Conditioned Inhibition in Sensory Preconditioning

by

Michael E. Saladin

A thesis presented to the University of Manitoba in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Psychology

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Abstract

Associative inhibition (i. e., acquired properties of a stimulus to actively suppress responding) has traditionally been studied with Pavlovian conditioning methodologies. A major defining feature of Pavlovian methodologies is their emphasis on the role of the US's response eliciting and motivational properties in the development of an association. One problem that results from this defining feature is that Pavlovian methodologies focus more on the performance correlates of inhibition than on inhibitory associations per se. One way to minimize the influence of US produced performance factors in the study of associative inhibition is to adopt a methodology which diminishes the role of the US in the development of associative inhibition. One approach is to use the sensory preconditioning (SPC) paradigm. The SPC paradigm for associative excitation involves (a) pairings of two neutral stimuli, S1 and S2, in a preconditioning phase, (b) subsequent pairings of S2 with a US, and (c) testing the response-eliciting properties of S1, the stimulus that was not paired with the US. The role of the US is minimized because the stimulus of interest (SI) never directly enters into association with the US. While the SPC paradigm has been used in the study of associative excitation, it has not yet been employed to study associative inhibition. The present studies are the first to identify associative inhibition using an SPC methodology. Experiment 1 and 2 demonstrated that Pavlovian-generated

associative inhibition does transfer to the associative structure created during the preconditioning phase of SPC. This phenomenon was designated transfer inhibition in SPC and involved the (a) sequential pairing of two neutral stimuli, CS1 and CS2, (b) unpaired inhibitory conditioning with CS2 and a US, and (c) application of an accepted test procedure for inhibition to assess the associative status of CS1 and CS2 (Experiment 1) or CS1 by itself (Experiment 2). Experiment 3 demonstrated that associative inhibition could be directly established in the preconditioning phase. This phenomenon was designated direct inhibition in SPC and involved (a) unpaired inhibitory training of two neutral stimuli, CS1 and CS2, (b) CS2-US excitatory conditioning, and c) testing for inhibition with CS1. The studies employed control groups that (a) provided excitatory (Experiment 1) and neutral associative (Experiment 1, 2 and 3) contrasts against which the level of inhibition accrued in the experimental groups was assessed, and (b) ensured that the effects of inhibitory training were due to associative rather than nonassociative processes (Experiment 1, 2 and 3). The results were not consistent with a perceptual learning account of SPC but rather, were consistent with the notion that there are general associative rules which are expressed similarly in Pavlovian conditioning and SPC. In addition, the implications for theories of conditioned inhibition and associative learning theories in general, were explored. It was concluded that Konorski's theory of conditioned

inhibition and Wagner & Brandon's (1989) AESOP associative learning theory provided the most complete account. Finally, the potential directions for future research on conditioned inhibition in SPC were discussed.

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Associative learning has been defined as an enduring change in the mechanisms of behavior as a consequence of an organism's experience with a relationship between perceptual events (cf. Domjan & Burkhard, 1986; Frolov, 1937; Gormezano & Marshall, 1983; Weisman & Dodd, 1979). To identify the defining characteristics of associative learning, conditioning researchers typically manipulate the relationship between perceptual events and observe the effect of these manipulations on behavior. Central to this experimental tactic is the assumption that associative learning is measured via performance changes that occur either during conditioning or during post-conditioning test procedures. That is, changes in behavior are assumed to correspond directly with changes in associative learning. However, the assumption of an isomorphic relation is not demanded by the definition of associative learning. Some researchers (e. g., Domjan & Burkhard, 1986; Hall, 1976; Hull, 1943) have argued, both logically and empirically, that changes in performance frequently do not reflect changes in the underlying organization and structure of associative learning. Acknowledgment of the sometimes independent nature of learning and performance is more consistent with the definition of associative learning because the definition asserts that learning constitutes a change in the mechanisms of behavior rather than behavior itself. Focusing on mechanisms of behavior instead of

instances of behavior allows both for learning to occur in the absence of performance and for performance changes to result from processes other than associative learning. The distinction between learning and performance is particularly relevant in defining two major classes of associative learning.

Since Pavlov's seminal works on conditioned reflexes, researchers have maintained that two fundamental classes of conditioning processes are associated with two distinct types of associative learning: associative excitation and associative inhibition (Pavlov, 1927; Weisman & Dodd, 1979). Prior to defining these two types of learning it is necessary to distinguish between conditioning processes and associative learning. Conditioning processes refer specifically to operations (i. e., manipulation of perceptual events) and behavioral effects (i. e., performance). The two basic types of conditioning processes are conditioned excitation and conditioned inhibition. Rescorla (1969) has defined conditioned excitation in terms of two operational criteria. These two criteria are (a) an operation relating a stimulus that does not produce a response (conditioned stimulus or CS), to a stimulus which does produce a response (unconditioned stimulus or US) such as the pairing of the two stimuli, and (b) a change in behavior that occurs as a consequence of the operation. A stimulus or CS which meets the two criteria is called a conditioned excitor. Conditioned inhibition was operationally defined (Rescorla, 1969) with respect

to conditioned excitation. Specifically, conditioned inhibition is produced by some operation relating a CS to a US, the ultimate result of which is a CS that controls a tendency opposite to that of a conditioned excitor (i. e., actively inhibits conditioned excitation). Thus, conditioned inhibition is defined only by its effects on conditioned excitation. A stimulus or CS which meets the above criteria is considered a conditioned inhibitor.

While the terms conditioned excitation and inhibition are anchored to the physical world by operations and behavioral effects, the terms associative excitation and associative inhibition refer to processes which are not directly measurable. Rather associative excitation and associative inhibition are inferences made from conditioned excitation and conditioned inhibition, respectively. The inferences form the organization and structure of associative processes. Thus, associative excitation and inhibition are theoretical terms which relate to the mechanisms of behavior. Learning theorists have offered a variety of positions concerning the theoretical mechanisms of behavior. For example, early theorists (e.g., Konorski, 1948; 1967; Pavlov, 1927) conceptualized associative excitation and inhibition in terms of neurological connections in the brain, whereas, more recently, the terms have referred to cognitive processes, such as memorial representations of relationships between stimuli (e.g., Wagner, 1981) or expectancies (e.g., Gibbon & Balsam, 1981). It is not the

intention of the present author to adopt one or the other of these theoretical definitions, but rather to accept that associative excitation and inhibition are concepts that relate imperfectly to a set of operations and behavioral effects. Thus, for the purposes of the present discussion, the terms associative excitation and associative inhibition refers to unobservable theoretical processes of learning that relate to the physical phenomena associated with conditioned excitation and conditioned inhibition, respectively.

In a general sense, the study of associative excitation and inhibition can actually be reduced to an investigation of the conditions that promote the development of conditioned excitation and inhibition. The conditions which promote associations (either type) have been elucidated through empirical studies which seek to manipulate variables that significantly impact on the development of conditioned excitation and inhibition. When the conditions have been well established through experimentation in a wide range of paradigms, the conditions are called associative rules (cf. Jensen, 1970). Thus, one of the major focuses of the study of association is the delineation of general rules that are important for the development of conditioned excitation and inhibition.

How are the conditions or general rules of association studied? There are two basic strategies. The first and most commonly adopted method of investigating associative rules is Pavlovian conditioning. In the typical Pavlovian conditioning

experiment, a neutral CS is repeatedly presented in some specified temporal relationship to a US. Determining whether the CS is an excitor is relatively easy because it is only necessary to monitor the development of responding as training proceeds (according to Rescorla's definition, all excitors impact directly on behavior). In contrast, the assessment of conditioned inhibition requires post-conditioning test procedures because, by definition, conditioned inhibition opposes the action of conditioned excitation and, therefore, normally has no observable effect on behavior during training. If behavior is unaffected as conditioned inhibition develops, then there can be no responding to monitor and hence the need for post-conditioning test procedures.

The second method to study association is sensory preconditioning (hereafter referred to as SPC). Although it was first studied in Pavlov's laboratory in 1931-1932 (Kimmel, 1977), W. Brogden (1939) is credited in North America with the first controlled demonstration of SPC. SPC consists of three distinct phases (Brogden, 1939; Prewitt, 1967; Seidel, 1959; Thompson, 1972). In the first phase, two CSs are repeatedly paired. For the purposes of the present discussion these two CSs are designated the "preconditioning CS" and the "to-be-conditioned CS". In the second phase, a response is conditioned to the to-be-conditioned CS by pairing it with a US. In the third phase, the preconditioning CS is presented alone in order to test for transfer of the response that

was trained in the second phase. Significant levels of responding to the preconditioning CS during the third phase indicates that response eliciting properties were transferred to the preconditioning CS via an association formed during the first phase of training.

Historically, SPC has been a methodology for studying associative excitation only. In fact, the prototypic procedure is defined in terms of the transfer of conditioned excitation to the preconditioned CS. The present studies intend to explore the potential of SPC for investigating associative inhibition. This point will be addressed more completely in a later portion of this introduction.

Because SPC is one of the central focuses of the present studies, it will be necessary to briefly discuss control procedures that permit the identification of SPC. The first point that needs to be considered here is that SPC is conceptualized as an associative chaining phenomenon involving the preconditioning CS, the to-be-conditioned CS, and a response. Specifically, in Phase 1 of SPC training, it is assumed that pairings of the preconditioned and to-be-conditioned CSs result in the development of an association between these stimuli (cf. Mercier & Baker, 1985). In the second phase, CS-US conditioning involving the to-be-conditioned CS and the US typically results in the development of CRs to the to-be-conditioned CS. CR acquisition to the to-be-conditioned CS is generally taken as evidence that the to-be-conditioned CS has

entered into an excitatory association with the US. At this point, it is assumed that the preconditioned CS and the US have become associated via a common mediational event, namely the to-be-conditioned CS (i. e., the linkage takes the form: preconditioned CS-> to-be-conditioned CS-> US). During testing, the associative linkage between the preconditioned CS, the to-be-conditioned CS, and the US is demonstrated if the preconditioning CS controls some level of responding (cf. Holland & Ross, 1983, Kimble, 1961). Thus, one of the most important functions that a control can serve is to ensure that the responding controlled by the preconditioned CS is due to the associative chain and not some other source of responding. The most commonly recommended control group is a group of subjects that receives the same treatment as the subjects receiving SPC with the exception that the CSs in the first phase are unpaired (Kimble, 1961; Seidel, 1959; Thompson, 1972). Because the unpaired control group breaks the temporal contiguity between the preconditioned and to-be-conditioned CSs which is needed to make the associative chain, it is an ideal control for demonstrating the associative chaining properties of SPC.

On logical grounds, a case could be made for including a control group in which the to-be-conditioned CS and US are unpaired during the second or conditioning phase. This type of control would also be an ideal control for demonstrating the associative chaining properties of SPC as it would break the

temporal contiguity between the to-be-conditioned CS to the US. However, this type of unpaired control procedure is rarely included in studies of SPC. Accordingly, the remaining remarks on the unpaired control group will pertain especially to the unpaired control which receives unpairings of the preconditioning CS and the to-be-conditioned CS.

Another problem for which the unpaired group controls is the possibility that familiarity with the test stimulus can account for the SPC effect (i. e., responding to the preconditioning CS in subjects given SPC training). If responding during testing is greater in SPC subjects than in the unpaired control subjects (indicating an SPC effect), then this difference can't be attributed to familiarity with the test stimulus because the unpaired control has equivalent stimulus experience but did not show the same effect during testing.

The unpaired group also controls for the possibility that stimulus generalization may account for the SPC effect. Stimulus generalization occurs when a response conditioned to a specific stimulus is elicited by other stimuli, normally of the same sensory modality. In the case of SPC, it could be argued that test responding (to the preconditioned CS) is simply a consequence of stimulus generalization. However, if an unpaired control is employed and the SPC group shows greater responding during the test phase, then the results can not be explained by appealing to

stimulus generalization because there is no reason why unpairings in the control group should diminish a stimulus generalization effect.

On a final note regarding control procedures, two studies by Pfautz, Donegan and Wagner (1978) have provided data which suggested that protection from habituation may be another problem that needs to be controlled when investigating SPC. Briefly, habituation is the waning of behavioral and/or electrophysiological responses with repeated stimulation (Harris, 1943; Sokolov, 1963; Thompson & Spencer, 1966). Protection from habituation occurs when the temporally first stimulus in a sequential stimulus compound is protected from habituation as a consequence of being followed by the other stimulus (Pfautz, Donegan & Wagner (1978). In SPC, protection from habituation can influence test results in response systems where, for example, the presumed neutral stimuli (CSs) to be used in the first phase of SPC actually elicit the target response prior to conditioning. This is most notable in the case of the conditioned emotional response (CER) where the measure of SPC is the preconditioning CS's capacity to suppress ongoing instrumental responding. This is a problem because, in cases like CER, it would be difficult to know whether or not the SPC effect was due to associative transfer or to protection from habituation.

Phautz et al (1978) have provided one method for determining the contribution of the protection from habituation effect. In their studies, subjects were trained with two pairs of

potential CSs, namely, S1-S2 and S3-S4. Subsequently, only S2 was paired with an effective US. The test phase consisted of presentations of both S1 and S3. The level of responding observed to S1 was taken as an index of the combined effects of SPC and protection from habituation; whereas S3 responding was taken as an index of protection from habituation alone. The results indicated a substantial level of responding to S3 but an even greater level to S1. This finding suggests that, for some response systems, protection from habituation contributes only partially to the observed SPC effect.

It does not seem likely that protection from habituation would pose a problem in SPC studies that make use of response systems in which neutral CS presentations do not yield appreciable levels of responding. If neutral CS presentations do not produce significant levels of responding in the targeted response system, then it follows that there can be no appreciable levels of behavior to be protected from habituation and therefore no protection from habituation. One example of a response system which is unlikely to yield an appreciable protection from habituation effect in the study of SPC is the rabbit nictitating membrane (NM) response system. It is well known that NM responding occurs on less than 5% of neutral CS presentations and that this is not appreciably different from the 3% baseline rate of NM responding observed for this response system (Gormezano, Kehoe & Marshall, 1983). Thus, the study of SPC will

be unencumbered by the protection from habituation effect when response systems such as the rabbit NM response system are employed.

Returning to the topic of associative learning, it is noteworthy that Pavlovian conditioning and SPC are not entirely equivalent means of studying association. For example, one reason that Pavlovian conditioning, as contrasted with SPC, has been used so widely is that it generally produces more robust effects on behavior (Weisman, 1977). However, one major problem with the Pavlovian approach is that the robust behavioral effects rather than associative learning per se become the focus of the research. The trouble with emphasizing behavioral effects is that they can sometimes be the result of nonassociative rather than associative processes. For example, habituation is a nonassociative process which can produce changes in behavior. In addition, it is generally the case that more physically intense USs and CSs produce faster CR acquisition (Moore & Gormezano, 1977). However, these manipulations are assumed to impact on the motivational aspects of CR performance rather than impacting on the actual development of the CS-US association (cf., Moore & Gormezano, 1977). In both the above examples, placing an emphasis on performance is misleading because changes in performance are actually the result of nonassociative processes.

The important point here is that behavioral or performance-related factors are only part of the associative learning process. In fact, our understanding of associative learning would be greatly enhanced by research methodologies in which performance factors are not so pronounced. SPC offers a unique view of association not obtainable with Pavlovian conditioning methodology--a view of association that is less encumbered by performance variables. The stimuli in SPC, being essentially neutral, do not exhibit any appreciable motivational or response-eliciting properties for many behavioral systems -with a notable exception being the CER. Because SPC, as contrasted with Pavlovian conditioning, allows for the development of association in the absence of motivational and response-eliciting properties of the US, it is a superior method for studying "pure" association.

One of the central objectives of the study of association is to use conditioned excitation and inhibition as a vehicle for determining general rules for the development of excitatory and inhibitory associations, respectively. If the rules are truly general, then they should be manifest in a similar manner in both SPC and Pavlovian conditioning studies. Are there any data pertaining to this particular supposition ? In fact, a considerable body of evidence (see Appendix A for a review) examines the effects of a number of important variables (e.g., number of training trials, temporal order, CS intensity, partial pairings, differential

conditioning) and training procedures on subsequent development of conditioned excitation in both Pavlovian conditioning and SPC. What is most remarkable about these studies is that independent variables generally produce parallel outcomes in Pavlovian conditioning and SPC. This adds considerable strength to the position that the rules which govern the development of excitatory associations are general.

Given the extensive evidence for the generality of associative rules based on the development of conditioned excitation, it might be expected that a similar line of evidence exists for associative inhibition based on the development of conditioned inhibition. However, no such evidence exists. While a considerable number of Pavlovian conditioning studies of conditioned inhibition exists, there is no systematic demonstration of conditioned inhibition in SPC. This leaves open the question of whether or not conditioned inhibition occurs in SPC. If it does, a parallel between the associative rules that govern conditioned excitation and inhibition would be suggested, thereby affording additional credibility to the notion that the rules of association are general.

Investigations of conditioned inhibition in SPC would also address the question of the theoretical relationship between associative excitation and inhibition. Consider four major types of theoretical statements about this relationship. First, Konorski's final

thesis on conditioned inhibition (Konorski, 1967), as opposed to his initial perspective ((Konorski, 1948), asserted that inhibition was essentially the same as excitation insofar as both involve excitatory connections between gnostic units (gnostic units are essentially psychological representations of individual events, for example CSs and USs) of the CS and the US. Excitatory conditioning results in CS gnostic units entering into an excitatory association with US gnostic units while, inhibitory conditioning results in CS gnostic units entering into excitatory association with gnostic units that are antagonistic to those of the particular US (i. e., no-shock gnostic unit). In the case of a shock US, a conditioned inhibitor is a stimulus that has an excitatory association to a 'no-shock' gnostic unit that can inhibit a 'shock' gnostic unit, thereby reducing the probability of a CR when the latter is activated. Thus, Konorski views associative inhibition as developing out of the same process as conditioned excitation and that the antagonism between associative excitation and inhibition forms the basis for CR performance.

The second theoretical statement comes from Rescorla and Wagner (1972; Wagner & Rescorla, 1972) who posit that the strength of association between two events is represented by a single dimension or continuum. Associative excitation corresponds to a range of positive values within the associative dimension, while associative inhibition corresponds to a range of negative values.

Associative excitation and inhibition are viewed as essentially equivalent processes insofar as a single general rule describes the processes.

The third approach, a relatively informally developed position by Fowler, Kleinman, and Lysle (1985), conceptualizes associative inhibition as a slave process of associative excitation. More specifically, the model asserts that associative inhibition is functionally dependent on associative excitation, either in the form of a conditioned excitor or a mental representation of the US. In either case, associative inhibition exists only to the extent that associative excitation is maintained. It is noteworthy that this position is consistent with views promulgated by early learning theorists (Hull, 1943; Pavlov, 1927).

Fourth and finally, some theorists (e.g., Gibbon & Balsam, 1981; Miller & Schachtman, 1985) maintain that associative inhibition as a separate associative entity does not exist, but rather is the product of excitatory associative variables. For example, Miller and Schachtman's (1985) comparator hypothesis posits that associative inhibition, as evidenced by conditioned inhibition, is simply a performance phenomenon that is a product of the comparison of two independent sources of associative excitation (i. e., a discrete CS and a comparator stimulus, such as the context). Thus, these theorists maintain that associative inhibition exists only as a performance by-product of associative excitation. The view of

inhibition endorsed by these theorists is similar to that of Skinner (1938), who argued that inhibition is a superfluous concept that could be more parsimoniously explained in terms of reduced excitation.

To summarize, Konorski (1967) and Rescorla and Wagner (1972) argue that associative excitation and inhibition are essentially equivalent processes; Fowler et al. (1985) assert that associative inhibition is a slave of or dependent on associative excitation; and Miller & Schachtman's (1985) comparator hypothesis maintains that associative inhibition per se does not exist, and that conditioned inhibition is a performance by-product of conditioned excitation. What makes the study of associative inhibition in SPC so useful in assessing each model's view of associative inhibition is that SPC methodology, as opposed to Pavlovian conditioning methodology, provides a unique opportunity for studying inhibitory associations in the relative absence of performance variables (response eliciting and motivational properties) of the US. Accordingly, if associative inhibition exists in its own right and is equivalent to associative excitation as posited by Konorski (1967) and Rescorla-Wagner (1972), then one would more likely expect to find evidence of associative inhibition using SPC methodology. In contrast, if associative inhibition is either (a) dependent on conditioned excitation and hence dependent on the response-eliciting properties of the US (e. g. inhibition as a slave) or

(b) strictly a performance by-product of conditioned excitation, then one would be less likely to expect to find evidence of inhibition within SPC because SPC methodology minimizes performance variables such as the response eliciting and motivational properties of the US.

Prior to discussing how the present studies were designed to examine conditioned inhibition in SPC, it is necessary to provide a brief account of Pavlovian conditioning strategies for developing and assessing conditioned inhibition. Since the methods for developing and assessing conditioned inhibition are quite numerous, the present brief review will focus on the most commonly employed procedures for developing and assessing conditioned inhibition (see LoLordo & Fairless, 1985; Williams & Overmier, 1988). Specifically, the inhibitory conditioning procedures discussed are (a) Pavlovian conditioned inhibition, (b) differential conditioning, (c) long forward trace conditioning, (d) backward conditioning and, (e) explicitly unpaired stimulus presentations. The two assessment procedures to be described are the "summation" and the "retardation-of-learning" tests.

Pavlovian conditioned inhibition and differential conditioning are similar in that a conditioned excitatory stimulus (CS+) is paired with a US. They are also similar in that the putative inhibitory CS (hereafter referred to as CS-) is unreinforced. However, in Pavlovian conditioned inhibition the CS- is paired either simultaneously or

asynchronously with the CS+, while in differential conditioning the CS- is presented alone. The three remaining inhibitory training procedures involve the repeated presentation of just one CS and the US. In long forward trace conditioning, the CS offset antedates the US onset, leaving an empty interval between the stimuli. In order for long forward trace conditioning to be effective as an inhibitory conditioning procedure the empty period has to be sufficiently long to preclude CR acquisition in a specified response system (cf. Hinson & Siegel, 1980). In backward conditioning, the onset of the US reliably precedes the occurrence of the CS-, usually by some relatively small time period. Finally, explicitly unpaired presentations consist of CS- and US presentations which are separated by long variable time periods and occur in a random or predetermined irregular sequence. Each of these five procedures reliably produces conditioned inhibition.

To identify that conditioned inhibition has accrued to a putative inhibitory CS, two tests are used: the summation test and the retardation-of-learning test. A summation test for inhibition consists of two types of stimulus presentations: (a) compound presentations of a putative conditioned inhibitor with a previously trained conditioned excitor, and (b) presentation of the conditioned excitor alone. If the target CS is inhibitory (i. e., imbued with properties which oppose conditioned excitation), then it is expected that responding on the compound trials will be less than on the

conditioned-excitor-alone trials because inhibition will diminish response-eliciting properties of the conditioned excitor. The retardation-of-learning test requires that subsequent to inhibitory training, the putative conditioned inhibitor be repeatedly paired with the US in order to transform it into a conditioned excitor. The logic of this test is that if the CS has been imbued with inhibitory properties which are opposite to those of conditioned excitors, then relative to a relevant control stimulus, it will resist or retard any effort to convert it into a conditioned excitor. It is often recommended that both tests be used when assessing putative conditioned inhibitors (Hearst, 1972; Rescorla, 1969). However, this recommendation is infrequently put into practice. One reason for this is that the conditioning procedures described above are well established as inhibitory training procedures. Another reason is that some conditioning phenomenon (e. g., facilitation of inhibitory conditioning following repeated exposure to the US alone) simply do not permit, on methodological grounds, the use of both testing procedures (Saladin & Tait, 1986).

Having discussed how conditioned inhibition is demonstrated with Pavlovian conditioning methodology, it is now possible to consider how it might be demonstrated with SPC. Logically, conditioned inhibition might be studied in two ways. First, conditioned inhibition in SPC could be developed in the same manner that conditioned excitation has been demonstrated with

SPC in the past. For example, the initial phase would consist of pairings of the preconditioning and to-be conditioned CSs. The second phase would consist of inhibitory conditioning of the to-be-conditioned CS. In the test phase, either the summation, or retardation-of-learning test, could assess the inhibitory properties of the preconditioning CS. Retarded CR performance in either the retardation-of-learning or the summation test would suggest that the inhibitory association developed during the second phase had transferred to the preconditioned CS via the excitatory association developed during the first phase.

Alternatively, one could attempt to establish an inhibitory association during the first phase of SPC training. Specifically, the preconditioning and to-be-conditioned CSs could be placed in a relationship to each other which typically results in the development of inhibition in Pavlovian conditioning. For example, the first phase could consist of unpaired presentations of the two CSs. In the second phase, the to-be-conditioned CS would undergo excitatory conditioning with a US. As with the first method, a test(s) for inhibition would be performed with the preconditioning CS in the final phase. Retarded CR performance during testing would suggest that the preconditioning CS had accrued associative inhibition as a result of its inhibitory relationship to the to-be-conditioned CS, which subsequently acquired conditioned excitation during excitatory conditioning. Thus, the preconditioning CS could become

a conditioned inhibitor by (a) conducting inhibitory training in the second phase of SPC, or (b) conducting inhibitory training in the first phase of SPC. To clarify subsequent references to these two methods of studying inhibition in SPC, the present author has designated the first method discussed above as transfer inhibition and the second as direct inhibition. The identification of transfer inhibition would parallel the typical SPC observation in that the inhibition accrued to the preconditioning CS would be transferred from the conditioned inhibition trained in the second phase. The identification of direct inhibition would be a unique demonstration of associative inhibition because direct inhibition would only result if inhibitory properties of the preconditioning CS were established prior to the introduction of conditioned excitation (i. e., in the first phase of SPC).

As already noted, there are no systematic investigations of inhibition in SPC with either of these methods. However, two studies (Rescorla, 1984; Tait, Black, Katz, & Suboski, 1972) provide some indirect evidence that inhibition in SPC may be obtained with either the first or second method, respectively. These studies are briefly discussed below.

