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Posttraining Shifts in the Overshadowing Stimulus–Unconditioned Stimulus Interval Alleviates the Overshadowing Deficit

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ABSTRACT

Two conditioned lick suppression experiments explored the effects on overshadowing of a posttraining change in the temporal relationship between the overshadowing conditioned stimulus (CS) and the unconditioned stimulus (US). Rats received either trace (Experiment 1) or delay (Experiment 2) overshadowing training. Then pairings of the overshadowing CS and US were given with either a trace or delay temporal relationship. Overshadowing was alleviated by shifting the overshadowing CS–US temporal relationship so that it no longer matched the overshadowed CS–US temporal relationship. These outcomes are explicable in terms of an integration of the comparator hypothesis, which states that cue competition effects (e.g., overshadowing) will be maximal when the information potentially conveyed by competing CSs is equivalent, and the temporal coding hypothesis, which states that CS–US intervals are part of the information encoded during conditioning.

ARTICLE

The *comparator hypothesis* (Miller & Matzel, 1988; Miller & Schachtman, 1985) is a rule for the retrieval and expression of Pavlovian associations, which posits that responding to a conditioned stimulus (CS) is modulated by a comparison of its associative strength to the associative strength of other salient cues that are strongly associated with the CS (i.e., typically, other salient cues that were present during training with the CS, which we hereafter call *comparator stimuli*). More specifically, responding to a CS is assumed to be directly related to the strength of the CS–US (unconditioned stimulus) association and inversely related to the product of the CS–comparator stimulus association and the comparator stimulus–US association. For example, in overshadowing, in which a more salient CS (A) that is present during the training of a less salient CS (X) attenuates conditioned responding to X, A is X's comparator stimulus at test due to its presence as the most salient stimulus during training of X. Notably, the presence of A during training does not interfere with acquiring an X–US association;

rather, it interferes with expression of that association. Stimulus saliency and training history have been shown to influence which stimulus will serve as the comparator stimulus for a given CS (e.g., Grahame, Barnet, & Miller, 1992; Miller, Esposito, & Grahame, 1992).

Important to note, the comparator hypothesis states that the critical comparison between associations to a target CS and to its comparator stimulus occurs at the time of testing (Miller & Matzel, 1988). Consequently, changes in the associative status of the comparator stimulus after termination of target CS training and before testing should influence responding to the target CS. Supportive of this view, posttraining associative deflation of the comparator stimulus– US association (i.e., extinction of the comparator stimulus) has been found to enhance responding to the target CS (Blaisdell, Gunther, & Miller, in press; Cole, Barnet, & Miller, 1995a; Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985; Matzel, Shuster, & Miller, 1987), and posttraining associative inflation of the comparator stimulus (i.e., reinforcement of the comparator stimulus) has been found to attenuate responding to the target CS (e.g., Denniston, Miller, & Matute, 1996; Miller & Matute, 1996). Thus, the comparator hypothesis appears accurate in positing that responding to a target CS is inversely related to the strength of the comparator stimulus–US association. However, the comparator hypothesis anticipates this attenuation in conditioned responding only when the US predicted by the comparator stimulus is qualitatively similar to the US predicted by the target CS. Consequently, a posttraining change in any attribute of the US predicted by the comparator stimulus relative to the US predicted by the target CS should attenuate cue competition. For example, Blaisdell, Denniston, & Miller (1997) found that blocking was attenuated if different USs were used in Phase 1 and Phase 2 of blocking treatments, even if the two USs were equated for reinforcement value. Although the effects of the magnitude and type of US associated with a CS's comparator stimulus have been examined, little attention has been given to how temporal attributes of a comparator stimulus–US association might influence its potential to compete with the target CS–US association. According to the comparator hypothesis, cue competition will be maximal when the competing cues convey the same information as the target CS about their common outcome (usually the US) with which they have been paired.

Cues can convey many different types of information about an outcome. Well known among these are whether the outcome will occur and the qualitative and quantitative attributes of the outcome. The *temporal coding hypothesis* posits that the temporal location of the outcome relative to its cue is also part of the information encoded within the cue–outcome association (Barnet, Arnold, & Miller, 1991; Matzel, Held, & Miller, 1988; Miller & Barnet, 1993). More specifically, the temporal coding hypothesis states (a) Contiguity is sufficient for the formation of an association; (b) the temporal relationship between the associates is automatically encoded as part of the association; (c) this temporal information plays a critical role in determining the form and timing of the associatively based response; and (d) the temporal relationships between multiple cues can be integrated to yield temporal relationships between events that were never paired. In contrast with other temporal coding theories that focus on how organisms perceive time and examine behavior primarily to better understand the mechanisms of timing (e.g., Church, 1984; Desmond & Moore, 1988; Gibbon, 1977; Honig, 1981; Killeen & Fetterman, 1988), the temporal coding hypothesis focuses on how organisms use their timing ability to modulate behavior. Prior research has found that, if a target CS and its competing (i.e., comparator) stimulus have different temporal relationships with the US, cue competition (e.g., overshadowing, Blaisdell, Denniston, & Miller, 1998; blocking, Barnet, Grahame, & Miller, 1993; Schreurs & Westbrook, 1982; but see Maleske & Frey, 1979) is reduced relative to when both cues have the same temporal relationship with the US.

