential procedures), Wagner and Rescorla (1972) asserted that the acquisition of negative associative strength to an initially neutral CS occurs through the compounding, in the absence of a US, of an initially neutral cue with other cues that have positive associative strength. Under these conditions, a negative discrepancy will exist between the net associative strength of the compound and the zero associative asymptote that nonreinforcement supports, and the neutral cue will acquire a negative value of associative strength. Since the speed of acquisition and final aymptotic level of negative associative strength is directly related to the summed positive associative strength of the compounded cues, stronger inhibitory conditioning would be expected with conditioning paradigms that have more excitatory compounded cues.

In the present series of experiments, the Rescorla-Wagner model's predictions on associative inhibition were assessed in four commonly employed inhibitory conditioning paradigms: unpaired, differential, backward and conditioned inhibition. According to the model, unpaired, backward, and differential conditioning produce inhibitory effects because the inhibitor is paired with excitatory situational cues. These situations cues are thought to become excitatory in the unpaired and backward paradigms as a result of the pairing of situational cues with the unsignalled US. In the differential conditioning paradigm, both situational cues and a discrete CS+ are paired with the US. However, theoretically the explicit CS+ overshadows the conditioning of the situational cues, with the result that only a weak excitatory associative strength accrues to the situational cues. Since the situational cues in the differential paradigm should have less positive

associative strength than situational cues in the backward and unpaired paradigms, the levels of inhibitory conditioning should also be weaker. In contrast, associative inhibition develops in the conditioned inhibition paradigm, because the CS- is simultaneously compounded with a nonreinforced CS+. Since the CS+ is usually paired with a US at least 50 percent of the time, the CS+ in the condition inhibition paradigm should have more associative strength than the situational CSs of the backward, unpaired, and differential paradigms which are reinforced only a small proportion of the time. Since the rate and level of associative inhibition is a function of the strength of the cue with which it is compounded, inhibitory conditioning should be strongest in the conditioned inhibition paradigm, weaker in the unpaired and backward paradigms, and weakest in the differential paradigm.

Experiment 1

In Experiment 1, unpaired, differential, conditioned inhibition and backward pairing procedures were employed to develop associative inhibition of the rabbit's nictitating membrane (NM) response. Subsequently, the relative levels of associative inhibition produced were measured by means of a retardation of learning test (Rescorla, 1969) in which the putative inhibitory stimulus was paired with the US. Associative inhibition is inferred from slower acquisition of a CR relative to control groups, and differential levels of associative inhibition inferred from differentially retarded CR acquisition.

Method

Subjects. The subjects were 42 experimentally naive male and female New Zealand white rabbits (Oryctolagus cuniculus) approximately

100 days old and weighing between 2.0 - 3.0 kg. The rabbits were purchased from the Kleefeld Rabbitry at Tourond, Manitoba.

Apparatus. The nictitating membrane (NM) transducing and conditioning apparatus has been described in detail elsewhere (Gormezano, 1966). Briefly, each rabbit was restrained in a Plexiglas box equipped with an adjustable stock and a foam padded ear clamp, in order to minimize head and gross body movement. The right eyelids were held open by Newey tailor hooks secured to an adjustable Velcro strap tightened around the rabbit's head. A 2mm loop of 00 Ethicon monofilament, sutured through the epithelium layer of the NM, was attached via a staple and thread to a piano wire armature that was mechanically fixed to the shaft of a 10K microtorque potentiometer. The potentiometer was fastened to a headmount, which was positioned tightly over the rabbit's head. The Plexiglas restraining box was enclosed within one of eight identical, legal size, fire-proof, filing cabinet drawers, equipped with a stimulus panel in the front and a ventilation fan at the back. White noise was presented continuously at 72 dB in the experimental room. NM response transducers were interfaced through a 10-bit, 16 channel analog-to-digital (A/D) converter to a 8K Raytheon 703 computer. Response identification and stimulus presentations were accomplished by a software system that was modified from Tait and Gormezano (1974). A voltage change of .2 in the A/D converter, which corresponded to a .5mm extension of the NM, was scored as a response. Trials in which a NM response was initiated in a 500 msec period prior to CS onset were not scored.

The US was a 2.5 mA, 50 msec, 60 Hz AC shock delivered to two stainless steel 9mm Autoclips implanted 10mm caudal to, and 10mm above

and below the horizontal plane of the right eye. The CSs were a 500 msec, 80dB, 1000 Hz tone, and a 500 msec, 80 dB, 10 Hz clicker, delivered through speakers located on the right side of the stimulus panel as viewed by the subject. All forward CS-US intervals were 450 msec.