In the Rescorla (1984) study, pigeons were given preconditioning pairings of (a) a "marbled" context CS (designated the preconditioning CS-) and a white discrete key light CS (designated the to-be-conditioned CS-) and, (b) pairings of a

"striped" context CS (designated the preconditioning CS+) and a dotted discrete key light CS (designated the to-be-conditioned CS+). In the subsequent conditioning phase, subjects received differential conditioning, a known inhibitory training procedure (Moore, 1972; Pavlov, 1927; Reberg & Black, 1969; Saladin & Tait, 1986; Wessels, 1973; Williams & Overmier, 1988). Specifically, subjects received both excitatory conditioning trials (CS+), consisting of pairings of food and the dotted discrete CS (i. e., to-be-conditioned CS+) and inhibitory conditioning trials (CS-), consisting of white light discrete CS alone (i. e., to-be-conditioned CS-). The differential conditioning was conducted in the unlined context.

The data of interest comes from two test conditions that were designed to evaluate associations between the discrete CSs (to-be-conditioned CSs) and contextual stimuli (preconditioning CSs). In one test condition, the subjects were returned to the two preconditioning contexts (i. e., the contexts which should have acquired excitatory and inhibitory properties through differential SPC), and their activity level was monitored. In line with previous research (cf. Kaplan & Hearst, 1985), it was expected that if a context-CS association was formed during preconditioning, then subjects would evidence higher levels of activity in the "striped" context (preconditioning CS+) that was paired with the dotted CS, which <u>was</u> subsequently reinforced (to-be-conditioned CS+), than in

the "marbled" context (preconditioning CS-) that was paired with a CS, which was not subsequently reinforced (to-be-conditioned CS-). This expectation was confirmed. The subjects tested in the context for which the key light had been reinforced were active on 60 % more of the observations than were the subjects tested in the other, presumably inhibitory, context.

Most relevant to the present discussion is the observation that subjects exhibited what might be called an excitatory response tendency (i. e., relatively high levels of general activity) in the context associated with a reinforced discrete CS (i. e., preconditioning CS+) and an inhibitory response tendency (i. e., relatively low level of general activity) in the context that was associated with an unreinforced discrete CS (i. e., preconditioning CS-). Although these data do not provide conclusive evidence of transfer inhibition in SPC, it does suggest that contextual stimuli which undergo differential inhibitory SPC exhibit a behavioral discrimination which appears similar to the type of discrimination that develops with discrete cue Pavlovian differential conditioning.

Rescorla's (1984) second test condition was also indicative of transfer inhibition accruing in SPC. Although this test phase was not designed specifically as an inhibition test, it did have some of the characteristics of a summation test for inhibition. Specifically, key peck responding was monitored when the previously trained CS+ was presented in each of the contexts that had undergone

differential SPC. Thus, a discrete conditioned excitor was presented in (i. e., compounded with) the context that was assumed to have excitatory properties (i. e., the preconditioning CS+ or striped context) and in the other context that was assumed to have inhibitory properties (i. e., the preconditioning CS- or marbled context). Consistent with the outcome of a positive summation test, key peck responding was substantially lower on trials where the conditioned excitor was presented in the putative inhibitory context (i. e., preconditioned CS-) than on trials where it was presented in the putative excitatory context (i. e., preconditioned CS+). The difference was statistically significant (i. e., 40 vs. 80 mean responses/minute).

While the Rescorla (1984) study provides some interesting <u>suggestive</u> data, it by no means constitutes a well controlled demonstration of transfer inhibition in SPC. In reference to the findings of the first test condition, the fact that birds were less active in the putative inhibitory versus excitatory context is not conclusive evidence that inhibition had developed as a result of differential SPC. A better case for inhibitory SPC could have been made if the preconditioned putative inhibitory and excitatory contexts were shown to control lesser and greater responding, respectively, relative to a group of naive control subjects. Even if such results were obtained, the best evidence for inhibition would

come from accepted inhibitory test procedures. This brings to immediate consideration, the results of the second test condition.

The Rescorla (1984) study was not intended to identify inhibition in SPC and consequently the second test condition was not designed to be an appropriate summation test. The greatest weakness of this test was the failure to identify the level of responding controlled by a discrete CS in a neutral context. The only evidence for inhibition was a lower level of responding on compound trials of a discrete excitatory CS and the putative inhibitory context (i. e., preconditioning CS-) than on compound trials of a discrete excitatory CS and a putative excitatory context (i. e., preconditioning CS+). The problem with this observation is that there is no way of knowing whether the difference on the combined stimulus trials is due to inhibitory properties accrued to the putative inhibitory context or to the response-potentiating properties accrued to the putative excitatory context. Clear evidence for inhibition would have been obtained if it had been demonstrated that the compound trial of the excitatory CS and inhibitory context (i. e., preconditioning CS-) controlled significantly less responding than trial presentations of the excitatory CS in a novel (neutral) context. Unfortunately, test comparisons of this nature were not conducted, and consequently the results of the second test condition are only suggestive of inhibition in SPC. In addition, the results are complicated by the absence of any

independent evidence showing that the differential conditioning parameters in the study were effective inhibitory training parameters. Again, this deficiency can be attributed to the fact that the study was not designed to demonstrate transfer inhibition in SPC.

A study by Tait et al. (1972) also provided some limited evidence for direct inhibition in SPC. During preconditioning, rats received either 7, 14, 28, or 56 pairings of a tone and light and an equal number of unpaired tone presentations of a different frequency. CER training with the light stimulus followed preconditioning. Differential SPC was assessed by measuring the level of lick suppression to the paired and unpaired preconditioning stimuli. The most significant observation in this study was that the paired preconditioning tone controlled more suppression than did the unpaired preconditioning tone. In Pavlovian differential conditioning a similar pattern of performance is observed between two CSs, one of which is directly paired with the US and one that is unpaired (e. g., Saladin & Tait, 1986). Most importantly, it is known that differential Pavlovian conditioning studies often reveal the unpaired CS to have inhibitory properties (e. g., Saladin & Tait, 1986). Since the Tait et al. (1972) study identified discriminative performance between the paired and unpaired preconditioning CS which parallels that commonly found in Pavlovian conditioning studies between the CS paired with the US and unpaired CS, it seems possible that the

unpaired preconditioning CS accrued appreciable levels of associative inhibition. Unfortunately, no explicit test for inhibition was performed on the unpaired preconditioning CS, and, thus, definitive evidence of inhibition was not obtained. Again, this deficiency can be attributed to the focus of the study, which was to document the acquisition of differentiation in SPC rather than inhibition.

In summary, Rescorla (1984) and Tait et al. (1972) study offer some suggestive evidence of inhibition in SPC. However, because the studies did not use the appropriate control and test procedures to identify inhibition in SPC, they can not be construed as strong evidence. Strong evidence for inhibition would come from SPC studies which have two important procedural criteria that were absent in the Rescorla (1984) and the Tait et al. (1972) study. First, they would employ the retardation-of-learning and/or summation test procedures for identifying inhibition. Second, they would have to provide independent evidence that the specified inhibitory conditioning parameters are capable of producing behavioral inhibition.

The present studies meet these procedural criteria. Experiments 1 and 2 employed the transfer method to study inhibition in SPC while Experiment 3 employed the direct method. All three studies employed the unpaired inhibitory conditioning procedure and the retardation-of-learning test for inhibition. The

rationale for adopting the unpaired inhibitory conditioning procedure and the retardation-of-learning test for inhibition is briefly discussed below.

Recent empirical findings which suggest that the unpaired procedure may be one of the purest methods of developing inhibition. Briefly, Williams and Overmier (1988) have reported two studies which indicate (a) that conditioned inhibitors, in addition to having inhibitory properties, also have collateral excitatory properties which sometimes mask the presence of inhibition during testing, and (b) that the Pavlovian, backward, and trace conditioned inhibitors, as opposed to unpaired and differential conditioned inhibitors, are more likely to have significant collateral excitatory properties. Therefore, based on these observations, the unpaired or the differential training procedures should maximize the probability of demonstrating inhibition in SPC. The choice of the unpaired over the differential procedure was based on the logic that the former is a purer conditioned inhibitor because it is not trained in the presence of a discrete conditioned excitor (i. e., a CS+) and, therefore, is less likely to develop collateral excitatory properties that might interfere with the expression of conditioned inhibition.

Having described the reasoning behind the selection of the unpaired procedure as a means of producing conditioned inhibition, it is now necessary to consider the most appropriate means of assessing inhibition. The retardation-of-learning test was chosen

over the summation test because of practical and methodological considerations. The retardation-of-learning test is a simple assessment procedure that involves the pairing of the putative inhibitor with the US subsequent to all training protocols. In contrast, the summation test requires the training of an independent conditioned excitor to be used during testing. The fulfillment of this requirement would necessitate an increase in the complexity of the present studies because it would call for an additional phase of training either before or after the preconditioning phase. Training a conditioned excitor prior to conducting SPC would be undesirable insofar as any training prior to Phase 1 pairings (i. e., pairings of the preconditioning and to-be-conditioned stimuli) is not consistent with accepted SPC training protocols and could affect the associative dynamics of the context (cf. Baker, Singh & Bindra, 1985). Additionally, the excitatory properties of a conditioned excitor trained at any other time during SPC could possibly interact with the processes that develop during SPC and interfere with the expression of inhibition in SPC (see Saladin & Tait, 1986 for a comparable argument regarding the use of the summation test). Thus, the retardation-of-learning test circumvents the practical and methodological difficulties associated with the summation test.

Experiment 1

The goal of Experiment 1 was to identify transfer inhibition in sensory preconditioning of the rabbit's nictitating membrane

response (hereafter referred to as NM response). To achieve this goal, six different training protocols were used. One was designed to promote the development of inhibition in SPC, while the remaining five protocols served as controls.

One of the control groups (SPC control) received traditional excitatory SPC training. The group served two purposes. First, if the experimental group failed to show evidence of inhibition but the traditional SPC effect was observed in this control group, then the failure to observe transfer inhibition could not be attributed to insufficient training in the preconditioning phase. Second, the assumed opposing nature of conditioned excitation and conditioned inhibition could be assessed by comparing the control with the experimental group during testing.

A second control group, called the no-treatment group, was employed to establish an associative null point. This group did not receive any training in the preconditioning and conditioning phases but did participate in the test phase. The no-treatment group served as a reference point from which both the level of inhibition accrued in the experimental group and the level of excitation accrued in the SPC control group could be determined.

Two other control groups were designed to interrupt the transfer of the association to the preconditioning stimulus. For one group, the disruption was accomplished by replacing the to-be-conditioned stimulus with a different CS during the preconditioning

phase. For the other group, the replacement of the to-be-conditioned CS occurred in the second phase of SPC. If the experimental group, but not these two controls, showed evidence of inhibition during testing, then the performance of the experimental group could be more confidently interpreted as evidence of associative transfer rather than as a nonassociative consequence of it's training history.

Finally, a fifth group served as a control for the potential confounding effects of latent inhibition. Latent inhibition is characterized by retarded conditioning to a CS as a result of preconditioning exposure to that CS alone (cf. Reiss & Wagner, 1972). If the test performance of the experimental group showed greater evidence of inhibition than this control group which has received only exposure to the to-be-conditioned CS during the second phase, then the inhibition in the experimental group would be due to conditioned inhibition rather than latent inhibition.

The present studies were concerned with a new area of research in associative learning. Consequently, the training parameters employed in these studies were not based on any previous research pertaining specifically to inhibition and SPC but rather were based on (a) the findings of a recent series of rabbit NM studies dealing with traditional excitatory SPC (Port & Patterson, 1984; Port & Patterson, 1985; Port et al. 1987) and, (b) pilot

research on SPC conducted in the laboratory with which the author is affiliated.

Method

Subjects

Forty-eight naive male and female New Zealand white rabbits (Oryctolagus cuniculus), approximately 100 days old and weighing between 1.5 and 2.5 kg, were obtained from Central Animal Care Services at the University of Manitoba. All subjects were housed in separate 60 x 45 cm wire mesh cages located in a ventilated colony room which was maintained on a 12-hr light cycle and at temperature of 23° C ± 1° C. All subjects were given free access to Landmark rabbit food and to water.

Apparatus

Conditioning of the rabbit's third eyelid or nictitating membrane (NM) response was conducted in an apparatus which has been described elsewhere (Gormezano, 1966). Briefly, each subject was placed in a Plexiglas restraining box equipped with an adjustable, foam-padded headstock, which minimizes gross head movement. The head of each subject was fixed in an upright position by a foam-covered pinnae clamp. The right outer eyelids was held open by two Newey tailor hooks which were attached to a bipartite Velcro strip that was adjusted to fit securely around the rabbit's head once each of the eyelids was inserted in the Newey hooks.

Extensions of each rabbit's NM was transduced in the following manner. A 10K ohm rotary microtorque potentiometer was mounted on a light-weight metal frame which was positioned securely on top of the subject's head. A piano wire armature was mechanically fixed to the rotary axle of a potentiometer. A thread leading from the armature was tipped with a staple that was carefully hooked through a 2.0 mm loop of 00 Ethicon suture embedded in the outer edge of the NM. The tautness of the thread was adjusted while the NM was in its retracted state such that there was no slack in the thread.

The experiment was conducted in one of eight identical fireproof, legal-size, file-cabinet chambers (66.3 x 32.5 x 27.5 cm). A ventilation fan was located at the back of, and external to, each sound-attenuating chamber. Electrical wires, speakers, and chamber light were concealed by an off-white stimulus panel (32.5 x 27.5 cm) which formed the false front of each chamber unit. Mounted behind each stimulus panel were two 8.75 cm, 8-ohm Jana speakers. The speaker on the right side of the panel was used for the delivery of a 500-ms, 80-dB, 10-Hz clicker (scale C, SPL), which served as CS1. CS2 was a 500 ms, 137895.14 N/m^2 airpuff delivered through a blunted 16-ga. needle which was fixed approximately 3.0 cm from each animal's left abdominal region. A 24-V, 6-W incandescent light bulb which was recessed behind a translucent white plastic cover that was on the left side of stimulus

panel provided both CS3 and the ambient chamber illumination throughout the experimental sessions. CS3 was a 500-ms, 10-Hz flashing of the houselight. A three-pin female stereo socket located at the center top of each stimulus panel allowed the electrical interfacing of each potentiometer. Female banana plugs, which were located in the lower right hand corner of the stimulus panel, permitted the delivery of a 3.0-mA, 100-ms, 60-Hz constant current shock US delivered to two stainless steel 9 mm Autoclips implanted 10 mm behind and 10 mm above and below the horizontal plane of the right eye. Continuous masking white noise was presented in the experimental room at 72 dB (scale C, SPL).

A software system modified from Tait & Gormezano, (1974) and implemented on a 8K Raytheon 703 computer controlled the timing and delivery of the stimuli and the recording of responses. The voltage output of each potentiometer was sensed by one channel of a 10-bit, 16-channel analog-to-digital converter interface of the computer. The criterion for a CR or UR was set at 0.5 mm of movement of the NM response during the CSs or the US, respectively. In all cases, a minimum 0.5 mm extension of NM corresponded to a change of 0.2 volts in the potentiometer, which, in turn, corresponded to a hexadecimal output of 10 in the analog-to-digital conversion system. Throughout the experiment, NM responses were scored during the 500 ms interval following the onset of any individual CS. Nictitating membrane responses

occurring during the CSs were designated as conditioned responses (CRs).

Procedure

The experimental protocol consisted of the following phases: (a) a preconditioning phase that consisted of one daily session of preconditioning pairings of CS1 and CS2, (b) a conditioning phase that consisted of four daily sessions of inhibitory conditioning in which the to-be-conditioned CS and the US were unpaired and, (c) a test phase that consisted of two daily sessions. The first session of the test consisted of unreinforced presentations of the preconditioning CS (CS1) followed by a retardation-of-learning test for inhibition, while the second session was a continuation of the retardation-of-learning test. Thus, the total duration of Experiment 1 was 7 days.

One day after arrival, the subjects were randomly assigned to one of six groups, each containing 8 subjects. The six groups were given the following group designators or reference names. The transfer inhibition group and the control group that received traditional excitatory SPC were designated Groups TI and E, respectively. The no treatment control was referred to as Group N. The control groups which were similar to the experimental group with the exception that the transfer of inhibition was interrupted by replacing the to-be-conditioned CS (CS2) with an irrelevant CS (CS3) in either the preconditioning phase or the conditioning phase

were designated Groups C1 and C2, respectively. Finally, the latent inhibition control group was designated Group LI.

Next, the subjects were brought to the preparation room where they were individually restrained. The paraorbital region of the right eye was shaved and any remaining hair was removed with a depilatory lotion (Nair). Then, the subjects' right eyelids were retracted with eyestraps and the NM was lifted off the surface of the cornea with a cotton swab (Life) and a 2.0- mm loop of suture was implanted in the epithelial layer of the NM. Following implantation of the suture and at the end of each day or conditioning session thereafter, the subjects' eyes were treated with Vetropolycin antibacterial gel to reduce the probability of infection. In addition, the subjects in each group had a numbered from one to eight inscribed on their backs with a felt marker to ensure that each of them was suited with the same recording equipment, experimental chamber, and home cage for the duration of the experiment.

Following one day of recovery from surgery, the preconditioning phase commenced. During the preconditioning phase, the subjects in groups TI, E, C2 and LI received 10 CS1-CS2 pairings, where the onset of the 500-ms clicker CS1 preceded the onset of the air puff CS2 by 500 ms. Group C1 received the same preconditioning experience as the aforementioned groups with the exception that the flashing houselight CS3 was substituted for CS2.

The mean intertrial interval for all of the groups was 90 s (i. e., a randomized sequence of values of 60, 90 or 120 s was used). Group N remained in the home cage for the duration of the preconditioning phase.

For the conditioning phase, Groups TI and C1 received four daily sessions of unpaired conditioning consisting of 30 CS2 and 30 US presentations. Unpaired training for Group C2 was similar to that of Groups TI and C1 except that CS3 was substituted for CS2. For each group the CS and US were separated by a mean intertrial interval of 45 s (i. e., randomly scheduled intervals of 30, 45 or 60 s) and neither stimuli occurred more than twice consecutively. For group E, subjects received daily sessions of excitatory conditioning consisting of 30 CS2-US pairings. In this group, the mean intertrial interval was 90 s (i. e., values of 60, 90 or 120 s were used) and CS onset preceded US onset by 500 msec. Group LI received the same pattern and number of CS2 presentations as Groups TI and C1, but no US presentations were given. Thus, the mean intertrial interval for group LI was 90 secs (i. e., values of 60, 90 or 120 s were used). As in the preconditioning phase, subjects in Group N remained in their home cages.

In the test phase, all six groups received 10 CS1-alone presentations followed by a retardation-of-learning test for inhibition. The CS1-alone presentations were conducted during the initial 10 trials of the first day of testing. The retardation-of-

learning test for inhibition commenced immediately after CS1-alone test trials and consisted of (a) 20 CS1-US pairings, and (b) 20 CS2-US pairings. The onset of the CS preceded US onset by 500 ms. On the following day of testing, each group received an additional 30 CS1-US pairings and 30 CS2-US pairings. The CS1-US trials were conducted to assess whether transfer inhibition was obtained; whereas, the CS2-US trials were conducted to assess whether conditioned inhibition developed during Phase 2 training. The presentation of the two different trial types during the retardation-of-learning test occurred in a predetermined, irregular sequence with the stipulation that no one trial type could occur more than twice consecutively. During testing, the mean intertrial interval was 90 s (i. e., 60, 90 or 120 s were used). The response measures (i. e., dependent measures) for each phase of Experiment 1 are described in the appropriate section of the results below. A diagrammatic summary of the design and experimental protocol of Experiment 1 is presented in Table 1.

Insert Table 1 about here

Results and Discussion

To promote clarity of presentation, the results of each phase of Experiment 1 will be discussed in the order in which they were

Table 1: Diagrammatic summary of the design and experimental protocol of Experiment 1.

Phase / Group	Preconditioning (ONE DAILY SESSION)	Conditioning (FOUR DAILY SESSIONS)	Testing: CS Alone & Retardation Test (ONE DAILY SESSION)	Testing: Retardation Test (ONE DAILY SESSION)
TI	10 CS1-CS2 pairings	30 CS2/US* unpairings	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings
E	10 CS1-CS2 pairings	30 CS2-US pairings	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings
C1	10 CS1-CS3 pairings	30 CS2/US* unpairings	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings
C2	10 CS1-CS2 pairings	30 CS3/US* unpairings	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings
LI	10 CS1-CS2 pairings	30 CS2-alone	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings
N	No Treatment (home cage)	No Treatment (home cage)	10 CS1-alone & 20 CS1-US pairings 20 CS2-US pairings	30 CS1-US pairings 30 CS2-US pairings

Temporal Parameters:
CS1=500-ms, 80-dB, 10-Hz clicker
CS2=500-ms, 137895.14-N/m2 airpuff
CS3=500-ms, 10-Hz flashing light
US=100-ms, 60-Hz, 3.0-mA shock

All CS-CS intervals=500-ms
All CS-US intervals=500-ms
Mean Intertrial interval=90-s but * indicates 45-s

executed. Prior to discussing the results in detail, the general statistical procedures used in Experiment 1 are described below.

The overall design of Experiment 1 is a mixed model repeated measures design with groups as the between factor and days as the within factor. The data obtained during each of the four phases were initially statistically analyzed via BMDP 1V one-way ANOVA and BMDP 2V repeated measures ANOVA with concurrent analysis of orthogonal components for trend.

Several a priori outcomes were postulated for the various phases of Experiment 1 and each is described and evaluated in the appropriate subsection below. Orthogonal comparisons (Hays, 1981) were used to evaluate the a priori hypotheses. In addition, several a posterior orthogonal comparisons were conducted to explore group differences that were apparent following data collection. Trend analysis was used to identify the emergence of group differences across within factors (i. e., days).

Preconditioning Phase

In this phase, rates of NM responding were obtained for Groups TI, E, C1, C2 and LI by measuring the occurrence of NM responses during the 500-ms CS1 presentation of each of the 10, CS1-CS2 preconditioning pairings. No NM response measure was obtained for Group N as this group of subjects remained in their home cages during the preconditioning phase. Because all subjects received pairings of neutral stimuli during this phase, it was

expected that NM responding would be very low (ie., near or below a baseline rate of 3%) and similar between Groups TI, E, C1, C2 and LI.

The mean percentages of NM responding for Groups C2, TI, C1, E and LI were 1.4, 1.3, 0.0, 0.0 and 0.0, respectively. The means of 1.4 and 1.3 for Groups C2 and TI, respectively, correspond to the occurrence, in each group, of 1 NM response across 8 subjects and 10-trials. The difference in the means is due to the fact that for Group C2 one of the CS1 presentations for one subject was not scorable because the subject had responded just prior to the CS1 presentation. Accordingly, the denominator for the mean of Group C2 was 79 instead of 80, as in the case of Group TI.

The lack of variability between the groups precluded statistical analyses. Nonetheless, an inspection of the means indicated that NM responding was below 3% and uniform between groups. Thus, the means confirmed that baseline NM responding was uniformly low in Groups TI, E, C1, C2 and LI.

Conditioning Phase

During the 500-ms CS presentations, the occurrence of NM responses was assessed for subjects in Groups TI, E, C1, C2 and LI. In particular, rates of NM responses were obtained for Groups TI, E, C1, and LI during CS2 presentations while the same measure was obtained for Group C2 during CS3 presentations. The measures were taken over four daily sessions during which subjects received

either 30 conditioning trials (i. e., Groups TI, E, C1, C2) or CS alone presentations (i. e., Group LI). No measure of NM responding was obtained for subjects in Group N as they remained in their home cages during this phase. Only Group E received CS-US pairings during this phase and consequently, it was expected that it would be the only group to evidence CR acquisition.

The mean percentages of NM responding collapsed across days for Groups E, C1, TI, LI and C2 were 60.2, 2.0, 1.8, 1.1 and .6, respectively. Clearly, Group E showed much higher levels of CR performance as compared to the other groups. A repeated measures ANOVA revealed a significant Group main effect [$F(4,35) = 169.17$, $p < .01$] and a planned comparison in which Group E was compared to Groups C1, TI, LI and C2, combined, revealed that the CR performance of Group E was greater than that observed in Groups C1, TI, LI and C2 [$F(1,35) = 676.25$, $p < .01$]. Since Group E contributed 99% of the variance to the Group main effect, it was concluded that the NM responding in Groups C1, TI, LI and C2 did not differ statistically. Thus, the analysis confirms that, (a) Group E was the only group to evidence significant levels of CR performance, and (b) there were no appreciable differences in the low levels of NM responding attained by Groups C1, TI, LI and C2.

