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Conditioned Inhibition Produced by Extinction-Mediated Recovery From the Relative Stimulus Validity Effect: A Test of Acquisition and Performance Models of Empirical Retrospective Revaluation

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Empirical retrospective revaluation is a phenomenon of Pavlovian conditioning and human causal judgment in which posttraining changes in the conditioned response (Pavlovian task) or causal rating (causal judgment task) of a cue occurs in the absence of further training with that cue. Two experiments tested the contrasting predictions made by 2 families of models concerning retrospective revaluation effects. In a conditioned lick-suppression task, rats were given relative stimulus validity training, consisting of reinforcing a compound of conditioned stimuli (CSs) A and X and nonreinforcement of a compound of CSs B and X, which resulted in low conditioned responding to CS X. Massive posttraining extinction of CS A not only enhanced excitatory responding to CS X, but caused CS B to pass both summation (Experiment 1) and retardation (Experiment 2) tests for conditioned inhibition. The inhibitory status of CS B is predicted by the performance-focused extended comparator hypothesis (J. C. Denniston, H. I. Savastano, & R. R. Miller, 2001), but not by acquisition-focused models of empirical retrospective revaluation (e.g., A. Dickinson & J. Burke, 1996; L. J. Van Hamme & E. A. Wasserman, 1994).

Pavlovian conditioned responding and human causal judgments can be modified in the absence of further training with the target stimulus. Some examples of these posttraining retrospective-revaluation effects include backward blocking (e.g., Chapman, 1991; Denniston, Miller, & Matute, 1996; Miller & Matute, 1996; Shanks, 1985; Wasserman, Kao, Van Hamme, Katagiri, & Young, 1996; Williams, Sagness, & McPhee, 1994), recovery from overshadowing mediated by extinction of the overshadowing cue (Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985; Matzel, Shuster, & Miller, 1987; Wasserman & Berglan, 1998; but see Wasserman et al., 1996), and backward conditioned inhibition (Chapman, 1991; Williams & Docking, 1995). Also, Jenkins and Lambos (1983) found that increasing the probability of the unconditioned stimulus (US) in the absence of a conditioned stimulus (CS; i.e., increasing p[USI ~ CS]) after CS–US pairings attenuated conditioned responding to the CS. Furthermore, decreasing p(USI ~ CS) after positive-contingency training strengthens excitatory conditioned responding (e.g., Kaplan & Hearst, 1985; Matzel et al., 1987), whereas the same manipulation after negative-contingency training attenuates responding indicative of inhibition (Hallam, Matzel, Sloat, & Miller, 1990; Kasprow, Schachtman, & Miller, 1987; but see Rescorla & Holland, 1977). Analogous retrospective changes have been reported in the operant literature (Dickinson & Charnock, 1985; Reynolds, 1965).

A variety of models have recently been formulated to account for these phenomena. One family of models is acquisition focused and includes the revised Rescorla-Wagner (1972) model of Van Hamme and Wasserman (1994) and the revised standard operating procedure (SOP; Wagner, 1981) model of Dickinson and Burke (1996). These models attribute posttraining changes in conditioned responding to the target CS to the revaluation of the CS's associative status in the absence of further presentations of that CS itself. Another family of theories of empirical retrospective revaluation emphasizes performance rules for the expression of acquired associations, rather than posttraining changes in the associative status of the target CS. These models assume that subjects acquire the target CS–US association, but fail to express this association until some competing association is altered after completion of training with the target CS.

In the original acquisition-focused models (i.e., Rescorla & Wagner, 1972; Wagner, 1981) from which the models of Dickinson and Burke (1996) and Van Hamme and Wasserman (1994) were derived, only CSs that are physically present on a trial can undergo changes in associative strength as a result of the events experienced on that trial. That is, the associative strengths of CSs not presented on a trial are unaffected by the events that occur on that trial. In the Rescorla-Wagner model, changes in the associative strength of CS X on a given trial are a function of a learning rate parameter for CS X that reflects the associability of CS X (α_x). According to the model, a CS that is not present on a trial has an associability of zero (i.e., $\alpha = 0$) and therefore will undergo no

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changes in associative strength. Thus, the model cannot account for retrospective-revaluation effects, such as the recovery from overshadowing of CS X produced by posttraining extinction of the overshadowing CS (A), because CS X is absent during the revaluation treatment that follows training of CS X. Van Hamme and Wasserman (1994; see also Wasserman & Berglan, 1998) revised the Rescorla-Wagner model by assuming that an absent CS has a negative associability on that trial (i.e., $\alpha < 0$), provided some stimulus previously paired with the absent CS is present. This modification results in two important predictions. First, if both the target CS and US are absent on a given trial, the associative strength of the CS should increase. Second, if the target CS is absent but the US is present on a given trial, the associative strength of the CS should decrease. Through these mechanisms, the model is able to explain empirical retrospective revaluation, including backward blocking and recovery from various cue-competition effects such as those that result from posttraining extinction of competing stimuli including overshadowing (Kaufman & Bolles, 1981; Matzel et al., 1985, 1987), the relative stimulus validity effect (Cole, Barnet, & Miller, 1995a), blocking (Blaisdell, Gunther, & Miller, 1999), and overexpectation (Blaisdell, Denniston, & Miller, 2000). It is important that this account depends on *new learning* occurring to the target CS retrospectively. This clever modification corrects a major failure of the Rescorla-Wagner model, namely the recovery from cue competition that results from extinction of the competing stimulus (see Miller, Barnet, & Grahame, 1995).

Dickinson and Burke (1996) formulated a revision of Wagner's (1981) SOP model that provides a similar account of retrospective-revaluation effects. Stimulus representations are assumed to be distributed among three states: inactive (I), and two active states, a primary state (A1) and a secondary state (A2). Upon physical presentation of a stimulus, the representation of that stimulus enters into the A1 state, then decays into the A2 state, and finally decays back into the I state. However, when a stimulus' associate is presented in the absence of the stimulus itself, the stimulus' representation enters directly into the A2 state (i.e., an associate can activate, retrieve, a memory of the target stimulus). In Wagner's original model, excitatory associations are formed between cues that are in the A1 state, but no learning occurs to cues that are in the A2 state of activity; thus, the model fails to account for retrospective-revaluation effects. In contrast, Dickinson and Burke's modification of Wagner's SOP model proposes that excitatory connections are formed not only when representations of the potential associates are both in the A1 state, but also when both stimulus representations are in the A2 state. This modification of SOP allows the model to account for retrospective-revaluation effects. For example, in recovery from blocking in which CS A blocks CS X, subsequent nonreinforced presentation of CS A activates both CS X and the US into the A2 state of activity, thereby resulting in an increase in the excitatory connection between CS X and the US.[1] Thus, both Van Hamme and Wasserman (1994) and Dickinson and Burke view retrospective revaluation as a consequence of further learning with respect to the target CS.