Procedure. All animals received 1 day of preparation, 1 day of adaptation, 3 days of forward click-shock pairings, 4 days of inhibitory training to the tone, 3 days of forward tone-shock reacquisition testing, and 1 day of tone extinction testing. With the exception of the inhibitory training phase, the phases were common for all rabbits. On the preparation day, the rabbits had the region around the right eye shaved, depilated and swabbed with isopropyl alcohol. A 2mm loop of 00 Ethicon monofilament was sutured into the epithilium layer of the NM, and the two Autoclip sutures were implanted. The rabbits were then randomly assigned to one of 6 groups (n=7). On the next day, the rabbits were adapted to the experimental chambers and NM transducing apparatus for 100 min. No stimuli were presented, and spontaneous NM responding was monitored. During the next 3 days, all rabbits were given 100 daily forward click-shock acquisition trials. The mean intertrial interval for this and all subsequent phases was 1 min.

Following click-shock acquisition, the 6 groups received varied treatments during the 4 days of tone inhibitory training. One group of rabbits received daily conditioned inhibition training (Group CI) in which 50 click-shock trials were interspersed amongst 50 tone-click simultaneous compounds trials. A second group received differential conditioning (Group D) in which 50 click-shock trials were interspersed amongst 50 tone-alone presentations. The third group received daily

unpaired presentations (Group UP) of 50 tone-alone trials and 50 shock-tone presentations. The fourth group received backward conditioning (Group B), with tone onset immediately following shock offset. Finally, two control groups in which no tones were presented were employed to be used as contrasts against which inhibitory effects could be assessed. One control group was given extended excitatory training to the click (Group E), in which 50 daily click-shock forward pairings were presented, while the other group (Group N) received no stimuli and were simply restrained in the conditioning chambers.

During tone inhibitory training, the groups were matched, where possible, for the timing of events. All groups except Group N, received USs at the same points in time during a conditioning session. The schedule of, and the intertrial intervals between US and no US trials were identical for Groups U, D and CI. In Groups B and E which did not receive "no US" trials, baseline responding was monitored in the intervals that corresponded to tone presentations in Groups U, D, and CI. For Group N, baseline responding was monitored in all time periods that corresponded to either click or tone presentations in Groups D and CI. The matching of groups in this fashion was undertaken to equate for any temporal dynamics that might affect the associative strength of situational cues.

Following tone inhibitory training, all groups received 3 days of 100 tone-shock pairings as a retardation of learning test for inhibition. On the next day the rabbits received a single 60 trial tone-alone extinction test.

Results

Repeated measures ANOVAS, orthogonal trend analysis, and Newman-Keuls post-hoc tests (=.05) were applied to the mean percent NM CRs in this and all subsequent data analysis. The within-subject factors included days, four 25-trial blocks within each day, and trial-type (reinforced or nonreinforced).

During adaptation, base rates of NM responding was less than 3 percent and did not differ between groups ($F(5,36)$ 1). The left-hand side of Figure 12 depicts the mean percent NM CRs made by Groups E, N, D, CI, UP, and B over the three days of click acquisition. Over days NM CR acquisition was rapid and reached a common high terminal value. No Group differences existed in this phase ($F(5,36)=1.59$).

The right-hand portion of Figure 1 presents the mean percent CRs of tone inhibitory training. For clarity of presentation, only performance on click-shock trials (Groups E, CI & D) and tone trials (Groups CI, D, UP) was included and not the base rate responding monitored for Groups E, N, UP and B. Base rate responding was at adaptation levels and did not differ between groups.

During tone inhibitory training, Groups E, CI and D maintained the same level of NM responding to the click that was established during the click-shock acquisition phase. Thus, the differing inhibitory treatments did not appear to directly affect responding on the reinforced click trial. A repeated measures ANOVA applied to the reinforced trials of Groups E, CI, and D, confirmed that the groups did not differ ($F(2,18)=1.07$), and did not change over days (Days effect ($F(3.54)=2.53$). On the other hand, large group differences in percent NM CRs on the nonreinforced trials were apparent. Group CI maintained a

much higher level of NM responding to the tone-click nonreinforced compound than Group D did to the nonreinforced tone, which in turn was slightly higher than NM responding to the tone in Group UP. In addition, NM responding on the nonreinforced trials in Group UP decreased over days whereas levels of responding Groups CI and D did not change appreciably over days. A repeated measures ANOVA applied to the nonreinforced trials groups, CI, D and UP confirmed this graphical depiction by yielding a significant Group main effect ($F(2,18)=39.98$, and a Days main effect ($F(3,54)=3.54$, $p < .05$). Post hoc tests indicated that Group CI maintained a higher level of NM responding on nonreinforced trials than Group D, which was higher than Group UP. In addition, trend analysis applied to the days effect for each group identified a significant linear decrease in NM CRs for Group UP ($F(1,18)=10.48$, $p < .01$) but not for Groups D ($F(1,18) < 1$) or CI ($F(1,18) < 1$).