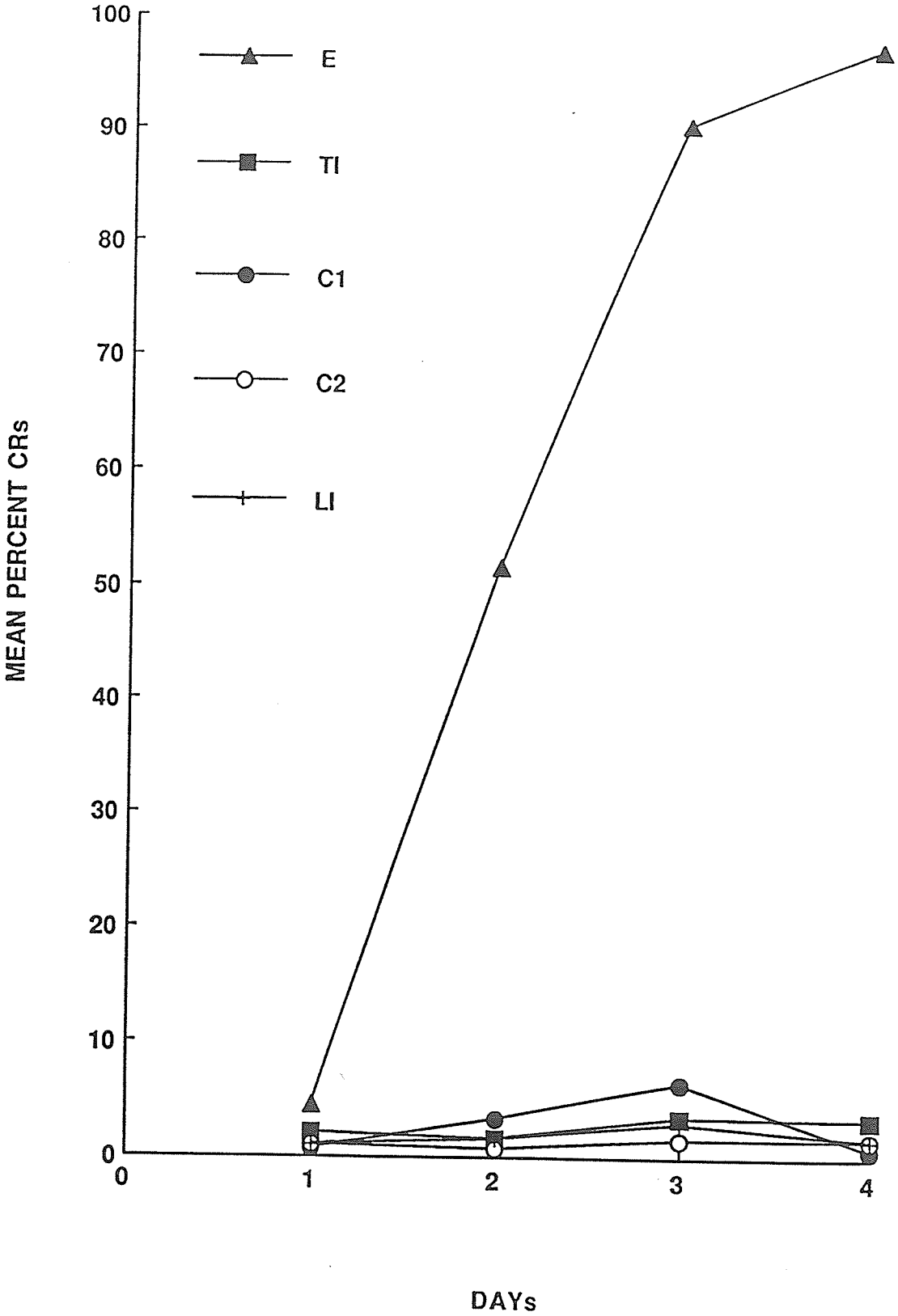
Figure 1 depicts the mean percentages of CRs as a function of the four daily sessions of Phase 2 conditioning for Groups E, C1, TI, LI and C2. The figure shows that CR acquisition reached high

asymptotic levels in Group E but not in Groups C1, TI, LI and C2, which failed to show any appreciable levels of CR acquisition. This observation was confirmed by the previous repeated measures ANOVA which yielded both a significant Days main effect [$F(3,105) = 46.69$, $p < .01$] and Group x Days interaction [$F(12,105) = 40.16$, $p < .01$]. The presence of a Days main effects indicates that CR acquisition occurred over the course of the conditioning phase. The Group x Days interaction confirms that CR acquisition occurred differentially among the groups.

Insert Figure 1 about here

Orthogonal components for trend applied to the days effect of each group revealed significant linear and quadratic components for Group E [linear - $F(1,7) = 391.72$, and quadratic - $F(1,7) = 7.23$, both $p's < .01$] but not for Groups TI, C1, C2, and LI [linear - $F's(1,7) = .37, .67, .89$ and 1.74, respectively, and quadratic - $F's(1,7) = .02, 4.25, .09$ and $.79$, respectively, all $p's > .07$]. The presence of linear and quadratic trend components in Group E but not Groups TI, C1, C2, and LI confirms that asymptotic CR acquisition occurred only in the former group. Additional evidence of asymptotic CR acquisition in Group E is indicated by the observation that it contributed 99.9% and 95.8% of the linear and quadratic variance, respectively, to the Group x Days interaction.

Figure 1: Mean percentages of NM CRs as a function of the four daily sessions of Phase 2 conditioning for Groups E, TI, C1, C2 and LI in Experiment 1.


100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
E
TI
C1
C2
LI
MEAN PERCENT CRs
DAYs

Conversely, Groups TI, C1, C2, and LI, combined, contribute only .1% and 4.2% of the linear and quadratic variance, respectively, to the Group x Days interaction. This finding clearly indicates that these groups evidenced minute levels of NM responding during the conditioning phase. Thus, the statistical analyses confirm the graphical interpretation that Group E showed asymptotic CR acquisition while Groups TI, C1, C2, and LI showed similar and minute levels of NM responding during conditioning.

CS1-Alone Test

In this initial phase of the test protocol, subjects in Group E, TI, C1, C2, LI and N were monitored for NM responses during each of 10, 500-ms CS1 presentations. This test condition was included to determine whether or not the traditional excitatory SPC protocol administered to Group E would result in the basic SPC effect (i. e., CR responding to CS1-alone presentations). Since this Group was the only group to receive training that could potentially generate excitatory CRs, it was expected that only Group E would show excitatory CRs during the CS1-alone test.

Figure 2 depicts the mean percentages of CRs/NM responses during the 10-trial block of CS1-alone presentations for Groups E, TI, C1, C2, LI and N. The means were 36.9, 2.8, 1.3, 0.0, 0.0 and 0.0, respectively. The figure indicates that Group E showed a high level of CR performance as compared to the other groups. The figure also indicates that Groups TI, C1, C2, LI and N showed similar

and low levels of NM responding during the test. A one-way ANOVA applied to the test data yielded a significant Group main effect [$F(4,42) = 7.68$, $p < .01$] and a planned comparison of Group E with Groups TI, C1, C2, LI and N, combined, revealed that the CR performance of Group E was greater than that observed in other groups [$F(1,42) = 38.19$, $p < .01$]. Since Group E contributed 99.5 % of the variance to the Group main effect, it was concluded that the NM responding in Groups TI, C1, C2, LI and N did not differ statistically. Thus, the analysis confirms the graphical interpretation that, (a) Group E was the only group to evidence significant levels of CR performance, and (b) there were no appreciable differences in the low levels of NM responding attained by Groups TI, C1 C2, LI and N.

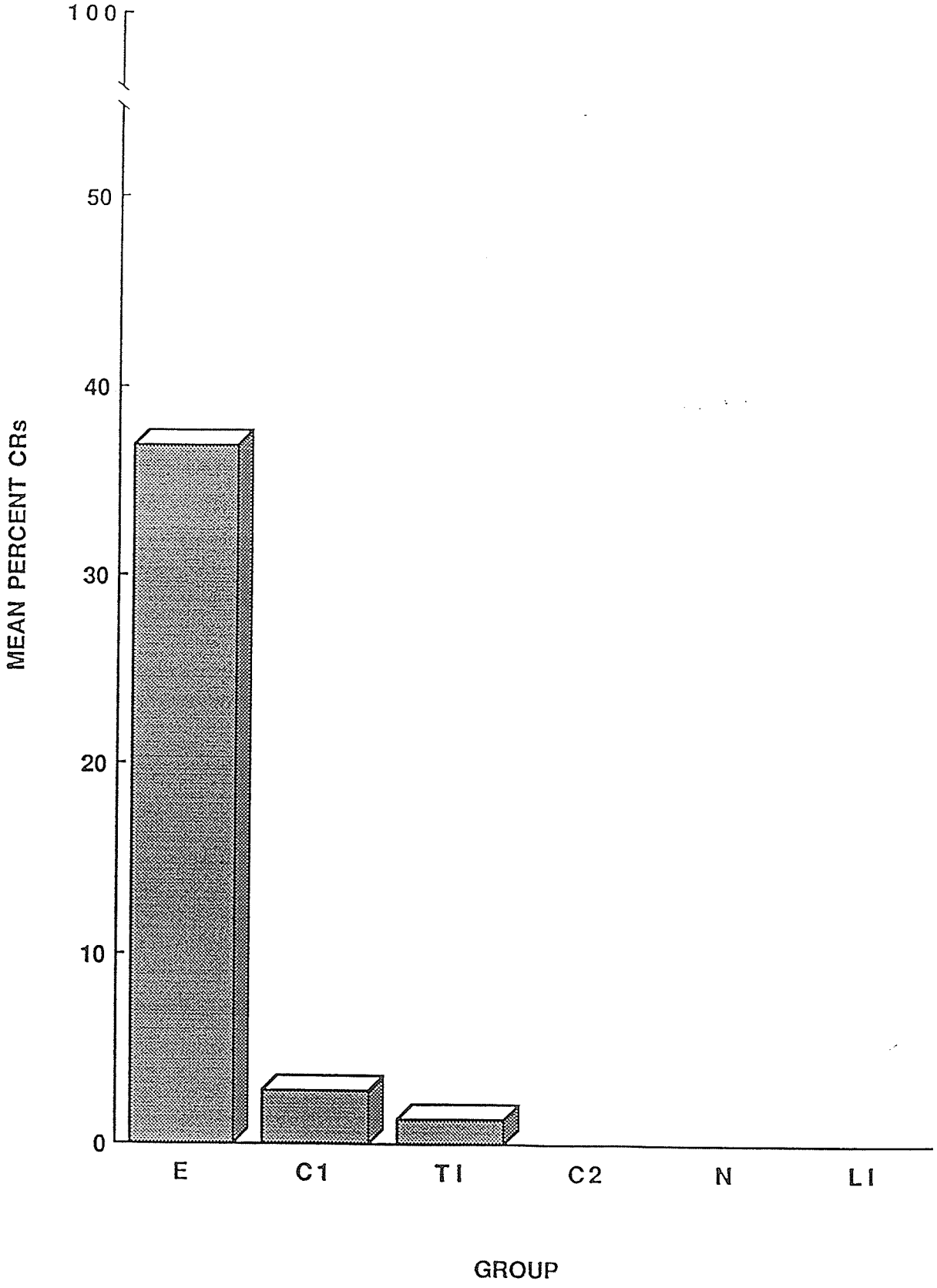
Insert Figure 2 about here

Retardation-of-Learning Test

To-be-conditioned CS (CS2). Over the two test sessions, the occurrence of NM CRs during 500-ms CS2 presentations was assessed for subjects in Groups TI, E, C1, C2, LI and N. The initial session consisted of 20 CS2-US pairings while the second session contained 30 CS2-US pairings.

A number of a priori hypotheses were postulated regarding the outcome the retardation-of-learning test on CS2. In particular,

Figure 2: Mean percentages of NM CRs during the 10-trial block of CS1-alone presentations for Groups E, C1, TI, C2, N and LI in Experiment 1.


100
50
40
30
20
10
0
MEAN PERCENT CRs
E
C1
TI
C2
N
LI
GROUP

the hypotheses were that (a) the highest level of CR performance would be evidenced by Group E as a consequence of prior excitatory conditioning (i. e., savings effect), and (b) due to their prior inhibitory training with CS2, Groups TI and C1 should evidence lower CR performance than the inhibitory control groups, Groups C2, LI and N. These hypotheses are evaluated below.

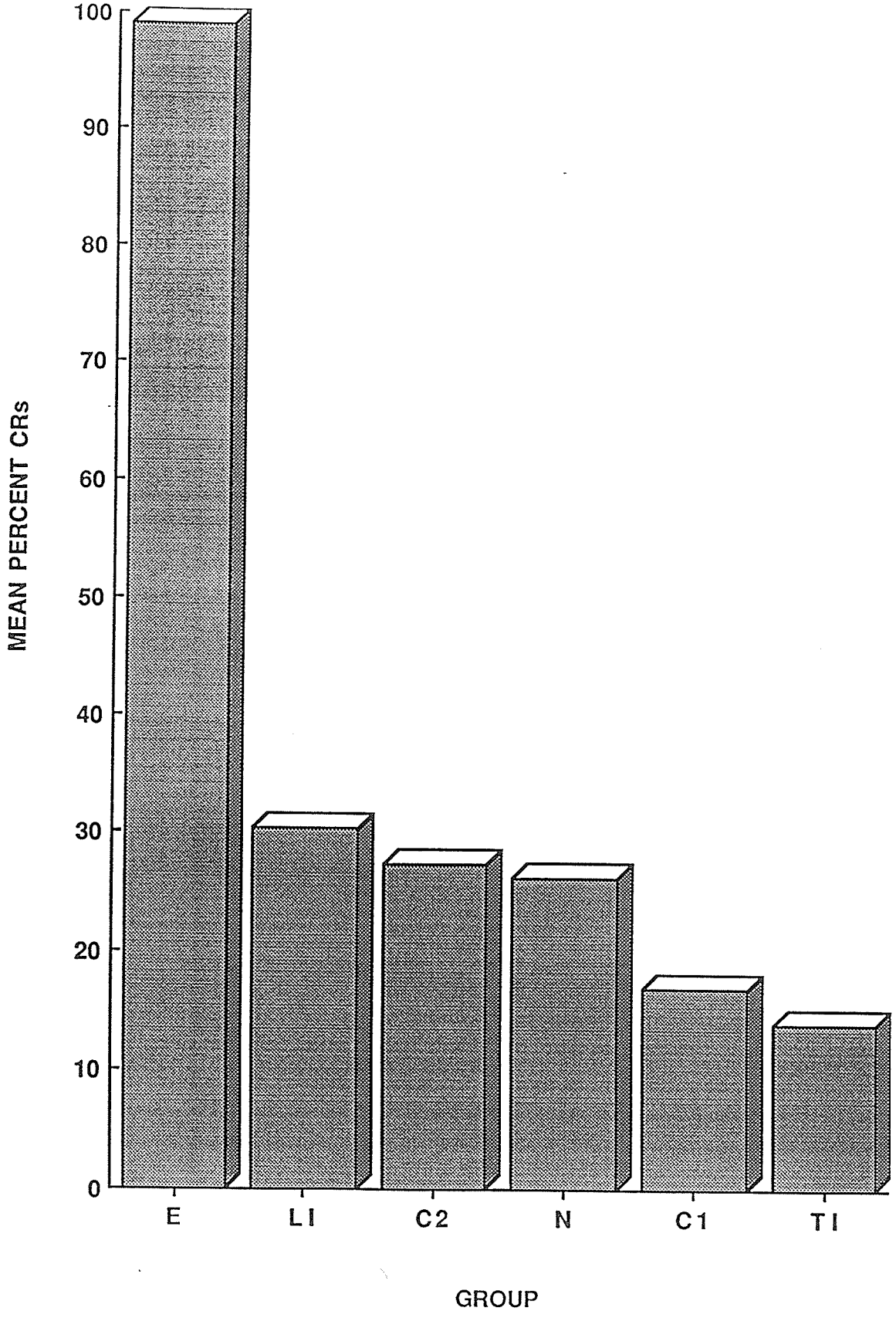
Figure 3 depicts the mean percentages of NM CRs to CS2 for Groups E, LI, C2, N, C1 and TI collapsed across the two sessions of the retardation-of-learning test. The means for Groups E, LI, C2, N, C1 and TI were 99.1, 30.3, 27.3, 26.3, 17.0 and 13.9, respectively. The figure indicates that Group E showed a higher level of CR performance than the other groups. This interpretation was supported by a planned comparison of Group E and the other five groups combined [$\underline{F}(1,42) = 133.83$, $\underline{p} < .01$]. The figure also suggests that the CR performance of Groups TI and C1 was lower than that of Groups LI, C2 and N. The planned comparison of Groups TI and C1 with Groups LI, C2 and N confirmed this graphical interpretation [$\underline{F}(1,42) = 5.16$, $\underline{p} < .05$]. A posterior pairwise comparisons between (a) the inhibitory controls, Groups C2, LI and N, and (b) between the inhibitory groups, Groups TI and C1, were all nonsignificant [All $\underline{F's}(1,42) < 1$]. Thus, the analysis confirms that (a) due to prior excitatory conditioning, Group E attained the highest level of CR performance, and (b) inhibition resulted from prior unpaired inhibitory conditioning with CS2 as indicated by the similar

and lower level of CR performance attained by Groups TI and C1. Logically, it would be impossible to discuss the transfer of inhibition to the preconditioned CS in the absence of evidence that the unpaired conditioning resulted in the to-be-conditioned CS becoming a conditioned inhibitor. The observation that conditioned inhibition did develop to the to-be-conditioned CS fulfills this necessary precondition for the assessment of transfer inhibition.

Insert Figure 3 about here

On a final note, a repeated measures ANOVA applied to the mean percentage of CRs to CS2 obtained over the two daily sessions of the retardation-of-learning test for Groups TI, E, C1, C2, LI and N revealed both a significant Days main effect [$\underline{F}(1,42) = 70.04$, $\underline{p} < .01$] and Group x Days interaction [$\underline{F}(5,42) = 3.86$, $\underline{p} < .01$]. The presence of a Days main effects indicates that CR acquisition occurred over the course of the conditioning phase. The Group x Days interaction indicates that CR acquisition occurred differentially among the groups. More specifically, the interaction appeared to be due to the similar pattern of CR acquisition evidenced by Groups TI, C1, C2, LI and N as contrasted with the high stable asymptotic values maintained by Group E. Orthogonal components for trend applied to the days effect of each group confirmed this interpretation by yielding a significant linear trend

Figure 3: Mean percentages of NM CRs to CS2 for Groups E, LI, C2, N, C1 and TI collapsed across the two sessions of the retardation-of-learning test in Experiment 1.


MEAN PERCENT CRs
100
90
80
70
60
50
40
30
20
10
0
E
LI
C2
N
C1
TI
GROUP

component for Groups TI, C1, C2, LI and N [all F's(1,7) > 7.0, all p's < .05] but not for Group E [F(1,7) < 1.0]. A repeated measures ANOVA applied to Groups TI, C1, C2, LI and N failed to identify a Group x Days interaction [F(4,35) = 1.23, p = .31] which further substantiates the similar pattern of CR acquisition in these five groups.

Preconditioning CS (CS1). Given that this test was conducted during the same sessions as the CS2 test, it follows that the occurrence of NM CRs during CS1 presentations were collected as described above for CS2. Again, the initial session consisted of 20 CS1-US pairings while the second session contained 30 CS1-US pairings.

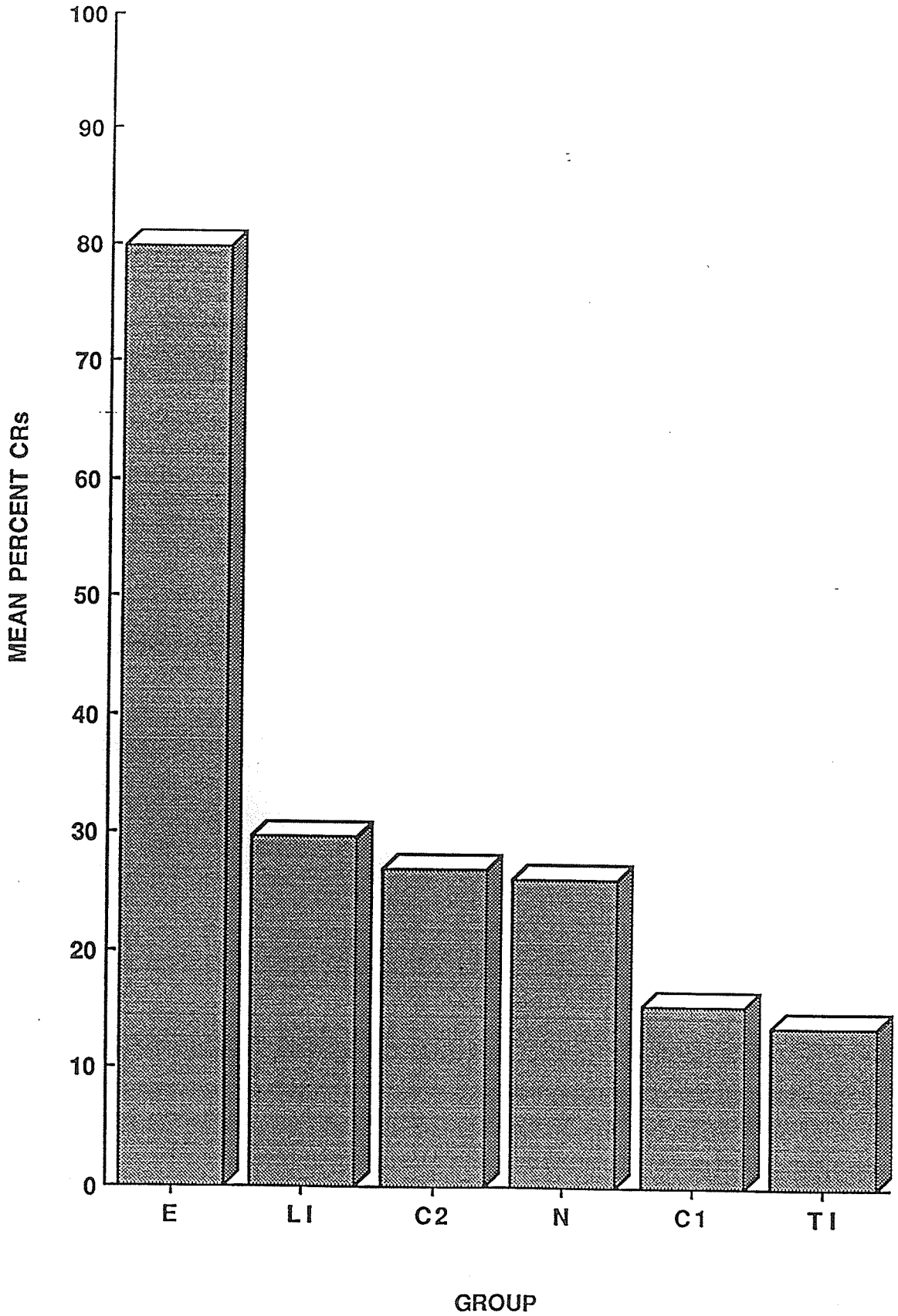
A number of a priori hypotheses were postulated regarding the outcome the retardation-of-learning test on CS1. In particular, the hypotheses were that (a) the highest level of CR performance would be evidenced by Group E as a consequence of prior excitatory SPC conditioning (i. e., a savings effect), and (b) due to its prior inhibitory SPC training with CS1, Group TI should evidence lower CR performance than the inhibitory control groups, Groups C1, C2, LI and N. These hypotheses are evaluated below.

Figure 4 depicts the mean percentages of NM CRs to CS1 for Groups E, LI, C2, N, C1 and TI collapsed across the two sessions of the retardation-of-learning. The means for Groups E, LI, C2, N, C1 and TI were 79.9, 29.7, 26.9, 26.1, 15.5 and 13.7, respectively.

The figure indicates that Group E showed a higher level of CR performance than the other groups. This interpretation was supported by a planned comparison of Group E and the other five groups combined [$F(1,42) = 66.85$, $p < .01$]. Contrary to expectation, the figure does not appear to indicate that Group TI attained a lower level of CR performance than Groups C1, C2, LI and N. Consistent with this graphical interpretation was the nonsignificant planned comparison of Group TI with Groups C1, C2, LI and N, combined [$F(1,42) = 2.31$, $p > .05$]. However, the figure does suggests that the CR performance of both Groups TI and C1 was lower than that of Groups C2, LI and N. This apparent difference was confirmed by an a posterior comparison of Groups TI and C1 with Group C2, LI and N [$F(1,42) = 4.93$, $p < .05$]. A posterior pairwise comparisons between (a) the controls, Groups C2, LI and N and (b) between Groups TI and C1 were all nonsignificant [All $F's(1,42) < 1$]. Thus, the analysis indicates that (a) due to prior excitatory SPC, Group E attained the highest level of CR performance , and (b) both Groups TI and C1 evidenced inhibition as indicated by the similar and lower level of CR performance attained by these groups as compared to the control Groups C2, LI and N, which also performed similarly.

Insert Figure 4 about here

Figure 4: Mean percentages of NM CRs to CS1 for Groups E, LI, C2, N, C1 and TI collapsed across the two sessions of the retardation-of-learning test in Experiment 1.


100
90
80
70
60
50
40
30
20
10
0
MEAN PERCENT CRs
E
LI
C2
N
C1
TI
GROUP

On a final note, a repeated measures ANOVA applied to the mean percentage of CRs to CS1 obtained over the two daily sessions of the retardation-of-learning test for Groups TI, E, C1, C2, LI and N revealed a significant Days main effect [$F(1,42) = 71.06$, $p < .01$] but not a significant Group x Days interaction [$F(5,42) = 1.33$, $p = .27$]. The presence of a Days main effect and absence of a Group x Days interaction indicates that similar rates of CR acquisition occurred over the course of the test phase for all groups.

In summary, evidence of excitatory SPC in Experiment 1 was indicated by the high level of CR performance of Group E, as compared to the other groups, in both the CS1-alone test and retardation-of-learning test with CS1. These findings are consistent with previous observations of excitatory SPC in the rabbit NM conditioning preparation (e. g., Port, Beggs and Patterson, 1987) and with the excitatory SPC literature in general (see Appendix A for a review). In fact, the level of CR performance on CS1-alone test trials obtained in the present study is virtually identical to that obtained in previous studies using the rabbit NM conditioning preparation (e. g., Port, Beggs and Patterson, 1987). In addition, the lower levels of CR performance evidenced by Groups TI and C1 as compared to Groups C2, LI and N in the retardation-of-learning test with CS2 indicates that the unpaired conditioning did produce associative inhibition. This observation is also consistent with

previous demonstrations of inhibition following unpairings of a CS and US in the rabbit NM conditioning preparation (e. g., Bromage & Scavio, 1978) as well as others (e. g., see LoLordo & Fairless, 1985 for a review).

