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An Extended Comparator Hypothesis Account of Superconditioning

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Three conditioned taste aversion experiments with rats investigated superconditioning. In each experiment, alternate exposures of 2 flavor compounds with a common element (i.e., AB/AS) were administered to establish an inhibitory relationship between the 2 unique elements, B and S, and prior to testing, S was paired with lithium chloride (LiCl). In Experiment 1, pairings of a neutral cue (X) with S in compound with B after the AB/AS exposures resulted in superconditioning between X and S. Extinction of the common element (A) just before the S−LiCl pairing attenuated both the inhibitory relationship between B and S (Experiment 2) and superconditioning between X and S (Experiment 3). These observations suggest that superconditioning consists of enhanced performance rather than enhanced associative acquisition.

Pavlovian responding has long been known to be enhanced by various procedures, such as increasing the physical intensities of the associates, conducting more pairings of the associates, and pairing the associates with greater spatiotemporal contiguity (Pavlov, 1927). More recently, on the basis of increasing interest in phenomena and mechanisms of interaction between cues trained in compound, another procedure to enhance Pavlovian responding, *superconditioning* (also known as supernormal conditioning; Rescorla, 1971), was proposed. Superconditioning, which was originally predicted by the Rescorla and Wagner (1972) model, is defined as an extraordinary augmentation of behavioral control by a conditioned stimulus (CS) resulting from its being paired with an unconditioned stimulus (US) in compound with a CS that was previously trained as a conditioned inhibitor for the US. In a typical superconditioning paradigm, conditioned inhibition training (e.g., A+/AB−)[1] is conducted (making CS B inhibitory). Next, excitatory conditioning of a target CS (X) in compound with the conditioned inhibitor is conducted (e.g., BX+). Superconditioning is demonstrated when the behavioral control by the target CS in the superconditioning condition (e.g., A+/AB− followed by BX+) is superior to that observed in two appropriate control conditions. The first necessary control condition is one that receives conditioning of the target CS in compound with a neutral CS (e.g., A+/AB− followed by CX+; hereafter called the *overshadowing control condition*). The second necessary control condition is one that receives conditioning to the target CS alone (e.g., A+/AB− followed by X+; hereafter called the *elemental-acquisition control condition)*. Omission of the first control condition would leave open the possibility that greater conditioned responding to X after the superconditioning treatment results from its being conditioned in compound with B independent of B's inhibitory potential (i.e., *potentiation*; e.g., Clarke, Westbrook, & Irwin, 1979; Rusiniak, Hankins, Garcia, & Brett, 1979). Omission of the second control condition would leave open the possibility that greater responding to X in the superconditioning condition relative to the overshadowing control condition results from attenuated overshadowing due to B being inhibitory (see Navarro, Hallam, Matzel, & Miller, 1989). The omission of either control condition prevents an unequivocal interpretation of superconditioning.

Although there are several early reports that claimed to demonstrate a superconditioning effect (e.g., Blanchard & Honig, 1976; Rescorla, 1971; Taukulis & Revusky, 1975), Navarro et al. (1989) pointed out that these studies did not use the elemental-acquisition control condition, but only the overshadowing control condition. Thus, these studies did not address the possibility that the observed enhanced responding was merely an attenuation of the overshadowing effect. Moreover, Navarro et al. conducted four experiments on superconditioning using a conditioned lick suppression preparation with rats and found no evidence of superconditioning. Although they observed greater responding in the superconditioning group relative to the overshadowing control group, responding in the superconditioning group never exceeded that of the elemental-acquisition control condition. The authors concluded that the superconditioning-like effects obtained in both their own and prior studies were no more than effects of protection from overshadowing caused by nonreinforced presentations of the overshadowing cue (B) during conditioned inhibition training (due to latent inhibition of the potential overshadowing stimulus). Therefore, Navarro et al. questioned the very existence of a genuine superconditioning effect.

Following the research conducted by Navarro et al. (1989), Pearce and Redhead (1995) showed, in an appetitive conditioning

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[1] Here and hereafter, capital letters represent different CSs, "+" represents presentation of a US, "−" represents no presentation of the US on that trial, and "/" indicates that trials of stimuli preceding and following the slash are interspersed during training.

preparation using rats, that BX+ training following A+/AB− training endows X with the potential to elicit superasymptotic responding (i.e., responding greater than that in a condition trained with the target cue alone). However, Pearce and Redhead's results cannot be viewed as conclusive because their experimental design was somewhat different from those used in prior studies of superconditioning. Specifically, they used the training excitor of the initial conditioned inhibition training as the target CS (i.e., a single cue served as both A and X). Although they certainly showed that excitatory conditioning of a target CS in compound with an inhibitory stimulus can produce greater responding than that produced by conditioning to the target cue alone, the effect they showed was explicable in terms of stimulus generalization. As the authors suggested, the observed high responding to their target stimulus could have reflected summation of the response potential of the X stimulus with the generalized response potential from the BX compound stimulus (see Pearce, 1987).

Aitken, Larkin, and Dickinson (2000) reported a corresponding finding, superlearning, in a human causal judgment preparation. In one of their experiments (Experiment 3), they compared three conditions, a superlearning condition (A+/AB− followed by BX+), an overshadowing control condition (no prior experience with B followed by BX+), and an exposure condition (A−/AB− followed by BX+), in a within-subject design and found that the causal rating of the target cue (X) in the superlearning condition was greater than those in the two control conditions. Their finding refuted the possible alternative explanation of superconditioning suggested by Navarro et al. (1989) that apparent superconditioning is just a failure of overshadowing caused by nonreinforced presentation of the overshadowing cue during conditioned inhibition training. However, as they did not include an elemental-acquisition control in their experiments, it is still difficult to accept their data as clear evidence of superconditioning. That is, one cannot determine whether the reinforcement of the target cue in compound with a conditioned inhibitor genuinely facilitated responding to it or was just more effective than the procedures in the other two conditions in preventing any overshadowing effect.

Recently, Williams and McDevitt (2002) reported a superconditioning effect in an autoshaping preparation using pigeons. They observed greater responding after superconditioning treatment relative to both appropriate control conditions previously mentioned. However, there is also a potential problem with their experiment. Williams and McDevitt used a partial reinforcement procedure during conditioning with the target CS; that is, they conducted BX+/BX− trials following A+/AB− trials. Therefore, the result they obtained is also explicable in terms of protection from extinction caused by the presentation of a conditioned inhibitor (B) in compound with a to-be-extinguished CS during nonreinforced BX trials (e.g., Chorazyna, 1962; Soltysik, Wolfe, Nicholas, Wilson, & Garcia-Sanchez, 1983). In other words, what they observed conceivably might not be an enhancement of excitatory conditioning itself, but instead, an attenuation of the decremental effect on responding otherwise produced by the nonreinforced presentations of the target CS during Phase 2 training.

On the basis of an autoshaping preparation with pigeons, Rescorla (2004; Experiments 1, 2, and 3) reported evidence of superconditioning in designs similar to that of Pearce and Redhead (1995), but he used a weaker US in the superconditioning phase than that in the previous conditioned inhibition training phase (i.e., X++/BX− followed by BX+, where ++ represents strong US and + represents moderate US). In Rescorla's Experiment 4, he also found that A++/AB− training followed by BX+/Y+ training resulted in stronger responding to X than to Y. On the face of it, this is good evidence of superconditioning. However, throughout all of his experiments, Rescorla used an immediate presentation of the grain hopper as the stronger US and a 5-s delayed presentation of it preceded by 5-s presentation of another stimulus as the weaker US. That is, the USs used in both conditioned inhibition training and superconditioning phases were physically identical but differed in the temporal relationship to the CS (i.e., delayed conditioning procedure or a trace conditioning procedure with a gap filler). Moreover, in Experiment 4, the stimulus that was presented before the US in the superconditioning phase also served as the training excitor during inhibition training (i.e., A+/AB− followed by BX → A+/Y → A+). Taking into consideration that stimulus A had already been trained with the US prior to the superconditioning phase, this procedure leaves open the possibility of A blocking both X and Y. If this occurred, this potential blocking effect might have been decreased when A was presented with B as the result of A+/AB− discrimination training in the previous phase. Thus, there is a possibility that presentation of B did not enhance responding to X but decreased the ability of A to block X more than Y. Thus, the complexity of Rescorla's procedures in these experiments prevents interpretation of his result as clear evidence of superconditioning.

As reviewed above, there are many lines of evidence of superconditioning since Navarro et al. (1989) pointed out the problem concerning the superconditioning phenomenon. There seems to be good reason to believe that the phenomenon of superconditioning really exists, especially taking into consideration the results of Williams and McDevitt (2002) and Rescorla (2004), despite the minor problems with their procedures that we described. The first purpose of the present series of experiments was to seek unambiguous evidence of genuine superconditioning in a well controlled preparation, avoiding the procedural problems that we pointed out in the studies of Rescorla and of Williams and McDevitt. Thus, we conducted a series of experiments designed to produce a superconditioning effect relative to the two previously mentioned control groups and without using a partial reinforcement procedure. The second purpose of the present series was to investigate the associative mechanism underlying any observed superconditioning effect.

Currently there are two theoretical accounts of superconditioning. Most contemporary learning theories explain superconditioning as enhanced learning of a target CS–US association caused by the presentation of a conditioned inhibitor in compound with the CS at the time of training. For example, the Rescorla-Wagner (1972) model states that the increment in associative strength between a CS and a US on a given trial is a positive function of the discrepancy between the asymptotic associative strength supported by the US and the total associative strength of all CSs that are present on that trial. Thus, when the pairing of a novel CS and a US is conducted in the presence of a conditioned inhibitor, the increment in associative strength between the CS and the US is larger than when the pairing is conducted with the CS alone because the total associative strength of the CS and the conditioned inhibitor is smaller than the associative strength of the CS alone. In the framework of this model, a CS reinforced in compound with a

conditioned inhibitor acquires associative strength more quickly than does an elementally trained CS.