In contrast, retrospective-revaluation effects can also be explained in terms of performance deficits. One such performance-focused model is the (original) comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985). The comparator hypothesis is a response rule for Pavlovian responding that states that responding to a CS is directly related to the associative strength of the target CS–US association and also inversely related to the associative strength to the US of the stimulus that has the strongest within-compound association with the target CS (excluding the US itself). This stimulus is hereafter called the target CS's *comparator stimulus* and may be the training context or discrete stimuli, such as competing CSs. In the framework of the comparator hypothesis (see Figure 1), the subject acquires three associations, (a) a target CS–US association, (b) a target CS–comparator-stimulus association, and (c) a comparator-stimulus–US association. Presentation of the target CS at test directly activates a representation of the US as a result of the target CS–US association (Link 1). This directly activated US representation is compared with the US representation that is indirectly activated in proportion to the product of the strengths of the target-CS–comparator-stimulus association (Link 2) and the comparator-stimulus–US association (Link 3). The degree of excitatory responding is directly related to the directly activated US representation and inversely related to the indirectly activated US representation. Alternatively stated, responding depends on the associative strength of the target CS relative to that of its comparator stimulus.

According to the comparator hypothesis, cue competition occurs at the time of testing. Therefore, any manipulation (including those that occur after completion of target CS training) that decreases the CS–comparator stimulus or comparator-stimulus–US associations (Links 2 or 3, respectively) will diminish the magnitude of the indirectly activated US representation, thereby augmenting excitatory responding to the CS. Conversely, any manipulation that strengthens Links 2 or 3 will enhance the indirectly activated US representation, thereby attenuating excitatory responding to the CS. Thus, extinction of the comparator stimulus (attenuating the strength of Link 3 and possibly Link 2) should attenuate the

[1] Another family of acquisition-focused associative models that permits changes in the associative status of absent cues is that proposed by Holland (1981, 1990) and revisited by Hall (1996). In the framework of these models, cues have positive associability on trials in which they are absent. Thus, after initial experience in which two cues are paired (and hence, associated), subsequent presentation of one cue (the associate) will activate in memory the representation of the other cue, allowing this representation to enter into associations similar in valence to those formed by the associate. For example, after sensory preconditioning in which two initially neutral CSs are presented together nonreinforced, pairings of one of the preconditioned associates with a US allows the other, nonpresent associate to enter into an excitatory association with the US as well. By attributing a positive associability to absent cues, these models can explain the excitatory responding to the nonpresent associate after the two phases of sensory preconditioning (e.g., Brogden, 1939; Holland, 1981; Rescorla, 1980). This family of models can also account for a plethora of related phenomena, such as backward sensory preconditioning (Cole, Barnet, & Miller, 1995b; Ward-Robinson & Hall, 1996), representation-mediated conditioning (Holland, 1981; Nakajima & Kawai, 1997), representation-mediated extinction (Holland & Forbes, 1982b), representation-mediated overshadowing and potentiation (Holland, 1983), representation-mediated occasion setting of conditioned flavor aversions (Holland & Forbes, 1982a), and the acquired equivalence and distinctiveness of cues (Hall, Ray, & Bonardi, 1993; Honey & Hall, 1989). However, this family of models cannot account for recovery from cue competition resulting from extinction of the target cue's competing stimulus, the focus of the current research question, and therefore will receive no further attention here.

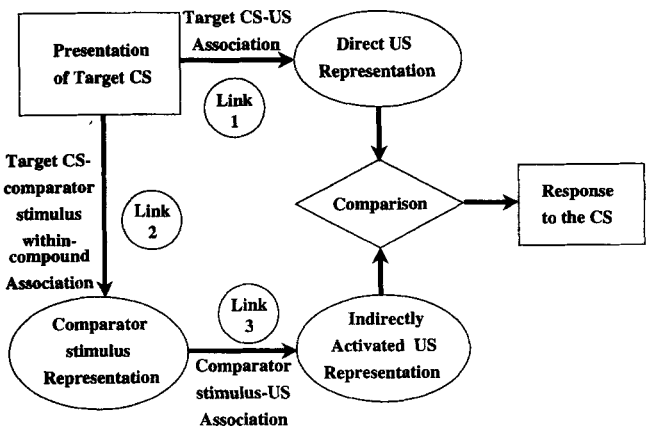

Figure 1. The original comparator hypothesis. CS = conditioned stimulus; US = unconditioned stimulus.

indirectly activated US representation, allowing greater expression of the target CS–US association. Empirical retrospective revaluation effects have been viewed as favoring the performance-focused comparator hypothesis over traditional acquisition-focused models of Pavlovian responding (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981). However, the emergence of recently modified versions of the Rescorla-Wagner model (e.g., Van Hamme & Wasserman, 1994) and Wagner's SOP model (Dickinson & Burke, 1996) allow acquisition-focused models to account for many of these phenomena, and thereby challenge the uniqueness of the comparator hypothesis' account of empirical retrospective revaluation.

The potential of the revised acquisition-focused models (hereafter called retrospective revaluation models) to explain retrospective revaluation prompted Denniston, Savastano, and Miller (2001) to extend the comparator hypothesis in search for new contrasting predictions that would differentiate it from the retrospective revaluation models. Within the framework of the extended comparator hypothesis, just as the effective strength of Link 1 (which determines response strength) is modulated by the product of Links 2 and 3 (see Figure 1), so too are the effective strengths of Links 2 and 3 each modulated by their own comparator processes that depend upon what we will call *second-order comparator stimuli* (see Figure 2). These second-order comparator stimuli (second order with respect to the target CS, but first order with respect to the target CS's first-order comparator stimuli) determine the effectiveness of the first-order comparator stimulus in modulating conditioned responding to the target CS.