In terms of the primary objective of the present study, transfer inhibition in SPC was not unequivocally demonstrated. A clear demonstration of transfer inhibition in SPC necessitated that Group TI show a lower level of CR performance than Groups C1, C2, LI and N on the retardation-of-learning test with CS1. This outcome was not obtained in the present study. However, Group TI and C1 together did evidence significantly lower CR performance than Groups C2, LI and N.

The question of most interest is why did Group C1 also show greater retardation-of-learning on the test? One possibility is that, during the compound retardation-of-learning test of CS1 and CS2, the inhibition that had accrued to CS2 generalized to CS1 in Group C1. Since both CS1 and CS2 were tested together, it is conceivable that generalization did occur in Group C1 and that this generalization resulted in CR performance in Group C1 that was comparable to that of Group TI. Alternatively, if the action of the inhibitory CS is to lower the activity in the representation of the US (Konorski, 1967; Rescorla, 1979), then, on the retardation-of-learning test the reduction produced by CS2 in both groups C1 and TI might reduce the subsequent effectiveness of CS1-US pairings.

This could result in slower acquisition to CS1. Both of these scenarios suggest that the failure to demonstrate transfer inhibition may have been an artifact of the compound testing protocol used in Experiment 1. Experiment 2 was conducted to investigate this possibility.

Experiment 2

If the simultaneous testing of CS1 and CS2 in the retardation-of-learning test of Experiment 1 affected the evaluation of inhibition to CS1, then it follows that testing only CS1 should reduce the generalization effect. If differences between C1 and TI emerged, then it would allow identification of transfer inhibition in SPC. Thus, the goal of Experiment 2 was to determine if testing only CS1 would result in the identification of transfer inhibition.

Essentially, Experiment 2 is a scaled down replication of Experiment 1. However, there are a number of important deletions from the overall experimental design outlined in Experiment 1. First, three of the control groups from Experiment 1 were not employed in the present study. Specifically, the traditional SPC group (Group E) was not included since the purpose for using this group in Experiment 1 was to verify the effectiveness of the preconditioning CS-CS pairings. Since Group E demonstrated the effectiveness of the preconditioning pairings in Experiment 1 and since the preconditioning phase of Experiment 2 was identical to that in Experiment 1, there was no need to include the traditional SPC

group. In addition, no latent inhibition control (Group LI) was employed in the present study because Experiment 1 demonstrated that latent inhibition was not a factor in evaluating the inhibition that accrued to the to-be-conditioned CS (i. e., Groups C1 and C2 evidenced lower CR performance and Group E higher CR performance than did Group LI). Also, this study did not include the control group from Experiment 1 that received the same treatment as the transfer inhibition group but was trained with an irrelevant CS during inhibitory conditioning (i. e., Group C2). Originally, this group was included to show that the breaking of the associative chain during the conditioning phase would preclude the observation of transfer inhibition thereby indicating that the presence of transfer inhibition in the experimental group must be due to an intact associative chain. Since the performance of this group indicated that the breaking of the associative chain did preclude the observation of transfer inhibition (i. e., Group C2 performed similar to the no treatment group in Experiment 1), it did not appear necessary to again demonstrate this effect in Experiment 2.

There were also two deletions from the experimental protocol used in Experiment 1. The first and most significant is that no retardation-of-learning test was performed on the putative inhibitor CS2. The only reason that CS2 was tested in Experiment 1 was to demonstrate that the unpaired conditioning procedure produced significant inhibition. Since Experiment 2 will make use of the

identical inhibitory conditioning procedure, it will not be necessary to conduct this test again. The second deletion is the preconditioning CS-alone test. Recall, that this test was administered in Experiment 1 to determine the relative levels of CR performance supported by the preconditioning CS following either excitatory or inhibitory SPC. Since it was shown that only the preconditioning CS of the traditional excitatory SPC group supported CRs and since this group was not included in the present replication of Experiment 1, the preconditioned CS-alone test was also deleted.

In Experiment 2, three groups of rabbits were administered a three phase experimental protocol similar to Experiment 1. One of the three groups, the experimental group, received training that was identical to that of Group TI in Experiment 1. Specifically, preconditioning pairings of two CSs were followed first by inhibitory conditioning and then by a retardation-of-learning test to the preconditioning CS only. The second group was a control group that was identical to Group C1 in Experiment 1. This Group received the same treatment as the experimental group with the exception that the to-be-conditioned CS was replaced by a different CS in the preconditioning phase. Finally, the third group was identical to the no treatment control group used in Experiment 1 to establish an associative null point. As in Experiment 1, this group received the retardation-of-learning test only and was instrumental

in establishing the level of transfer inhibition evidenced by the experimental group.

Method

Subjects

Twenty-four naive male and female New Zealand white rabbits (Oryctolagus cuniculus) similar to those described in Experiment 1 served as subjects. In addition, the living quarters and conditions were the same as described above.

Apparatus

The apparatus was the same as described in Experiment 1. In addition, all stimuli and temporal specifications were the same as those described in Experiment 1.

Procedure

Surgical preparation, recovery and group assignment procedures were the same as described in Experiment 1. The criterion for CRs and URs were also the same.

As in Experiment 1, a group designator was assigned to each of the three groups as a quick reference term for the group and it's respective treatment. The group designators in Experiment 2 were: TI-2, C1-2, and N-2. The group designator TI-2 references the transfer inhibition (TI) group for Experiment 2 (-2) while the designator C1-2 references the control group which is similar to the experimental group with the exception that there was an explicit attempt to disrupt the development of an inhibitory association by

using a different to-be-conditioned CS in the preconditioning phase. Finally, N-2 denotes the no treatment control group which participated only in the test phase.

The experimental protocol consisted of the following three phases: a) a preconditioning phase which consisted of 1 daily session of paired presentations of two CSs, b) a conditioning phase consisting of 4 daily sessions of the inhibitory conditioning involving unpairings of the to-be-conditioned CS and the US and, c) a retardation-of-learning test phase where all subjects received one daily sessions of testing. Thus, the duration of this study was 6 days.

After one day of recovery from surgery, the preconditioning phase commenced. In this phase, the subjects in group TI-2, received 10 paired presentations of the 500-ms clicker CS1 and the 500-ms airpuff CS2 with a mean intertrial interval set to 90-s (ie., a randomized sequence of values of 60, 90 or 120-s was used). Group C1-2 received the same preconditioning experience as the aforementioned group with the exception that the 500-ms flashing light CS3 was substituted for CS2. The interstimulus interval for Groups TI-2 and C1-2 was 500-ms. Group N-2 remained in the home cage for the duration of the preconditioning phase.

For the conditioning phase, Groups TI-2 and C1-2 received four daily sessions of unpaired inhibitory conditioning consisting of 30 CS2 and 30 US presentations. In these groups, the CS and the

US were separated by a mean intertrial interval of 45-s (ie., 30, 45 or 60-s were used). Subjects in Group N-2 remained in their home cage for the duration of the conditioning phase.

In the test phase of this study, all groups received a retardation-of-learning test for inhibition. This test for inhibition consisted of one daily sessions of 60 CS1-US pairings. The onset of the 500-ms CS1 preceded US onset by 500-ms. During testing, the mean intertrial interval was 60-s (ie., 45, 60 or 90-s were used). The response measures (i. e., dependent measures) for each phase of Experiment 2 are described in the appropriate section of the results below. A diagrammatic summary of the design and experimental protocol of Experiment 2 is presented in Table 2.

Insert Table 2 about here

Results and Discussion

As with Experiment 1, the results of each phase of Experiment 2 will be discussed in the order in which they were executed. The overall design and statistical procedures are the same as those described in Experiment 1. Like Experiment 1, the present study contains a number of a priori outcomes and each is described in the appropriate section of the results below.

Table 2: Diagrammatic summary of the design and experimental protocol of Experiment 2.

Phase / Group	Preconditioning (ONE DAILY SESSION)	Inhibitory Conditioning (FOUR DAILY SESSION)	Retardation Test CS1 (ONE DAILY SESSION)
TI-2	10 CS1-CS2 pairings	30 CS2/US unpairings	60 CS1-US pairings
C1-2	10 CS1-CS3 pairings	30 CS2/US unpairings	60 CS1-US pairings
N-2	No Treatment (home cage)	No Treatment (home cage)	60 CS1-US pairings

Temporal Parameters:

CS1=500-ms, 80-dB, 10-Hz clicker
CS2=500-ms, 137895.14-N/m2 airpuff
CS3=500-ms, 10-Hz flashing light
US=100-ms, 60-Hz, 3.0-mA shock
All CS-US intervals=500-ms
Mean Intertrial interval=90-s except during testing where it was 60-s

Preconditioning and Conditioning Phases

In the preconditioning phase, rates of NM responding were obtained for Groups TI-2 and C1-2 by measuring the occurrence NM responses during the 500-ms CS1 presentation on each of the 10, CS1-CS2 or 10, CS1-CS3 preconditioning pairings, respectively. No NM response measure was obtained for Group N-2 as this group of subjects remained in their home cages during the preconditioning phase. Because all subjects received pairings of neutral stimuli during this phase, it was expected that NM responding would be very low and similar between Groups TI-2 and C1-2.

The mean percentages of NM responding for Groups TI-2 and C1-2 were 0.0 and 1.3, respectively. The means indicate that NM responding was absent in Group TI-2 and only one subject in Group C1-2 emitted one NM response during the preconditioning phase. As with Experiment 1, statistical analysis was precluded by the virtual absence of variability between Groups TI-2 and C1-2. However, an inspection of the means clearly indicated that NM responding was below the expected baseline rate of 3% and was similar between the groups. Thus, the means confirmed that baseline NM responding was uniformly low in Groups TI-2 and C1-2.

During the 500-ms CS2 presentations of the unpaired conditioning phase, the occurrence of NM responses was assessed for subjects in Groups TI-2 and C1-2. The measures were taken over four daily sessions during which subjects in Groups TI-2 and

C1-2 received 30 unpaired conditioning trials. No measure of NM responding was obtained for subjects in Group N as they remained in their home cages during this phase. Since unpaired conditioning does not result in the development of CRs, it was expected that neither of the two groups would evidence CR acquisition.

The mean percentages of NM responding collapsed across days for Groups TI-2 and C1-2 were 0.3 and 1.9, respectively. The means of 0.3 and 1.9 correspond to a total of 3 and 18 NM responses out of a total of 960 unpaired trials (4 daily sessions x 8 subjects x 30 trials) for Groups TI-2 and C1-2, respectively. As in the preconditioning phase, the near absence of variability during the conditioning phase precluded statistical analysis. An inspection of the means and the corresponding overall level of NM responding clearly indicated that there was no appreciable difference in the low levels of NM responding attained by Groups TI-2 and C1-2. Thus, no appreciable levels of CR performance were observed for Groups TI-2 and C1-2 during the unpaired conditioning phase.

The mean daily percentages of CRs during the four days of unpaired conditioning for Groups TI-2 and C1-2 were 0.0, 0.8, 0.0, 0.4. and 0.0, 0.4, 3.5, 3.8, respectively. The means indicate that Groups TI-2 and C1-2 failed to show any appreciable levels of CR acquisition. While statistical analysis was not applicable, an inspection of the daily means clearly confirm that CR acquisition

was absent in both groups over the course of the conditioning phase.

Retardation-of-Learning Test

The occurrence of NM CRs during the 500-ms CS1 presentations was assessed for subjects in Groups TI-2, C1-2 and N-2 over the six, 10- trial blocks of CS1-US pairings. The a priori hypotheses to be evaluated in this phase was that Group TI-2 should evidence lower CR performance and/or a retardation of CR acquisition onset than the inhibitory control groups, Groups C1-2 and N-2, with the latter two groups performing similarly.

The mean percentages of NM CRs for Groups C1-2, N-2 and TI-2 collapsed across the six, 10-trial blocks of the retardation-of-learning test were 20.2, 14.6 and 3.1, respectively. The means suggest that Group TI-2 showed a lower level of CR performance than Groups C1-2 and N-2 which did not appear to differ. However, this interpretation was not supported by a planned comparison of Group TI-2 with Group C1-2 and N-2, combined [$F(1,21) = 2.62$, $p > .05$]. In addition, a planned comparison of Group C1-2 with Group N-2 did not identify any performance difference [$F(1,21) < 1.0$]. Thus, although Group TI-2 appeared to attain a lower overall level of CR performance than Group C1-2 and N-2 during the test, the analysis indicates that this difference did not reach significance.

Figure 5 depicts the mean percentages of CRs as a function of the 6, 10-trial blocks of the retardation-of-learning test for Groups TI-2, C1-2 and N-2. The figure shows that the onset of CR acquisition was retarded in Group TI-2 relative to Groups C1-2 and N-2, with the former group failing to evidence any CR acquisition prior to the final block of the test. A repeated measures ANOVA applied to the block means yielded a significant Blocks main effect [$F(5,105) = 8.25$, $p < .01$] but not a Group x Blocks interaction [$F(10,105) = 1.43$, $p > .05$]. Orthogonal components for trend applied to the Blocks main effect revealed only a significant linear component [$F(1,105) = 39.41$, $p < .01$]. The presence of a linear Blocks main effect indicates that CR acquisition occurred over the course of the test phase while the absence of a Group x Blocks interaction indicates that CR acquisition occurred in all three groups.

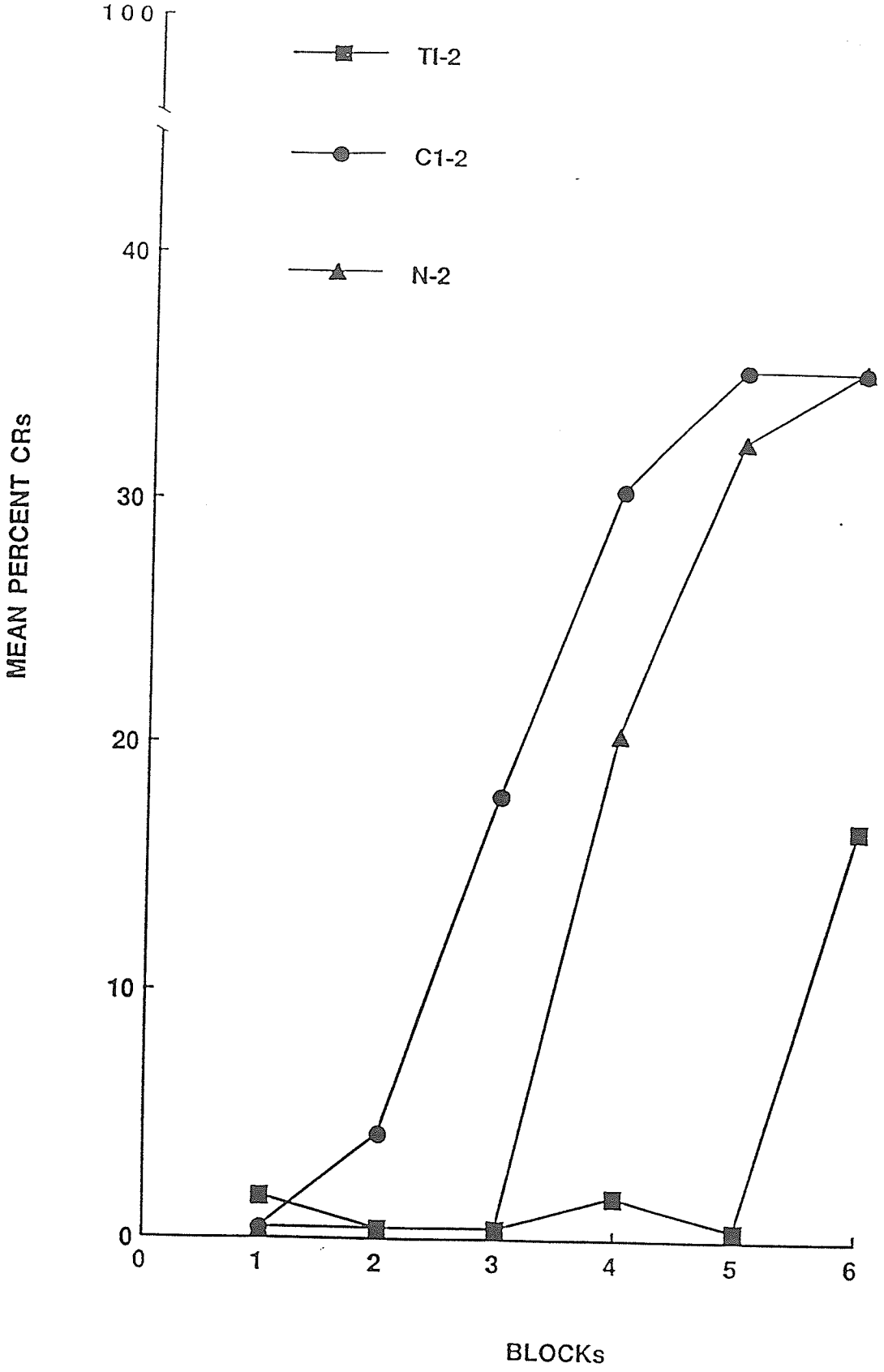
However, because Group TI-2 did not evidence any CR acquisition until the sixth and final block of the test, it was speculated that a repeated measures ANOVA applied to the group means of the first five blocks of test might detect the apparent acquisition difference among the groups. In fact, an ANOVA did yield both a significant Blocks main effect [$F(4,84) = 8.25$, $p < .01$] and Group x Blocks interaction [$F(8,84) = 1.99$, $p = .05$]. Orthogonal components for trend applied to the Block main effect and Group x Block interaction revealed significant linear

components only [$F(1,84) = 24.85$, and $F(2,84) = 6.55$, respectively, both $p's < .01$]. The presence of a linear Blocks main effect indicates that CR acquisition occurred over the course of the first five blocks of the test phase while the linear Group x Blocks interaction indicates that CR acquisition occurred differentially in the three groups.

Insert Figure 5 about here

Orthogonal components for trend applied to the blocks effect of each group failed to reveal linear component for Group TI-2 [$F(1,28) < 1.0$] but did identify a significant linear component in Groups C1-2 and N-2 [$F(1,28) = 13.63$ and $F(1,28) = 7.23$, respectively, both $p's < .01$]. The absence of a linear trend component in Group TI-2 indicates that CR acquisition was absent in this group over the first five blocks of the test while the presence of a linear trend component in Groups C1-2 and N-2 confirms that CR acquisition occurred in these two groups. It is also noteworthy that (a) Group TI-2 contributed only 0.01% of the linear variance to the Group x Days interaction, and (b) Groups C1-2 and N-2 contributed 56.2% and 43.8%, respectively, of the linear variance to the Group x Days interaction. These findings corroborate the observation that Group TI-2 did not evidence CR acquisition during the first five blocks of the test but Groups C1-2 and N-2 did

Figure 5: Mean percentages of NM CRs as a function of the 6, 10-trial blocks of the retardation-of-learning test for Groups TI-2, C1-2 and N-2 in Experiment 2.


100
40
30
20
10
0
0
1
2
3
4
5
6
TI-2
C1-2
N-2
MEAN PERCENT CRs
BLOCKs

evidence essentially equivalent rates. Thus, the statistical analyses confirm the graphical interpretation that (a) the onset of CR acquisition was retarded for Group TI-2 relative to Groups C1-2 and N-2, and (b) Groups C1-2 and N-2 evidenced similar acquisition rates.

To summarize, two major observations are apparent from the findings of the retardation-of-learning test in Experiment 2. First, it was observed that Group TI-2 showed an overall lower CR performance as compared to Groups C1-2 and N-2, combined. However, this difference did not reach statistical significance. Second, it was observed and statistically confirmed that the onset of CR acquisition in Group TI-2 was retarded relative to the control groups, Groups C1-2 and N-2 and that the control groups evidenced a similar rate of CR acquisition. Logically, positive evidence of conditioned inhibition in SPC can take the form of either overall lower levels of CR performance and/or retarded onset of CR acquisition. Since Experiment 2 provides clear evidence that the onset of CR acquisition was retarded in Group TI-2 only, it can be concluded that Experiment 2 has demonstrated conditioned inhibition in SPC. Thus, the combined findings of Experiments 1 and 2 suggest two major conclusions.

First, the findings of Experiments 2 are consistent with the explanation of the observation in Experiment 1 that both Groups TI and C1 evidenced a transfer inhibition-like effect. Recall the

speculation in Experiment 1 that the failure to observe differences between Group TI and C1 on the compound retardation-of-learning test of CS1 and CS2, may have resulted because the inhibition accrued to CS2 may have generalized to CS1 in Group C1. Interestingly, a comparison of the inhibition test performance observed for Group TI-2 and Group TI from Experiment 1 indicates that the performance of Group TI-2 was lower than for Group TI. This suggests the possibility that collateral excitation (cf. Williams & Overmier, 1988) may have generalized to Group TI in Experiment 1 rather than inhibition having generalized to Group C1. In either case, given that this potential generalization effect was eliminated in Experiment 2 by testing the preconditioning CS alone, and that transfer inhibition was observed, it appears that the foregoing explanation for the results of Experiment 1 was supported.

Second and most importantly, Experiments 1 and 2 constitute the first well-controlled demonstration of unpaired transfer inhibition in SPC. More specifically, it was shown that, following unpaired inhibitory conditioning in the conditioning phase of a SPC protocol, conditioned inhibition can be transferred to a CS that had never entered into a direct inhibitory relation with the US.

Experiment 3

The purpose of Experiment 3 was to identify direct inhibition in sensory preconditioning. To achieve this goal, three groups of rabbit subjects received different training protocols. The

experimental group received training that was designed to promote the development of inhibition during the preconditioning phase. More specifically, the experimental group received unpaired presentations of two CSs, CS1 and CS2, followed by CS2-US conditioning which was in turn followed by a retardation-of-learning test for inhibition with CS1. On logical grounds, it seemed possible that if the unpairings in the first phase resulted in an inhibitory association between CS1 and CS2, the latter stimulus of which subsequently became a conditioned excitor, then it might also be the case that CS1 would have a inhibitory relation to the US even though the two were never explicitly unpaired. Of course, the inhibitory relation of CS1 to the US would be expressed as retarded CR performance in the test.

The two remaining groups of subjects served as controls. The first of the two control groups was an inhibitory control group. This group received a treatment similar to the experimental group with the exception that it did not receive preconditioning inhibitory training. Thus, this group was instrumental in determining the impact of inhibitory preconditioning in the experimental group. The second control group received the same treatment as the experimental group except that the to-be-conditioned CS was replaced by a different CS in the preconditioning phase. This treatment was designed to disrupt the associative chain that is presumed to produce the SPC effect. Thus, the second control

group was similar to the C1 and C1-2 control group used in Experiments 1 and 2, respectively.

Based on the findings of Experiments 1 and 2, three control groups that were included in Experiment 1 were not included in the present study. First, since the latent inhibition control group in Experiment 1 (Group LI) did not show any evidence of retarded acquisition following 130 CS2 presentations (i. e., a latent inhibition effect), it seems unlikely that such an effect would be expected to occur in the present study where CS alone presentations were much fewer in number. Accordingly, no latent inhibition control group was included in the present study.

Second, recall that Group C2 from Experiment 1 received treatment similar to the transfer inhibition group (i. e., Group TI) except that it was trained with an irrelevant CS during inhibitory conditioning. This group was originally employed to demonstrate interruption of the transfer of the association to the preconditioning stimulus, thereby underscoring the associative nature of the transfer effect in Group TI. Since the expected interruption effect was clearly demonstrated in Experiment 1, it seemed highly improbable that such an effect would not be observed in the present study (this was the same rationale for not using Group C2 in Experiment 2). In addition, the present study employed a control group, similar to Group C1 in Experiment 1, that would adequately control for the expected associative transfer effect in the

experimental group and which would also serve as a good control for training history because the group would receive a training protocol essentially equivalent to the experimental group. This being the case, it was decided that a Group C2 control was unnecessary for the present study.

Finally, it was decided that the demonstration of yet a second traditional excitatory SPC effect would not serve to further our understanding of the primary process under investigation, namely conditioned inhibition in SPC. In Experiment 1, a traditional excitatory SPC group was used to verify that an association had developed between the preconditioning CSs. Since the general protocol used in the preconditioning phase of the present study is essentially the same as that used in Experiment 1, it is unnecessary to demonstrate again that an association would be formed between the preconditioning stimuli.

Unlike Experiments 1 and 2, the training parameters for Experiment 3 are based on previous research. In particular, the training parameters are based partly on the findings of Experiments 1 and 2 and partly on pilot research from our lab that pertained specifically to direct inhibition in SPC.

Method

Subjects

Twenty-four naive male and female New Zealand white rabbits (Oryctolagus cuniculus) similar to those described in

Experiment 1 and 2 served as subjects. However, three subjects were discarded from the study because they failed to meet a performance criterion imposed during the conditioning phase (the performance criterion is described in the procedure section). Thus, twenty-one subjects participated in the present study. Lastly, the living quarters and maintenance regimes were the same as described in Experiments 1 and 2.

Apparatus

The apparatus was the same as described in Experiment 1. In addition, all stimuli and temporal specifications were the same as described for Experiment 1.

Procedure

Surgical preparation, recovery and group assignment procedures were the same as described in Experiment 1. The criterion for CRs and URs were also the same.

As in Experiments 1 and 2, the author has provided a group acronym or designator was assigned to each group as a quick reference term for the group and it's respective treatment. The group designators in Experiment 2 are: DI, C1-3 and IC. Group designators DI and IC reference the direct inhibition (DI) and the inhibitory control (IC) groups, respectively. C1-3 is the designator for the control group which was similar to the experimental group with the exception that there was an explicit attempt to disrupt the development of an inhibitory association.

The experimental protocol consisted of the following phases: (a) a preconditioning phase that consisted of 2 daily session of unpaired presentations of two CSs, (b) a conditioning phase that consisted of 3 daily sessions of the excitatory training involving pairings of the to-be-conditioned CS and the US and, (c) a retardation-of-learning test phase where all subjects received 1 daily session of inhibition testing. Thus, the duration of Experiment 3 was 6 days.