Insert Figure 12 about here

Figure 13 displays the mean percent NM CRs of Groups E, CI, and D on the reinforced trials, and of Groups CI, D, and UP on the nonreinforced trials as a function of the four 25-trial blocks within each of the four days of inhibitory training. For the reinforced trials, percent CRs were maintained at high levels for all groups over each daily session. However, a slight decrease of about 4 percent over each daily session produced a Blocks main effect ($F(3,54)=4.58$, $p < .05$). On the other hand, percent CRs dropped over each session on the nonreinforced trials. The pattern of responding differed according to

the nonreinforced treatment. For Group CI, the within session decrease in percent CRs developed over days and there was substantial recovery in CR level between days. For Group UP the within session decrease appeared on each day and there was little recovery between days. The different patterns of responding yielded significant Blocks main effect ($F(3,54)=5.62$, $p < .01$) with a reliable linear component for trend ($F(1,54)=16.02$, $p < .01$) and a significant linear component ($F(2,54)=4.48$, $p < .05$) to the Group X Block interaction in the ANOVA that was applied to the nonreinforced trials.

Insert Figure 13 about here

The results of the 3 day retardation of learning test to the tone indicates that the control groups, E and N, showed almost immediate transfer of responding to a new signalling cue. Similarly, Group CI showed the same immediate acquisition of NM responding to the inhibitory trained tone. However, Groups D and B initially responded at much lower levels, and gradually increased responding over days to the same level as Groups E, N, and CI. Finally, Group UP showed the slowest acquisition of NM responding and only attained an 84 percent level of CRs on the final day of acquisition. A repeated measures ANOVA confirmed these observations by yielding a significant Group x Days interaction ($F(10,72)=10.44$, $p < .01$) which contained a significant linear orthogonal component for trend ($F(5,72)=17.29$, $p < .01$). Post hoc tests indicated that on Day 1 of tone reacquisition, Group UP had a lower level of percent CRs than Groups B and D, which were lower than Groups E, N, and CI. On Day 2, Group UP was significantly lower than

Groups E and N, but not lower than Groups CI, D, or B. No other comparisons were significant during the three days of tone-shock pairings.

A repeated measures ANOVA applied to the 10-trial blocks of extinction yielded a significant Group x Blocks interactions ($F(25,100)=1,87$, $p < .05$) which contained a significant linear orthogonal component for trend ($F(5,100)=3.63$, $p < .01$). Trend analysis applied to the mean percent CR of each group indicated that there was a significant decrease in percent CRs across the 10-trial blocks of extinction in Groups CI, D, N, and B but not Groups E or UP. Thus, Group E maintained a high level of responding over extinction while the inhibitory groups decreased responding over the six blocks of extinction. One exception to the pattern was Group UP, which started extinction at a lower level than the other inhibitory groups, and maintained this level over extinction.

Discussion

If a decrease in responding on nonreinforced trials during tone inhibitory training is considered an index to the development of associative inhibition, then inhibition developed differentially, with Group UP showing the fastest rate of acquisition of inhibition, Group CI the slowest and Group D a moderate rate. However, since Group CI received nonreinforced tone-click compound trials while Group UP and D received nonreinforced tone trials, the level of inhibition demonstrated in Group CI should be underestimated by looking at nonreinforced trial performance. Both the presence of the excitatory click and the possibility that within compound associations developed (Rescorla, 1981a, 1981b) should lead to higher responding in Group CI. Thus, the

development of associative inhibition on the nonreinforced trials can be directly compared only for Groups D and UP. The more rapid development of inhibition in Group UP relative to Group D is consistent with the Rescorla-Wagner model, and may reflect the hypothesized blocking of excitatory conditioning to situational cues in Group D by the discrete CS+.

However, the within session patterns of responses observed during the inhibitory training phase are not consistent with the Rescorla-Wagner model. The model asserts that associative inhibition develops gradually and incrementally. Accordingly, as inhibition develops a systematic decrease in responding on nonreinforced trials should be observed. While the pattern of responding in Group UP is consistent with the model, the recovery of responding between session in Groups D and CI is not. While it would be argued that the between session recovery of responding on nonreinforced trials reflects the observation that associative inhibition is more readily forgotten than excitation (e.g., Hendersen, 1978), this argument also leads to conclusions that are inconsistent with the Rescorla-Wagner model. Since Group UP did not show a between-session recovery, whereas Group CI did, it would follow that the associative inhibition developed in Group UP was not forgotten. This could occur only if Group UP acquired associative inhibition more rapidly than Group CI. However, the model predicts that associative inhibition should be acquired most rapidly by Group CI. Thus invoking differential forgetting to explain the between session recovery would yield an account that is inconsistent with the Rescorla-Wagner model. And finally, the decrease in responding on nonreinforced trials can not be attributed to performance decrements

observed with multitrial experimental sessions (e.g., Tait, Kehoe & Gormezano, 1983). For in the present experiment, large within session decrements were observed on nonreinforced trials, but only slight decreases were observed on reinforced trials that occurred late in the session. It appears that the decrements were specific to the operations employed to create inhibition. Thus, the development of associative inhibition does not always follow the predictions of the Rescorla and Wagner model.