The comparator processes for Link 2 work in the following manner. When the target CS is presented at test, it directly activates a representation of the first-order comparator stimulus by way of the association between the target CS and the first-order comparator stimulus (Link 2.1 of Figure 2) and also indirectly activates a representation of the first-order comparator stimulus through the conjoint activation of the association between the target CS and the second-order comparator stimulus (Link 2.2) and the association between the second-order comparator stimulus and

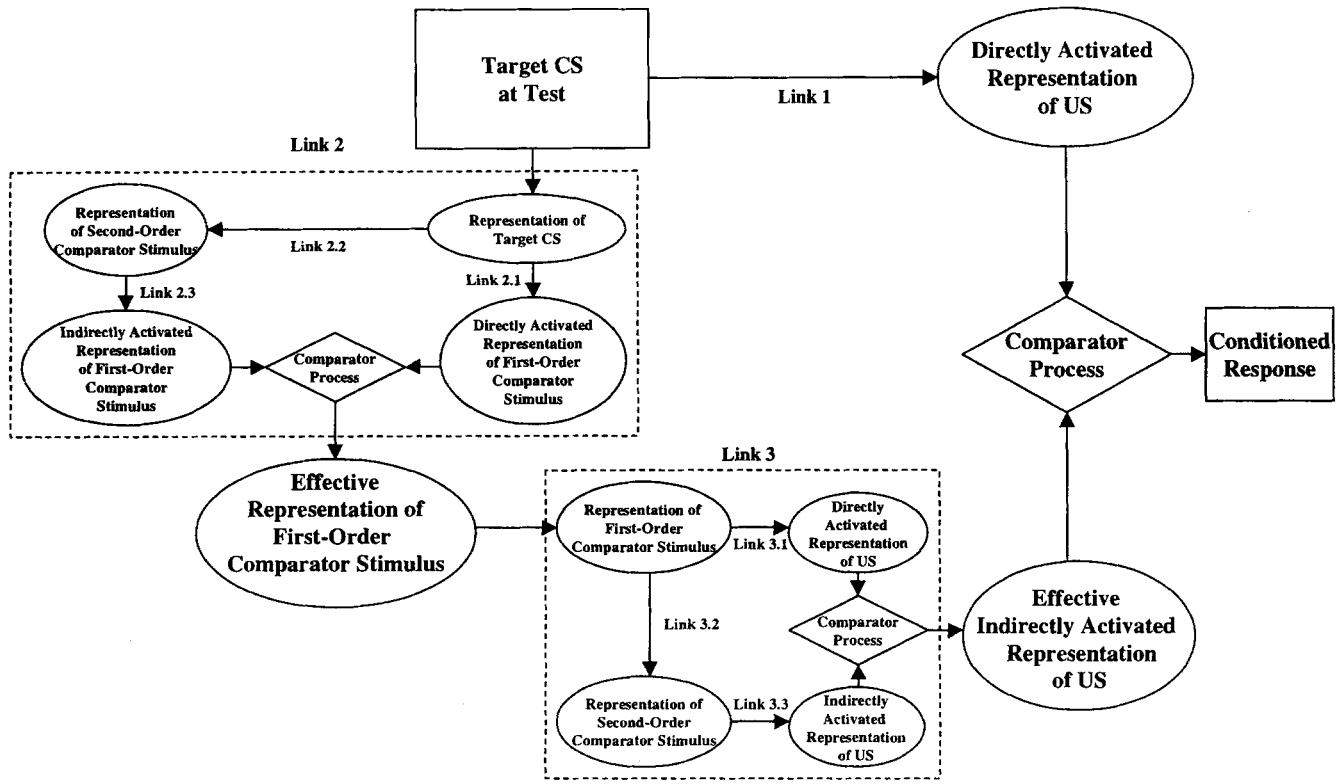

Figure 2. The extended comparator hypothesis. CS = conditioned stimulus; US = unconditioned stimulus.

the first-order comparator stimulus (Link 2.3). These directly and indirectly activated representations of the first-order comparator stimulus are compared. The output of this comparison determines the strength of activation (i.e., effectiveness) of the representation of this particular first-order comparator stimulus, which then serves as the input for Link 3. The effectiveness of this first-order comparator stimulus increases directly with the strength of Link 2.1 and inversely with the strengths of Link 2.2 and Link 2.3. An analogous process is assumed for Link 3.[2]

A variety of predictions differentiate the acquisition-focused models of retrospective revaluation from the extended comparator hypothesis (see Blaisdell, Bristol, Gunther, & Miller, 1998; Blaisdell, Savastano, & Miller, 1999; Denniston et al., 2001; Friedman, Blaisdell, Escobar, & Miller, 1998). One prediction involves the effects of retrospective-revaluation treatment on Pavlovian conditioned inhibition. In conventional Pavlovian conditioned inhibition training, subjects receive trials during which an excitatory CS (X) is paired with a US, interspersed with nonreinforced trials on which this training excitor and another stimulus (B) are presented in compound. Both retrospective-revaluation models and the extended comparator hypothesis predict that CS B will become inhibitory (through acquisition of negative associative strength [Van Hamme & Wasserman, 1994], the accrual of inhibition to CS B [Dickinson & Burke, 1996], or a comparator process in which the US representation activated directly by CS B is weak relative to the US representation activated indirectly by CS X, which is the first-order comparator stimulus for CS B). The present research used a variant of the standard Pavlovian conditioned inhibition procedure. The X–US pairings occurred in the presence of a more valid predictor (CS A) of the US, so that little excitatory responding was expected to develop to CS X (i.e., this procedure should have produced the relative stimulus validity effect; cf. Wagner, Logan, Haberlandt, & Price, 1968; Wasserman, 1974). With this modification of Pavlovian inhibition training, the two families of models make contrasting predictions concerning the effect of posttraining extinction of CS A on the inhibitory status of CS B.

The relative stimulus validity effect specifically refers to the deficit in responding to CS X seen in a group (correlated) trained with AX+ and BX− trials interspersed (+ indicating reinforcement and − indicating nonreinforcement), relative to a group (control) trained with comparable AX and BX trials but each reinforced on 50% of the trials (AX±/BX±). Both families of models predict that the low responding to CS X in the correlated condition will prevent CS B from eliciting behavior indicative of conditioned inhibition, albeit through different mechanisms.

According to the Van Hamme and Wasserman (1994) model, CS X acquires little associative strength due to the presence of CS A. As a result of CS X having little associative strength, CS B acquires little negative associative strength, thereby preventing CS B from becoming a strong conditioned inhibitor. In the framework of the extended comparator hypothesis, the strong A–US association masks the excitatory X–US association. Because the X–US association is not effective, it fails to support excitatory or inhibitory responding to CS B. That is, the effectiveness of the X–US association in modulating responding to CS B is itself mediated by the A–US association, which is strong. Therefore, CS B should have little inhibitory value. Decrements in the A–US association should increase CS X's effectiveness in activating a representation of the US. In the present situation, this means that extinction of CS A should increase CS X's effective link to the US (e.g., Cole et al., 1995a), and thus (because CS X is CS B's comparator) decrease responding to CS B. Van Hamme and Wasserman and Dickinson and Burke (1996), however, fail to predict any effect of extinction of CS A on stimulus control by CS B. According to the retrospective-revaluation models, extinction of CS A will either have no effect on the associative strength of CS B (i.e., it will remain at zero because presentation of CS A should not activate the representation of CS B due to the absence of any A–B association) or, if second-order activation is postulated, with both CS B and the US absent on extinction of CS A trials, CS B's associative strength should increase.