The extended comparator hypothesis, recently proposed by Denniston, Savastano, and Miller (2001), explains superconditioning in quite a different way. In the framework of the original comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985; see upper right part of Figure 1), conditioned responding to the target cue is determined at the time of testing by the interaction of three associations, which are formed at the time of training. The first association is between the target CS and the US (Link 1), the second association is between the target CS and another stimulus (i.e., comparator stimulus) that is associated with the target CS (Link 2), and the third of these associations is between the comparator stimulus and the US (Link 3). At test, conditioned responding to the target CS is assumed to reflect a comparison of the US representations directly and indirectly activated by the target CS. Excitatory responding to the target CS is positively correlated with the strength of the direct activation of the US representation (Link 1) and negatively correlated with the strength of the indirect activation of the US representation (the product of the strengths of Links 2 and 3). All cues maintaining a within-compound association with either the target CS or with an associate of the target CS act conjointly as comparator stimuli (Figure 1 depicts only one among what might be many comparator stimuli). It is noteworthy that, in the comparator framework, cue-competition phenomena are explained as the behavioral consequence of an interaction among different associations at the time of testing.

Furthermore, in the framework of the extended comparator hypothesis of Denniston et al. (2001), not only is Link 1 modulated by Links 2 and 3, but further comparator processes are presumed to modulate both Links 2 and 3 (see Figure 1). Thus, the effective strength of the target CS−first-order comparator stimulus associ-

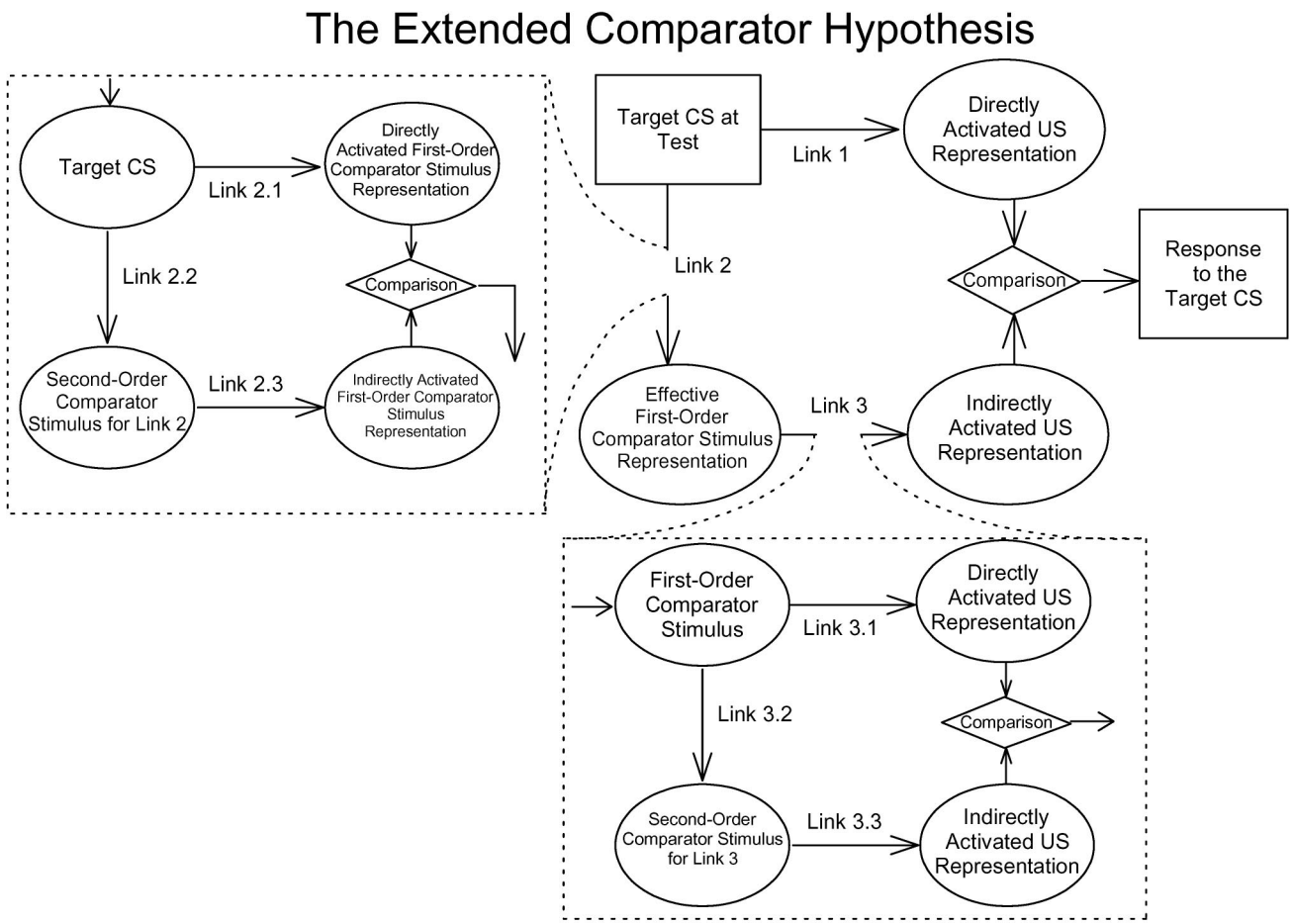

Figure 1. The extended comparator hypothesis (Denniston, Savastano, & Miller, 2001). Ovals represent stimulus representations, and rectangles represent the physical test stimulus and response. Conditioned responding to the target conditioned stimulus (CS) is determined by both the directly and the indirectly activated unconditioned stimulus (US) representation at the time of testing: Direct activation of the US representation (Link 1) is positively correlated, and indirect activation of the US representation (the product of Links 2 and 3) is negatively correlated with the magnitude of conditioned responding. In the original comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985), responding to the target CS is down-modulated only by the absolute strength of Links 2 and 3, whereas in the extended comparator hypothesis, the effectiveness of each of these two comparator links is potentially influenced by its own comparator processes.

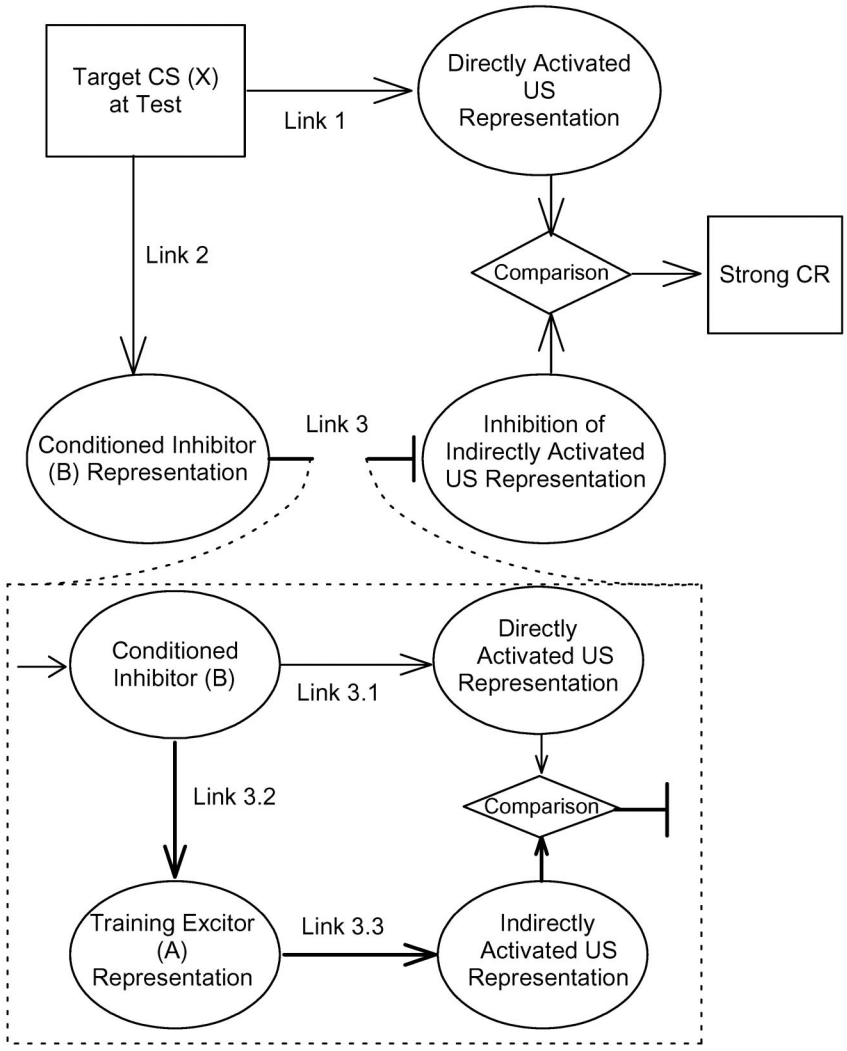

Figure 2. An extended comparator hypothesis account of superconditioning. Ovals represent stimulus representation, and rectangles represent the physical test stimulus and response. Arrows represent excitatory (positive) associative effects. Lines that end with a vertical bar represent inhibitory (negative) associative effects. Thickness of each line represents strength of associative effect. The higher order comparator effect on Link 2 is omitted because it is negligible in this situation. CS = conditioned stimulus; US = unconditioned stimulus; CR = conditioned response.