The major observation of Experiment 1 was the order of the inhibitory groups during the retardation of learning test. Unpaired, backward, and differential training to the tone retarded the subsequent development of an overt CR to that same tone, leading to the conclusion that reliable associative inhibition was established with these procedures. Moreover, since greater retardation was observed in Group UP relative to Groups B and D, the terminal level of inhibition created in Group UP was higher than in Groups B and D. In contrast, the conditioned inhibition training did not retard NM acquisition, and consequently there was no evidence that the tone in Group CI acquired inhibitory tendencies. Thus, the terminal level of associative inhibition was greatest in Group UP, weaker in Groups B and D, and weakest in Group CI. Since the Rescorla-Wagner model predicted that the terminal levels of associative inhibition should be rank-ordered CI B = UP D, the prediction was <u>not</u> confirmed.

In tone extinction, Groups N, B, UP, CI, and D showed more rapid extinction than did Group E. Even though the terminal level of percent CRs for Groups E, B, D were identical, the test did not have an extended postasymptotic phase. Therefore the Rescorla-Wagner model might predict

that the excitatory associative strength to the tone in Groups B and D was less than in Group E at the end of the test, and consequently, more rapid extinction followed. A similar interpretation could account for the extinction performance of Group UP buttressed by the observation of a lower terminal level of percent CRs for Group UP on the retardation test. However, the model has difficulty in accounting for the performances of Groups E, N, and CI. Throughout the retardation test percent CRs were at a high level and no differences between the three groups were observed. Accordingly, the assertion of differential associative strengths amongst the groups would be tenuous. If no differences in associative strength exists between the groups, then the path independent assumption of the model leads to an expectation of no differences between the groups in extinction.

An examination of the experimental protocols of Groups E and N indicates that the Groups differed only in the number of click-shock pairings; Group E received click-shock pairings during the inhibitory training phase whereas Group N did not. Thus, Group E should have a greater excitatory associative strength to the click. To produce differences in tone extinction, the differences in excitatory associative strength would have to be transferred to the tone during tone-shock pairings and be maintained. The high level of CRs to the tone for both Groups on the first day of the retardation test is consistent with this hypothesis. A similar transfer of associative strength from the click to the tone could have occurred in Group CI. While Groups E and CI received the same number of click-shock pairings, the net associative strength to the click would be lower in Group CI since the click was reinforced on only 50% of its presentations.

Alternatively, since Groups CI showed within-session decreases in percent CRs on nonreinforced trials, it could be argued that some associative inhibition developed to the tone. The more rapid extinction in Groups CI relative to Group E would then be due to the past inhibitory history of tone. For this hypothesis to be true, either the retardation of learning test would have to be an insensitive test for the inhibition developed in Group CI, or a path dependent rather than a path independent model is required to account for associative inhibition.

Experiment 2

A surprising observation of Experiment 1 was the rank orderings of the Groups on the terminal level of associative inhibition. The weakest evidence of inhibition was in the conditioned inhibition group, the group that the Rescorla-Wagner model predicted should have the highest level of associative inhibition. However, since it was possible that the retardation test in Experiment 1 was not sensitive to the inhibition established in Group CI, it was necessary to confirm the rank ordering of Experiment 1 using a different technique to measure terminal inhibitory strengths. Accordingly, Experiment 2 was designed to assess associative inhibition first with a summation test (Hearst, 1972; Rescorla, 1969) and then with a retardation test.

Since a summation test requires two CS+s, two auditory CSs, a tone and a click were separately conditioned to the US prior to the inhibitory manipulations. The tone was then used during inhibition training, and the click was used as the summation stimulus. In addition, to establish stronger terminal levels of associative inhibition, more extensive inhibitory training was conducted and the ratio of nonreinforced to reinforced trials was changed from 1:1 to 2:1.

Method

Subjects and Apparatus. The subjects were 35 experimentally naive male and female New Zealand white rabbits (Oryctolagus cuniculus). The CSs were a 500 msec, 80 dB 1000 Hz tone; a 500 msec, 80 dB 10 Hz clicker; and, a 500 msec, 20 p.s.i. airpuff delivered to the left rib area beneath the front paw. Otherwise the apparatus was identical to that used in Experiment 1.