Experiment 1

Experiment 1 was designed to examine the consequences of extinguishing CS A, after correlated relative validity (RV) training (i.e., AX+/BX−), on the inhibitory status of CS B, using a summation test for conditioned inhibition (see Table 1). Groups RV.A, Acq, and RV received correlated relative validity training. A fourth group (Con) received a control treatment consisting of AX± trials interspersed with BX± trials in place of relative validity training during Phase 2. In addition, all of the groups received CY+ trials interspersed among this training to better equate nonassociative factors between groups (see below). Subsequent to this treatment, subjects in Groups RV.A and Acq received massive extinction of CS A, whereas subjects in Groups RV and Con received extinction of CS C (an irrelevant stimulus included to control for nonassociative effects of extinction of an excitor previously trained in compound with another stimulus). Finally, Groups RV.A, RV, and Con were assessed on how well CS B attenuated conditioned suppression to a separately trained (i.e., transfer) excitor (CS D; i.e., test on compound BD), compared with suppression to CS D alone by Group Acq. The extended comparator hypothesis, but not the retrospective revaluation models, predicts that extinction of CS A in Group RV.A would result in CS B passing a summation test of conditioned inhibition (i.e., CS B should attenuate conditioned responding to CS D) relative to Group RV. This prediction arises from the extended comparator hypothesis' claim that responding to CS B will be modulated by its own comparator stimulus (X), which itself is modulated by its own comparator stimulus (A). Group Acq was intended to document the excitatory value of CS D.

In a second test, all of the subjects were presented with CS X alone. This test was expected to demonstrate weaker suppression in Group RV compared with Group Con, thereby replicating the relative stimulus validity effect with our procedure. Both the extended comparator hypothesis and the retrospective-revaluation models predict more responding to CS X in Group RV.A than in Group RV, that is, recovery from the relative stimulus validity effect as a result of extinction of CS A.

[2] Although we could postulate on the actions of even higher order (third, fourth, etc.) comparator processes modulating the second-order comparator links, the effects of such processes presumably dampen with each higher order; therefore, we concern ourselves here only with the first- and second-order comparator loops.

Table 1
Design Summary for Experiment 1: Summation Test

Group	Phases 1 + 3	Phase 2	Phase 4	Test	Predictions RR	Predictions ECH	Test	Predictions RR	Predictions ECH
RV.A	D+	AX+/BX−/CY+	A−	BD	CR[a]	cr[a]	X	CR	CR
Acq	D+	AX+/BX−/CY+	A−	D	CR	CR	X	CR	CR
RV	D+	AX+/BX−/CY+	C−	BD	CR	CR	X	cr	cr
Con	D+	AX±/BX±/CY+	C−	BD	CR	CR	X	CR	CR

Note. A, B, and C were noise, light, and clicks, counterbalanced within group. X and Y were high-frequency tone and buzzer, counterbalanced within group. CS D was a low-frequency tone. Group names: RV = relative validity; Acq = acquisition of behavioral control by CS D; Con = relative validity control; A = extinction-of-A treatment; BD = testing occurred on the BD compound; D = testing occurred on CS D. CR denotes a large conditioned response, and cr indicates a small conditioned response. + and − denote presence and absence of the footshock US, respectively, and ± denotes 50% reinforcement.
[a] Contrasting predictions of the extended comparator hypothesis (ECH) and the retrospective-revaluation (RR) models.

Method

Subjects

Twenty-four male (290–390 g) and 24 female (200–280 g) naive, Sprague-Dawley–descended rats, bred in our colony, served as subjects. Subjects were individually housed in wire-mesh cages in a vivarium maintained on a 16-hr light–8-hr dark cycle. All of the experimental manipulations were performed approximately midway through the light portion of the cycle. Food was freely available in the home cage. A progressive water-deprivation schedule was imposed over the week prior to the beginning of the experiment, until water availability was limited to 30 min per day. All of the rats were handled 3 times a week for 30 s, from time of weaning to the initiation of the study. Subjects were randomly assigned to one of the four groups (*n*s = 12).

Apparatus

Twelve identical chambers, each measuring 30 × 25 × 32 cm (l × w × h), were used. The walls of each chamber were made of Plexiglas, and the floor was constructed of 0.5 cm diameter rods, spaced 2 cm center-to-center and connected by NE-2 neon bulbs that allowed a 1.0-mA constant-current footshock to be delivered by means of a high voltage AC circuit in series with a 1.0-MΩ resistor. Each chamber was housed in an environmental isolation chest that was dimly illuminated by a No. 1820 incandescent house light mounted on the ceiling of the experimental chamber. Each chamber was equipped with a water-filled lick tube (opening = 0.3 mm in diameter) that extended 1 cm from the rear of a cylindrical niche, 4.5 cm in diameter, 5.0 cm deep, that was left–right centered on one wall, with its axis perpendicular to the wall and positioned with its bottom 4 cm above the grid floor. An infrared photobeam was projected horizontally across the niche, 1 cm in front of the lick tube. In order to drink from the tube, subjects had to insert their heads into the niche, thereby breaking the horizontal infrared photobeam. Thus, the amount of time the photobeam was disrupted could be monitored; this served as our dependent measure. A 45-Ω speaker mounted on the interior back side of each environmental chest could deliver a high-frequency complex tone (3000 and 3200 Hz) and a low-frequency complex tone (300 and 320 Hz) each 8 dB(A-scale) above background. A second 45-Ω speaker mounted on the ceiling of the experimental chamber could be used to deliver a click (C) train stimulus (6/s) 8 dB(A) above background. A third 45-Ω speaker mounted on the side wall of the chamber was used to deliver a white noise (N) stimulus 8 dB(A) above background. A small speaker attached to the outside wall of each experimental chamber could deliver a buzzing sound, 8 dB(A) above background. A 75-W (nominal at 120 VAC, driven at 100 VAC) incandescent bulb was mounted on the back wall of each environmental chest 30 cm from the floor of the conditioning chamber. This bulb could be flashed (0.25 s on, 0.25 s off). The white noise, flashing light, and clicks, counterbalanced within groups, served as CSs A, B, and C. The high-frequency tone and buzzer served as CSs X and Y, counterbalanced within groups. The low-frequency tone always served as CS D. Ventilation fans in each enclosure provided a constant 76-dB(A) background noise. All CSs during treatment were 10 s in duration. The 1.0-mA footshock US was 0.5 s in duration.