ation is determined not only by the absolute strength of that association (Link 2.1) but also by the product of the strength of the association between the target cue and any other potential comparator stimulus (second-order comparator stimulus for Link 2; Link 2.2) and the strength of the association between the other comparator stimulus and the first-order comparator stimulus (Link 2.3). In a similar manner, the effective strength of the association between the first-order comparator stimulus and the US association is determined not only by the absolute strength of that association (Link 3.1) but also by the product of the strength of the association between the first-order comparator stimulus and its own comparator stimulus (second-order comparator stimulus for Link 3; Link 3.2) and the strength of the association between the second-order comparator stimulus and the US (Link 3.3).[2]

The phenomenon of superconditioning is explained by the extended comparator hypothesis as a result of higher order comparator effects (see Figure 2). In the case of superconditioning (i.e., A+/AB− followed by BX+), strong associations between A and the US and between A and B and a weak (or no) association

[2] This comparator process presumably summates over all possible first-order comparator stimuli and, for reiterations in which a stimulus is other than a first-order comparator stimulus, it must be considered as a potential second-order comparator stimulus. Effectiveness of higher order comparator stimuli in modulating responding to the target cue is hypothesized to be reduced through a damping factor that increases with the order of the comparator process.

between B and the US should be formed as the result of Phase 1 training. In Phase 2, moderate associations between X and the US, between X and B, and between B and the US should be formed. When X is presented at test, responding to X is proportional to the strength of the direct activation of the US, which is determined by the strength of the X-US association (Link 1) and inversely related to the strength of indirect activation of the US, which is determined by the product of the strength of association between X and B (Link 2) and between B and the US (Link 3). Here, the effective strength of the association between X and B (Link 2) should be great because there is no effective comparator stimulus for X other than B. However, the strength of the association between B and the US (Link 3) should be strongly down-modulated (perhaps down to effectively negative values) because B should have a strong association with the training excitor A (Link 3.2), which in turn should have a strong association with the US (Link 3.3). If the product of Links 3.2 and 3.3 are greater than the strength of Link 3.1, then responding to X should be up-modulated, resulting in behavior indicative of superconditioned responding to X.

The main difference between the explanation provided by the extended comparator hypothesis and that provided by the other models such as the Rescorla-Wagner model is that the extended comparator hypothesis views superconditioning as enhanced performance, whereas the other models view it as enhanced acquisition. The most obvious consequence of this difference concerns the impact of a posttraining change in the associative status of the inhibition training excitor on superconditioning. The extended comparator hypothesis predicts a loss of superconditioning if the inhibition training excitor (A) is extinguished after the establishment of superconditioning because a decrement of the training excitor-US association should cause a loss of the inhibitory potential of the conditioned inhibitor (B) and consequently cause a loss of superconditioning by the target CS (X). The acquisition-focused models of conditioned behavior do not predict this loss of superconditioning because they view superconditioning as resulting from a stronger association between the target CS and the US, which was already established prior to the posttraining extinction of A. According to these models, there is no reason why manipulations performed on the inhibition training excitor after the establishment of superconditioning should alter the associative status of the target CS. In this series of experiments, we investigated the mechanism of superconditioning by assessing the effectiveness of posttraining extinction of the training excitor.

Three experiments were conducted in a conditioned taste aversion preparation. Experiment 1 sought empirical evidence of genuine superconditioning. Experiment 2 investigated the effectiveness of the conditioned inhibition treatment used in Experiment 1 and the effect of posttraining extinction of the training excitor on conditioned inhibition. Experiment 3 examined the effect of posttraining extinction of the training excitor on the superconditioning effect. The two studies directly concerning superconditioning (Experiments 1 and 3) were embedded within sensory preconditioning paradigms rather than in first-order conditioning paradigms. The reason we used a sensory preconditioning preparation is that some posttraining manipulations, specifically those that might cause a decrement in responding to a target CS, are not as effective outside of a sensory preconditioning paradigm (e.g., Denniston, Miller, & Matute, 1996; Miller & Matute, 1996b; Oberling, Bristol, Matute, & Miller, 2000). Thus, at least Experiment 3 had to be embedded within a sensory preconditioning paradigm. To maintain consistency throughout the series, Experiment 1 was also conducted within sensory preconditioning. On the one hand, the use of sensory preconditioning potentially limits the generality of the results of this research to instances in which the outcomes are of low biological significance (see Miller & Matute, 1996a, for a discussion). On the other hand, most learning, particularly by humans, involves outcomes of low biological significance; thus, a demonstration of superconditioning effect between neutral stimuli is important, and as far as we know, this is the first examination of the superconditioning effect in a sensory preconditioning paradigm.

We should point out, however, a theoretical problem introduced by our use of sensory preconditioning. Although we think that sensory preconditioning is necessary to investigate with high sensitivity the effect of posttraining manipulations and contrast predictions from different models, most learning theories, including the Rescorla-Wagner model and the extended comparator hypothesis, cannot explain the basic sensory preconditioning effect. When multiple cues are trained in compound, the response potential of one cue is not always inversely related to the response potential of the other cue as is the case in cue-competition phenomena (a relationship that we call *negative mediation*). Instead, the response potentials can be positively correlated (a relationship that we call *positive mediation)*. For example, feature-negative discrimination training (i.e., A+/AX−) can result in either conditioned inhibition to X (i.e., negative mediation of responding to X by A) or excitatory second-order conditioning or sensory preconditioning to X (i.e., positive mediation of responding to X by A). However, most learning theories are designed to explain only negative mediation (i.e., competition) among multiple stimuli trained together, despite overwhelming empirical evidence for the occurrence of both positive and negative cue mediation. This means that associative theories might be able to explain how neutral stimuli associatively interact with each other with respect to an outcome within a sensory preconditioning paradigm but are not able to explain why the sensory preconditioning effect itself occurs. This problem notwithstanding, our use of sensory preconditioning here is consistent with past efforts in which higher order conditioning procedures such as sensory preconditioning and second-order conditioning have proven to be useful tools in investigating the nature of associative learning phenomena (e.g., Rescorla, 1980).

Experiment 1

The purpose of Experiment 1 was to seek evidence of genuine superconditioning. As already noted, the experimental design was embedded within a sensory preconditioning paradigm using a conditioned taste aversion preparation. Several recent studies using flavor cues have shown that alternating exposures to two compound flavors sharing a common element results in inhibitory learning between the two unique elements (e.g., Artigas, Chamizo, & Peris, 2001; Dwyer, Bennett, & Mackintosh, 2001; Dwyer & Mackintosh, 2002; Espinet, Iraola, Bennett, & Mackintosh, 1995; see also Leonard & Hall, 1999, for a replication with audiovisual stimuli, and Artigas et al., for a replication with human subjects). Theoretically, this can be viewed as being similar to a Pavlovian conditioned inhibition design (e.g., A+/AB−), with a surrogate

outcome substituted for the US (e.g., A-S/AB-no S). In our experiments, we took advantage of this phenomenon to produce reciprocal conditioned inhibition between neutral stimuli in a sensory preconditioning paradigm.

The design of Experiment 1 is summarized in Table 1. In Phase 1, the subjects in Group Superconditioning (SC) received alternating exposures to two compound flavors that shared a common element, AB and AS, which were expected to produce inhibitory learning between the two unique elements, B and S. Flavor S was treated as a surrogate outcome in the sensory preconditioning paradigm, which was later paired with a lithium chloride (LiCl) US. B was treated as a conditioned inhibitor for the outcome (S) in the remaining part of the experiment. Following inhibitory training, the subjects in Group SC received pairings of a target cue (X) and the surrogate outcome (S) in the presence of the conditioned inhibitor (B; i.e., BX-S); this was expected to produce superconditioning of the X-S association. Subjects in Group Elemental-Acquisition Control (Acq-Ctrl) received the same manipulation as Group SC in Phase 1 followed by X-S pairings without stimulus B in Phase 2 (i.e., X-S). Subjects in Group Overshadowing Control (OV-Ctrl) received alternating exposures to two compounds without a common element (AS and DB), which was not expected to produce inhibitory learning between B and S, and then received the same procedure as Group SC. In these three groups, conditioned taste aversion to X was tested after S was paired with the LiCl US.

Method

Subjects

The subjects were 18 male and 18 female, experimentally naïve, Sprague-Dawley descended rats obtained from our own breeding colony. Their body weights ranged from 302 g to 358 g for males and from 201 g to 254 g for females. Subjects were individually housed in stainless steel, wire mesh cages in a vivarium maintained on a 16-hr light, 8-hr dark cycle. All experimental manipulations were conducted near the middle of the 16-hr light period. Subjects were allowed free access to food in the home cage. All rats were handled for 30 s three times per week from weaning until the end of the study. Subjects were randomly assigned to one of the three groups described above (*n*s = 12), counterbalanced for sex and body weight prior to the initiation of the study.

Table 1
Design Summary of Experiment 1

Group	Phase 1	Phase 2	Phase 3	Test
SC	AS/AB	BXS	S → US	X
OV-Ctrl	AS/DB	BXS	S → US	X
Acq-Ctrl	AS/AB	XS	S → US	X

Note. A and D were an almond odor and a banana odor, counterbalanced. S was a sodium saccharin solution. B was a sodium chloride solution. X was a mint odor. The unconditioned stimulus (US) was a 13 ml/kg body weight intraperitoneal injection of 0.15M lithium chloride. Arrows represent intraperitoneal injection of the US immediately following 5-min access to the solution. Slashes represent interspersed treatments. In Phases 1 and 2, odors and tastes were presented as compound solutions for 15 min. In Phase 1, two sessions using different solutions were conducted on each day with the two sessions separated by 5 hr. SC = superconditioning; OV-Ctrl = overshadowing control; Acq-Ctrl = elemental-acquisition control.