The studies described above found cue competition to depend on the target CS and its competing stimulus having the same temporal relationship to the US. More recently, Denniston, Blaisdell, and Miller (1998) suggested that conditioned inhibition could be altered by posttraining modification of the temporal relationship between the excitatory CS used in inhibition training (i.e., the inhibitor's comparator stimulus) and the US. The present research extends this paradigm to cue competition. We studied the effect on cue competition (specifically, overshadowing) of a posttraining shift in the temporal relationship between an overshadowing CS (i.e., the overshadowed CS's comparator stimulus) and US (i.e., updating the overshadowing stimulus–US temporal relationship), whereas the temporal relationship of the overshadowed (i.e., the target) CS and US was left unchanged. If the overshadowed CS and the overshadowing stimulus share the same temporal relationship with the US as a result of initial training (i.e., AX → US), then updating the overshadowing stimulus–US temporal relationship so that it no longer matches the overshadowed CS–US temporal relationship should reduce the overshadowing stimulus's potential to compete with the overshadowed

CS. Thus, after completion of overshadowing training in which A and X have the same temporal relationship to the US and the presence of A during training of X is seen to impair responding to X, updating the temporal relationship between A and the US, such that the A–US temporal relationship is now different from the X–US temporal relationship, should reduce the effectiveness of A in overshadowing X. This prediction is based on the validity of the following two assumptions: (a) the comparator hypothesis's view that responding to the target (i.e., overshadowed) CS is inversely related to the strength of the association between the CS's comparator (e.g., overshadowing) stimulus and the US at the time of testing, and (b) the temporal coding hypothesis' view that the temporal relationship between the CS and the US is part of the information encoded within an association (also see Gibbon & Balsam, 1981).

In the present research, we gave overshadowing treatment with either a trace (Experiment 1) or a delay (Experiment 2) relationship between the competing cues (A and X) and the US in Phase 1, followed by temporal updating training of A with a delay or trace relationship with the US in Phase 2. In the conjoint framework of the comparator hypothesis and the temporal coding hypothesis, dissimilar A–US intervals in Phases 1 and 2 should enhance responding to X (i.e., reduce overshadowing of X) relative to groups for which the A–US interval remains unchanged between Phase 1 (i.e., overshadowing treatment) and Phase 2. Such an outcome would support the notion that cue competition effects are dependent on (among other factors) the current status of the temporal attributes of the overshadowed CS and the overshadowing stimulus with respect to the US at the time of testing.

Reduced overshadowing by updating the A–US temporal relationship would also provide evidence for an expression deficit, rather than an acquisition deficit, account of cue competition effects. Acquisition deficit accounts (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) posit that the overshadowing effect is a failure to encode a target CS–US association. If this were the case, temporal updating treatment of A (as well as any other modification of the A–US association) should not enhance responding to X (i.e., reduce overshadowing). However, if the overshadowing effect results from a failure to express an acquired target CS–US association, then shifting the temporal relationship between A and the US away from that between X and the US, such that A and X no longer convey the same information, may restore responding to X. Updating of the temporal relationship between a competing stimulus and a US (presumably a qualitative change in the A–US association) is in some ways analogous to posttraining quantitative changes of the competing stimulus–US association (i.e., effectively decreasing or increasing the strength of the target CS–US association). Decreasing the strength of this association has been demonstrated to enhance responding to the target CS (e.g., see Blaisdell, Gunther, & Miller, in press; Cole et al., 1995a; Kaufman & Bolles, 1981; Matzel et al., 1985; Matzel et al., 1987) and increasing the strength of this association has been demonstrated to attenuate responding to the target CS (e.g., Denniston et al., 1996; Miller & Matute, 1996). Thus, it would not be surprising if a posttraining manipulation of the temporal attributes of a competing stimulus also affects the way it modulates responding to the target CS. Note, however, that in the framework of the comparator hypothesis posttraining changes in the strength of the comparator stimulus–US association presumably influence the outcome of the comparison, whereas changes in the nature of the US associated with the CS's comparator stimulus (e.g., ice water vs. footshock in Blaisdell et al., 1997), or when the US is expected following the CS's comparator stimulus presumably influence the degree to which the comparator process is activated; comparator processes are presumably most effective when the US representations being compared are qualitatively similar and expected at the same time after their respective CSs.