Procedure

Table 1 summarizes the critical aspects of the treatment procedure. For any given subject, all of the training and testing occurred in the same chamber.

Acclimation. On Day 1, all of the subjects received 60 min of acclimation to the experimental chambers with the lick tubes in place. This session served to establish and stabilize baseline licking behavior and acclimate the rats to the experimental context. In addition, each subject received two nonreinforced presentations each to the noise, clicks, high tone, flashing light, and buzzer. These presentations were given to reduce the likelihood of the subjects configuring stimuli presented in compound during training. Such configuring could potentially work against our observing the effects predicted by either family of models, which depend on each stimulus being processed as a separate element.

Phases 1 and 3 (transfer excitor treatment). After acclimation, the lick tubes were removed from all of the chambers. On Days 2 and 15, all of the subjects received four daily exposures to Stimulus D followed immediately upon termination by the footshock US in a daily 60-min session.

Phase 2 (relative validity treatment). On Days 3–14, subjects in Groups RV.A, Acq, and RV received 6 AX+, 6 BX−, and 6 CY+ (with + signifying the US and − the absence of the US) trials within each daily 90-min session, for a total of 72 trials of each trial type across the 12 days of training. Subjects in Group Con received 3 AX+, 3 AX−, 3 BX+, 3 BX−, and 6 CY+ trials per session. The mean intertrial interval was 5 min (range = 3–7 min).

Phase 4 (extinction). On Days 16–19, subjects in Groups RV.A and Acq received 50 A− trials within each daily 60-min session, whereas subjects in Groups RV and Con received 50 C− trials per session, for a total of 200 extinction trials. The mean intertrial interval was 90 s (range = 60–120 s).

Reacclimation. Prior to reacclimation, lick tubes were returned to each chamber. On Days 20 and 21, subjects were allowed to drink during daily 60-min sessions to reestablish baseline levels of drinking, which is com-

monly disrupted by administration of footshock. These sessions did not include any nominal stimulus presentations.

Testing. On Day 22, subjects in Groups RV.A, RV, and Con were tested for conditioned lick suppression to the compound BD stimulus by presenting the compound immediately after completion of each subject's first five cumulative seconds of licking behavior (as measured by the total amount of time the infrared beam was disrupted). Thus, all of the subjects were licking at the time of compound CS onset. Subjects in Group Acq were tested in a similar manner, but on CS D. Times to complete the first five cumulative seconds of licking (i.e., prior to CS onset) and times to complete an additional five cumulative seconds of licking in the presence of the test CS were recorded. A 10-min ceiling was imposed upon subjects for completing five cumulative seconds of drinking in the presence of BD or D. On Day 23, all of the subjects were similarly tested for conditioned lick suppression to CS X.

In all of the experiments reported, suppression data were transformed to log (base 10) scores to better meet the assumptions of parametric inferential statistics. Also, after the convention of our laboratory, subjects that took more than 60 s to complete their first five cumulative seconds of licking on either test day (i.e., prior to CS onset) were scheduled to be eliminated from the study because of their exhibiting unusually great fear toward the experimental context. In Experiment 1, no subject took more than 60 s to complete its first five cumulative seconds of licking on either test trial. An alpha level of .05 was adopted for all tests of statistical significance.

Results and Discussion

The central observations from Experiment 1 were the demonstrations of suppression to CS X of the relative stimulus validity response deficit (Groups RV vs. Con) and recovery from this effect as a result of extinction of CS A (Groups RV.A vs. RV; see Figure 3). Most important, extinction of CS A resulted in CS B passing a summation test for inhibition (Groups RV.A vs. Acq; see Figure 4).

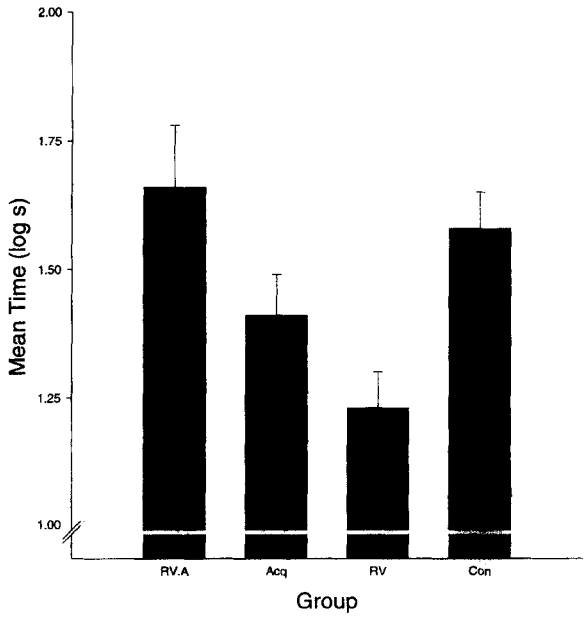

Figure 3. Experiment 1: Mean times (log s) to complete five cumulative seconds of licking in the presence of target CS X. RV = relative validity; Acq = acquisition of behavioral control; Con = relative validity control. *Error bars represent standard errors of means.*

Conditioned Suppression to CS X

A one-way analysis of variance (ANOVA) conducted on pre-CS times to complete five cumulative seconds of licking prior to CS onset found no significant differences between groups in baseline drinking behavior, $F(3, 44) < 1.0$. A one-way ANOVA conducted on suppression to X scores revealed an effect of treatment, $F(3, 44) = 4.71$, $p < .01$. Planned comparisons were conducted on the suppression scores for CS X using the overall error term from the latter one-way ANOVA. Subjects in Group RV suppressed less than did subjects in Group Con, demonstrating the relative stimulus validity effect, $F(1, 44) = 7.91$, $p < .01$. Extinction of CS A resulted in strong suppression to CS X in Group RV.A compared with the low suppression in Group RV, $F(1, 44) = 12.04$, $p < .005$. Moreover, consistent with the findings of Cole et al. (1995a), this recovery was specific to extinction of CS X's comparator stimulus A (Group RV.A), and did not generalize from the extinction of an alternate excitor (C) with which CS X had never been paired (Group RV). This stimulus specificity indicates that comparator effects are limited to stimuli that share a within-compound association with the target CS. Responding to CS X in Group Acq was lower than in Group RV.A, $F(1, 44) = 3.91$. Although this difference was only marginally significant ($p = .054$), it was unexpected because the two groups differed only on the first test day of treatment. Perhaps, extinction of CS D alone in Group Acq (as a result of the 10-min test exposure) generalized more to CS X than did extinction of the BD compound. Whatever the source of this marginal difference, it does not undermine our interpretation of the results.