Apparatus

All experimental manipulations were conducted in the rats' home cages. Each solution was provided at room temperature in graduated plastic bottles, fitted with a stainless steel lick tube with a 0.2-cm opening. The amount of each liquid ingested was indexed by the difference in the levels of liquid measured before and after each presentation.

As shown in Table 1, five different flavor stimuli, three odors and two tastes, were used in this study: A 1.0% (vol/vol) almond odor solution (Pure Almond Extract, McCormick & Co.) and a 1.0% (vol/vol) banana odor solution (Artificial Ripe Banana #112, Virginia Dare Extract Co.) served as A and D, counterbalanced within groups. A 0.5% (vol/vol) mint odor solution (Mint Extract, McCormick & Co.) served as X. A 0.6% (wt/vol) salt solution served as B, and a 0.15% (wt/vol) sodium saccharin solution served as S. The US was a 13 ml/kg of body weight intraperitoneal injection of 0.15M LiCl.

Procedure

A progressive water deprivation schedule was imposed over 4 days before the beginning of the experiment until water availability was limited to 30 min per day.

Pretraining. On Days 1–4, all subjects experienced two 15-min pretraining sessions per day. Water was presented in the graduated plastic bottle, and consumption by each subject was recorded. The first of these sessions started at approximately 11:00 a.m. and the second at approximately 4:00 p.m. This treatment acclimated subjects to drinking from the lick tubes at the daily treatment times.

Phase 1. On Days 5–16, all subjects experienced two 15-min Phase 1 sessions per day. These two sessions were conducted in the same manner as in the pretraining phase, except flavored solutions were presented. All subjects were provided with two different taste−flavor compounds on each day. Subjects in Groups SC and Acq-Ctrl were given the AS-compound solution in one session and the AB-compound solution in the other session. Subjects in Group OV-Ctrl were given the AS-compound solution in one session and the DB-compound solution in the other session. The order of presentation of these compounds was counterbalanced within groups. Consumption during each session was recorded.

Phase 2. On Days 17–19, all subjects experienced one 15-min Phase 2 session per day at approximately 1:00 p.m. Subjects in Groups SC and OV-Ctrl were given the BXS-compound solution, and subjects in Group Acq-Ctrl were given the XS-compound solution. Consumption during each session was recorded. All subjects received an additional 15 min of access to water soon after the experimental session.

Phase 3. On Day 20, all subjects experienced Phase 3 treatment. The session started at approximately 3:30 p.m. on that day. All subjects received a 5-min presentation of the S solution, followed immediately by an intraperitoneal injection of LiCl. The amount of the S solution was limited to 5 ml, which was completely consumed by all subjects during the 5-min exposure period. All subjects received an additional 25 min of access to water approximately 1 hr after this treatment.

Recovery. On Days 21 and 22, all subjects experienced one 15-min recovery session with water per day, starting at approximately 1:00 p.m. Consumption during these sessions was recorded. All subjects received an additional 15 min of access to water soon after the session.

Test. On Day 23, a test session was conducted. The X solution was presented to all subjects for 15 min, and consumption was recorded. For all statistical analyses, the criterion for rejection of the null hypothesis was .05.

Results and Discussion

The means of the AS-compound solution consumption across all sessions in Phase 1 were 14.7, 15.0, and 14.5 ml in Groups SC,

OV-Ctrl, and Acq-Ctrl, respectively, and those of the AB- or DB-compound solution were 15.1, 14.4, and 14.7 ml in Groups SC, OV-Ctrl, and Acq-Ctrl, respectively. A 2 (solution: AS vs. AB or DB) × 3 (group) analysis of variance (ANOVA) revealed no significant effect or interaction (Fs < 1). The means of the BXS- or XS-compound solution consumptions across all sessions in Phase 2 were 11.6, 12.3, and 10.4 ml in Groups SC, OV-Ctrl, and Acq-Ctrl, respectively. A one-way ANOVA revealed no significant effect of group, $F(2, 33) = 1.41$, *ns*. In Phase 3, all the subjects consumed all 5 ml of the S solution within 5 min.

Mean consumption of the test solution in each group in the test phase is depicted in Figure 3. Group SC showed greater suppression of consumption than the other two groups, suggesting a superconditioning effect. A one-way ANOVA conducted on these data revealed a difference among the groups, $F(2, 33) = 13.05$. Planned comparisons using the error term of this ANOVA found that the mean consumption in Group SC was lower than in Groups OV-Ctrl and Acq-Ctrl, Fs(1, 33) = 19.99 and 19.13, respectively. These results clearly show that the superconditioning effect was observed in this study compared with the two superconditioning control groups, OV-Ctrl and Acq-Ctrl. There was no significant difference between Groups OV-Ctrl and Acq-Ctrl ($F < 1$), suggesting the absence of overshadowing in Group OV-Ctrl. It seems that this is because the concentration of B used in this experiment accorded it a relatively low salience.

The results of Experiment 1 provide evidence of superconditioning, which excludes the alternative explanations possible in prior studies. Enhancement of responding was observed relative to two appropriate controls without using a partial-reinforcement procedure. However, Experiment 1 provided no direct evidence of inhibitory learning between B and S as the result of alternating exposures to AB and AS. This leaves the results of Experiment 1 open to a different alternative explanation. If AB and AS pairings in Phase 1 caused excitation rather than conditioned inhibition between B and S (i.e., second-order conditioning between neutral stimuli), and that excitation between B and S was positively rather than negatively mediated to target cue X (i.e., potentiation of the X-S association by an X-B-A-S higher order association), then the results of Experiment 1 could be explained without recourse to the construct of superconditioning. This possibility is far less likely than the possibility of superconditioning because third-order conditioning between neutral stimuli is assumed in this explanation, and as far as we know, no such empirical evidence has ever been reported. Nevertheless, the possibility still makes it important for us to seek evidence of conditioned inhibition between neutral stimuli resulting from our procedure in Experiment 1. This point was addressed in the next experiment.

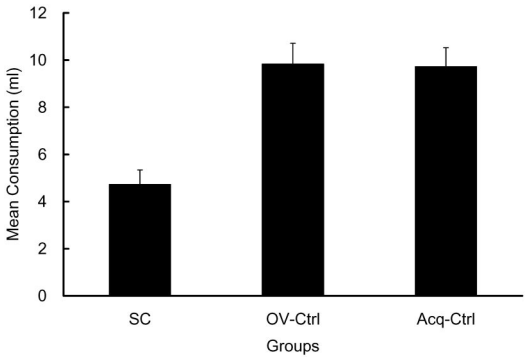

Figure 3. Mean consumption of the test solution in Experiment 1. In all groups, the X solution (mint) was presented at test. In Phase 1, Groups SC (superconditioning) and Acq-Ctrl (elemental-acquisition control) received alternating exposures of two compound flavors, AS and AB, and Group OV-Ctrl (overshadowing control) received alternating exposures of AS and DB. A and D were an almond odor or a banana odor (counterbalanced), S was saccharin, and B was a salt solution. In Phase 2, Groups SC and OV-Ctrl received presentation of the BXS compound, and Group Acq-Ctrl received presentation of the XS solution. Subjects were tested after first-order conditioning of S with an intraperitoneal injection of lithium chloride as an unconditioned stimulus. Lower consumption represents greater conditioned aversion. Error bars represent standard errors of the mean.

Experiment 2

The primary purpose of Experiment 2 was to complement the results of Experiment 1 by assessing inhibitory learning between the two unique elements (B and S), which theoretically should have been established through the alternating presentations of the two compound stimuli (AS and AB) that shared a common element (A) in Phase 1. To achieve this, Experiment 2 used a first-order conditioning procedure (see Table 2) rather than the sensory preconditioning procedure of Experiment 1. This was done because pilot data collected in our laboratory showed that summation- and retardation-test evidence of conditioned inhibition was difficult to obtain within sensory preconditioning. However, published studies (e.g., Artigas et al., 2001; Espinet et al., 1995) have shown that inhibitory learning between two neutral stimuli results in first-order conditioned inhibition to one unique element when the other unique element was conditioned with the US (i.e., the Espinet effect). In this experiment, we used exactly the same parameters that were used in Phase 1 of Experiment 1 and tried to evidence conditioned inhibition by replicating the Espinet effect. The Phase 2 treatment of Experiment 1 was omitted because we were not pursuing superconditioning in this experiment.

We should point out, however, that the use of the Espinet effect raises the same problem as the use of sensory preconditioning. Most contemporary learning theories can explain why alternating exposures of two compound stimuli that share a common element results in inhibitory learning between two neutral stimuli, whereas they cannot explain why the Espinet effect itself occurs, that is, why first-order excitatory conditioning of a unique element results in first-order conditioned inhibition to the other unique element (but see McLaren & Mackintosh, 2000, for a model that can account for the Espinet effect). Nevertheless, because occurrence of the Espinet effect is now taken as evidence of inhibitory learning between neutral stimuli, we made use of this phenomenon in Experiment 2.