After one day of recovery from surgery, the preconditioning phase commenced. In this phase, the subjects in groups DI received 20 presentations of the 500-ms, 80-dB, 10-Hz clicker CS1 and the 500-ms, 137895.14/m ^{2} airpuff CS2 in an explicitly unpaired arrangement with a mean intertrial interval set to 90-s (ie., randomized sequence of values of 60, 90 or 120-s was used). Group C1-3 received the same preconditioning experience as Group DI with the exception that the 500-ms, 60-Hz, flashing light CS3 will be substituted for CS2. Subjects in Group IC remained in the home cage for the duration of the preconditioning phase.

In the conditioning phase, groups DI, C1-3 and IC received daily sessions of excitatory conditioning consisting of 50 CS2-US pairings.[1] The US was a 100-ms, 60-Hz, 3.00-mA shock. In all of the above groups, CS2 onset preceded US onset by 500-ms. The mean intertrial interval for all groups was 60-s (ie., a randomized sequence of values of 45, 60 or 90-s was used).

A performance criterion was imposed on all subjects during this phase. The performance criterion was implemented to ensure that uniformly high terminal levels of CR performance were observed for the three groups. This was important for two reasons. First, assuming that second phase conditioning is positively related to the magnitude of the SPC effect, high levels of CR performance during the conditioning phase would increase the likelihood of identifying direct inhibition in SPC. Second, uniform terminal levels of conditioning would ensure that any between group performance differences observed in the test phase could not be attributed to performance differences observed during the conditioning phase, especially on the last daily session of conditioning. Accordingly, all subjects were required to meet a criterion of greater than 80% CRs to CS2 on the third and final day of conditioning. As a consequence of this criterion, one subject from each of the three groups was discarded. Thus, N = 7 for each group.

In the test phase of this study, all groups received a retardation-of-learning test for inhibition. This test for inhibition consisted of 50 CS1-US pairings. The onset of CS1 preceded US onset by 500-ms. During testing, the mean intertrial interval was 60-s (ie., a randomized sequence of values of 45, 60 or 90-s was used). A diagrammatic summary of the design and experimental protocol of Experiment 3 is presented in Table 3.

Insert Table 3 about here

Results and Discussion

As with Experiments 1 and 2, the results of each phase of Experiment 3 will be discussed in the order in which they were executed. The overall design and statistical procedures are the same as those described in the previous experiments. Like Experiments 1 and 2, the present study contains a number of a priori comparisons and each is described in the appropriate section of the results below.

Preconditioning Phase

During the two daily preconditioning sessions, rates of NM responding were obtained for Groups DI and C1-3 by measuring the occurrence of NM responses during the 500-ms CS1 presentations in the preconditioning phase. No NM response measure was obtained for Group IC as this group of subjects remained in their home cages during the preconditioning phase. Because subjects in Groups DI and C1-3 received pairings of neutral stimuli during this phase, it was expected that NM responding would be very low and similar between Groups DI and C1-3.

The mean percentages of NM responding collapsed across the two preconditioning sessions for Groups DI and C1-3 were 0.7 and 0.3, respectively. The mean of Group DI corresponds to two

Table 3: Diagrammatic summary of the design and experimental protocol of Experiment 3.

Phase / Group	Preconditioning (TWO DAILY SESSIONS)	Conditioning (THREE DAILY SESSIONS)	Retardation Test (ONE DAILY SESSIONS)
DI	20 CS1/CS2 unpairings	50 CS2-US pairings	50 CS1-US pairings
C1-3	20 CS1/CS3 unpairings	50 CS2-US pairings	50 CS1-US pairings
IC	No Treatment (home cage)	50 CS2-US pairings	50 CS1-US pairings

Temporal Parameters:

CS1=500-ms, 80-dB, 10-Hz clicker
CS2=500-ms, 137895.14-N/m2 airpuff
CS3=500-ms, 10-Hz flashing light
US=100-ms, 60-Hz, 3.0-mA shock

All CS-US intervals=500-ms
Mean Intertrial interval=
a) 90-s Preconditioning
b) 60-s Conditioning & Retardation Test

subjects in seven emitting one NM response each over the course of the preconditioning phase (i. e., 2 NM responses over the course of 278 scorable CS1 presentations). In Group C1-3, the mean corresponds to one subject in seven emitting one NM response over the course of the preconditioning phase (i. e., 1 NM response over the course of 280 CS1 presentations). The means indicate that NM responding was very low and similar between both groups during the preconditioning phase. As with the previous studies, statistical analysis was precluded by the virtual absence of variability between Groups DI and C1-3. Nonetheless, the near total absence of responding leads to the conclusion that NM responding to CS1 was uniformly low in Groups DI and C1-3 during the preconditioning phase.

Conditioning Phase

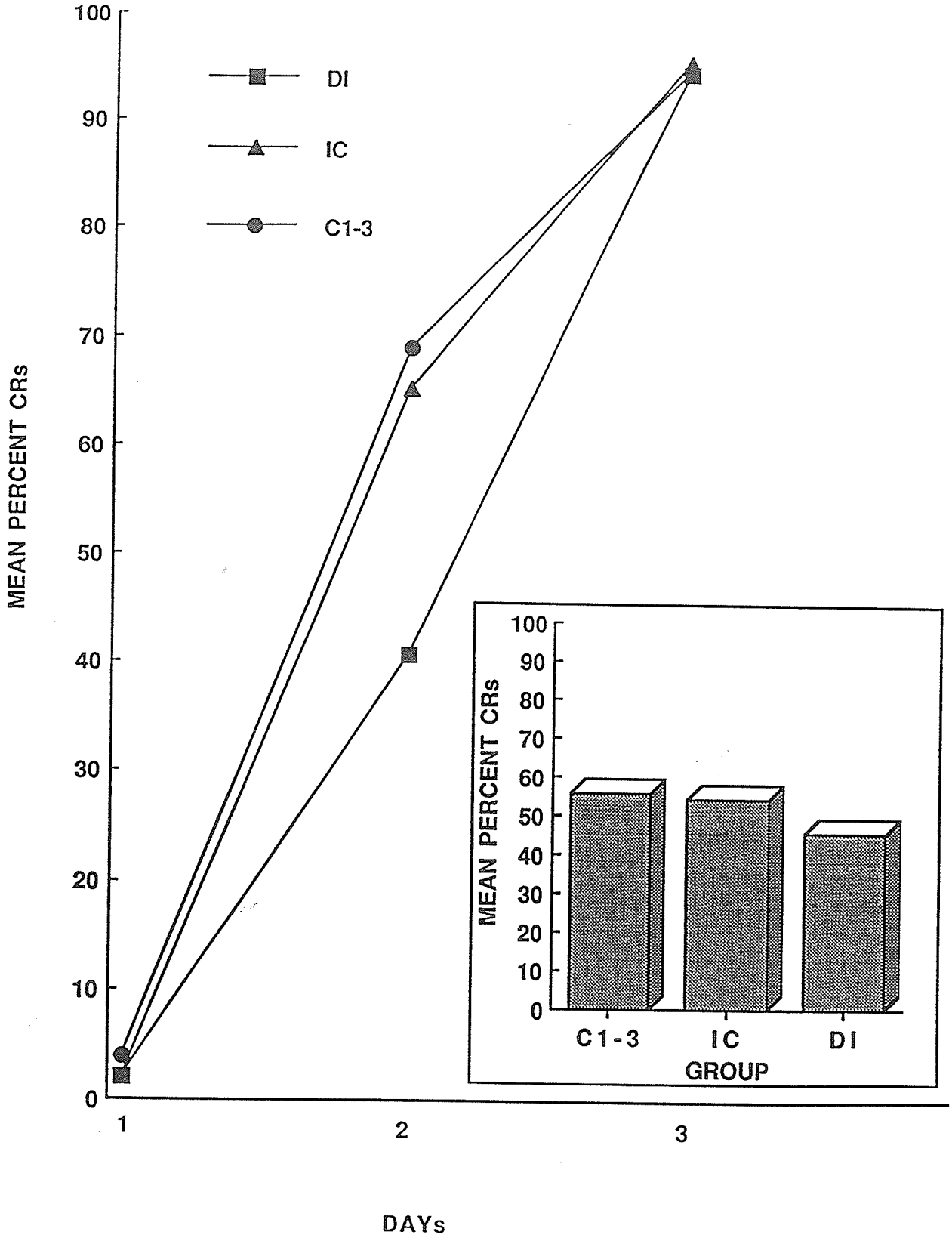
During the 500-ms CS2 presentations of the conditioning phase, the occurrence of NM CRs was assessed for subjects in Groups DI, C1-3 and IC. The measures were taken over three daily sessions during which subjects in Groups DI, C1-3 and IC received 50 paired conditioning trials. Since paired conditioning does result in the development of CRs, it was expected that Groups DI, C1-3 and IC would evidence (a) a similar overall levels of CR performance, (b) a high common terminal level of CR performance on the final day of conditioning, and (c) parallel rates of CR acquisition.

The framed histogram in Figure 6 depicts the mean percentages of CRs collapsed across days for Groups DI, IC and C1-3. The figure indicates that the overall level of CR performance of Groups C1-3 and IC was very similar, with Group DI showing only a slightly lower level than the former two groups. In short, the figure suggests that Groups DI, IC and C1-3 evidenced similar overall levels of CR performance. This graphical interpretation was confirmed by three a priori comparisons in which Group DI was separately compared to Groups C1-3 and IC and the latter two groups were compared [F's(1,18) = 2.08, 1.48 and .05, respectively, all p's > .05]. Thus, the analyses confirm that the overall levels of CR performance attained by Groups DI, C1-3 and IC were similar.

Insert Figure 6 about here

The line plot in Figure 6 depicts the mean percentage of CRs as a function of the three daily sessions of conditioning with CS2 for Groups DI, IC and C1-3. The figure clearly shows that parallel and high rates of CR acquisition occurred Groups DI, IC and C1-3. This observation was confirmed by a repeated measures ANOVA which yielded a significant Days main effect [F(2,36) = 134.36, p < .01] but not a Group x Days interaction [F(4,36) = 1.54, p > .05]. Orthogonal components for trend applied to the Days main

Figure 6: The framed histogram depicts the mean percentages of NM CRs collapsed across the three daily sessions of conditioning for Groups C1-3, IC and DI in Experiment 3. The line plot depicts the mean percentages of NM CRs as a function of the three daily sessions of conditioning with CS2 for Groups DI, IC and C1-3 in Experiment 3.


100
90
80
70
60
50
40
30
20
10
0
MEAN PERCENT CRs
DI
IC
C1-3
1
2
3
DAYs
MEAN PERCENT CRs
C1-3
IC
DI
GROUP

effect revealed a significant linear component only [$F(1,18) = 4015.18$, $p < .01$]. The presence of a linear Days main effect indicates that a high level of CR acquisition was attained over the course of the conditioning phase. The absence of a Group x Days interaction confirms that CR acquisition occurred at the same rate in Groups DI, IC and C1-3.

To further assess the CR performance of Groups DI, IC and C1-3 during the conditioning phase, the daily group means were inspected. The line plot of CR acquisition suggests that the CR performance of Group DI was somewhat lower than in the controls, Group C1-3 and IC, on day 2. However, a series of a posteriori comparisons of Group DI with Groups IC and C1-3, either separately or combined, failed to identify any statistically significant differences among the groups [$F's(1,18) = 1.85$, 2.45 and 2.85, respectively, all $p's > .05$]. Thus, although Group DI showed a somewhat lower level of CR performance than the control groups on the second day of conditioning, this difference was not statistically significant.

On a final note, it is apparent from the acquisition plot in Figure 6 that, despite any modest differences in CR acquisition early in the conditioning phase, the CR performance of the three groups was virtually identical on the final day of conditioning. This graphical interpretation was confirmed by a series of a posteriori comparisons in which Group DI was separately compared with

Groups C1-3 and IC and the latter two groups were compared [all F's(1,18) < 1.0]. Thus, the analyses establish that Groups DI, C1-3, and IC achieved a common terminal level of CR performance.

In sum, the above analyses confirm a number of expectations. First, it was observed that the overall levels of CR performance attained by Groups DI, IC and C1-3 were similar. Second, CR acquisition occurred at the same high rate in Groups DI, IC and C1-3. Third, a follow-up analyses performed on the mean percentages of CRs on day 2 of the conditioning phase failed to identify differences among Groups DI, IC and C1-3 even though Group DI did evidence a somewhat lower level of CR performance than Groups C1-3 and IC on day 2. Fourth and finally, Groups DI, IC and C1-3 evidenced virtually identical high levels of CR performance on the third and final day of the conditioning phase.

Retardation-of-Learning Test

The occurrence of NM CRs during 500-ms CS1 presentations was assessed for subjects in Groups DI, C1-3, and IC over the five, 10-trial blocks of CS1-US pairings. The a priori hypothesis to be evaluated in this phase was that Group DI should evidence lower CR performance and/or a retardation of CR acquisition onset than the inhibitory control groups, Groups C1-3 and IC, with the latter two groups performing similarly.

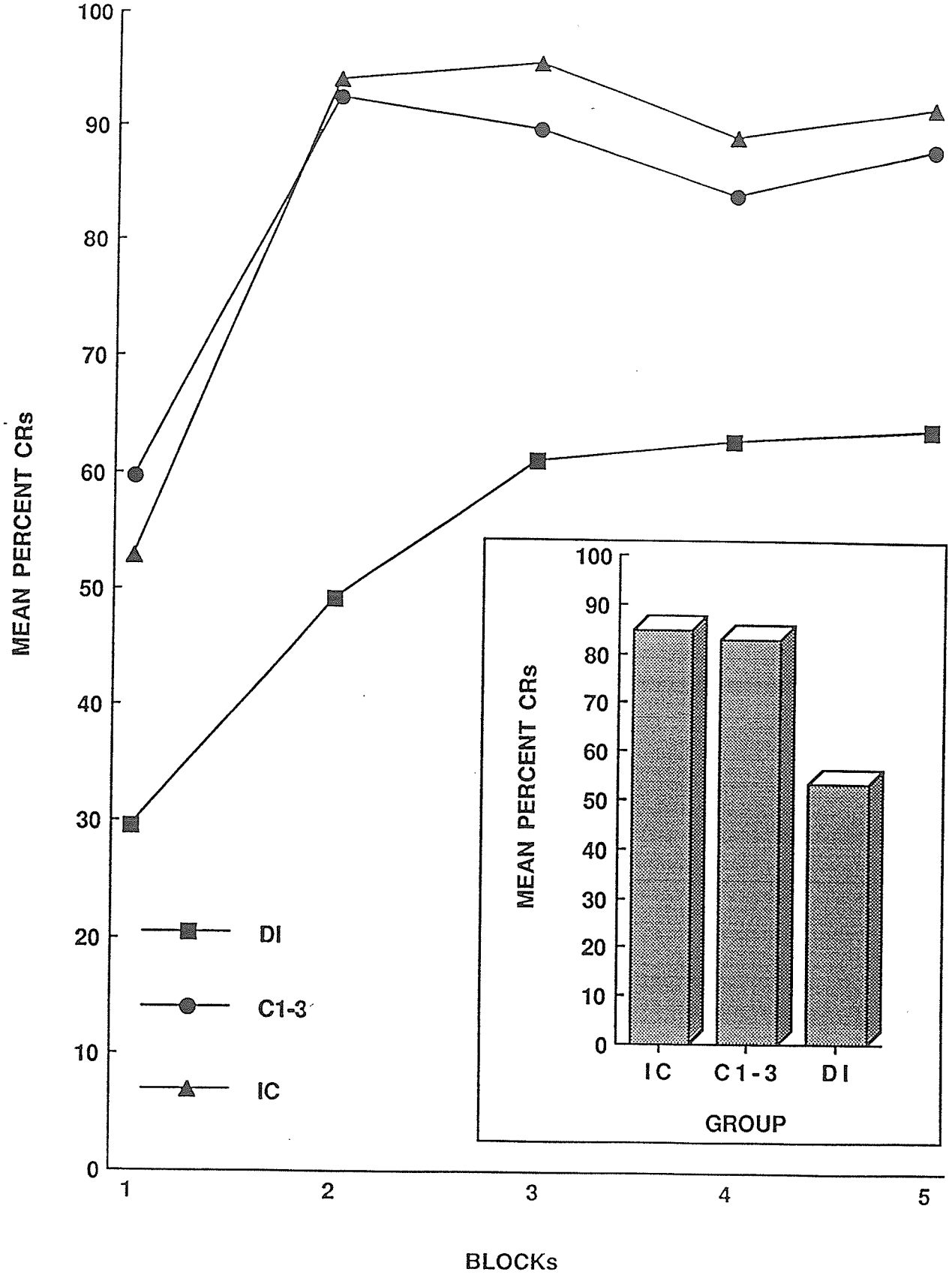
The framed histogram in Figure 7 depicts the mean percentages of CRs for Groups DI, C1-3, and IC collapsed across the

5, 10-trial blocks of the retardation-of-learning test. The figure clearly shows that Group DI evidenced a lower level of CR performance than either Groups C1-3 and IC, which attained similar levels of performance. This graphical interpretation was confirmed by a planned comparison of Group DI with Group C1-2 and N-2, combined [$F(1,18) = 9.63$, $p > .01$]. In addition, a planned comparison of Group C1-3 with Group IC did not identify any performance differences [$F(1,18) < 1.0$]. Thus, the lower overall level of CR performance of Group DI as compared to Group C1-3 and IC, which did not differ, indicates that direct inhibition in SPC was demonstrated.

Insert Figure 7 about here

The line plot in Figure 7 depicts the mean percentage of CRs as a function of the 5, 10-trial blocks of the retardation-of-learning test for Groups DI, C1-3 and IC. The figure shows that CR acquisition in Groups DI, C1-3 and IC shared a similar pattern of acquisition during the retardation-of-learning test. Specifically, the acquisition of all three groups was characterized by an initial rise in CR performance during the first two (Groups C1-3 & IC) or three blocks (Group DI) and this rise was followed by a stabilization of responding during the last three (Groups C1-3 & IC) or two (Group DI) blocks. A repeated measures ANOVA applied to the block

Figure 7: The framed histogram depicts the mean percentages of NM CRs for Groups IC, C1-3, and DI collapsed across the 5, 10-trial blocks of the retardation-of-learning test in Experiment 3. The line plot depicts the mean percentages of NM CRs as a function of the 5, 10-trial blocks of the retardation-of-learning test for Groups DI, C1-3 and IC in Experiment 3.


MEAN PERCENT CRs
100
90
80
70
60
50
40
30
20
10
0
1
2
3
4
5
BLOCKs
DI
C1-3
IC
MEAN PERCENT CRs
100
90
80
70
60
50
40
30
20
10
0
IC
C1-3
DI
GROUP

means yielded a significant Blocks main effect [$F(4,72) = 19.08$, $p < .01$] but not a Group x Blocks interaction [$F(8,72) < 1.0$]. Orthogonal components for trend applied to the Blocks main effect revealed significant linear [$F(1,18) = 27.81$, $p < .01$] and quadratic [$F(1,18) = 21.27$, $p < .01$] components. The presence of linear and quadratic Blocks main effect indicates that an initial rise in CR performance was followed by a stabilization of responding while the absence of a Group x Blocks interaction indicates that this acquisition pattern was common to all three group. Thus, the analyses confirm that Groups DI, C1-3 and IC shared a pattern CR acquisition that was characterized first by an initial rise and then by a stabilization of CR performance.

To summarize, the major observation yielded by the present study was that, during a test for inhibition, Group DI evidenced a lower level of CR performance than the control groups, Groups C1-3 and IC, which did not differ. This observation indicates that unpaired presentations of two neutral stimuli, CS1 and CS2, can result in the identification of associative inhibition accrued to one of the stimuli, CS1, following excitatory training with the other stimulus, CS2. In short, Experiment 3 constitutes a clear demonstration of direct inhibition in SPC.

In addition to yielding the first controlled demonstration of direct inhibition in SPC, the present study also has important implications for accepted control procedures employed in SPC

studies. As noted in the introduction section, one of the most common control groups used in the study of excitatory SPC is a group of subjects that receives the same treatment as the subjects receiving SPC except that the CSs in the first phase are unpaired. The logic of this control procedure is that the unpairings preclude the development of an 'association' by breaking up the associative chaining properties that produce an excitatory SPC effect. Thus, the unpaired control in SPC is presumed to represent an associative null point. However, if first phase unpairings result in the development of an inhibitory association, as suggested by the findings of Experiment 3, then an unpaired group is not an appropriate control for the demonstration of excitatory SPC effect because it would not constitute an associative null point against which an excitatory effect could be assessed.

To minimize interpretive problems in future research on inhibitory or excitatory SPC, I would suggest that the associative chaining effects presumed to underlie the basic phenomena could be controlled for by including control groups similar to Groups C1 and C2 from the present series of studies. Groups C1 and C2 received the same training as the experimental groups except that, in the former case, preconditioning training involved a to-be-conditioned CS that was different from the one actually used in the conditioning phase. For Group C2, conditioning was conducted with a CS that was different than the to-be-conditioned CS used in

the preconditioning phase. Thus, relative to the experimental group, the associative chain is broken either in the preconditioning or conditioning phase for Group C1 and C2, respectively.

It is noteworthy that, in conditioning preparations that are prone to the protection of habituation effect (Pfautz et al, 1978), the control protocol suggested above would also provide an excellent means of determining the relative contribution of protection of habituation and SPC observed in an experimental group. Recall that the protection from habituation effect occurs when habituation is prevented from occurring to the temporally first of two sequentially presented CSs as a result of it being followed by the second stimulus. In CER studies of a excitatory SPC, a control group like Group C1 or C2 could be instrumental in determining the contribution of protection from habituation because both of these groups would have the same experience with the preconditioning CS as the experimental group. However, since associative effects are not expected in these control groups (the chain is broken in both groups), it follows that any responding during a preconditioning CS-alone test in these groups could be attributable to a protection from habituation effect and that any difference in responding between the controls and the experimental group is likely due only to an associative SPC effect.

General Discussion

The general discussion is divided into five major sections. The first section is a summary and interpretation of the major findings of the present studies. Wherever appropriate, convergence between the results of the present studies and the extant SPC and Pavlovian conditioning literature will be identified. Second, the implications of the present results for a nonassociative account of SPC is considered. In the third section, the four associative theories of inhibition outlined in the introduction are discussed with respect to the present findings. Penultimately, four associative learning theories, each representing a distinct general class of associative learning theories, are examined in light of the present findings. In the fifth and final section, the future direction of research on conditioned inhibition in SPC is considered.

Summary and Interpretation of Present Studies

In Experiments 1 and 2, two significant observations were obtained. First, it was demonstrated that the unpaired presentations of a CS (to-be-conditioned CS) and a US resulted in retarded CR acquisition to that CS when it was later paired with the US in a retardation-of-learning test for inhibition. Retarded CR acquisition to the CS was taken as evidence conditioned inhibition. This replicates previous research which has demonstrated conditioned inhibition following unpaired training (cf. LoLordo & Fairless, 1985). Second and most importantly, it was shown that

preconditioning CS-US conditioning was retarded if the preconditioning CS had been previously paired with a to-be-conditioned CS that was later made into a conditioned inhibitor (i.e., via unpairings). This observation indicates that conditioned inhibition had transferred to the preconditioning CS as a consequence of an association it formed with the to-be-conditioned CS that was later made into a conditioned inhibitor. Thus, Experiments 1 and 2 provide the first controlled demonstration of transfer inhibition in SPC. Transfer inhibition in SPC is the procedural and functional opposite of traditional or excitatory SPC in which excitatory rather than inhibitory conditioning is performed with the to-be-conditioned CS.

Experiment 3 yielded one observation of major importance. In particular, it was found that unpaired presentations of a preconditioning CS and a to-be-conditioned CS prior to excitatory conditioning of the to-be-conditioned CS, resulted in retarded CR acquisition to the preconditioning CS in a retardation-of-learning test for inhibition. Since retarded CR acquisition in a retardation-of-learning test is evidence of conditioned inhibition, Experiment 3 represents the first controlled demonstration of direct inhibition in SPC. What is most notable about this latter observation is that it strongly suggests that an inhibitory association can be established between two CSs prior to any conditioning manipulation involving a US. Furthermore, it is possible that the inhibitory association

between the two CSs had bidirectional properties (i. e., each CS may have had an inhibitory connection to the other).

The identification of transfer inhibition in Experiments 1 & 2 is consistent with a previous study by Rescorla (1984) which provided some findings that were suggestive of transfer inhibition in SPC. Similarly, the observation of direct inhibition in SPC in Experiment 3 is consistent with a previous study by Tait et al. (1972) which was suggestive of direct inhibition in SPC.

A recent review of the SPC literature following the completion of the present studies revealed a study by Martinez, Aguado and Rosales (1989/1991) which reports findings consistent with Experiment 3 reported here. Because the article was published in an inaccessible Spanish psychology journal (Revista de Psicologia General y Aplicada), the specifics of this study were not available. However, the English abstract of the report suggested that direct inhibition in SPC may have been demonstrated with rats in a CER paradigm. Specifically, following unpaired presentations of the preconditioning CS with a to-be-conditioned CS and excitatory conditioning of the to-be conditioned CS, retarded conditioning of a preconditioning CS was observed in a retardation-of-learning test for inhibition. While the reported results are consistent with an observation of direct inhibition in SPC, the absence of any specifics regarding control procedures must necessarily make any conclusions about outcome tentative. For example, it is unknown whether or

not the study contained controls that would allow the authors to attribute performance deficits in the retardation-of-learning test to associative chaining effects that are presumed to underlie SPC (i. e., a control similar to Group C1 in the present studies would be needed). However, if the reported results are the product of a well-controlled research design, then the fact that parallel findings can be generated in two different laboratories using two different conditioning preparations with two different species speaks well of the robustness of direct inhibition in SPC.