Procedure. The rabbits received 1 day of preparation, 1 day of adaptation, 5 days of click-shock and tone-shock pairings, 7 days of inhibitory conditioning to the airpuff CS, 3 days of summation testing and 1 day of airpuff reacquisition testing. All rabbits were treated identically over each experimental phase except during airpuff inhibitory training phase. Following adaptation, the rabbits were divided into 5 groups (n=7), and received 5 days of forward acquisition to the tone and the click CS. On one-half of the trials, shock was paired with the tone, while on the other half shock was paired with the click. All forward CS-US intervals in this and subsequent phases were 450 msec. The two types of trials were randomly interspersed at a mean intertrial interval of 1 min, for a combined total of 96 trials on each day.

The differing inhibitory treatments were begun the day after the final day of click and tone forward acquisition. One group of rabbits (Group CI) received 32 daily tone-shock trials randomly interspersed amongst 64 simultaneous presentations of the airpuff and tone. Group D received 32 daily tone-shock trials randomly presented amongst 64 airpuff-alone presentations. Group UP received 32 shock-alone presentations intermixed amongst 64 airpuff-alone trials. Group B

received 64 backward shock-airpuff presentations that were matched temporally to the nonreinforced trials of Groups UP, CI and D. The onset of the airpuff immediately followed US offset. Finally, Group E received 32 daily tone-shock presentations at the same time as the paired trials in Groups D and CI. In Groups B and E, spontaneous responses were monitored during trials in which no stimuli were presented.

Following tone inhibitory training, all groups received 3 days of summation tests. Thirty-two click-alone, 32 airpuff-alone, and 32 click-airpuff simultaneous trials were randomly interspersed on each day of testing at an intertrial interval of 1 min. On the next day all rabbits were given 96 airpuff-shock forward reacquisition trials.

Results

During the adaptation session, response levels were low (ranging from 2% to 4%) and did not differ between Groups ($F(4,33)$, $p < .01$. During the 5 days of excitatory training, all groups acquired CRs to both the click and tone. Acquisition rates were indistinguishable both between groups (percent CRs for the fifth day: 97.1, 93.1, 95.0, 92.6, 92.2 for Groups E, CI, D, UP, and B, respectively) and between the two cues (percent CRs for the fifth day were 93.5 and 93.2 for the tone and click CSs respectively). A repeated measures ANOVA confirmed that no differences existed between Groups ($F(4,33) < 1$), CS Type ($F(1,33) < 1$) and the interaction between the two ($F(4,33)=1.19$). In addition, no interactions of groups or CS Type were observed with either days, or blocks within days. Thus, at the beginning of the inhibitory training phase, the tone and click were equally excitatory for all groups.

During the inhibitory phase for Groups E, CI, and D on the reinforced trials the percent CRs increased gradually from 90.1% to 96.9% over the seven days. However within each session there was an overall drop of 4.9 in percent CRs that primarily resulted from the large drop in CRs in Group CI. A repeated measures ANOVA applied to the reinforced trials confirmed the interpretation by yielding significant Days ($F(6,114)=3.11$ $p < .01$), Blocks within sessions ($F(3,57)=3.78$ $p < .05$) and Group x Blocks interaction ($F(6,57)=3.78$ $p < .05$) effects. The interaction contained a significant linear trend component ($F(2,57)=11.17$ $p < .01$) that resulted from the decrease across Blocks for Group CI ($F(1,57)=22.00$ $p < .01$) and the nonsignificant linear trends for Group NI ($F(1,57)=1.11$) and D ($F(1,57)=2.48$). For the nonreinforced trials, the percent responding: was lower in Groups D and UP relative to Group CI; decreased over days for Groups CI and D but not UP; and decreased within sessions for all groups, although this effect was observed only on the first four days for Group UP the first six days for Group D and on all days for Group CI. A repeated measures ANOVA confirmed the interpretation by yielding significant: Groups ($F(2,18)=10.03$, $p < .01$), linear orthogonal trend component ($F(2,108)=4.26$, $p < .05$) to the Group x Days interaction; Blocks within a session ($F(3,54)=22.29$, $p < .01$); Days x Blocks ($F(18,324)=1.66$, $p < .05$) and Group x Days x Blocks ($F(36,324)=1.97$, $p < .01$) effects.

On the three days summation test, the mean percent CRs were 47.7 to CS+, 23.2 to CS- and 42.0 on CS+CS- summation trials. A repeated measures ANOVA identified a significant trial type effect ($F(2,60)=35.45$, $p < .01$) and Newman-Keul's tests ($\alpha=.05$) confirmed that performance on each type of trial significantly differed from the other

two. This pattern was not found for all groups. For all groups, responding to CS- was less than the two other types of trials. However only in Groups CI, D and UP was responding on the summation trials reliably lower than on CS+ trials. The difference in performance to the two types of trials did not significantly vary between the three groups.