The secondary purpose of Experiment 2 was to assess a potential mechanism underlying inhibitory learning between two neutral stimuli. Several prior studies of first-order conditioned inhibition have revealed that, following Pavlovian conditioned inhibition training (i.e., A+/AB−), extinction of the inhibition training excitor (A) can cause a loss of conditioned inhibition (e.g., Hallam, Matzel, Sloat, & Miller, 1990; Lysle & Fowler, 1985). The alternating exposures to two compounds with a common element (e.g., the alternating AB/AS exposures used in Phase 1 of Experiment 1)

Table 2
Design Summary of Experiment 2

Group	Phase 1	Phase 2	Phase 3	Phase 4	Summation test	Retardation test
Cl	AS/AB	Water	S → US	Y → US′	Y	B → US′ B
					BY	— —
Ctrl	AS/DB	Water	S → US	Y → US′	Y	B → US′ B
					BY	— —
Cl A−	AS/AB	A	S → US	Y → US′	Y	B → US′ B
					BY	— —

Note. A and D were an almond odor and a banana odor, counterbalanced. S was a sodium saccharin solution. B was a sodium chloride solution. Y was a mint odor. The unconditioned stimulus (US) and the US′ were intraperitoneal injections of 0.15M lithium chloride of 13 ml/kg and 10 ml/kg body weight, respectively. Arrows represent intraperitoneal injection immediately following 5-min access to the solution. Slashes represents interspersed treatment. Dashes represent no experimental manipulation in that phase. In Phase 1, odors and tastes were presented as compound solutions for 15 min. In Phase 2, three identical sessions of 10 min were conducted in 1 day with three sessions separated by 4-hr intervals. CI = conditioned inhibition; Ctrl = control.

constitute a Pavlovian conditioned inhibition procedure, with the US replaced with a more innocuous unique stimulus element (S). Our interest was whether the same manipulation, posttraining extinction of the inhibition training excitor (A), would cause loss of the expression of inhibitory learning between two neutral stimuli.

In Phase 1, two groups (Conditioned Inhibition [CI] and Conditioned Inhibition A− [CI A−]) received alternating presentations of AS and AB, which were procedurally identical to those received by Groups SC and Acq-Ctrl in Experiment 1. An additional group (Control [Ctrl]) received alternating presentations of AS and DB, which were procedurally identical to those received by Group OV-Ctrl in Experiment 1. In Phase 2, Groups CI and Ctrl received presentations of water, and Group CI A− received extinction presentations of the A solution alone. Then first-order conditioning to S and a novel CS (Y) was conducted in Phases 3 and 4, respectively; the former was intended to induce conditioned inhibition to B, and the latter was intended to create a transfer excitor for summation testing of conditioned inhibition. In the summation-test phase, subjects in each group were divided into two subgroups and tested either with Y alone or with the BY compound. After this summation test session, retardation-of-acquisition testing was conducted with subjects in those subgroups that had been tested with Y alone during the summation-test phase (i.e., the subjects that were not exposed to B during the summation test). Specifically, first-order conditioning to B was conducted, and conditioned suppression of B was measured. Our interests were whether subjects in Group CI would show inhibitory behavioral control by B (i.e., less suppression to B) in comparison to those in Group Ctrl and whether A-alone presentations in Phase 2 in Group CI A− would degrade the effect of conditioned inhibition to B in comparison to Group CI.

Method

Subjects

The subjects were 36 male and 36 female, experimentally naïve, Sprague-Dawley descended rats obtained from our own breeding colony. Their body weights ranged from 164 g to 274 g for males and from 138 g to 182 g for females. Subjects were reared and maintained in the same manner as in Experiment 1. Subjects were randomly assigned to one of the three groups (*n*s = 24), counterbalanced for sex and body weight before the initiation of the study.

Apparatus

All experimental manipulations were conducted in the home cages as in Experiment 1. Presentation and measurement of the solutions was conducted in the same manner as in Experiment 1. As shown in Table 2, five different cues were used in this study. Cues A, B, D, and S were the same as those used in Experiment 1. The 0.5% (vol/vol) mint flavor solution, which served as stimulus X in Experiment 1, was stimulus Y in this study. This experiment used two doses of 0.15M LiCl, 13 ml/kg and 10 ml/kg of body weight.

Procedure

As in Experiment 1, a progressive water deprivation schedule was imposed 4 days before the beginning of the experiment until water availability was limited to 30 min per day.

Pretraining. On Days 1–4, all subjects experienced two 15-min pretraining sessions per day in the same manner as in Experiment 1.

Phase 1. On Days 5–16, all subjects experienced two 15-min sessions per day in the same manner as in Experiment 1. Subjects in Groups CI and CI A− received the AS-compound solution in one session and the AB-compound solution in the other session, and subjects in Group Ctrl received the AS-compound solution in one session and the DB-compound solution in the other session.

Phase 2. On Days 17–23, all subjects experienced three 10-min Phase 2 sessions per day. The three sessions started at approximately 9:00 a.m., 1:00 p.m., and 5:00 p.m. Subjects in Groups CI and Ctrl received water during these sessions, and subjects in Group CI A− received the A solution. The consumption of solution during each session was recorded.

Phase 3 and recovery. On Day 24, subjects experienced Phase 3 treatment at approximately 1:00 p.m. All subjects received one 5-ml presentation of the S solution followed by an intraperitoneal injection of LiCl in the same manner as in Phase 3 of Experiment 1. The 13 ml/kg body weight injection of 0.15M LiCl was administered immediately after the S presentation. One hour after the treatment, the subjects received additional water presentation for 25 min. On Days 25 and 26, all subjects experienced one 15-min recovery session plus 15 min of additional water per day in the same manner as in Experiment 1.

Phase 4 and recovery. On Day 27, all subjects experienced Phase 4 treatment. Subjects received two sessions, one in the morning (at approximately 9:00 a.m.) and the other in the afternoon (at approximately 2:00 p.m.). In the morning session, 5 ml of the Y solution was presented until

the subjects consumed all of it. This nonreinforced presentation of the Y solution in the morning session was intended to minimize neophobic responding to Y in the afternoon session. In the afternoon session, 5 ml of the Y solution was presented for 5 min and was immediately followed by a 10 ml/kg body weight intraperitoneal injection of 0.15M LiCl. A lower dose of LiCl was used at this time to avoid complete suppression to Y during the subsequent summation test and, thus, to allow for the detection of conditioned inhibition. Water was presented for 20 min 1 hr after the afternoon experimental session. On Days 28–30, all subjects experienced one 15-min recovery session per day in the same manner as in Experiment 1.

Summation test. On Day 31, a summation test of conditioned inhibition was conducted. The test solution was presented to all subjects for 15 min, and consumption was recorded. Half of the subjects in each group were tested with the Y solution (i.e., the transfer excitor), and the other half were tested with the BY-compound solution. Water was made available for 15 min after the session.

Retardation-of-acquisition test. In this phase, only the subjects that had received the Y solution on Day 31 were used. On Day 32, each subject received 5 min of access to 5 ml of the B solution, which was immediately followed by a 10 ml/kg body weight intraperitoneal injection of 0.15M LiCl. The session was conducted at approximately 1:00 p.m. Water was presented for 25 min 1 hr later. On Days 33 and 34, subjects received one recovery session per day in the same manner as in Experiment 1. On Day 35, two sessions were conducted, one in the morning (at approximately 10:00 a.m.) and the other in the afternoon (at approximately 3:00 p.m.). In the morning session, the B solution was presented to the subjects in each group for 15 min, and the consumption was recorded to assess potential retardation of behavior control arising from the Day 32 B-LiCl pairing (Test 1). To assess retardation across multiple training trials, in the afternoon session, 5 ml of the B solution was presented for 5 min and again immediately followed by a 10 ml/kg body weight intraperitoneal injection of 0.15M LiCl. One hour following the afternoon session, the subjects were given water for 10 min. On Days 36 and 37, one recovery session was conducted daily in the same manner as in Experiment 1. On Day 38, testing of the B solution was again conducted in the same manner as in the morning session on Day 35 (Test 2). Because of a procedural mistake made by the experimenter in Phase 3, data from 6 subjects, 1 from each subgroup, were lost.

Results and Discussion

The means of the AS solution consumption across all sessions in Phase 1 were 12.9, 12.7, and 13.6 ml, in Groups CI, Ctrl, and CI A−, respectively, and those of the AB or DB solution were 12.9, 12.8, and 13.5 ml in Groups CI, Ctrl, and CI A−, respectively. A 2 (solution) × 3 (group) ANOVA revealed that no main effect or interaction was significant (Fs < 1). The means of the solution consumption across all sessions in Phase 2 were 8.2, 8.5, and 8.5 ml in Groups CI, Ctrl, and CI A−, respectively. These data were analyzed with a one-way ANOVA, which revealed no significant effect of group ($F < 1$). In Phase 3, all the subjects consumed 5 ml of the S solution within 5 min. In the afternoon session of Phase 4, all the subjects consumed 5 ml of the Y solution within 5 min.