In my introductory remarks, the position was adopted that there are general rules governing the development of associative excitation and inhibition (cf. Weisman, 1977). It was further stated that SPC and Pavlovian conditioning are two unique and independent methods that researchers have employed to elucidate general associative rules. Given that the laws of association are indeed general, it would be logical to conclude that the rules of association should manifest themselves in a similar manner regardless of the whether one uses Pavlovian conditioning or SPC to identify them. In fact, Appendix A provides considerable evidence that associative rules governing the development of conditioned excitation do manifest themselves in highly similar manner in Pavlovian conditioning and SPC. However, there is no such parallel set of observations regarding the generality of rules governing the development of conditioned inhibition. Remarkably, conditioned

inhibition has only been studied using Pavlovian conditioning. The results of the present series of investigations provide clear demonstrations of conditioned inhibition in SPC thereby filling the gap in the SPC literature. The most important implication of the present observations of conditioned inhibition in SPC is that they lend additional credibility to the notion that there are general learning processes that give rise to general associative rules. While the issue of whether or not there are general associative rules can hardly be decided by one series of studies, the identification of conditioned inhibition in SPC fills a gap that, until now, left a considerable hole in the argument favoring the existence of general associative learning processes.

Implications of the Present Results for a Nonassociative Account of SPC

The findings of the present studies have some clear implications for a recent theoretical account of SPC (Honey and Hall, 1991) which relies on nonassociative mechanisms. Honey and Hall (1991) present a 'perceptual' account of SPC, based on an earlier and similar statement by Rescorla (1980), which they claim is superior to an associative explanation (as outlined in the introduction). Briefly, the perceptual explanation of excitatory SPC (Honey and Hall, 1991; Rescorla, 1980) asserts that preconditioning exposure to a simultaneous compound of CS1 (preconditioning stimulus) and CS2 (to-be-conditioned CS) is

presumed to result in subjects forming a "unified representation" of the compound that is distinct from but similar to either of the elements, CS1 and CS2. When CS2 acquires associative strength during CS2-US conditioning, the associative strength of CS2 generalizes to the unified representation of the compound CS1+CS2 because the representation of the compound stimulus contains a CS2 element. Furthermore, because the unified representation of the stimulus compound CS1+CS2 contains a CS1 element, it is asserted that the associative strength of the stimulus compound CS1+CS2 generalizes to the CS1. When CS1 is presented alone in the test, the foregoing sequence of generalization of associative strength allows for the occurrence of a response to CS1.

There are three major problems with attempting to apply the perceptual account to the results of the present studies. The first is that a "unified representation" of two stimuli is much less likely to develop under conditions of stimulus asynchrony (Rescorla, 1980). In Experiments 1 and 2, CS1 and CS2 were sequentially rather than simultaneously presented and as a result, subjects are much less likely to form unified representation of the CS1 and CS2. If a unified representation of CS1 and CS2 did not form during preconditioning, then there would be no way for the perceptual account to explain the transfer of inhibition in Experiments 1 and 2. Even if one were to assume that a CS1+CS2 representation did

form in Experiments 1 and 2, it would be extremely unlikely that a similar representation was formed in Experiment 3 because CS1 and CS2 were explicitly unpaired in this experiment. If subjects did not form the unified representation in Experiment 3, then the perceptual account could not provide mediational mechanism to explain the observed inhibitory effect.

The second major problem with the perceptual analysis is that even if one could conclude that a unified representation could result from the CS1 and CS2 unpairings in Experiment 3 (and it's doubtful), then the "unified representation" would have to have a valence or sign to explain the inhibitory outcome. Yet a "unified representation" is, by definition, unitary and there is no reason for it to have a sign because a sign implies a qualitative relationship between one or more units of something, as in the case of associations between two or more stimulus elements. Since the results of Experiment 3 strongly suggest that inhibition (i. e., a negative sign) developed in the preconditioning phase where the "unified representation" would also presumably originate, it would appear that the perceptual analysis is inapplicable.

And finally, the perceptual account assumes that "unified representation" are vehicles through which associative strength generalizes and are not themselves determinants of the sign of the associative strength (i. e., excitatory versus inhibitory). This being the case, the perceptual account can not anticipate an inhibitory

outcome in Experiment 3 since only excitatory conditioning was performed with CS2. Consequently, only excitatory associative strength could generalize first to the representation and then to the CS1, accepting the unlikely possibility that a subjects could form a unified representation of two unpaired CSs. With only excitatory associative strength available for generalization, the perceptual account can not explain the inhibitory effect observed in Experiment 3.

Thus, the perceptual account of traditional or excitatory SPC effects offered by Rescorla (1980) and Honey and Hall (1991) is not viable as an explanation of conditioned inhibition in SPC. In fact, the traditional notion of associations between separate stimulus units is still the most reasonable explanation of conditioned inhibition in SPC. This being the case, it now seems reasonable to examine the implications of the present studies first for associative model of inhibition and second, for associative learning theories in general.

Implications of Present Results for Associative Theories of Inhibition

In the latest review of Pavlovian conditioned inhibition, LoLordo and Fairless (1985) examined the results of a wide range of inhibitory training procedures in an attempt to delineate the determining factors that yield conditioned inhibition. Their assessment indicated that concurrent conditioned excitation was necessary and sufficient for the development of conditioned

inhibition. LoLordo and Fairless noted that it was not possible to exclude the presence of concurrent excitation, either in the form of discrete cues or excitatory background/contextual cues during inhibitory training. Put another way, they found no conclusive evidence that conditioned inhibition could be produced in the absence of some source of conditioned excitation.

Contrary to the conclusions of LoLordo & Fairless (1985), the results of Experiment 3 strongly suggest conditioned inhibition can be acquired using an unpaired inhibitory training protocol which precluded a concurrent source of conditioned excitation. In Experiment 3, the inhibitory training consisted of the unpaired presentations of two CSs in the preconditioning phase. Because the subjects did not experience the US during the preconditioning phase and because US occurrence is necessary for the development of conditioned excitation, conditioned excitation could not be present at the time of the unpairing procedure. That conditioned inhibition was subsequently identified to have resulted from the unpairings suggests that the relation between the stimuli, rather than the presence of concurrent excitation, was the operative process. While the generality of the effect has yet to be determined, Experiment 3 provides the first evidence that conditioned inhibition can develop in the absence of a concurrent source of conditioned excitation.

So what implication does this have for the four associative theories of conditioned inhibition outlined in the introduction? To address this point, let us consider each of the positions in turn.

Fowler et al, (1985), conceptualize conditioned inhibition as a slave process of associative excitation. The position asserts that associative inhibition is functionally dependent on some form of conditioned excitation. This conclusion was derived from extensive research on conditioned inhibition (via the Pavlovian procedure) conducted in Fowler et al's laboratory and which yielded the following observations. First, it was found that conditioned inhibition was deactivated to a putative inhibitor, say S1, if a discrete cue excitor, say S2, that was concurrently trained with S1 was extinguished. Second, conditioned inhibition was maintained to S1 if S2 was extinguished as long as some attempt was made to develop some alternative source of conditioned excitation, for example, by giving US presentations in a different context and then test for inhibition in that alternate context. Third and finally, conditioned inhibition was reactivated to S1 following extinction of S2 if either S2 or some other discrete cue was paired with the US prior to testing for inhibition with S1. Taken together, the studies of Fowler et al suggest that conditioned inhibition was maintained to the extent that some source of concurrent conditioned excitation was also maintained. From this, Fowler et al concluded that conditioned inhibition is a slave to conditioned excitation.

Fowler et al's position has difficulty with the present results. Given Fowler's assumption that the US is the source of all conditioned excitation and noting that the US was absent during the unpairings of the two CSs in the preconditioning phase, Fowler et al., (1985) would not expect conditioned inhibition to develop in Experiment 3. Since conditioned inhibition was observed, Fowler et al's., (1985) view of conditioned inhibition as a slave process to conditioned excitation remains, at best, an incomplete account of the dynamics of conditioned inhibition.

Second, the perspective on conditioned inhibition offered by Miller and Schachtman (1985) maintains that associative inhibition does not exist as an independent associative entity. They view the effects noted with conditioned inhibition procedures as performance phenomena that result from the comparison of two independent sources of conditioned excitation (i. e., a discrete CS and a comparator stimulus, such as the context). Specifically, a CS will acquire an inhibitory response potential under conditions where the probability of the US occurrence is greater in the absence of the CS than in it's presence. Thus, conditioned inhibition exists only as a performance by-product of associative excitation. Because contrasting sources of conditioned excitation accrue only through the action of a US, conditioned inhibition can not result in the absence of the US. Such a view of inhibition is not consonant with the findings of Experiment 3 because USs were not present during

the inhibitory unpaired training conducted in the preconditioning phase and inhibition was obtained. Since the conditioned inhibition noted in Experiment 3 can not be a performance artifact, the clear implication is that associative inhibition is a real phenomenon.

The theory of Rescorla and Wagner (1972; Wagner & Rescorla, 1972) provides a third perspective on conditioned inhibition. Briefly, they assume that (a) associative strength falls along a dimension with positive (excitatory) and negative (inhibitory) values symmetrically ranged about the null point, (b) a unitary mechanism or rule determines associative change, (c) associative inhibition results when the associative potential provided by the US is less than the combined associative strength of all stimuli occurring on a trial (this is a more general statement of the LoLordo and Fairless (1985) position that conditioned inhibition results when the CS is nonreinforced in the presence of a conditioned excitor), and (d) CR performance is a linear transform of associative strength. This latter assumption provides the basis for inferring associative inhibition from conditioned inhibition. The inhibition assumption (c above) makes the Rescorla and Wagner perspective vulnerable to the same criticism leveled against the two foregoing models. Since the inhibitory training in the preconditioning phase of Experiment 3 did not involve a US, there is no possibility that the putative inhibitor occurred in compound with a conditioned excitor. Thus, the Rescorla & Wagner (1972) perspective on conditioned inhibition

is not congruent with the finding that conditioned inhibition can develop in the absence of conditioned excitation.

Lastly, Konorski (1967) viewed conditioned inhibition and conditioned excitation as essentially equivalent processes in that they both entailed connections between the gnostic units of stimulus events. He also promoted the idea that the associative structure of conditioned inhibition is independent of that of conditioned excitation by asserting that the connections formed during the two types of conditioning involve different types of gnostic units. In classical conditioning, pairing a CS with a US results in a connection between the CS and US gnostic units, whereas conditioning with an inhibitory configuration of the CS and US develops connections between CS gnostic units and "no-US" gnostic units. Konorski's (1967) view has two unique features. First, excitatory and inhibitory connections are conceptually independent. That is, a CS could have both types of connections at the same time as demonstrated by Tait and Saladin (1986). Secondly, the connections are between representations of the stimuli (i. e., gnostic units). Consequently, Konorski's analysis could be applied to the representation of the CSs that occur during the preconditioning phase. Accordingly, in the preconditioning phase of Experiment 3, Konorski would assume that the gnostic units of the preconditioning CS1 would form an inhibitory connection with the "no-CS2" gnostic units of the to-be-conditioned CS2 and vice versa.

Thus, Konorski's (1967) perspective on conditioned inhibition is consonant with the observation that inhibition can develop without concurrent excitation.

To summarize, it has been shown that the results of Experiment 3 question the commonly held assumption that concurrent conditioned excitation is necessary for the development of conditioned inhibition (cf., LoLordo & Fairless, 1985). The results of Experiment 3 also call into question the generality of theories of associative inhibition that endorse only variations of the above assumption. The explanatory power of any theory of inhibition is limited if it views conditioned inhibition (a) as a slave to conditioned excitation (Fowler, et al, 1985), (b) as a behavioral by-product of conditioned excitation (e. g. Miller & Schachtman, 1985), or (c) as resulting exclusively from a stimulus being nonreinforced in the presence of a conditioned excitor (Rescorla & Wagner, 1972). Finally, it was shown that Konorski's (1967) perspective was consonant with the observation that conditioned inhibition can develop without conditioned excitation. The greater explanatory power of Konorski's view is derived from its emphasis on the independence of conditioned inhibition and conditioned excitation, a property that allows motivationally inert stimuli to form an inhibitory association with each other.

Implications of Present Results for Associative Learning Theories

One of the most important functions of any theory of associative learning is that it should be able to accommodate new findings that relate to associative learning. Since the present series of studies have provided the first demonstration of transfer and direct inhibition in SPC, it seems fitting to consider whether or not contemporary learning theories can account for them. In particular, the present discussion will pose two questions to theories of associative learning. The first is- can the theories account for the traditional SPC effect? and the second is- can the theories explain the observations of conditioned inhibition in SPC? These two questions will be posed to four general classes of conditioning theories. The four classes of theories are (a) models based on the Rescorla-Wagner (1972) associative axiom (b) comparator models, (c) attentional models, and (d) opponent-process models. A discussion of every conditioning model within each of the four classes would not only be astonishingly overlong and complex but would also prove to be quite redundant as many of the models share core explanatory assumptions. This being the case, one model was selected as a representative of it's class. The rationale behind each selection is provided in each of four subsections below.

Models Based on the Rescorla-Wagner (1972) Associative Axiom

The Rescorla & Wagner model (1972) of Pavlovian conditioning has been the most influential learning theory over the

past 19 years. The greatest virtue of the model is the mathematical precision with which it has been formulated, a precision which has allowed for the articulation of many specific predictions. This theory has not only been fruitful in generating new research to test its many predictions, it has also given rise to several conditioning theories that represent extensions or modifications of its core assumptions (Frey & Sears, 1978; Mackintosh, 1975; Pearce & Hall, 1980;- with the latter two theories emphasize attentional factors related to CS processing). These theories, despite their important differences, comprise a class theories that share the basic mathematical structure of the Rescorla-Wagner model. This mathematical structure can be concisely referred to as the Rescorla-Wagner associative axiom.

The Rescorla & Wagner axiom (1972) asserts that $\Delta V_n = \alpha \beta (\lambda - V_{n-1})$ where the change in associative strength on trial N (ΔV_n) is equal to the product of the salience of the CS and the US (α and β, respectively where $0 \leq \alpha, \beta \leq 1$) multiplied by the discrepancy between the maximum amount of conditioning that a specific US will support (λ where $\lambda \geq 0$) and the sum of the associative strength that has accrued to the CS over N-1 trials (V_{n-1}). As conditioning proceeds, the value of V will grow progressively larger resulting in the gradual reduction in the level of associative strength (ΔV) acquired on each successive trial. Eventually, V will equal λ and therefore, there will be no gains in associative strength

($\Delta V = 0$). At this point in conditioning, learning is complete and CR performance is stable and asymptotic. Although it is not immediately apparent, this mathematical model predicts the outcomes of experiments which employ multiple CSs. The effectiveness of the model with respect to multiple CS processing, develops out of the basic assumption that CSs compete for the associative strength that the US will support (ie., $V = V_{cs1} + V_{cs2} + ... V_{csn}$). The outcome of the competition will depend on the relative salience and/or the conditioning history of the CSs.

Despite its highly influential status, the Rescorla-Wagner (1972) model encounters considerable difficulty in attempting to explain traditional excitatory SPC or conditioned inhibition in SPC as observed in the present studies. Technically, the Rescorla-Wagner (1972) model is not congruent with the commonly held assumption that SPC involves the development of associations between two CSs. To illustrate, take the case of excitatory SPC or transfer inhibition in SPC. It is unlikely to be a contestable point that the first phase pairing of two CSs in both cases results in a change in the associative status of CS1. Interestingly, the Rescorla & Wagner model has no mechanism to express the development of associative strength to CS1 because changes in the associative status of a CS require that λ be some value greater than 0. Given that λ is a US parameter and that no US is presented during preconditioning, the model does not allow that CS1-CS2 pairings to produce an increase

in associative strength to CS1. Thus, contrary to the commonly held notion that SPC results in the development of associations between two CSs, the Rescorla-Wagner associative axiom, $\Delta V = \alpha \beta (\lambda - V)$, does not permit learning (i. e., increases in associative strength) to occur during the preconditioning phase of SPC.

The inability of the Rescorla-Wagner model to accommodate the development of associations between two CSs also prevents the model from accounting for excitatory or transfer inhibition effects. If no association can be formed during preconditioning, then the only effect that the preconditioning CS1-CS2 pairings could have is to minimally reduce the salience of the two CSs (i. e., decrement in α). Since there is no possibility of any associative link between the first and second phase of SPC training, the model would clearly expect that second phase training would not impact on the associative status of CS1. Therefore, regardless of the type of second phase conditioning given, the model would expect a slight retardation effect, due to reduced CS1 salience, in retardation-of-learning test. However, Experiment 1 and 2 showed very clearly that excitatory training in the second phase of SPC results in a large savings effect during a retardation-of-learning test while inhibitory training results in a significant retardation effect. Thus, the expectations of the Rescorla-Wagner model are clearly at

odds with regard to observed excitatory and inhibitory transfer effects in SPC.

The model does not fair much better in attempting to explain the observation of direct inhibition in SPC. Again the theory does not allow for the establishment of an association during the preconditioning CS1/CS2 unpairings and again it would expect that the only effect of the unpairings would be a slight reduction in CS salience. Since, the second phase CS2-US conditioning would be expected to have no effect on the associative status of CS1, the model would further expect only a mild retardation effect during the retardation-of-learning test. This expectation is consistent with the observed performance of Group DI in Experiment 3. However, the model would make an identical prediction for the control Group C1-3 because this group had the same experience with CS1 as did Group DI. Consequently, the observed absence of retardation in Group C1-3 is inconsistent with the model's prediction. Overall, the appeal of the Rescorla-Wagner model to nonassociative factors (i. e., CS salience reduction) is insufficient to accommodate the excitatory or inhibitory SPC effects observed in the present studies.

Comparator Models

The comparator model of performance offered by Miller and his associates (Miller & Schachtman, 1985; Miller & Matzel, 1988) is a probability-based response rule for the expression of Pavlovian conditioned associations. The model addresses issues of

association by operationally defining associations in terms of two independent probabilities. One of the probabilities pertains to the likelihood of occurrence of reinforcement in the presence of a CS while the other pertains to the likelihood of reinforcement in the presence of stimuli other than the target CS (i. e., the comparator stimuli). Comparisons involving these probabilities are presumed to relate directly to response generation. Variations of these notions can be found in other comparator models such as scalar expectancy theory (Gibbon, 1981, Gibbon & Balsam, 1981) and the relative waiting time hypothesis (Jenkins, Barnes, and Barrera, 1981). However, the present discussion will focus on comparator theory as representative of this general class of theories because it is the one that has been most broadly applied as a model for Pavlovian conditioning. Outlined below is a more detailed description of the comparator model which will be followed by an examination of how the model addresses the two questions of interest.

Borrowing from Rescorla's (1968) early contingency analysis, the comparator hypothesis assumes that subjects extract from their experiences in Pavlovian conditioning experiments, two important probabilities about the occurrence of the CS and US. The first of these is the probability of the occurrence of the US given the presence of the CS P(US|CS) and the second is the probability of the occurrence of the US given the absence of the CS (i. e., P(US|noCS). The former of these two probabilities corresponds to

the associative strength of the CS while the latter reflects the associative strength of the background cues and/or any other punctate stimuli present at the time of the training of the CS. Consequently, both probabilities can assume positive values between 0 and 1 as a result of the training protocol. In contrast to Rescorla's model, the comparator model asserts that these two probabilities represent two independently formed associations, the comparison of which determines the subjects response to the CS. Specifically, the model asserts that the response potential of a target CS is determined by a comparison between the (a) associative strength of the target CS, and (b) the associative strength that the relevant comparator baseline (i. e., background cues or discrete CS) has at the time of testing rather than at the time of training (cf. Miller & Schachtman, 1985; Miller & Matzel, 1988).

The comparator model has an elegantly simple rule for determining when a CS will be either excitatory or inhibitory. In particular, the model stipulates that conditioned excitation develops to a CS when P(US|CS) > P(US|noCS) whereas conditioned inhibition results when P(US|CS) < P(US|noCS). What is most notable about this position is that both conditioned excitation and inhibition are assumed to result from the comparison of two associations that can assume only positive values (i. e., probabilities). Thus, conditioned inhibition is presumed to be the

behavioral consequence of the positive association between the CS and US being weaker than the positive association between the background cues and the US. However, there is no underlying negative or inhibitory association. Having outlined the essential features of the comparator hypothesis, it is now possible to consider the model in light of the results of the present studies.

Like the Rescorla-Wagner model above, the comparator hypothesis has very little to say about the development of associations between two CSs. It is clear from the probability aspects of the model (i. e., P(US|CS) & P(US|noCS)) that the only types of associations that develop are those involving some form of relationship between a CS and US. Nonetheless, if the model is applied in the strictest sense, then it would assume that preconditioning pairings of two CSs would not result in a change in their neutral associative status (i. e., P(US|CS1) = P(US|CS2) = P(US|background cues) = 0). However, the model does permit that the associability of the CSs will diminish during preconditioning pairings (Matzel & Miller, 1988). Depending on whether one does excitatory or inhibitory conditioning during the second phase, the comparator hypothesis makes two quite different predictions about the outcome of the retardation-of learning test. Each of these two scenarios is considered below.

According to the model, when the to-be-conditioned CS (CS2) is paired with the US in phase 2, the conditions exist for the

posttraining inflation of a potential comparator (in this case CS2) for the preconditioning CS (CS1). The model would suggest that the CS2 rather than the background cues would acquire most of the associative strength. At the beginning of a CS1-alone or retardation-of-learning test (i. e., savings test) with the CS1, the relevant comparator, the background cues, would be expected to be approximately associatively neutral (with the exception of a somewhat reduced associability as a result of prior nonreinforced presentations). This being the case, the comparator model would assert that (a) CR performance on CS1-alone test trial would be near zero, and (b) conditioning to CS1 during the savings test should proceed at a rate comparable to subjects who have no prior training history (i. e, naive subjects). However, these expectations were not confirmed either in Experiment 1 or in numerous other studies which have demonstrated excitatory SPC effects (i. e., appreciable levels of CRs on CS1-alone test trials and substantial savings in a savings test). Thus, excitatory SPC effects can not be accommodated by the comparator model.

The comparator hypothesis also fails to explain the transfer of inhibition in SPC. Again, unpaired inhibitory conditioning following CS1-CS2 pairings would be viewed as posttraining inflation of the background cues which are the comparator stimuli (i. e., P[US|background] is increased). At the commencement of the retardation-of-learning test, the comparator hypothesis would

expect that because CS1 is associatively neutral it would evidence retarded acquisition due to the excitatory background cues that serve as a comparator (i. e., P(US|CS1) < P(US|background)). Accordingly, the comparator hypothesis would correctly expect that CS1 conditioning be retarded. However, according to the model a similar level of retardation in the control Group C1 would occur because it received the same experience with CS1 in the preconditioning phase and with the US in the conditioning phase. While this expectation was true for the findings of Experiment 1, the comparator model can't explain why the simple change of testing protocols in Experiment 2 would eliminate the retardation effect observed for Group C1 in Experiment 1. The reason the comparator model can not explain this differential outcome is that there is no theoretical reason why a change in the testing protocol between the Experiments 1 and 2 should effect the overall associative value of the comparator (i. e., background cues) established during unpaired conditioning. Accordingly, if the background cues retain their excitatory associative status regardless of the differential training protocol, then the comparator hypothesis would expect that the C1 control groups in both Experiments 1 and 2 should have evidenced retardation. The failure of this expectation to be confirmed in Experiment 1 and 2 highlights the inability of the comparator hypothesis to provide a complete account of transfer inhibition in SPC.

The comparator model is also conceptually unable to accommodate the observation of direct inhibition in SPC. This follows because the comparator model does not allow for the development of an inhibitory response potential when two CSs are unpaired (i. e., P(US|CS1) = P(US|CS2) = P(US|background cues) = 0). After the posttraining inflation to CS2 that occurs in the second phase (i. e., [P(US|CS2) > 0] but not to the background cues [P(US|background cues) ≈ 0]), the comparator hypothesis would contend that, at the beginning of the retardation-of-learning test, the background cues (i. e., comparator) would be as associatively neutral as CS1. Given the model's expectation of associative equivalence between the CS1 and the comparator background cues, it would further expect that CR performance should not be retarded during the test. Yet, this expectation lies in direct opposition to the findings of Experiment 3. Thus, the comparator hypothesis is unable to account for the excitatory and inhibitory SPC effects because it fails to adequately characterize CS-CS associations and discrete cue/comparator associative dynamics.

Attentional Models

Although several attentional models of conditioning have been proposed (e. g. Lovejoy, 1968, Sutherland & Mackintosh, 1971), the most contemporary and comprehensive attentional model is the conditioned attention theory (or CAT) offered by

Lubow, Schnur and Rifkin (1976). As such, this theory was chosen as the prototypical representative for this general class of theories and will be discussed with respect to the target questions.

Although the conditioned attention theory of Lubow, Schnur and Rifkin (1976; Lubow, Weiner & Shnur, 1981; Lubow 1989) was originally developed to address the phenomenon of latent inhibition, it has also been extended to apply to a wide range of conditioning phenomenon, including SPC (Lubow, Schnur and Rifkin, 1976). One of the basic assumptions of the model is that attention and inattention (both are hypothetical constructs) have many of the properties of conditioned responses with the exception that they are unobservable. The level of attention is viewed as a unidimensional variable that includes attention and inattention. It is also posited that levels of attention that are higher than the level of attention elicited on the first presentation of a stimulus are called attentional whereas levels of attention that are lower than the level of attention initially elicited by a stimulus are called inattentional . And finally, the model asserts that the likelihood of an attentional response occurring during a given stimulus presentation is increased when the stimulus is followed by reinforcement but decreased when it is not. The latter condition represents the development of inattention.