Conditioned Suppression to CS D and Compound BD

A one-way ANOVA conducted on pre-CS times to complete five cumulative seconds of licking prior to CS onset found no significant differences between groups in baseline drinking behavior, $F(3, 44) < 1.0$. A one-way ANOVA conducted on suppression to BD or D scores revealed an effect of treatment, $F(3, 44) = 5.89$, $p < .005$. Planned comparisons were conducted on the suppression to BD or D scores using the overall error term from the latter one-way ANOVA. Subjects in RV.A suppressed less than did subjects in Group Acq, demonstrating that extinction of CS A resulted in CS B passing a summation test of conditioned inhibition, $F(1, 44) = 7.04$, $p < .05$. Moreover, behavior indicative of conditioned inhibition to CS B was specific to extinction of CS A (Group RV.A) and was not produced by extinction of CS C (Group RV, which exhibited high suppression to the BD compound), $F(1, 44) = 9.42$, $p < .005$.

Experiment 2

Because both summation and retardation tests are thought to be necessary to establish the existence of conditioned inhibition (Hearst, 1972; Rescorla, 1969), our next experiment was largely equivalent to Experiment 1, except for the use of a retardation test to assess inhibition (see Table 2). Three groups (RV.A, Acq, and RV) received correlated relative stimulus validity treatment con-

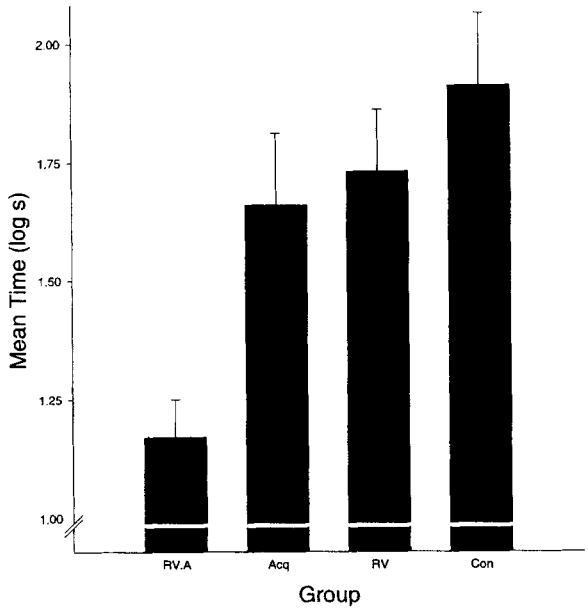

Figure 4. Experiment 1: Mean times (log s) to complete five cumulative seconds of licking in the presence of the BD compound test stimulus, except for Group Acq, which was tested on CS D alone. RV = relative validity; Acq = acquisition of behavioral control; Con = relative validity control. Error bars represent standard errors of means.

sisting of AX+ trials interspersed with BX− (or CX− in the case of Group Acq, with this change serving to avoid any learning with respect to B). A fourth group (Con) received uncorrelated relative stimulus validity treatment consisting of AX± trials interspersed with BX± trials. In addition, Groups RV.A, RV, and Con received CY+ trials interspersed among the other trials, whereas Group Acq received Y+ trials interspersed among the other trials (with this difference serving to allow CS C to substitute for CS B during relative stimulus validity training for this group). Subsequent to this treatment, subjects in Groups RV.A and Acq received extinction of CS A, whereas subjects in Groups RV and Con received extinction of irrelevant excitatory cue CS C. Next, all of the groups received three retardation test pairings of CS B and the US (i.e., B+); note that for Group Acq, CS B was a novel stimulus at the initiation of these pairings. Finally, all of the groups were tested for conditioned suppression of drinking by CS B. The extended comparator hypothesis, but not the retrospective-revaluation models, predicted that extinction of CS A in Group RV.A would result in CS B passing a retardation test of conditioned inhibition (i.e., CS B would produce little conditioned suppression relative to the novel control Stimulus B in Group Acq). Transfer excitor training (D+) was given as in Experiment 1, despite its seeming irrelevance to the retardation test, because we wanted to maintain identical treatment in the summation and retardation experiments until the time of the retardation test pairings, for reasons compellingly stated by Papini and Bitterman (1993).

Method

Subjects and Apparatus

Twenty-four male (292–400 g) and 24 female (185–260 g) Sprague-Dawley–descended rats, bred in our colony, served as subjects. Rats were randomly assigned to one of four groups (*ns* = 12), counterbalanced for sex. The rats were housed and maintained as in Experiment 1. The apparatus and stimuli were identical to those used in Experiment 1.

Procedure

Table 2 summarizes the critical aspects of the treatment procedure.

Acclimation, transfer excitor training, relative stimulus validity training, and extinction. These treatments were identical to Experiment 1, except that Group Acq received Y+ trials (instead of the CY+ trials of Experiment 1) interspersed with AX+ and CX− trials during relative stimulus validity training. This ensured that Stimulus B would be novel during the retardation test pairings; we planned to compare conditioned responding elicited by this novel Stimulus B to responding elicited by CS B in Group RV.A as our measure of conditioned inhibition. Our counterbalancing within groups the physical stimuli that served as CSs B and C validates our substituting CS C for CS B during training in Group Acq.

Retardation conditioning with CS B. On Day 20, all of the subjects received three B–US trials, 15, 27, and 45 min into a 60-min session.

Reacclimation. This was identical to Experiment 1 except that it occurred on Days 21 and 22.

Testing. On Day 23, all of the subjects were tested for conditioned lick suppression to CS B. Other than the use of CS B, testing was identical to

Table 2
Design Summary for Experiment 2: Retardation Test

						Predictions	
Group	Phases 1 + 3	Phase 2	Phase 4	Phase 5	Test	RR	ECH
RV.A	D+	AX+/BX−/CY+	A−	B+	B	CR[a]	cr[a]
Acq	D+	AX+/CX−/Y+	A−	B+	B	CR	CR
RV	D+	AX+/BX−/CY+	C−	B+	B	CR	CR
Con	D+	AX±/BX±/CY+	C−	B+	B	CR	CR

Note. A, B, and C were noise, light, and clicks, counterbalanced within group. X and Y were high-frequency tone and buzzer, counterbalanced within group. D was a low-frequency tone. Group names: RV = relative validity; Acq = acquisition control; Con = relative validity control; A = extinction-of-A treatment. CR denotes a large conditioned response, and cr indicates a small conditioned response. + and − denote presence and absence of the footshock US, respectively, and ± denotes 50% reinforcement.