Group mean consumption of the Y-alone and BY-compound solutions in the summation-test phase is depicted in Figure 4. Subjects in all subgroups that were tested with the Y solution alone showed strong suppression of consumption, which confirms good conditioning to the Y solution. Among subjects tested with the BY-compound solution, Group CI showed greater consumption than did Groups Ctrl and CI A−. These results indicate that Group CI passed the summation test of conditioned inhibition but that

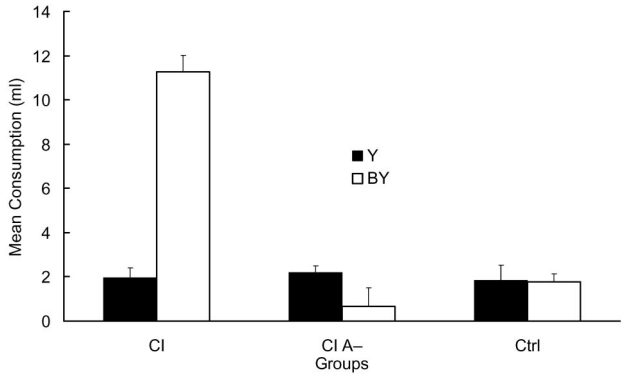

Figure 4. Mean consumption of the test solution in the summation-test phase of Experiment 2. In Phase 1, Groups CI (conditioned inhibition) and CI A− received alternating exposures of two compound flavors, AS and AB, and Group Ctrl (control) received AS and DB. A and D were an almond odor or a banana odor (counterbalanced), S was saccharin, and B was a salt solution. In Phase 2, Groups CI and Ctrl received presentations of water, and Group CI A− received presentations of the A solution. Subjects received first-order conditioning to Y (mint) following first-order conditioning of S with an intraperitoneal injection of lithium chloride as an unconditioned stimulus. Half of the subjects in each group were tested with Y and the other half with the BY compound. Lower consumption represents greater conditioned aversion. Error bars represent standard errors of the mean.

Group CI A− did not. A 3 (group) × 2 (stimulus: Y vs. BY) ANOVA revealed a main effect of group, $F(2, 60) = 41.50$, and stimulus, $F(1, 60) = 24.88$, and an interaction of Group × Stimulus, $F(2, 60) = 43.00$. Planned comparisons using the error term of this ANOVA revealed no significant difference in consumption of the Y solution among groups (Fs < 1), whereas the consumption of the BY-compound solution was greater in Group CI than in Groups CI A− and Ctrl, Fs(1, 60) = 139.50 and 112.28, respectively, which did not differ from each other, $F(1, 60) = 1.48$.

On both Days 32 and 35, all subjects consumed 5 ml of the B solution, which was presented immediately before the injections, within 5 min. The test data showing consumption of the B solution, which were obtained on the first session of Day 35 (Test 1) and on Day 38 (Test 2), are depicted in Figure 5. Group CI consumed more than did Group Ctrl, whereas Group CI A− showed a level of consumption similar to that of Group Ctrl. These results suggest that Group CI exhibited retarded stimulus control of behavior, whereas Group CI A− did not. The data were analyzed with a 3 (group) × 2 (day) ANOVA. This ANOVA revealed a main effect of group, $F(2, 30) = 6.54$, and day, $F(1, 30) = 23.95$. The interaction was not significant, $F(2, 30) = 2.18$. Planned comparisons using the error term from this ANOVA revealed that the consumption by Group CI was greater than that by Groups CI A− and Ctrl, Fs(1, 30) = 11.62 and 7.55, respectively, which did not differ significantly from each other ($F < 1$).

The results of Experiment 2 demonstrate the effectiveness of the parameters used in Phase 1 training, which were identical to those of Experiment 1, in establishing an inhibitory relationship between B and S. This suggests that Phase 1 training in Experiment 1 produced inhibitory learning between two neutral cues. Furthermore, Experiment 2 successfully replicated Espinet et al. (1995;

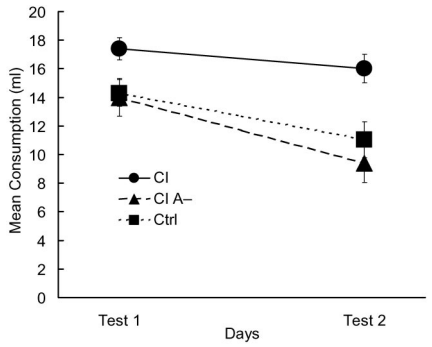

Figure 5. Mean consumption of B (salt) in the retardation-of-acquisition test phase in Experiment 2. In Phase 1, Groups CI (conditioned inhibition) and CI A− received alternating exposures of two compound flavors, AS and AB, and Group Ctrl (control) received AS and DB. A and D were an almond odor or a banana odor (counterbalanced), and S was a saccharin solution. In Phase 2, Groups CI and Ctrl received presentations of water, and Group CI A− received presentations of the A solution. Subjects received first-order conditioning to Y (mint) following first-order conditioning of S with an intraperitoneal injection of lithium chloride as an unconditioned stimulus. For those subjects initially tested on the Y solution alone, the consumption of B was measured twice, each following a first-order conditioning trial of B with an intraperitoneal injection of lithium chloride as an unconditioned stimulus. Lower consumption represents greater conditioned aversion. Error bars represent standard errors of the mean.

i.e., the Espinet effect) with a new control condition. We alternated exposures to two compounds sharing a common element in Group CI and alternated exposures to two compounds without a common element in Group Ctrl. Neither Espinet et al. (1995) nor its replications (e.g., Artigas et al., 2001; Leonard & Hall, 1999) used a control condition similar to ours. To demonstrate that alternating exposures to two compounds results in the formation of stronger inhibitory learning when these two compounds share a common element than when they do not, they compared the effectiveness of alternating exposures of two compound stimuli (e.g., AB/AS) to that of alternating exposures of elemental stimuli (e.g., B/S). The current replication with a new control condition illuminates the nature of this effect and confirms the assumption of prior studies.

It is noteworthy that Experiment 2 also assessed the effect of posttraining extinction of the common element in inhibitory learning between neutral cues. Massive extinction of A in Phase 2 caused a loss of the Espinet effect in Group CI A−, which was otherwise obtained in Group CI. This indicates that inhibitory learning between the neutral stimuli vanishes if their associations with the common element are extinguished, even if this extinction occurs after the establishment of inhibitory learning. The loss of conditioned inhibition caused by posttraining extinction of a training excitor in first-order conditioning situations has been reported in several prior studies (e.g., Hallam et al., 1990; Lysle & Fowler, 1985). Our finding suggests a common mechanism underlying both first-order conditioned inhibition and inhibitory learning between neutral cues. In Experiment 3, we examined the effect of posttraining extinction of a common element in superconditioning, which presumably relies on inhibitory learning between neutral cues.

Experiment 3

The purpose of Experiment 3 was to further investigate the mechanism underlying the superconditioning effect observed in Experiment 1. In Experiment 2, inhibitory learning between neutral cues was reduced when the common element shared between the two compounds was extinguished. Experiment 3 investigated whether posttraining extinction of the common element can also cause a retrospective loss of superconditioning.

Most associative theories explain superconditioning as an enhancement of associative acquisition at the time of training; that is, reinforcement of a target cue in compound with a conditioned inhibitor should result in the acquisition of a stronger association between the target cue and the outcome (e.g., Rescorla & Wagner, 1972; Wagner, 1981). In contrast, the extended comparator hypothesis (Denniston et al., 2001) explains this phenomenon as enhanced responding resulting from a higher order comparator process at the time of testing (see Figure 2). In this framework, the target cue elicits stronger responding as a result of a comparison with a conditioned inhibitor, which has an inhibitory potential due to down-modulation by its own comparator stimulus, the excitor from inhibition training (Links 3.2 and 3.3 in Figure 2). The extended comparator hypothesis generates a prediction different from that of the acquisition-enhancement account concerning the effectiveness of posttraining manipulations of the inhibition training excitor in a superconditioning paradigm. The prediction of most associative theories is that extinguishing the association between the training excitor and the US after the establishment of superconditioning should have no effect on responding to the target CS because the already acquired strong association between the target cue and the US should remain intact regardless of the posttraining extinction of the training excitor. In contrast, according to the extended comparator hypothesis, posttraining extinction of the training excitor should have a detrimental effect on responding to the target CS because the resulting decrement in effective associative strength between the training excitor and the US produces a loss of the inhibitory potential of the conditioned inhibitor, thereby eliminating its ability to support behavior indicative of superconditioning at the time of testing. Thus, examination of the effect of posttraining extinction of the training excitor on superconditioning could indicate whether superconditioning is better viewed as an enhancement of associative acquisition or performance.

The design of Experiment 3 is summarized in Table 3. All four groups, Superconditioning (SC), Superconditioning A− (SC A−), Superconditioning D− (SC D−), and Elemental-Acquisition Control (Acq-Ctrl), received interspersed exposures to three compounds in Phase 1; two of them were compounds with a common element (AB/AS), which was expected to result in an inhibitory relationship between B and S as demonstrated in Experiment 2. The other stimulus compound was DS, which was used to create a control group for extinction of A in Group SC A− during Phase 3. In Phase 2, the three superconditioning groups (SC, SC A−, and SC D−) received pairings of a target cue (X) and S in compound with B, while the other control group (Acq-Ctrl) received pairings of X and S without B. In Phase 3, the three superconditioning groups received different treatments. Group SC A− received presentations of A alone, which were expected to extinguish the associations between A and S and between A and B and, conse-

Table 3
Design Summary of Experiment 3

Group	Phase 1	Phase 2	Phase 3	Phase 4	Test
SC	AS/AB/DS	BXS	Water	S → US	X
SC A−	AS/AB/DS	BXS	A	S → US	X
SC D−	AS/AB/DS	BXS	D	S → US	X
Acq-Ctrl	AS/AB/DS	XS	Water	S → US	X

Note. A and D were an almond odor and a banana odor, counterbalanced. S was a sodium saccharin solution. B was a sodium chloride solution. X was a mint odor. The unconditioned stimulus was a 13 ml/kg body weight intraperitoneal injection of 0.15M lithium chloride. Arrows represent intraperitoneal injection immediately following 5-min access to the solution. Slashes represent interspersed treatment. Odors and tastes were presented as compound solutions for 10 min and 15 min in Phases 1 and 2, respectively. In Phase 1, three sessions featuring different solutions were conducted on each day separated by 4-hr intervals. In Phase 3, three identical 10-min sessions were conducted in a day separated by 4-hr intervals. SC = superconditioning; Acq-Ctrl = elemental-acquisition control.

quently, cause a loss of the inhibitory relationship between B and S (an effect observed in Experiment 2). Group SC D− received presentations of D alone, which were expected to cause extinction of the association between D and S without having any effect on the inhibitory potential of B or the excitatory potential of X. Lastly, Groups SC and Acq-Ctrl received equivalent presentations of water. The degree of aversion to the target cue was measured after first-order conditioning to S. Our main interest was whether the aversion exhibited by Group SC A− would differ from that in the other two superconditioning groups, SC and SC D−.