CAT makes four important assumptions regarding the dynamic properties of attentional responses. First, the probability of the

occurrence of an attentional responses (R_a) to a stimulus is inversely related to stimulus exposure. That is, R_a decreases as experience with the stimulus increases. Second, increases and decreases in R_a are conditionable like other Pavlovian responses. All that is necessary for R_a to be conditioned to a stimulus, say S1, is that it occur contiguous to another event, say S2, that also elicits an R_a. It is noteworthy that conditioning of an R_a will occur whether or not S2 is able to support conditioning of an overt response (Lubow, 1989). Third, repeated presentation of a stimulus, under certain conditions, may result in an initial increase of R_a. For example, during initial and contiguous presentation of two CSs, say CS1 and CS2, the R_a of the temporally first CS, CS1, will be increased. The reason that there is an initial increase in R_a is that, during compound CS presentations, CS1 acquires the capacity to elicit not only its own R_a but also the R_a of the CS1-CS2 stimulus complex. Over the course of many presentations, the CS1 and the CS1-CS2 complex both lose there ability to elicit R_a thereby resulting in an overall decrement in R_a for both stimuli (i. e., development of inattention). This build up of inattention is what allows CAT to explain conditioning phenomenon such as latent inhibition and conditioned inhibition. Fourth, a stimulus must elicit a requisite minimum level of R_a before that stimulus will be effective in entering into an association. In short, stimulus associability is positively related to R_a magnitude.

On a final note regarding CAT, the reader should be aware of the hypothesized relationship between conditioned attention and excitation and conditioned inattention and inhibition. Dealing first with conditioned attention and excitation, Lubow (1989) states that attention is conditionable in the Pavlovian sense but is quite independent of conditioned excitation and it's resultant CR acquisition. In fact, Lubow (1989) asserts attention can be conditioned prior to the occurrence of a CR but that CR performance can not be established in the absence of conditioned attention. For example the pairing of two CSs can result in the conditioning of attention, but no CR performance would be expected in the absence of a response eliciting stimulus (i. e., US). Inattention is also conditionable in a Pavlovian sense but it is different from conditioned inhibition in the way it is expressed. For example, a stimulus which has received training that imbues it with conditioned inattention will pass a retardation-of-learning test for inhibition but fail a summation test for inhibition whereas a stimulus that has received training which imbues it with conditioned inhibition will pass both test (Reiss & Wagner, 1972). In sum, one can think of conditioned attention as having properties similar (but not identical) to conditioned excitation and inattention as having some of the properties of conditioned inhibition.

How does this model explain the development of a Pavlovian conditioned response? Conditioning to a CS will occur only when

the magnitude of the CS-elicited R_a that occurs during the first CS presentation is maintained. The maintenance of the CS-elicited R_a ensures that attention will be conditioned to the CS. The primary role of the US is to maintain the R_a to the CS. Given that the CS-elicited R_a is maintained by the US, a CR will soon develop to the CS. Thus, as already noted, conditioning of attention to the CS is a prerequisite to CR acquisition.

Unlike the previous two models, CAT can explain both excitatory and inhibitory transfer SPC effects. In the case of excitatory SPC, a limited number of CS1-CS2 pairings result in the development of conditioned attention R_a to CS1. In the conditioning phase, CS2 has both attention (CS2-elicited R_a) and a response conditioned to it. If a CS1-alone test is performed, then CS1 presentations will result in the elicitation of the R_a of CS1 and the CS1-CS2 complex, the latter of which will, in turn, elicit the CS2-elicited R_a followed by the evocation of the CR. CAT explains transfer inhibitory SPC in the same manner with the following exceptions. During the unpaired inhibitory conditioning phase, the repeated presentation of CS2 alone results in the accrual of substantial levels of conditioned inattention (i. e., R_a decrement) and conditioned inhibition. In the retardation-of-learning test with CS1, CS1 elicits the CS1-CS2 complex, which in turn elicits the conditioned inattention of CS2, thereby by slowing the acquisition

of CRs to CS1. This retardation effect constitutes the transfer inhibition effect.

While CAT explains the transfer inhibition effect quite nicely, it does not at all accommodate the observation of direct inhibition. The problem is that CAT assumes that the retardation effect during the retardation-of-learning test is a product of inattention accrued to CS1 during preconditioning unpairings (i. e., latent inhibition effect). Since both Groups DI and C1-3 have identical experience with CS1 during the preconditioning phase, and since the attentional status of CS2 is irrelevant to the test with CS1, CAT would have to assume that similar level of inhibition would be observed in Groups DI and C1-3. This expectation is contrary to the findings of Experiment 3. Thus, while CAT can explain the basic phenomenon of excitatory SPC and the observation of transfer inhibition in SPC in Experiment 2, it cannot explain the finding of direct inhibition in Experiment 3 largely because it views the direct inhibition effect as entirely due to latent inhibition. This view puts CAT in the awkward position of requiring both Groups DI and C1-3 to evidence the direct inhibition effect.

Opponent-process Models

Early opponent-process theories such as those proposed by Solomon and Corbit (1974) and Schull (1979) emphasized the role of two opposing US-elicited hedonic processes in the development of Pavlovian conditioning. Wagner's (1981; Mazur & Wagner,

1982) sometimes-opponent-process or SOP theory outlined the application of the concept of opponent-processes to an information processing model of Pavlovian conditioning. The model represented a significant extension of the earlier opponent-process models in that it is a highly detailed and specific formulation which allows for the articulation of clear predictions. Recently, Wagner and Brandon, (1989) have presented a "dual-representation" version of SOP, called AESOP, which they view as a significant evolutionary step in the development of the earlier SOP. Since AESOP represents the most highly evolved and conceptually elaborate model of learning in the opponent-process class, it is the model that has been selected to represent this class.

Wagner and Brandon's (1989) learning model conceptualizes learning as a series of dynamic connections among stimulus nodes or memorial representations of stimuli that are activated by the presentation of stimuli. More specifically, AESOP asserts that the presentation of stimulus events (CSs and USs) result in the direct activation of multi-element nodal representations of those stimulus events. The nodal elements can also be activated indirectly by associative connections. The elements of each node can occupy any one of three states which were labeled inactivity (I) or resting state, primary activity state (A1) and secondary activity state (A2). Each time a node is activated by the stimulus it represents, some portion of the elements (p_1) in the I state will be elevated to the A1

state, from which they will subsequently decay, first to the A2 state and finally to inactivity (I). The model asserts that the rate of decay from A1 to A2 (denoted p_{d1}) occurs faster than the rate of decay from A2 to I (denoted p_{d2}). The proportion of elements activated from I to A1 (p_1) will increase as a function of stimulus intensity. When nodal activation occurs indirectly, which is assumed to occur when the nodal representation of a different stimulus to which the node has an excitatory associative connection is activated, some portion of the nodal elements (p_2) will first be elevated to the A2 state followed by decay to inactivity. The proportion of elements activated from I to A2 (p_2) is assumed to be directly related to the strength of the associative connection.

The key difference between the earlier SOP (Wagner, 1981; Mazur & Wagner, 1982) and AESOP (Wagner and Brandon, 1989) is that only the latter theory postulates that all USs have both sensory and emotive nodal attributes and therefore separate sensory and emotive nodal systems. It is further assumed that excitatory and inhibitory conditioning with any given US can involve either or both of the sensorial and emotive nodal attributes of the US. The only functional difference between these two US attributes is that the elemental decay rate is slower in the emotive than the sensory attributes.

According to AESOP, an excitatory association between the representations of a CS and US occurs only when the activation of

the respective nodes results in overlapping temporal distributions of A1 elemental activity. Following the establishment of an excitatory link between a CS and US, the presentation of the CS will first elicit A1 elemental activity in the CS node followed by an increase in A2 elemental activity in the US node (i. e., increase in p_2) and a corresponding increase in the probability of a CR. AESOP also asserts that an inhibitory link between two nodes occurs only when one nodes A1 elemental activity overlaps with the other nodes A2 elemental activity. After establishing an inhibitory link between a CS and US, presentation of the inhibitory CS will first elicit A1 elemental activity in the CS node followed by a decrease in A2 elemental activity in the US node (i. e., decrease in p_2) and a corresponding decrease in the probability of a CR. To apply the rules of SOP and therefore AESOP to sensory preconditioning, Mazur and Wagner (1982) have acknowledged that the associative rules must allow certain instances of CS-elicited A2 elemental activity to increase A2 elemental activity in the US node and hence increase the probability of a response. While this variation in the rules of AESOP was clearly intended to assist the model in explaining phenomena such as SPC and second-order conditioning, the proponents of the model have been silent regarding a decision rule that would clearly distinguish circumstances where the rule variation applies from those where it does not apply.

What is particularly relevant to the findings of the present studies, and to the SPC literature in general, is that AESOP explicitly allows that two CSs can have excitatory and inhibitory links or associations with each other (cf. Mazur & Wagner, 1982). Let us now examine the explanatory rigor of AESOP with respect to excitatory SPC, transfer inhibitory SPC, and direct inhibitory SPC.

In the first phase of excitatory SPC, CS1-CS2 pairings are presented and AESOP would predict that A1 elemental activity in the CS1 node would overlap with A1 elemental activity in the CS2 node resulting in the development of an excitatory link between the two CSs. In the conditioning phase, the pairing of CS2 and the US results in the overlapping of A1 elemental activity in the nodes of both the CS and US, giving way to the development of an excitatory link and CR acquisition. In the test phase, CS1-alone presentations would increase A2 elemental activity in the CS2 node followed by a rise in A2 elemental activity in the US node which would give rise to a CR probability appreciably greater than zero (Mazur & Wagner, 1982). If CS1 were paired with the US, as in the case of the savings test in Experiment 1, the increased A2 elemental activity of the US node would result in initial levels of CR activity appreciably above zero and the need for fewer pairings to yield asymptotic CR performance. Both of these expectancies were confirmed in Experiment 1 and in numerous previous investigations of excitatory SPC.

For transfer inhibition, the preconditioning phase nodal dynamics are identical to those for excitatory SPC: an excitatory link between CS1 and CS2 is developed. In the unpaired conditioning phase (Experiments 1 and 2), the CS acquires an inhibitory link to the US as a result of A1 elemental activity in the CS node overlapping with the more temporally distant A2 elemental activity in the US node. The net result is a decrease in the probability of CR acquisition. In the retardation-of-learning test, the initiation of A1 elemental activity in the CS1 node following presentations of CS1 would precipitate A2 elemental activity in the CS2 node which would in turn reduce A2 activity in the US node. The reduction in the A2 elemental activity of the US node precipitated by the CS1 would, at least temporarily, impede the establishment of an excitatory link between CS1 and the US (i. e., retardation would be observed). Thus, AESOP can account for transfer inhibition in SPC.

The question that remains is- does AESOP adequately account for direct inhibition in SPC? The answer appears to be "yes". In the first phase of direct inhibition training, CS1 and CS2 are explicitly unpaired, resulting in the overlap of distal A2 elemental activity of the CS2 node with the A1 elemental activity of the CS1 node when CS1 is presented (and vice versa for CS2). This results in an inhibitory link between CS1 and CS2. In the subsequent conditioning phase, the pairing of CS2 and the US

results in the establishment of an excitatory link between the two stimuli as well as the development of CRs to CS2. When the retardation-of-learning test is performed, CS1 presentations initiate a decrease in the A2 elemental activity of the CS2 node which prevents the initiation of A2 elemental activity in the US node. This slows the development of an excitatory link between CS1 and the US. The slowing of the development of the excitatory link between CS1 and the US is observed as retarded CR acquisition (i. e., behavioral inhibition). Thus, AESOP provides an adequate account of direct inhibition in SPC.

To summarize, it was established that three associative learning model's, each representing a influential class of theories, were unable to provide a complete account of traditional excitatory SPC or of the findings of conditioned inhibition in SPC observed the present studies. The Rescorla-Wagner (1972) and Miller and Schachtman's comparator hypothesis are both unable to account for either the traditional excitatory SPC effect or transfer and direct inhibition in SPC. The explanatory weakness of these models stems directly from their failure to allow for CS-CS associations and from their overemphasis on the role of US processing in the development of excitatory and inhibitory associative (Rescorla-Wagner, 1972) or behavioral (comparator hypothesis) processes. On the other hand, the conditioned attention model or CAT (Lubow, Schnur and Rifkin, 1976), provides an elegant account of transfer excitatory and

inhibitory effects in SPC. However, the model is unable to account for the observation of direct inhibition in SPC. The model cannot explain the finding largely because it views the direct inhibition effect as entirely due to latent inhibition and as a consequence cannot explain the differential performance Groups DI and C1-3 in Experiment 3. Finally, AESOP (Wagner & Brandon, 1989) is the one theory that can provide a complete account of the full range of excitatory and inhibitory SPC effects observed in the present studies. The major strengths of this model in comparison to the other three is that it explicitly permits the formation of excitatory and inhibitory associations between CSs and precisely defines the dynamics that give rise to such associations.

Future Directions of Research on Conditioned Inhibition in SPC

The present research documents the occurrence of two new associative phenomena: transfer and direct associative inhibition. What the experiments do not do is provide great insight into the mechanisms that control the phenomena. This is a task that needs to be pursued in future research. In the discussion that follows, a number of potential avenues of research pertaining to conditioned inhibition in SPC are outlined. The ideas expressed below do not constitute a complete catalogue of all possible future research but rather represent a small and select sample of ideas that the present author expects would be fruitful if explored. Specifically, the research ideas presented below address (a) the generality of

conditioned inhibition in SPC using other common inhibitory training protocols, (b) the generalizability of established empirical rules for conditioned inhibition derived from the Pavlovian conditioning to conditioned inhibition in SPC, and (c) the mediational role of contextual cues in the expression of conditioned inhibition in SPC. These three potential areas of future research are discussed in turn below.

First, if the rules of association are general, then it follows that the variety of training protocols which have been used to produce conditioned inhibition in Pavlovian conditioning should also result in conditioned inhibition in SPC. As noted earlier, some of the protocols used to produce conditioned inhibition in Pavlovian conditioning (cf. LoLordo & Fairless, 1985) have been (a) Pavlovian conditioned inhibition, (b) differential conditioning, (c) long forward trace conditioning, (d) backward conditioning, and (e) unpaired conditioning. Since only the unpaired procedure was used to produce conditioned inhibition in the present studies, a question that remains to be answered is whether both transfer and direct conditioned inhibition in SPC would be observed with the other four conditioned inhibition protocols. Confirmation of conditioned inhibition in SPC using these other training protocols would provide further support for the generality of the rules of association.

Empirical rules that govern the expression of conditioned inhibition in Pavlovian conditioning are generated by manipulating

conditioning variables and observing their effects on measures of conditioned inhibition. Some of the empirical rules identified to date are: (a) the development of conditioned inhibition is facilitated by protracted exposure to the US prior to inhibitory conditioning (Hinson, 1982, Experiment 4; Saladin & Tait, 1986, Experiment 1), (b) a change in the contextual cues will eliminate the facilitative effects of US preexposures on the development of conditioned inhibition (Saladin & Tait, 1986, Experiment 2), (c) repeated presentations of a putative inhibitor following inhibitory conditioning does not result in extinction of the inhibitor (e. g. Pearce, Nicholas & Dickinson, 1982; Williams, 1986; Witcher & Ayres, 1984; Zimmer-Hart & Rescorla, 1974), (d) conditioned inhibition generalizes to stimuli that resemble the target inhibitory stimulus (i. e., evidence a generalization gradient- Moore, 1972), and (e) the acquisition function of conditioned inhibition follows or lags behind that of conditioned excitation (see Weisman & Litner, 1969). If a parallel set of empirical rules were established for conditioned inhibitors developed in SPC, then the argument for the generality of associative and empirical rules of conditioned inhibition would be further supported. Although failure to identify parallel empirical outcomes in SPC might be construed as inconsistent with the existence of general associative and empirical rules, such findings might be even more interesting in that they would suggest the

possibility that conditioned inhibitors developed in SPC are a unique class of conditioned inhibitors.

Finally, in recent years there has been a growing interest in trying to understand the mediational role of the context (i. e., background cues) in Pavlovian conditioning (Balsam & Tomie, 1985). Likewise, it would be important to explore the extent to which the context mediates the development and maintenance of conditioned inhibition in SPC. In a recent report by Matzel, Held and Miller (1988), four possible linkages or associative chains were postulated that would allow a preconditioning stimulus to control responding (they were examining excitatory SPC effects). What is most interesting about their four hypothetical associative paths is that not one of them suggests the possibility that contextual cues might mediate the development of a CR to the preconditioning stimulus.

If, in the case of conditioned inhibition in SPC, the contextual cues do play an important mediational role in the expression of conditioned inhibition, then a change of context in any one of the three stages of SPC training could elucidate at what point in the associative chaining process, if any, is the context most important in producing conditioned inhibition. For example, if the inhibition test of a SPC study of transfer inhibition was performed in a context different from the one in which preconditioning and conditioning was conducted and conditioned inhibition was not

observed in the test, then one might conclude that the contextual cues are important for the expression of conditioned inhibition in SPC. Building on this observation, the question might be posed- is a change in either the preconditioning context or the conditioning context sufficient to eliminate the observation of conditioned inhibition in the test? This is a slightly more subtle question because now the investigator is trying to assess if the contextual cues at the time of preconditioning verses conditioning contribute uniquely to the expression of conditioned in the test. Accordingly, if conditioned inhibition is not observed when preconditioning is done in a different context but is observed when conditioning is done in a different context, then one could conclude that the contextual cues during preconditioning are important in maintaining the integrity of conditioned inhibition in SPC. Conversely, if conditioned inhibition in not observed when conditioning is done in a different context but is observed when preconditioning is done in a different context, then one could conclude that the contextual cues during the conditioning phase are important in maintaining the integrity of conditioned inhibition in SPC. Alternatively, if you don't observe conditioned inhibition following either of the above manipulations, then one could assume that maintaining the same contextual cues throughout SPC is essential to the preservation of conditioned inhibition in SPC. No matter what the obtained outcome of this line of research, the research would identify the

mediational role provided by the context in SPC and conditioned inhibition.

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Appendix A

Generality of the Associative Rules Governing the Development of Associative Excitation: A Comparison of SPC and Pavlovian Conditioning Research

A principle focus of the study of association is the establishment of general rules which allow for the specification of the relationship between learning and performance. Researchers who pursue such general rules are often referred to as general process learning theorists (Seligman & Hagar, 1972).

In attempting to establish general rules of association, general process researchers have manipulated various conditioning variables and observed the consequences on the behavior of an organism. If a specific behavior change reliably occurs when a specific variable is manipulated, then it is argued that a functional associative rule has been established for the specified behavior and conditioning parameter under consideration. When a functional associative rule has been demonstrated with a number of different types of training procedures, the associative rule becomes a general associative rule. While these general rules of association are not rules in the 'absolute' sense that they apply to all species in all conditioning situations at any time (Seligman & Hagar, 1972), the rules do specify relationships between experience and behavior that occur with substantial consistency. If there are general rules of association in the sense described above, then different methods

for studying association need to be used to identify the rules. Pavlovian conditioning and SPC are two such methods that could be employed to identify associative rules. Accordingly, one would expect that the manipulation of conditioning variables should yield similar behavioral effects in both Pavlovian conditioning and SPC studies. The following pages constitute a review of the literature in Pavlovian conditioning and SPC where the effects of similar conditioning variables and procedures have been examined. As already noted, this review pertains specifically to conditioned excitation. The review covers a number of areas where research findings revealed notable parallels between Pavlovian conditioning and SPC including: a) the generality of both Pavlovian conditioning and SPC across response systems and species, b) number of training trials, c) temporal relationship between training stimuli, d) CS intensity e) other behavioral phenomenon common to Pavlovian conditioning and SPC which suggest the generality of associative rules.

Response systems and species. If excitatory associative learning is a general process, and if Pavlovian conditioning and SPC are equivalent means of studying such associations, then it follows that Pavlovian conditioning and SPC should be observed in a wide variety of species and response systems. There is voluminous evidence that Pavlovian conditioning effects occurs in a wide variety of response systems (e.g.s., pupillary response, salivary

response, conditioned licking, eyelid and nictitating membrane responses, jaw movement response, heart rate changes, electrodermal response or GSR, etc) and in a equally diverse range of species including humans, dogs, cats, rabbits, rats, pigeons, monkeys (see reviews by Hall, 1976; Schwartz, 1989; Mazur, 1986). If SPC and Pavlovian conditioning access the same associative structure, then it would be expected that SPC should be observed in the same response systems and species in which Pavlovian conditioning has traditionally been observed. This expectation has been substantiated. Specifically, SPC has be identified in the following response systems and species: a) leg flexion with dogs (early studies from Pavlov's and Brogden's laboratories), b) nictitating membrane response (NMR) conditioning with rabbits (Port & Patterson, 1984; 1985; Port, Beggs, & Patterson, 1987), c) heart rate conditioning with rabbits (Pfautz, Donegan & Wagner, 1978) d) taste aversion conditioning with rats (Archer, Cotic & Jarbe, 1986; Archer & Sjoden, 1982; Fudim, 1978; Lavin, 1976; Rescorla, 1980; Revusky, 1980; but see Galef & Osborne, 1978) e) autoshaping with pigeons (eg., Rescorla, 1981; Rescorla, 1984; Rescorla & Cunningham, 1978); f) GSR conditioning with human subjects (Wickens & Cross, 1963) and g) CER with rats (egs., Prewitt, 1967; Rizley & Rescorla, 1972; Tait, Marquis, Williams, Weinstein & Suboski, 1969). Thus, conditioned excitation as a measure of excitatory associative learning does

appear to be a general process that is observed across various response measures and species in both Pavlovian conditioning and SPC studies.

Number of training trials. One conditioning variable that has been shown to significantly impact on the development of conditioned excitation in Pavlovian conditioning is the number of training trials. At first sight, the effects of this variable on the development of conditioned excitation in Pavlovian conditioning and SPC studies appears to be quite different. For example, Thompson (1972) has asserted that in Pavlovian conditioning, CR performance (conditioned excitation) increases as a function of the number of CS-US pairings while in SPC studies, CR performance during testing does not increase as a function of the number of preconditioning pairings. However, a closer analysis the data actually suggests that there considerable congruity between the two types of studies. These congruities are described below.

To date there are only 5 SPC studies which have explicitly examined the effects of the number of preconditioning pairings on CR performance during testing. In one study, Brogden & Gregg (1951) gave 2, 10 and 20 preconditioning pairings to human subjects in an auditory acuity procedure. The results indicated that a) all groups showed an SPC effect, b) the 2 and 10 trial groups evidenced more responding than the 20 trial group on the test and c) no statistically reliable differences existed between the groups.

While this study provided some evidence into the effects of the number of preconditioning pairing, the range of training values employed was too small to offer any definitive conclusions.

In a subsequent study by Hoffeld, Kendall, Thompson, & Brogden, (1960) groups of six cat subjects received 0, 1, 2, 4, 8, 10, 20, 80, 200, 400 and 800 preconditioning pairings prior to avoidance conditioning and testing. The results of the study showed an inverted U-shaped function where the maximum level of SPC occurred with 4 pairings. In agreement with Thompson (1972), the authors of this study concluded that SPC and Pavlovian conditioning are dissimilar in that they are affected differently by the amount of training. There is however some concern about the conclusions one can draw from this study. Specifically, it seems possible that the differential magnitude of SPC may be a function of the amount of second phase conditioning. Because the animals were trained to a criterion level of responding, some groups received substantially more acquisition training than others. In fact, those groups that received the greatest level of acquisition training also exhibited the greatest SPC effect.

In the third study, Prewitt (1967) examined the effects of the number of preconditioning trials on SPC in the CER training paradigm with rat subjects. This study compared groups of subjects that were given 1, 4, 16, and 64 preconditioning pairings with either a light or tone serving as the preconditioning stimulus. Control groups

for this study received either a) unpaired presentations during the preconditioning phase, or b) no preconditioning training. All groups received CER training and a test for SPC following the preconditioning phase. The results of the study indicated that SPC increased as a function of the number of trials up to 16 trials and that 64 trials did not result in significant change in the level of SPC.

A fourth study by Tait, Black, Katz and Suboski (1972) provided some data concerning the effects of the number of preconditioning pairings eventhough the study was actually designed to show evidence of discriminative SPC (discussed later). In this study, groups of rats received 7, 14, 28 or 56 preconditioning pairings of a tone and light stimulus. As in the Prewitt (1967) study above, the preconditioning phase was followed by CER training and a test for SPC. The results indicated that tone suppression was an increasing function of the number of preconditiong pairings, reaching asymptote at 28 pairings. Tait et al (1972) replicated Prewitt's (1967) observation that SPC became asymptotic after a relatively small number of trials. Neither study found a curvilinear function.

The fifth and final study (Revusky, 1980) examined the effects of 1, 2, 4, 8, 16 preconditioning pairings on SPC of taste aversion response with rat subjects. The results of the study indicated that SPC was pronounced with one trial and did not change as a function of the number of pairings. This result was

consistent with an ealier unpublished study by Lavin that was reported by Revusky (1980).

Because the methodological weaknesses of the Hoffeld et al, (1960) study seriously compromise it's significance, only the other four studies described above should be considered in drawing conclusions about the effects of the number of trials on SPC. Collectively, these studies suggest that the number of preconditioning pairings required to produce asymptotic SPC is quite small. Additionally, it appears that SPC increases as a function of the number of preconditioning pairings but the range of this effect is rather small since very few pairings are required to produce asymptotic SPC. Consistent with the observation that SPC is asymptotic with relatively few preconditioning pairings are the findings of a recent series of rabbit nictitating membrane conditioning studies by Patterson and his associates (Port & Patterson, 1984; 1985; Port, Beggs, & Patterson, 1987) which showed that 10 preconditioning pairings produce a greater SPC effect than either 20 or 40 pairings. Overall, the most consistent observation yielded by SPC studies appears to be that SPC increases to asymptotic levels after only a small number of preconditioning pairings.

Contrary to the present author, Thompson (1972) views the relationship between SPC performance and the number of training trials as inconsistent with the basic findings of Pavlovian

conditioning. This is probably due to two factors. One is that Thompson's position is 17 years old and thus is not a reflection of the current research on classical conditioning. The second is that Thompson greatly overgeneralized and simplified the results of the Pavlovian conditioning research of his time. It is often the case that variables which affect Pavlovian conditioning produce complex effects and simplifications and generalizations are simply unwarranted.