[a] Contrasting predictions of the extended comparator hypothesis (ECH) and the retrospective revaluation (RR) models.

Day 22 of Experiment 1. One subject from Group RV failed to complete its first five cumulative seconds of licking (i.e., preCS) within 60 s; thus, its data were excluded from all statistical analyses.

Results and Discussion

The central observations from Experiment 2 were that extinction of CS A resulted in CS B passing a retardation test for conditioned inhibition (Group RV.A vs. Group Acq), and that equivalent extinction of an irrelevant excitor (CS C) failed to cause CS B to pass a comparable retardation test (Group RV.A vs. Group RV). Group means are illustrated in Figure 5.

A one-way ANOVA conducted on pre-CS times to complete five cumulative seconds of licking prior to CS onset found no significant differences between groups in baseline drinking behavior, $F(3, 43) < 1.0$. A one-way ANOVA conducted on suppression to CS B scores revealed an effect of treatment, $F(3, 43) = 3.36$, $p < .05$. Planned comparisons were conducted on the suppression to CS B scores using the overall error term from the latter one-way ANOVA. Subjects in RV.A suppressed less than did subjects in Group Acq, demonstrating that extinction of CS A resulted in CS B's passing a retardation test for conditioned inhibition, $F(1, 43) = 6.88$, $p < .05$. Moreover, behavior indicative of conditioned inhibition to CS B was specific to extinction of CS A (Group RV.A) and was not produced by extinction of CS C (Group RV).

General Discussion

Experiment 1 replicated Cole et al.'s (1995a) demonstration of attenuation of the relative stimulus validity response deficit through posttraining extinction of the competing stimulus (A, the most valid predictor of the US). Furthermore, this experiment showed that posttraining extinction-mediated recovery of excitatory responding to CS X also enhanced CS B's inhibitory potential, as assessed with a summation test of conditioned inhibition. Experiment 2 replicated this finding with a retardation test of conditioned inhibition. Although retrospective revaluation models (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994) and the comparator hypothesis (e.g., Miller & Matzel, 1988) adequately account for the recovery of excitatory responding to CS X by extinguishing CS A, only the extended comparator hypothesis can account for the accompanying increase in inhibitory value of CS B.[3]

In the framework of the extended comparator hypothesis, relative stimulus validity training (AX+/BX−) establishes CS X as CS B's first-order comparator stimulus by virtue of the strong within-compound association presumably formed between CSs B and X through their contiguous presentation.[4] Furthermore, the contiguous presentations of CSs A and X presumably resulted in the formation of a strong within-compound association between these cues. This should have established CS A as the first-order comparator stimulus for CS X, and consequently established CS A as the second-order comparator stimulus for CS B. The presentation of CS B at test presumably activated the representation of CS X. However, the extent to which CS X's representation was

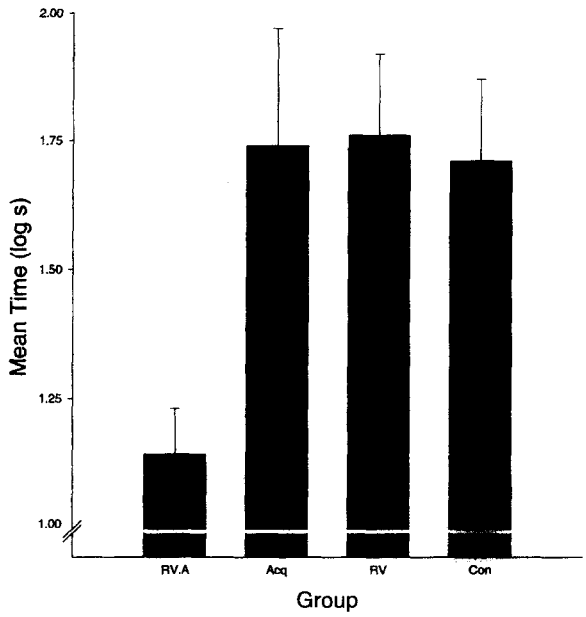

Figure 5. Experiment 2: Mean times (log s) to complete five cumulative seconds of licking in the presence of target CS B. RV = relative validity; Acq = acquisition of behavioral control; Con = relative validity control. Error bars represent standard errors of means.

[3] It is important to note that the comparator hypothesis's (original or extended) explanation of the basic relative validity effect is parameter dependent. In the framework of the comparator hypothesis, CSs A and B are expected to act equally as comparator stimuli for CS X, because CS X was equally contiguous with these two stimuli during training. In the correlated condition (AX+/BX−), in which all reinforcement occurred on AX trials, CS A should have strongly activated the indirect–US representation, whereas CS B should have had a negligible effect. In the control group (AX±/BX±), in which both AX and BX trials are reinforced 50% of the time, CSs A and B should have had an equal and moderate activating effect on the indirect–US representation. When two cues occupy equal comparator roles, these comparator stimuli appear to have an additive effect on activating the indirect–US representation (Grahame, Barnet, & Miller, 1992) and therefore on attenuating responding to the target CS. For the comparator hypothesis to predict weaker responding to CS X in a correlated (relativity validity) group than in a control group, reinforcement in the control condition must produce a summed value of the A–US and B–US associations of less than 50% of the value of the A–US association in the correlated group. This requires an ogival acquisition curve and is surely less pleasing than predictions that are not parameter dependent. (Notably, parameter dependence of predictions is a hallmark of both the original [Wagner, 1981] and revised [Dickinson & Burke, 1996] versions of SOP.) Regardless of the parametric constraint, the comparator hypothesis does predict an increase in responding to CS X in the correlated group through posttraining extinction of CS A, which is the only comparator of CS X that has a strong association with the US (Cole et al., 1995a). The central focus of the experiments reported here is the effect of extinction of CS A on responding to CS B and how this effect can differentiate between two families of models that have been designed to explain empirical retrospective revaluation.