Method

Subjects

The subjects were 24 male and 24 female, experimentally naïve, Sprague-Dawley descended rats obtained from our own breeding colony. Their body weights ranged from 297 g to 372 g for males and from 205 g to 273 g for females. Subjects were maintained in the same manner as in Experiments 1 and 2. Subjects were randomly assigned to one of the four groups (ns = 12), counterbalanced for sex and body weight prior to the initiation of the study.

Apparatus

As in Experiments 1 and 2, all experimental manipulations were conducted in the home cages. The presentation and measurement of solutions were conducted in the same manner as in Experiments 1 and 2. As shown in Table 3, five different cues were used in this study: Three flavors (almond, banana, and mint) and two different tastes (salt and saccharin) were used in the same roles as in Experiment 1. The US was a 13 ml/kg body weight intraperitoneal injection of 0.15M LiCl.

Procedure

The subjects were water deprived as in Experiments 1 and 2.

Pretraining. On Days 1–4, all subjects experienced three 10-min pretraining sessions per day. Water was presented using graduated plastic bottles, and the consumption by each subject was recorded. The sessions started at approximately 9:00 a.m., 1:00 p.m., and 5:00 p.m.

Phase 1. On Days 5–16, all subjects experienced three 10-min Phase 1 sessions per day. These three sessions were conducted in the same manner as in the pretraining phase except for the solutions provided to the subjects. On each day, all subjects received three different taste−flavor compounds: AS, AB, and DS. The order of presentation of these three different compounds was counterbalanced within groups. Consumption during each session was recorded.

Phase 2. On Days 17–19, all subjects experienced one 15-min Phase 2 session per day. Each session started at approximately 1:00 p.m. Subjects in Groups SC, SC A−, and SC D− received access to the BXS-compound solution, while subjects in Group Acq-Ctrl received access to the XS-compound solution. The consumption during each session was recorded. All subjects received an additional 15 min of access to water after the experimental session.

Phase 3. On Days 20–26, all subjects experienced three 10-min Phase 3 sessions per day, conducted in the same manner as in the pretraining phase and Phase 1 except for the solutions provided to the subjects. Subjects in Groups SC and Acq-Ctrl received three daily water presentations. Subjects in Group SC A− received access to A three times, and subjects in Group SC D− received the same access to D. The consumption during each session was again recorded.

Phase 4 and recovery. On Day 27, all subjects experienced Phase 4 treatment. The session started at approximately 1:00 p.m. on that day. All subjects received access to the S solution followed immediately by an intraperitoneal injection of LiCl in the same manner as in Phase 3 in Experiment 1. On Days 28 and 29, all subjects experienced one recovery session with water per day in the same manner as in Experiment 1.

Test. On Day 30, the test session was conducted. The target stimulus X was presented alone to all subjects for 15 min, and consumption was measured in the same manner as in Experiment 1.

Results and Discussion

The means of the AS-compound solution consumption across all sessions in Phase 1 were 11.7, 12.1, 12.0, and 11.5 ml, those of the AB-compound solution were 9.8, 11.0, 10.3, and 8.9 ml, and those of the DS-compound solution were 11.4, 11.8, 12.0, and 11.1 ml in Groups SC, SC A−, SC D−, and Acq-Ctrl, respectively. A 3 (solution) × 4 (group) ANOVA revealed a main effect of solution, $F(2, 88) = 26.58$. Neither the main effect of group nor the interaction was significant, $F(3, 44) = 1.08$ and $F < 1$, respectively. Planned comparisons using the error term of this ANOVA revealed that consumptions of the AS- and DS-compound solutions were greater than that of the AB-compound solution, Fs(1, 44) = 43.00 and 27.95, respectively, but did not differ from each other ($F < 1$). Presumably the palatability of the saccharin solution was higher than that of the salt solution. The means of solution consumption across all sessions in Phase 2 were 12.2, 12.2, 11.5, and 11.5 ml in Groups SC, SC A−, SC D−, and Acq-Ctrl, respectively. These data were analyzed with a one-way ANOVA, which revealed no difference among groups ($F < 1$). The means of solution consumption across all sessions in Phase 3 were 9.6, 10.3, 9.7, and 8.8 ml in Groups SC, SC A−, SC D−, and Acq-Ctrl, respectively. These data were analyzed with a one-way ANOVA, which revealed no significant effect of group, $F(3, 44) = 1.12$. In Phase 4, all the subjects consumed the full 5 ml of the S solution within 5 min.

Mean consumption of the X solution by each group on Day 30 is depicted in Figure 6. Group SC showed stronger suppression than did Group Acq-Ctrl, suggesting that the central finding of superconditioning in Experiment 1 was successfully replicated. More important, Group SC A− showed weaker suppression than did the other two superconditioning groups, SC and SC D−,

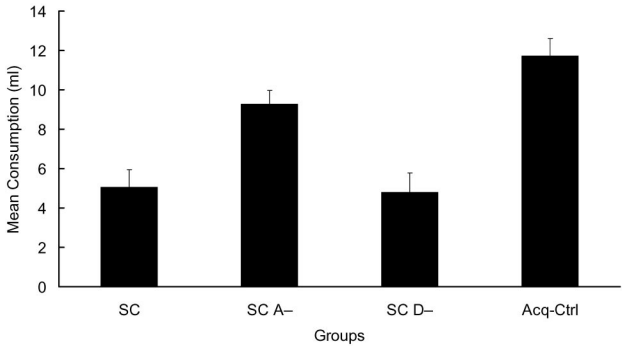

Figure 6. Mean consumption of X (mint) in the test phase in Experiment 3. In Phase 1, all groups received mixed exposures of three compound solutions, AS, AB, and DS. A and D were an almond odor or a banana odor (counterbalanced), S was saccharin, and B was a salt solution. In Phase 2, Groups SC (superconditioning), SC A−, and SC D− received presentations of BXS, and Group Acq-Ctrl (elemental-acquisition control) received presentations of the XS compound. In Phase 3, Groups SC and Acq-Ctrl received presentations of water, Group SC A− received presentations of A, and Group SC D− received presentations of D. Subjects were tested after a first-order conditioning trial of S with an intraperitoneal injection of lithium chloride as an unconditioned stimulus. Lower consumption represents greater conditioned aversion. Error bars represent standard errors of the mean.

suggesting that posttraining extinction of the training excitor, A, caused a loss of the superconditioning effect. Moreover, the weaker suppression by Group SC A− relative to Group SC D− demonstrates that the effect of the posttraining manipulation was stimulus specific.

A one-way ANOVA conducted on these data revealed a difference among groups, $F(3, 44) = 15.18$. Planned comparisons using the error term from this ANOVA revealed that the consumption in Groups SC and SC D− was less than that in Group Acq-Ctrl, Fs(1, 44) = 29.58 and 32.10, respectively, and also less than that in Group SC A−, Fs(1, 44) = 11.87 and 13.48, respectively. Additional planned comparisons revealed that the consumption of Group SC A− was marginally less than that in Group Acq-Ctrl, $F(1, 44) = 3.98$, $.05 < p < .06$. There was no significant difference between Groups SC and SC D− ($F < 1$).

The central finding in Experiment 3 was that the superconditioning effect is sensitive at the time of testing to the associative status of the training excitor that was used in conditioned inhibition training. Extinction of the training excitor, A, degraded responding to X in Group SC A− compared with Group SC. One cannot attribute this finding to a generalization of extinction from A to X because the third superconditioning group, SC D−, received an extinction treatment of another associate of S that was irrelevant to conditioned inhibition, D, but did not show any decrement in the superconditioning effect. This finding supports the prediction of the extended comparator hypothesis but is problematic for other models of learning that explain superconditioning as enhanced acquisition of the association between the target cue and the outcome at the time of target cue reinforcement. A detailed explanation of this theoretical conclusion is provided in the General Discussion section.

General Discussion

The first aim of this experimental series was to provide a demonstration of genuine superconditioning effect obtained under well controlled conditions. This aim was achieved: Experiment 1 found greater conditioned taste aversion in Group SC than in the two control groups, OV-Ctrl and Acq-Ctrl. Use of these two control groups refutes alternative explanations such as a failure of overshadowing (e.g., Navarro et al., 1989) or a result of potentiation (e.g., Clarke et al., 1979; Rusiniak et al., 1979). Furthermore, we avoided all of the problematic procedures that we discussed in the introduction. For example, we did not use a partial reinforcement procedure during training of the target CS (Phase 2), which was used in a prior study of superconditioning (Williams & McDevitt, 2002). Thus, we provided clear evidence of superconditioning. We have to point out, however, that our finding was achieved within a sensory preconditioning situation. On the one hand, this is an expansion of the phenomenon of superconditioning to a new situation; as far as we know, this is the first demonstration of super sensory preconditioning. On the other hand, this limits the generality of our finding to a sensory preconditioning situation. Although we conducted our experiments within a sensory preconditioning preparation to maximize the effects of posttraining extinction of the excitor used in inhibitory training, there is no apparent reason to suspect that the phenomenon of superconditioning is limited to sensory preconditioning situations.