Is the data presented above inconsistent with the literature on Pavlovian conditioning? In fact it is not. Notice that three of the four well designed studies discussed above made use of either CER (e.g., Prewitt, 1967) or taste aversion (e.g., Revusky, 1980) response systems to examine the effects of the number of training trials on SPC. Studies of CER conditioning have shown that asymptotic levels of conditioning can occur in as little as 5 to 16 trials and that pre-asymptotic conditioning is an increasing function of the number of training trails (e.g.s., Kamin, 1968; Randich & LoLordo, 1981). Even more dramatic is the well documented finding that taste aversion learning is near asymptotic with 1 CS-US pairing (e.g.s., Mazur, 1986; Revusky & Bedarf, 1972). Thus, the observation that SPC occurs with relatively few preconditioning pairings is consistent with a considerable portion of the Pavlovian conditioning literature in which conditioning occurs with relatively few CS-US pairings

In general, the above discussion suggests one general observation. Specifically, both Pavlovian conditioning and SPC studies yield converging evidence which indicates that there is a general rule regarding the effects of the number of training trials on excitatory associative learning. This rule could be stated as follows: excitatory associative learning develops to asymptotic levels with relatively few pairings.

Order of stimulus presentation and interstimulus interval. Studies in SPC and Pavlovian conditioning have been conducted to assess the effects of the temporal relationship between the conditioning and preconditioning stimuli, respectively, on subsequent excitatory conditioning. These studies have been performed in the interest of defining the conditions (i.e., rules) under which excitatory conditioning develops most optimally. The major point of interest for the present discussion is whether or not the Pavlovian conditioning and SPC studies yield similar outcomes when this variable is manipulated. Analogous outcomes will provide further evidence for the notion of general rules of association which can be determined using both Pavlovian conditioning and SPC.

In SPC, eight studies have evaluated the effects of the temporal arrangement between the preconditioning CSs. In general, these studies have examined either a) the relative strength of SPC that results from forward, simultaneous, and backward pairings of the preconditioning CSs (hereafter refered to as stimulus order) or,

b) the relative levels of SPC obtained when the interstimulus interval (ie., time between the onset of each of the preconditioning CSs) is varied in forward and backward SPC. However, several of the studies examined both stimulus ordering and interstimulus interval at the same time thereby making it difficult to discuss each of the studies with respect to one or the other of these categories. To eschew repetitive description of each of the studies under each of the above categories, the present author will first describe the SPC studies in their order of publication and then summarize their findings either with respect to stimulus ordering and interstimulus interval. Subsequently, the findings of the SPC studies will be contrasted with those yielded by Pavlovian conditioning studies.

In the first of these studies, Silver & Meyer (1954) examined the effects of forward, simultaneous and backward preconditioning pairings on the magnitude of SPC obtained in a runway escape task with rat subjects. In the preconditioning phase of the study, the two CSs, a light and a buzzer, were presented to three group of rats. In the forward group, the onset of the preconditioning CS preceded the onset of the to-be-conditioned CS by 1.5 secs while for the simultaneous group the CSs occurred at the same time. The backward group received the to-be-conditioned CS 1.5 secs prior to the preconditioning CS. During conditioning, all subjects received CS-US presentations in the runway. A CR was defined as at least 6-in. of movement down the runway prior to the onset of the US.

The results of the study showed that all three groups evidenced SPC and that the forward group was superior to the simultaneous and backward groups. However, it is doubtful that the Silver & Meyer (1954) study demonstrated backward excitatory SPC because the intertrial interval was so short (ie., 2.7 secs) that the backward preconditioning CS on any given trial was temporally contiguous with the to-be-conditioned CS on the subsequent trial. Accordingly, the backward training was actually inadvertent forward conditioning. Thus, this study only provides clear evidence of forward and simultaneous SPC, with a larger SPC effect being observed the former as compared to the latter.

Hoffeld, Thompson & Brogden (1958) examined simultaneous preconditioning and a variety of forward preconditioning interstimulus intervals in a cage turning avoidance task with cat subjects. In this study, groups of subjects received the preconditioning pairings of a tone and light CS at 0.0, 0.5, 1.2, 2.0 or 4.0 sec ISIs. A control group which experienced the training apparatus but which did not receive any preconditioning treatment was also included. The results showed that all of the preconditioning treatment groups evidenced SPC when compared to the control and that the 4.0 sec ISI group evidenced significantly greater SPC than the other groups. Thus, the study provided evidence of SPC at both simultaneous and forward ISIs and that SPC is optimal with a 4.0 sec forward ISI. The results of this study

should be considered in light of two weaknesses. First, the number of subjects in each group (n=4) was too small to detect any but the most profound group differences. Second, since the objective of the study was to identify an ISI function, the authors should have examined a greater range of ISI values.

During the same year, Coppock (1958) published a human galvanic skin response (GSR) study which provided evidence on the relative strength of SPC obtained with forward and backward preconditioning pairings. The results of the study showed that only the group preconditioned with a forward ISI evidenced significant SPC (ie., the backward group and unpaired control performed similarly on the test). Thus, this study found forward SPC to be robust but found no evidence of backward SPC.

As a follow-up to an earlier study (Hoffeld et al., 1958), Wynne & Brogden (1962) evaluated the effects of simultaneous and various forward and backward preconditioning interstimulus intervals on the level of SPC observed in a cage turning avoidance task with cat subjects. The interstimulus intervals employed in this study were -4, -2, -1, 0, 1, 2, 4, 8, 16 with the negative values being backward interstimulus intervals, the 0 ISI representing simultaneity and the remaining values being forward interstimulus intervals. Because the duration of the two preconditioning CSs was held constant at 2.0 secs, the forward and backward interstimulus intervals could be further differentiated into either trace or delay

interstimulus intervals. A trace interstimulus intervals is defined as a relationships between two CSs where there is stimulus free period between occurrences of the CSs. In the case of delayed interstimulus intervals, there is no stimulus free period and very often some overlap between the CSs. The -4, 4, 8,16 sec interstimulus intervals represent backward and forward trace conditioning parameters where, for example, the 16 sec. interstimulus interval indicates that the preconditioning CS would be presented for 2 sec. and then a 14 sec stimulus free period would follow and then the to-be-conditioned CS would be presented for 2 secs. The -2, -1, 1, 2 ISIs represent backward and forward delayed preconditioning intervals where, for example, the 2 sec. interstimulus interval indicates that preconditioning CS would be presented for 2 sec. and then the to-be-conditioned CS would immediately be presented for 2 secs. The results of this study indicated that the 4.0 sec interstimulus interval gave the greatest SPC, the 0, 1, and 2 sec interstimulus interval groups produced less pronounced, albeit significant SPC and, the -4, -2, -1, 8, and 16 sec interstimulus intervals did not result in SPC as indicated by comparison to a no preconditioning control group. Thus, four conclusions seem warranted based on this study. One is that there appears to be an optimal forward preconditioning interstimulus intervals in SPC and that deviations from this optimum value produces less pronounced SPC. Second, there is evidence that

simultaneous SPC does occur although the effect is not as pronounced with forward interstimulus intervals. Third, short but not long trace intervals result in SPC. Forth and finally, the failure to identify appreciable levels of backward SPC is consistent with Coppock (1958).

Using a human GSR training procedure similar to that of Coppock (1958), Wickens & Cross (1963) compared simultaneous preconditioning with 3 different forward preconditioning interstimulus intervals. The specific forward interstimulus intervals examined were 100, 400 and 600 msec. and a light and tone stimulus served as preconditioning CSs. Since no control group was included in this study it is difficult to determine the absolute level of SPC evidenced by the various experimental groups. Nonetheless, according to the authors the results of the study indicated that the maximum level of SPC was obtained with 400 msec interstimulus interval and that lesser levels of SPC were obtained with the simultaneous, 100 and 600 msec interstimulus intervals. Again, these results confirm that there is an optimal interstimulus interval for SPC and that deviations from that optimum value results in less pronounced SPC. This study also suggests that simultaneous SPC occurs but to a lesser degree than forward SPC.

Two studies published in 1969 offered data regarding the comparative strength of backward and forward SPC. One of these studies (Tait et al., 1969) used CER with rats to examine, among

other things, the level of CER obtained with 10 sec. backward and forward ISIs. These researchers found substantial SPC in the forward but not the backward conditioning groups. This conclusion was based on a comparison between the forward and backward trained groups of the Tait et al., (1969) study and the unpaired and no preconditioning control groups of Prewitt's (1967) study using similar CER training parameters. The other study by Brown & King (1969) used a human salivary training procedure in an attempt to identify backward and forward SPC under optimal conditions. In this study an unpaired control group was used to assess the effects of SPC training. Again, no evidence of backward SPC was obtained although a robust forward SPC effect was demonstrated. Taken together, the results provide substantial evidence for forward but not backward SPC. Note that the failure to identify backward SPC is consistent with Wynne & Brogden (1962) and Coppock (1958).

In the final study to be condidered here, Spiker & Ferraro (1977) used a CER training paradigm with rat subjects to examine the magnitude of SPC obtained with a variety of forward preconditioning interstimulus intervals. The interstimulus intervals examined in this study were 4.0, 10.5, 17.0 and 23.5 secs. The two preconditioning CSs employed were: a) a stimulus compound consisting of two 7-W white cue-lights (ie., CS1) and, b) a 70 dB, 1250 Hz tone (ie., CS2). This study also made use of an unpaired control group. The results of the study showed that the greatest

level of SPC was obtained with the 4.0 sec interstimulus interval followed next by the 10.5 sec ISI. A trend analysis showed supression to be an inverse function of interstimulus interval thereby indicating that SPC was an inverse function of interstimulus interval. Again, there appears to be considerable support for an optimal forward preconditioning ISI where deviations from that value result in diminished SPC.

Collectively, these eight studies offer a number of general observations about the effects of stimulus ordering and interstimulus interval on SPC. With regard to stimulus order effects, it would appear that SPC is more pronounced with forward pairings than with simultaneous pairings and that no SPC results from backward pairings. With regard to interstimulus intervals, it would appear that: a) relatively short forward delay and trace interstimulus intervals produce the most robust SPC effect; b) there appears to be an optimal forward CS-CS interval and that deviations from this optimal value result in less pronounced SPC; and, c) that the optimal ISI appears to vary across response systems (ie., SPC in human GSR studies is optimal at approximately 400 msec while optimal SPC in CER with rats occurs at approximately 4.0 secs [4000 msecs]).

Are the above observations consistent or inconsistent with the findings of the Pavlovian conditioning research? The answer to this question is clearly in the affirmative. In regard to stimulus order effects, Pavlovian conditioning is most effective when the

conditioning stimuli are in a forward temporal arrangement (eg., Hall, 1976; Pavlov, 1927). The Pavlovian literature also indicates that simultaneous conditioning has been reliably demonstrated (Burkhart & Ayers, 1978; Heth, 1976; Mahoney & Ayers, 1976; Heth & Rescorla, 1973) but is much less robust than the conditioning produced through forward pairings (Rescorla, 1980). Finally, the issue of backward Pavlovian conditioning has proved to be a very complex one. Indeed, studies in Pavlovian conditioning have shown backward pairings of a CS and US to result in excitatory (egs., Heth, 1976; Heth & Rescorla, 1973), inhibitory (egs., Seigel & Domjan, 1971;1974), both excitatory and inhibitory (egs., Overmier, Payne, Brackbill, Linder & Lawry, 1979; Tait & Saladin, 1986) and no associative effects (egs., Champion, 1962; Smith, Coleman & Gormezano, 1969). Not surprisingly, the SPC literature parallels one aspect of the Pavlovian conditioning in that backward SPC appears to produce no associative effects (ie., no SPC). However, it remains entirely possible that backward SPC does produce some excitatory or inhibitory effects which may be identified with other test procedures. For example, a retardation-of-learning test might be more useful in identifying inhibition. Alternatively, a positive transfer or savings in a retardation-of-learning test might indicate the presence of conditioned excitation. To date, there are no SPC studies examining the effects of

backward pairings which have reported the findings of alternative test strategies such as a retardation-of-learning test.

In regards to interstimulus interval, numerous empirical reviews of the Pavlovian conditioning literature (e.g.s., Hall 1976; Gormezano & Moore, 1969; Gormezano, Kehoe & Marshall, 1983) indicate that there is an optimal forward ISI and that conditioning involving non-optimal ISIs is generally less effective. It is also known that the optimal ISI in Pavlovian conditioning will vary from one response system to another (Schneiderman, 1972). For example, the optimal training ISI for GSR is approximately .5 sec. while effective CER conditioning is obtained with ISIs in the range of 4-10 secs., the optimal ISI being approximately 7 secs. (McAllister & McAllister, 1971). Thus, the findings of the SPC studies described above closely resemble those of Pavlovian conditioning studies.

In terms of the central concern of this review, it appears that once again SPC and Pavlovian conditioning studies indicate that they are equivalent means of pursuing the rules of excitatory associative learning. Specifically, they both yield similar behavioral effects in response to the manipulation of the temporal relationship between conditioning stimuli.

CS intensity. CS intensity is another variable which is known to effect the development of excitatory conditioning in both SPC and Pavlovian conditioning. In the pages that follow, a series of

four SPC studies will be described and the results summarized. Following the summary, the results of the SPC studies will be compared to those of Pavlovian conditioning studies.

The first of four studies examining the effects of the intensity of the preconditioning stimuli on SPC was conducted by Thornton (1956). Using a hurdle avoidance training procedure with rat subjects, Thornton assessed the relative level of SPC obtained with preconditioning CSs that were either low (L) or high (H) in intensity. The CSs used were a light (L) and a buzzer (B). The high intensity light was a 200 watt bulb and the low intensity light was a 10 watt bulb. The exact intensity properties of the high and low intensity buzzer stimuli were not specified by the author. In the preconditioning phase of the study, subjects received 240 L-B simultaneous presentations of either low or high intensity. The second phase consisted of 60 massed buzzer-shock US avoidance conditioning trials where the US onset followed the buzzer onset by 2.5 sec. In the third phase, the subjects received 60 massed light stimulus-US avoidance conditioning test trials (same temporal parameters as in phase 2). SPC was measured by the mean number of avoidance response performed to the light stimulus duri ng the third phase. Four control groups which received preconditioning training with only one of either LL, HL, LB or HB were also employed. The results of the study indicated that only the group given low intensity CS preconditioning pairings (ie., group LL-LB)

evidenced SPC. The author concluded that there is an optimal intensity beyond which SPC is not demonstrable.

Wokoun (1959) investigated the effects of varying the intensity of either the preconditioning CS or the to-be-conditioned CS in a shock avoidance task with rat subjects. The two stimuli used were a buzzer which was of constant intensity and a light which was presented in one of three intensities (15, 50 or 100 watt bulb). Of the six experimental groups, three received 600 forward preconditioning light-buzzer pairings and the remaining three groups received 600 buzzer-light pairings. Control groups received presentations of either of the stimuli or no preconditioning training at all. During conditioning, all experimental groups received pairings of the to-be-conditioned stimulus (or control stimulus) and a shock US to establish avoidance behavior. Two test conditions were used: one consisted of 10 presentations of the preconditioning CS alone and the other was a savings test where the preconditioning CS was paired with the US. The results of the study showed that SPC was a inverse function of the intensity of the preconditioning CS but a inverted U-shape function of the intensity of the to-be-conditioned CS. A subsequent analysis of the Wokoun (1959) data by Tait & Suboski (1972) suggested that there may have been a "serious problem" with the experiment. In particular, avoidance conditioning was not found to be affected by the various intensities of the light stimulus. Since the intensity of the light did not affect

avoidance conditioning, there is little reason to expect that it should affect SPC.

The remaining two studies which assessed the effects of CS intensity on SPC employed CER training with rat subjects. In the first of these studies, Holmes (1968, Experiment 3) gave groups of subjects 9 pairings of a light and noise CS of two intensities. The high intensity light stimulus was a 25 watt bulb powered by a 110 volt power supply while the low intensity light was the same bulb powered by a 40 volt power source. The high intensity noise was delivered at 90 db whereas the low intensity noise was delivered at 60 db. Control subjects received the same stimulus experience with the exception that the stimuli were unpaired. In the second phase, the subject received CER training with either a high or low intensity light or noise stimulus. The test phase consisted of unreinforced presentations of the preconditioning stimulus. The result of the study indicated that a) when both preconditioning stimuli were weak, there was little evidence of SPC, b) when one of the preconditioning CSs was strong and one weak, an intermediate level of SPC was observed, and c) when both CSs were strong, the highest level of SPC was identified. On the whole, Holmes' data suggests that SPC is a function of CS intensity.

Lastly, two studies performed by Tait & Suboski (1972) evaluated the effects of the intensity of the phase 1 CSs on SPC. In Experiment 1, the intensity of the to-be-conditioned CS was

varied while in Experiment 2 the intensity of the preconditioning CS was varied. In both studies, the CSs were a) a 5-watt light illuminated by 28 volt current with a 0, 34, 68 ohm resistor in series to determine intensity, b) a 73, 80, or 87 dB tone. Moreover, preconditioning training in both studies consisted of 16 forward CS-CS presentations, conditioning consisted of 10 CS-US pairings and testing consisted of 10 presentations of the preconditioning CS alone. The results of both experiments indicated that a) when the to-be-conditioned CSs intensity is varied, the level of SPC does not change although there was a numerically superior level of supression in the high intensity vs low intensity group, b) SPC is a positive function of the intensity of the preconditioning CSs.

Simply stated, it would appear that varying the intensity of the CS during preconditioning can have a multitude of effects on the level of SPC observed in testing. Summarizing the results above one finds that SPC is: a) unaffected by CS intensity manipulations (Thornton, 1956), b) an inverse function of CS intensity (Wokoun, 1959), c) an inverted U-shape function of CS intensity (Wokoun, 1959) and, d) a positive function of CS intensity (Holmes, 1968; Tait & Suboski, 1972, Experiment 2). It is also evident that the only effect of CS intensity on SPC which has been replicated is the finding of a positive relationship (Holmes, 1968; Tait & Suboski, 1972, Experiment 2). Therefore, while a number of different outcomes have been demonstrated, only the finding of a positive

relationship between SPC and CS intensity has been observed with any consistency.

Pavlovian conditioning studies which have examined the effects of CS intensity on CR development have also identified a diverse range of outcomes (Gray, 1965). In particular, Pavlovian conditioning studies have found that conditioning is either unrelated to CS intensity (Grant & Schneider, 1948,1949), an inverse function of CS intensity (Kimmel, 1959), or a positive function of CS intensity (Barnes, 1956; Imada, Yamazaki, Morishita, 1981; Kamin & Brimmer, 1963; Kamin & Schaub, 1963). The Pavlovian conditioning literature is also consistent with the SPC literature in that the observation of a positive relationship between CS intensity and CR development is the most consistently observed outcome. It is likely that the diversity of findings in both the Pavlovian conditioning and SPC literature is a product of different training procedures and paramenters employed by researchers to study the effects of CS intensity. This notion has gained some empirical support from studies which have shown that conditioning parameters such as US intensity do influence CS intensity effects (eg., Kamin & Brimer, 1963). In any case, the parallels between the Pavlovian conditioning and SPC literature are undeniable and lend further credence to the position that they are equivalent means of identifying rules that govern the development of excitatory associations.

Other parallels between SPC and classical conditioning. General process learning theorist have maintained that there are general associative rules that govern the development of conditioned excitation. Accordingly, if the mechanisms of association are general, it would follow that behavioral phenomenon (ie., patterns of behavior) which reliably result from specific training experiences in Pavlovian conditioning studies should also be observed in SPC studies that afford a similar training experience. In fact, there are a few SPC studies which have attempted to identify behavioral phenomenon (eg., extinction) that are well documented in the Pavlovian conditioning literature. These studies are briefly reviewed below.

Extinction is a behavioral phenomenon which has been identified as an integral part of Pavlovian conditioning since the seminal work of Pavlov (1927). Pavlov defined extinction as a "phenomenon of a rapid and more or less smoothly progressive weakening of the reflex to a conditioned stimulus which is repeated a number of times without reinforcement". Contemporary definitions of extinction are congruent with Pavlov's statement (see Gormezano & Moore, 1969; Gormezano, Kehoe & Marshall, 1983). Since extinction is a robust feature of Pavlovian conditioning, it might reasonably be assumed that SPC should also exhibit the capacity for extinction. This assumption was tested in three studies. Coppock (1958) found less SPC in a group of subjects that

had received unreinforced presentations of the preconditioning stimulus following preconditioning than for a group that received preconditioning treatment alone. Unfortunately, the significance of this finding was somewhat diminished by the authors failure to statistically evaluate the group differences. Nonetheless, the study did offer the first evidence of extinction in SPC. This led Tait et al., (1969) to evaluate the effects of various amounts of extinction training on subsequent test performance in a CER study of SPC. Although the study examined the effects of extinction training on both backward and forward SPC, only the finding of the forward SPC are relevant to the present discussion. In their study, rat subjects were given 16 preconditioning pairings followed by either 0, 1, 4, 16 or 64 extinction trials (ie., preconditioning stimulus alone) which in turn was followed by CER training. SPC, as indexed by suppression of rat licking behavior, was found to be a decreasing function of extinction. Finally, Rescorla & Freberg (1978, Experiment 1) used a taste aversion SPC procedure with rat subjects to study the effects of extinction of a preconditioning CS (sucrose flavored water) prior to aversion conditioning involving a to-be-conditioned CS (.005 M hydrochloric acid solution) and emetic US (LiCl). The results indicated that SPC, as measured by the consumption of the preconditioning CS, was less pronounced in subjects that received unreinforced presentations of the preconditioning CS than in subjects that received preconditioning

training alone. Thus, the findings of these three studies correspond closely to those commonly found in Pavlovian conditioning.

Discriminative or differential conditioning is said to occur when the level of performance (eg., CRs) controlled by a cue(s) that is reliably reinforced is substantially in excess of that observed for a cue(s) that is not reliably reinforced. This phenomenon has been observed repeatedly by Pavlovian conditioning researchers (Kimble, 1961; Kinkaide, 1974; Moore, 1972; Pavlov, 1927; Saladin & Tait, 1986; VanDercar & Schneiderman, 1967). Only one study has attempted to identify this reliable Pavlovian conditioning phenomenon in SPC. The study, by Tait, Black, Katz, Suboski, (1972), employed rat subjects in a CER training procedure. During preconditioning, the subjects received either 7, 14, 28, or 56 pairings of a tone and light and an equal number of unpaired tones of a different frequency. CER training with the light stimulus followed preconditioning. Discriminative SPC was assessed by measuring the level of lick suppression to the paired and unpaired preconditioning stimuli. Consistent with the results of Pavlovian discriminative conditioning studies (eg., Saladin & Tait, 1986), the results showed that the paired preconditioning tone controlled more suppression than did the unpaired preconditioning tone. In addition, suppression was positively related to the number of paired tone presentations but inversely related to the number of unpaired tones.

Subsequent to Pavlovian conditioning with a CS, other similar stimuli which have never been paired with the US will also elicit the behavior controlled by the CS (see Pavlov, 1927 and Moore, 1972). The transfer of response control to similar stimuli is known as generalization. Two SPC studies have provided some data which indicates that stimuli similar to the preconditioning CS can also elicit responding during testing. Kendall & Thompson (1960) gave groups of cat subjects 20 pairings of tones of 2000 cps and 250 cps prior to instrumental avoidance training with the 250 cps tone CS. When given unreinforced test trial of 250, 500, 1000, 2000, 4000 and 8000 cps tones it was found that: a) responding was very high to the conditioning CS (ie., 250 cps tone), b) SPC was significant as indicated by substantial responding to the 2000 cps in SPC subjects relative to control subjects and c) some generalization was evident as indicated by elevated levels of responding to a 4000 cps tone. A second study by Cho & Mitchell (1971) examined generalization in a avoidance task with human subjects. During preconditioning experimental subjects received 5 presentations of a light and either a 670 or 1850-Hz tone while control subjects received unpaired presentations of the same stimuli. Following preconditioning, all subjects received avoidance training with the light and a shock US. Testing consisted of unreinforced presentations of 670, 1000, 1400 and 1850-Hz tones. In addition to a basic SPC effect, test data showed that the highest level of responding was controlled by

the preconditioning tones (ie., 670 and 1850-Hz tones) and that appreciable levels of responding also occurred to the untrained 1000 and 1400-Hz tones. Thus, both of the above studies provide evidence of generalization effects in SPC which parallel those found in Pavlovian conditioning.

The partial reinforcement effect (PRE) is another Pavlovian conditioning phenomenon which has been identified in SPC. Briefly, the effect of partial reinforcement in Pavlovian conditioning is to retard acquisition and increase resistance to extinction (Gormezano & Moore 1969; Gomezano, Kehoe & Marshall, 1983; Holmes & Gormezano, 1970; Willis & Lundin, 1966). Tait, Simon & Suboski (1971) used a CER procedure with rats to identify PRE in SPC. In this study groups of rats received one of the following preconditioning treatments: a) 64 CS1-CS2 pairings, b) 32 CS1-CS2 and 32 CS1 alone presentations, c) 16 CS1-CS2 and 48 CS1 alone presentations and, d) random presentations of CS1 and CS2. Thus, treatments a), b), and c) correspond to 100%, 50% and 25% reinforcement schedules, respectively, and treatment d) is the unpaired control condition. Subsequent to CER training which consisted of 10 CS2-shock US pairings, 10 CS1 alone presentations were given for four days. Because acquisition can not be indexed directly in SPC, suppression ratios on the first 3 CS1 presentations were used as an index of the terminal level of SPC developed in each group. The results of the study indicated that groups

Footnotes

[1] Due to an equipment failure, one subject in Group C1-3 received a weaker US than did the remaining subjects during the first 2 daily sessions of the conditioning phase (US < 1.0 mA). However, because this subject met the performance criterion imposed on the third and final day of the conditioning session and because it seemed that training with a weaker US would only work against the identification of direct inhibition (i. e., weaker second phase conditioning in this subject would likely only result in slower/lower CR performance during the inhibition test), the subject was included in the study.