The ANOVA also continued significant Trial Type x Days ($F(4,120)=4.76$ $p < .01$), Trial Type x Blocks within session ($F(6,180)=5.64$ $p < .01$) and Group x Trial Type x Days ($F(16,120)=2.32$, $p < .01$) interactions. The interactions of Trial Type with Days and Blocks within session reflected a decrease in responding on CS+ and summation trials relative to a fairly constant response level on CS- trials. The triple interaction indicated that the extinction pattern was not similar for all groups over days. An examination of each group indicated that the extinction functions for the CS+ and summation trials were parallel for Groups E, B, and UP, converged for Group D and diverged for Group CI. Significant differences in percent CRs between CS+ and summation trials were found on only the first day for Group D, only the last day for CI and on all three days for Group UP.

During the retardation of learning test, the high percent CR levels indicated that reacquisition occurred rapidly for all groups with Groups E, CI and D being somewhat higher than Group UP and B. A repeated measures ANOVA applied to blocks of 24 trials during reacquisition failed to reveal Group differences ($F(4,30)=1.03$) but did identify that reacquisition occurred rapidly (Blocks within session: ($F(3,90)=2.67$, $p < .05$) and differentially between Groups (linear component to the Groups x Block interaction ($F(4,90)=2.56$, $p < .05$).

Since the rapid acquisition may have obscured initial group differences, comparisons between the groups were conducted on their performance during the first 24-trial block. The mean percent CRs for the first block were 89.8, 83.3, 67.5, 64.3 and 60.1 for Groups E, CI, D, UP and B. Post hoc tests indicated that the mean percent CRs for Groups E and CI did not differ and were higher than the initial levels of Groups D, UP and B which also did not differ.

Discussion

The inhibitory training phase of Experiment 2 replicated the observations of Experiment 1, performance levels on the inhibitory trials were rank ordered Group CI, Group D and Group UP from highest to lowest. With the airpuff cue, larger within session effects were observed in Group CI and better discrimination in Group D than was observed in Experiment 1. With the two extra days of training, performance in Groups D and UP converged.

The overall performance on the subsequent summation tests indicated that only Groups CI, D and UP had lower performance on the summation trials relative to the CS+ trials. Accordingly, the inference would be that these were the only groups to have developed inhibition. In addition, since the amount of suppression on the summation trials was about the same for the three groups, the level of inhibition acquired would be equal. However, an examination of the three days that made up the summation phase indicated that only Groups D and UP showed a reliable suppression on the first day of testing and that Group UP was the only Group to show an effect on all three days. It would appear that the highest level of inhibition was in Group UP followed by Group D. For Groups CI, the difference in performance on the two types of

trials developed over days in testing. It was as if the level of inhibition was increasing over the testing period. Since the summation trials were simultaneous pairings of a CS+ and a putative inhibitory cue, it is possible that the summation test served as further conditioned inhibition training and did lead to an increment in the level of inhibition to the CS-. On the basis of Experiment 2, it would appear that extended summation testing biases the test in favour of identifying associative inhibition in the conditioned inhibition paradigm.

The subsequent retardation of learning test confirmed this conclusion by revealing retarded acquisition in Groups D, B and UP but not with Group CI. This outcome replicated the observations of Experiment 1. However, unlike in Experiment 1, no difference existed between the three groups. Since the retardation test followed the summation test, it would be expected that the 96 CS- and 96 CS+CS- trials that occurred during summation testing would tend to homogenize the level of inhibition in the three groups. Despite a possible homogenizing effect, the ordering of the groups on the retardation test was similar to that found in Experiment 1. Percent CRs in Groups CI and E were highest and decreased over Groups D, UP and B.