[4] Although the context might have served as a first-order comparator stimulus for CS B, the effectiveness of the B–context association should have been down modulated by CS X, which would have been the comparator stimulus for the B–context association. Thus, the context should have little impact on responding to B.

activated presumably was modulated by the product of the strengths of the B–context association and the context–X association because the context was another cue present during the BX trials (i.e., the context was CS B's second-order comparator stimulus for Link 2). The representation of CS X in turn presumably activated the representation of CS A (CS B's second-order comparator stimulus for Link 2). CS A was a more valid predictor of the US than was CS X. Therefore, the product of the X–A and A–US associations should have been large relative to the X–US association, with the result that CS A should have down modulated the potential of CS X's representation to indirectly activate the US representation. This weakened, indirectly activated US representation should have resulted in CS B's failing to elicit behavior indicative of conditioned inhibition. However, extinction of CS A (Groups RV.A of Experiments 1 and 2) should have enhanced responding to CS X, and in so doing also should have made CS B a more effective conditioned inhibitor. That is, extinction of CS A should have attenuated the strength of the US representation CS A activated, thereby reducing the competition between this US representation and the one activated by CS X. As a result, the effective US representation activated indirectly by CS B's comparator stimulus (CS X) should have been strong relative to the negligible US representation directly activated by CS B. Note that the absolute X–US association is equally strong in groups that did and did not receive extinction of CS A, but the effectiveness of this X–US association in modulating inhibitory control by CS B was presumably attenuated by a strong A–US association. Only after extinction of the competing A–US association (i.e., Link 3) was the effective X–US association able to support CS B's serving as a conditioned inhibitor.[5]

The retrospective-revaluation models (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994) also predict that extinction of CS A should make CS X more excitatory. This increase in excitation to CS X is achieved as a result of the physical absence of both CS X and the US during the A− trials, and the assumption that absent stimuli have a negative associability. Therefore, the absence of both CS X and the US during extinction of CS A should have enhanced the excitatory association between them. (Note that in this framework the absolute X–US association is weak prior to extinction of CS A, and gains strength through the extinction [A−] treatment. That is, in contrast with the assumptions of the comparator hypothesis, there is no latent X–US association prior to extinction of CS A.) However, these models anticipate either no change in the associative status of CS B or a decrease in B's effective inhibitory status, depending on one's assumptions concerning how second-order relationships between stimuli map into these models. For example, Dickinson and Burke (see also Wasserman & Berglan, 1998) might anticipate no within-compound association between CSs B and A because they were never presented together, so presentation of CS A should not activate a representation of CS B. Consequently, there should be no change in the associative status of CS B (which should be near zero at the end of relative stimulus validity treatment) as a result of extinguishing CS A. Alternatively, one might argue that presentation of CS A would activate the representation of CS X in the A2 memory state of the revised SOP model, which in turn might activate a representation of CS B to State A2. According to this hypothetical extension of the Dickinson and Burke model, because CS A was presented alone during extinction, a representation of the US should be activated into State A2. With representations of both CS B and the US in State A2, the model would predict that CS B would gain excitatory strength, which should then summate with any previously acquired inhibitory value of CS B, making it less inhibitory. It is important that either prediction of the Dickinson and Burke model contrasts with that of the extended comparator hypothesis, which anticipates an increase in the inhibitory status of CS B. (A similar statement about the generality of extinction of CS A on all cues present during training, regardless of their contiguity to CS A during training, could be made in the framework of the Van Hamme & Wasserman model.)

Notably, a modification of the Dickinson and Burke (1996) model might posit that presentation of CS A during extinction could activate the representation of CS X into the A2 state, which in turn might activate a representation of CS B into the A1 state. That is, the representation of a stimulus that is directly activated by its associate (e.g., the representation of CS X activated by presenting CS A), might itself activate the representation of its associate (e.g., CS B) into State A1. Thus, although first-order representations are activated into the A2 state, second-order stimulus representations are activated into the A1 state. Such a modification could, in principle, account for the results reported here and predicted by the extended comparator hypothesis. However, such a modification of the Dickinson and Burke model is antithetical to the spirit in which Wagner's (1981) original and Dickinson and Burke's modified SOP models were proposed. Wagner (p. 10), implied that State A2 (the secondary state of activation) represents the type of active stimulus processing that occurs to reactivated memories, whereas the A1 state includes the perceptual or attentional processing of a stimulus representation that results from the physical presentation of the stimulus. This distinction is incompatible with the notion that a second-order, associatively activated representation would command perceptual or attentional processing similar to that of a physically presented stimulus. If anything, second-order representations of physically absent stimuli would be expected to be equal in strength to, or weaker than, first-order associatively activated representations of absent stimuli. Nevertheless, to suppose that second-order representations enter the A1 state violates the spirit of the original and revised SOP models.

A similar argument could be made regarding the Van Hamme and Wasserman (1994) model. As the model was formulated, absent stimuli have a negative associability. To account for the present observations, the model might be modified to assume that associatively activated, but physically absent stimuli carry a positive or negative associability that flip-flops alternatively with each successive order of activation (representations of physically present cues being positive, first-order retrieved representations being negative, and second-order retrieved representations being positive). We cannot refute this alternative account, but we leave

[5] Although inflation of CS X's effective association with the US increased CS B's inhibitory properties, previous attempts at inflation effects have been less successful (e.g., Miller, Hallam, & Grahame, 1990; but see Denniston et al., 1996; Miller & Matute, 1996). However, prior attempts have been unsuccessful only when the target cue was biologically significant prior to the inflation manipulation. In the current experiments, CS B had never been paired with the US, and thus was not biologically significant at the time of inflation treatment.

it up to those authors to contemplate such an extension of their theory.

It has been suggested that inhibition to CS B might ordinarily be masked in the relative stimulus validity design by collateral excitation through a secondary link with CS A. Hall (1996) provided converging evidence for the mediation of excitation between stimuli through their associations with a common element. Applied to the relative validity design, CS A and CS B could form an excitatory association by way of the common X element. This mediated link would confer excitatory value from CS A to CS B. Extinction of CS A might then unmask inhibition to CS B, which provides an alternative explanation for the inhibition observed in Group RV.A relative to Group RV (see Figures 4 and 5). Although there is nothing in our data to refute such an explanation, we do not find it very tenable. In our lab, we have found second-order relationships to be excitatory when few trials (e.g., 8) are given, whereas inhibitory relationships predominate after many trials (e.g., 48; Yin, Barnet, & Miller, 1994). Because our subjects received 72 AX and 72 BX trials, we would expect an inhibitory relationship to develop between CS A and CS B. An inhibitory A–B relationship should have no adverse effect on the difference in inhibition to X in these two groups.

In conclusion, the performance-focused extended comparator hypothesis can account for the empirical retrospective-revaluation effects observed in these experiments (i.e., recovery from the relative validity effect, as well as the enhanced inhibitory status of CS B through the massive posttraining extinction of a so-called second-order comparator stimulus). These findings are inexplicable in the frameworks of acquisition-focused retrospective revaluation models as currently formulated, such as those of Dickinson and Burke (1996) and Van Hamme and Wasserman (1994).

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