One problem with Experiment 1 was that it did not provide evidence of inhibitory learning between neutral cues, upon which the superconditioning effect theoretically depends. Experiment 2 addressed this omission by using exactly the same parameters during Phase 1 as those that were used in Experiment 1. Experiment 2 showed that these parameters were sufficient to generate inhibitory learning between two neutral stimuli when assessed with a first-order conditioned excitor (i.e., the Espinet effect). Moreover, Experiment 2 found that behavior indicative of inhibition was attenuated when the common element shared by the two compounds was presented alone, extinguishing its association to the outcome, after the compound exposures. Several prior studies have found that the extinction of a training excitor used in Pavlovian conditioned inhibition (i.e., A+/AB− followed by A−) degrades the inhibitory potential of the conditioned inhibitor (e.g., Hallam et al., 1990; Lysle & Fowler, 1985). The consequences of extinguishing A in our Experiment 2 were similar to these previous observations if we regard the unique element, S, that is later paired with the US, as the analog of the US in Pavlovian conditioned inhibition training. Therefore, the present results suggest that a common mechanism underlies first-order conditioned inhibition and inhibitory learning between two neutral stimuli. Our present observation broadens the generality of the effect of this posttraining manipulation upon inhibitory relationships. Furthermore, Experiment 3 demonstrated that posttraining extinction of the training excitor not only weakened the inhibitory relationship between two neutral stimuli but also attenuated any superconditioning effect that it presumably supports.

Acquisition-focused theories of learning (e.g., Rescorla & Wagner, 1972; Wagner, 1981) explain superconditioning as a result of enhanced acquisition of an excitatory association. These models hypothesize that the acquisition of associative strength by a CS is a function of the effectiveness of the US and the associative status

of any other CSs present during training. Specifically, the magnitude of associative strength accruing to a CS on a learning trial is based on the discrepancy between the effectiveness of the US and the associative status of all CSs present during that trial. Pavlovian conditioned inhibition training (i.e., interspersed A+/AB− trials) results in the formation of a strong excitatory association between the training excitor (A) and the US because, during A+ trials, the US is present and other effective CSs are absent. The AB− trials result in the formation of negative (inhibitory) associative strength between the conditioned inhibitor (B) and the US because another effective CS, A, is present and the US is absent. And if another stimulus is reinforced in compound with a previously established conditioned inhibitor (i.e., superconditioning treatment: e.g., A+/AB− followed by BX+), associative acquisition to X is expected to be greater than if B were not inhibitory because the presence of the conditioned inhibitor increases the discrepancy between the experienced US and the total associative strength of the CSs present in that trial.

More generally, acquisition-focused models attribute cue-interaction phenomena (e.g., blocking, overshadowing, superconditioning, conditioned inhibition) to differential acquisition of associative strength during training. Responding at the time of testing is assumed to directly reflect the associative strength of the target stimulus. In the framework of traditional acquisition-focused theories, the associative strength of a CS changes only when the CS itself is presented. This makes it difficult for these models to explain the effects of posttraining associative manipulations of stimuli other than the target cue, such as the posttraining extinction of A on superconditioning in the present studies. However, some recent revisions of acquisition-focused theories (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994) overcame this difficulty of traditional models in explaining many of the effects of posttraining manipulations. In the framework of the revised acquisition-focused theories, the associative strength of a CS can change not only when the CS itself is physically presented but also when it is absent but expected on the basis of the presence of other stimuli that have an excitatory association with the absent CS. When a stimulus is physically presented, it is hypothesized to activate an indirect representation of each of its associates. These indirectly activated stimuli representations are assumed to change their associative strengths in the opposite direction to that of stimuli that are present. For example, in the case of posttraining extinction of the training excitor following Pavlovian conditioned inhibition training (i.e., A+/AB− followed by A−), which was evidenced in Experiment 2, Phase 1 training should result in the formation of excitatory associations between the training excitor A and the US and between A and the conditioned inhibitor B, as well as an inhibitory association between B and the US. In Phase 2, exposure of the inhibition training excitor A, which has excitatory associations with B and the US, presumably results in acquisition of an excitatory association between B and the US as well as in a weakening of the A-US and A-B excitatory associations. Thus, unlike traditional acquisition-focused theories, these revised acquisition-focused theories predict the results observed in Experiment 2.

The original comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985) provides an alternative account for cue-interaction phenomena. In the framework of the original comparator hypothesis, three associations, that between the target CS and the US (Link 1), that between the target CS and another CS (comparator stimulus; Link 2), and that between the comparator stimulus and the US (Link 3), are formed at the time of training, and responding to the target CS is determined by the comparison between the directly activated US representation (Link 1) and the indirectly activated US representation (the product of Links 2 and 3) at the time of testing (see upper right part of Figure 1). Because it emphasizes the importance of the associative status of the competing stimulus at the time of testing rather than during training, the comparator hypothesis can explain the effect of posttraining manipulations. For example, in the case of Experiment 2, exposure to the training excitor (A) alone should result in a weakening of Link 3 (A-US association) due to extinction. Thus, at the time of testing, the indirect activation of the US representation is impaired, resulting in a loss of inhibitory control by the conditioned inhibitor (B). However, the phenomenon of superconditioning is quite problematic for the original comparator hypothesis. In the framework of the comparator hypothesis, responding to the target CS X in the superconditioning group is determined by the strength of association between X and the US (Link 1) and the product of the strength of association between X and B (Link 2) and the strength of association between B and the US (Link 3). As B presumably acquires no excitatory associative strength with the US in Phase 1 but some excitatory associative strength in Phase 2, responding to X should be (a) weaker than that in the elemental-acquisition control group, which lacks down-modulation by B, and (b) identical to that in the overshadowing control group. The limitation of the original comparator hypothesis arises from its taking into account only the absolute associative strength of one comparator cue. In other words, it ignores any comparator effects to which the target CS's comparator stimulus might be subject.

The extended comparator hypothesis (Denniston et al., 2001), a recent revision of the comparator hypothesis (Miller & Matzel, 1988), overcomes this problem of the original comparator hypothesis by hypothesizing the existence of higher order comparator processes that modulate the effectiveness of Links 2 and 3 (see Figure 1 in its entirety). In the case of superconditioning, the extended comparator hypothesis takes into account not only the absolute associative strength between B and the US but also those of B's companion cues such as training excitor A. By so doing, it can explain the observed greater excitatory responding to the target cue in the superconditioning condition (see Figure 2). The explanation of superconditioning by the extended comparator hypothesis is distinct from the explanation by the acquisition-focused theories in that it explains superconditioning as a result of the interaction of associations at the time of testing, that is, as a superresponding effect, as opposed to superacquisition of the target association.

As noted above, both the revised acquisition-focused theories and the extended comparator hypothesis can explain the results of Experiments 1 and 2, that is, both the superconditioning effect and the effect of posttraining extinction of A on conditioned inhibition. However, the results of Experiment 3 are highly problematic for the revised acquisition-focused theories, whereas they are explicable in terms of the extended comparator hypothesis. In the framework of the extended comparator hypothesis, extinction of the training excitor (A) is expected to cause a loss of the second-order comparator effect on the conditioned inhibitor (B), regardless of whether it is conducted before or after the supercondition-

ing trials (BX-S). The weakening of associative strength between the training excitor and the outcome (S) should result in attenuated conditioned inhibition and, consequently, a reduction in the superconditioning effect. However, in the framework of the revised acquisition-focused theories, presentations of the training excitor alone after superconditioning treatments are not expected to cause any change in the association between the target CS and the outcome because the target CS has not been paired directly with the training excitor and should have no association with it. Thus, even though the effect of posttraining extinction of the training excitor on conditioned inhibition is expected, these theories cannot explain the effect of this posttraining manipulation on superconditioning. In order for revised acquisition-focused theories to explain this kind of phenomena, which might be called second-order comparator effects (e.g., Denniston, Savastano, Blaisdell, & Miller, 2003), additional revisions seem to be necessary.

Finally, we should point out two problems with the extended comparator hypothesis (Denniston et al., 2001). First, because it is not a quantitative model, it does not make quantitative predictions based on mathematical parameters such as the predictions made by the acquisition-focused models. However, Stout, Savastano, and Miller (2005) have developed a mathematical implementation of the extended comparator hypothesis that begins to address this deficiency. Second, as we previously mentioned, the extended comparator hypothesis as well as the acquisition-focused models cannot explain the sensory preconditioning effect or the Espinet effect. Although there were good reasons to do so, both of the experiments on superconditioning were conducted in a sensory preconditioning paradigm, and in Experiment 2, the Espinet effect was used to assess inhibitory learning between two neutral stimuli. All of the preceding theoretical discussions were predicated on the outcome (S) being a mediating stimulus in sensory preconditioning paradigms (or in a mixed paradigm that examined the interaction of the Espinet effect with first-order conditioning of the summation test transfer stimulus and a retardation test; i.e., Experiment 2). To explain these phenomena, further modifications will have to be made to the extended comparator hypothesis as well as the acquisition-focused models, allowing them to explain the apparent coexistence of positive and negative cue mediation. Stout et al.'s (2005) mathematical implementation of the extended comparator hypothesis includes a modification that enables it to account not only for negative cue mediation but also for positive cue mediation, including sensory preconditioning. How the acquisition-focused models can be modified to explain coexistence of positive and negative mediation phenomena including the results of this study is a question for the future. But Dwyer (2001) has suggested a direction for the development for these models.

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