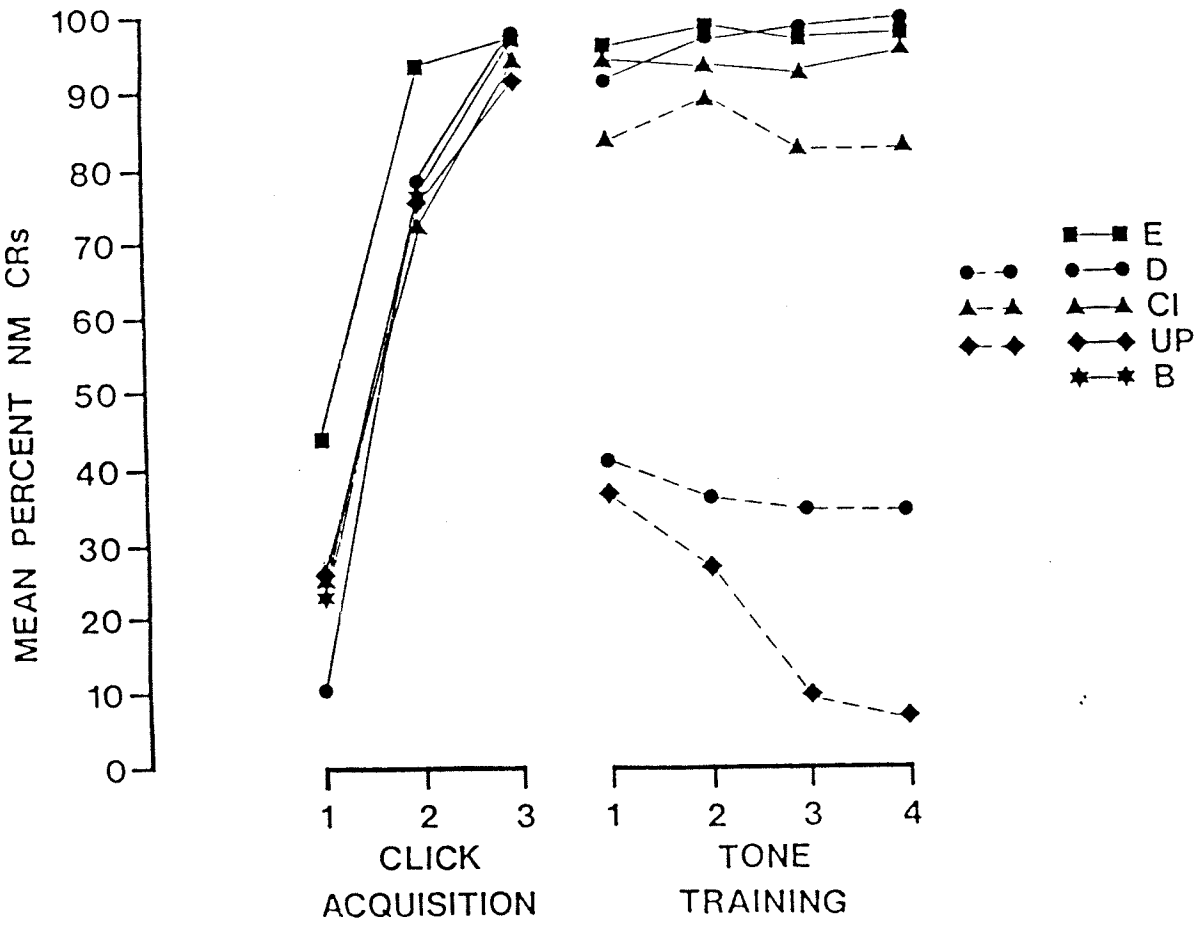
The results of Experiment 2 suggest that when subjects are matched on total number and timing of experiences, then the unpaired and differential procedure produced higher levels of associative inhibition that did the conditioned inhibition paradigm. Since the Rescorla-Wagner (1972) model predicts that associative inhibition should be highest with conditioned inhibition training, the results of Experiment 1 and 2 appear to contradict the model. However, the model can be used

postdictively to provide a rationale for the results. The Rescorla-Wagner model invokes the conditioning of associative strength to contextual cues to account for some inhibitory phenomena (Wagner & Rescorla, 1972). If it is assumed that contextual conditioning participates in all classical conditioning processes, then performance on the inhibition tests of Experiment 1 and 2 would result from the combined associative strengths of the context (V_c) and the inhibitory cue (V_x). If the groups differed in V_c, then performance on the inhibition tests would not be an index of the relative strengths of the V_xs. For example, if Groups CI and D are compared, the conclusion that greater inhibition was created in Group D are compared, the conclusion that greater inhibition was created in Group D could be in error. It is possible that V_x for Group CI was less than Groups D's V_x, and at the same time that the V_c for Group CI was much larger than the V_c for Group D. Since the associative strength controlling performance on the inhibition tests would be the sum $V_c + V_x$ if this sum were greater in Group CI then performance would be at a higher level. Is this a realistic possibility? It would appear that it is. During inhibitory training the background cues of Group CI would be protected on the inhibitory trial by the overshadowing of the excitatory CS and therefore not be lowered in associative strength to the same extent as the contextual cues of Group D. Thus, the V_c for Group CI could be higher than in Group D, or any of the other training procedures. Experiment 3 was designed to determine whether inhibitory contextual cues could have contributed to the differences observed in Experiment 1 and 2.

Figure Captions

Figure 12. Experiment 1, percent CRs to click during the acquisition phase and percent CRs to tone during the inhibitory training phase across daily sessions (E = Group Excitatory; D = Group Differential; CI = Group Conditioned Inhibition; UP = Group Unpaired; B = Group Backward; Dashed lines = nonreinforced trials; Solid lines = reinforced trials).

Figure 13. Experiment 1, percent CRs to reinforced and nonreinforced trials during the inhibitory training phase for each 25-trial block within a session and for each session (E = Group Excitatory; D = Group Differential; CI = Group Conditioned Inhibition; UP = Group Unpaired; Dashed lines = nonreinforced trials; Solid lines = reinforced trials).


MEAN PERCENT NM CRs
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TRAINING
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D
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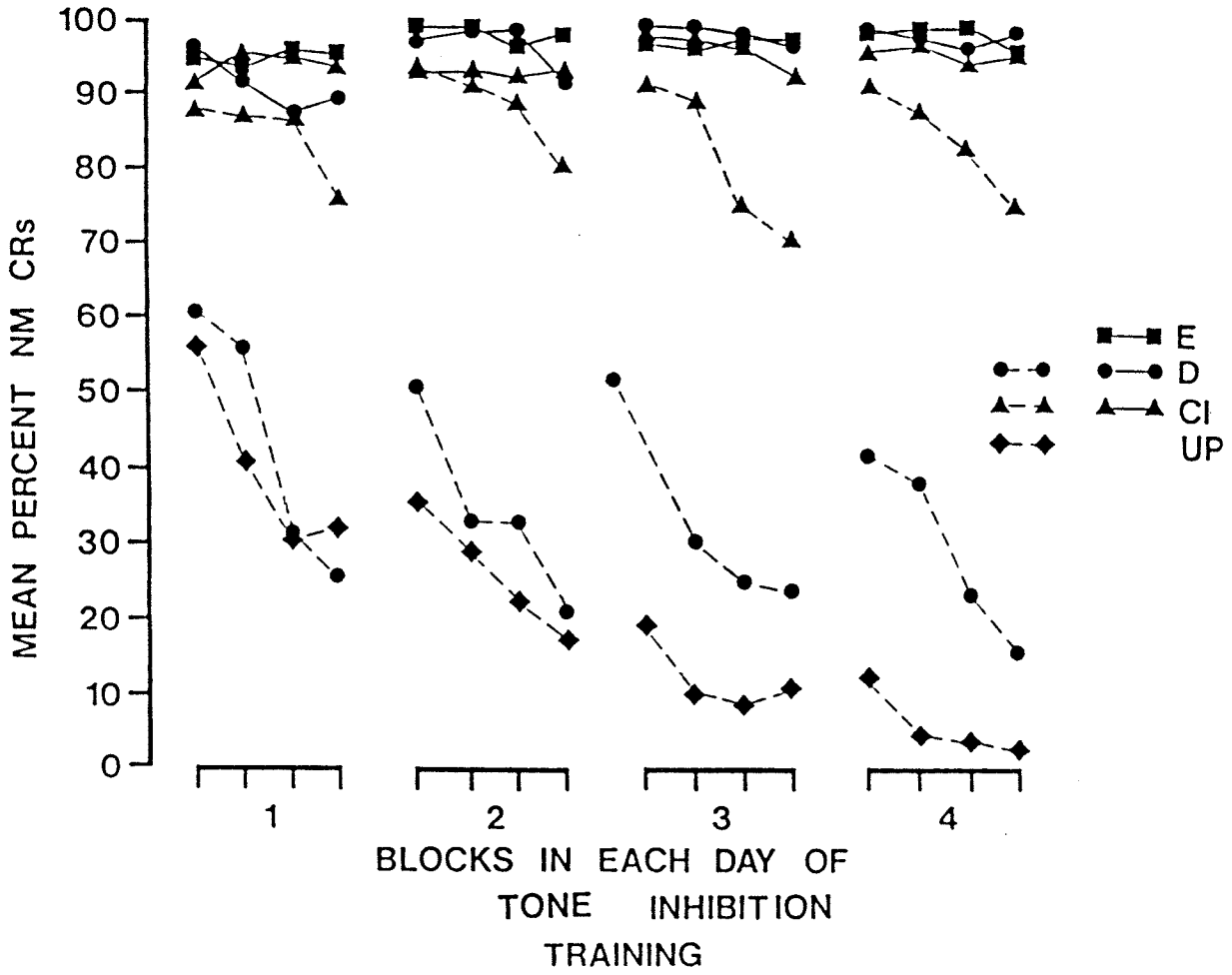
MEAN PERCENT NM CRs
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BLOCKS IN EACH DAY OF TONE INHIBITION TRAINING
E
D
CI
UP