Experiment 1

Experiment 1 manipulated the A–US temporal relationship following overshadowing training (i.e., AX → US, where A is more salient than X). Specifically, in Phase 1 we presented the AX stimulus compound in a trace relationship with the US (i.e., AX $\xrightarrow{5}$US; where 5 represents the gap [in seconds] between CS termination and US onset). Subsequently, in Phase 2 further A–US pairings were given with either a trace (A $\xrightarrow{5}$US) or a delay (A $\xrightarrow{0}$US) relationship between A and the US (see Table 1). This updating of the A–US temporal relationship, so that it no longer matched the X–US temporal relationship that was established during Phase 1, was expected to attenuate the overshadowing effect. Thus, stronger responding to X was expected after Phase

2 A $\xrightarrow{0}$US treatment than after A $\xrightarrow{5}$US treatment. In some sense, this prediction is counterintuitive because changing A from being a trace CS to a delay CS, if anything, should have enhanced responding to A, and consequently might be expected to reduce responding to X. But integration of the comparator hypothesis with the temporal coding hypothesis suggests that this revaluation of A should enhance responding to X.

Table 1
Design of Experiment 1

Group	Phase 1	Phase 2	Test
Acq.Same	X $\xrightarrow{5}$ *US*	A $\xrightarrow{5}$ *US*	X
Acq.Diff	X $\xrightarrow{5}$ *US*	A $\xrightarrow{0}$ *US*	X
OV.Same	AX $\xrightarrow{5}$ *US*	B $\xrightarrow{5}$ *US*	X
OV.Diff	AX $\xrightarrow{5}$ *US*	B $\xrightarrow{0}$ *US*	X
Update.Same	AX $\xrightarrow{5}$ *US*	A $\xrightarrow{5}$ *US*	X
Update.Diff	AX $\xrightarrow{5}$ *US*	A $\xrightarrow{0}$ *US*	X
GenDec.Same	X $\xrightarrow{5}$ *US*	B $\xrightarrow{5}$ *US*	AX
GenDec.Diff	X $\xrightarrow{5}$ *US*	B $\xrightarrow{0}$ *US*	AX

Note. Acq = acquisition control treatment; OV = overshadowing treatment with an alternate stimulus updated in Phase 2; Update = overshadowing treatment with the overshadowing stimulus updated in Phase 2; GenDec = generalization decrement control; Same = temporal relationship between A and unconditioned stimulus (US) during Phase 2 was the same as that of Phase 1; Diff = temporal relationship between A and US during Phase 2 was different than that of Phase 1; A = overshadowing stimulus; X = overshadowed stimulus; B = alternate stimulus. Superscripts (0 or 5) denote duration of gap (in seconds) between conditioned stimulus (CS) termination and US onset.

Design of Experiment 1

In Experiment 1, Condition Acquisition (Acq; i.e., Groups Acq.Same and Acq. Different [Diff]) served to demonstrate acquisition of conditioned responding to Stimulus X, against which overshadowing (OV) would be measured. Condition OV (i.e., Groups OV.Same and OV.Diff) was intended to demonstrate overshadowing when overshadowing treatment was followed by no further A–US training. These groups served as controls in assessing Condition Update (Groups Update.Same and Update.Diff) in which the temporal relationship between A and the US was manipulated following overshadowing treatment. For Group Update.Same, the Phase 2 pairings of A and the US occurred with the same temporal relationship as in Phase 1. However, for Group Update.Diff, the Phase 2 pairings of A and the US occurred with a different temporal relationship than in Phase 1. We expected this last group to exhibit a reduction in overshadowing relative to Group Update.Same. Finally, control Condition Generalization Decrement (GenDec; consisting of Groups GenDec.Same and GenDec.Diff) was included to show that any overshadowing in the other groups was not due to the failure to generalize from AX in training to X in testing (i.e., generalization decrement).

Method

Subjects

Thirty-six male (300–455 g) and 36 female (210–320 g) experimentally naive, Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Subjects were individually housed in wire-mesh cages in a vivarium maintained on a 16-hr light–8-hr dark cycle. Experimental manipulations occurred approximately midway through the light portion of the cycle. A progressive water

deprivation schedule was imposed during the week prior to the beginning of the experiment, until water availability was limited to 30 min per day at the beginning of the study. All animals were handled three times per week for 30 s from weaning until the beginning of the study. Subjects were randomly assigned to one of eight groups, *n*s = 12 for Groups Update.Same and Update.Diff, and *n*s = 8 for Groups Acq.Same, Acq.Diff, OV.Same, OV.Diff, GenDec.Same, and GenDec.Diff. Groups were counterbalanced for sex.

Apparatus

Two types of experimental chambers were used. Chamber R was rectangular in shape and measured 24 × 9 × 16 cm (length × width × height). The walls and ceiling of the chamber were constructed of clear Plexiglas, and the floors were constructed of stainless steel rods measuring 0.5 cm in diameter, spaced 1.5 cm center-to-center, connected by NE-2 neon bulbs, which allowed for the delivery of constant-current footshock produced by a high-voltage AC circuit in series with a 1.0-MΩ resistor. Each of six copies of Chamber R were housed in separate sound- and light-attenuating environmental isolation chests. Enclosure R was dimly illuminated by a 2-W (nominal at 120 VAC) driven at 60 VAC incandescent house light mounted on an inside wall of the environmental chest, approximately 30 cm from the experimental chamber.

Chamber V was a 25.5-cm long box in the shape of a vertical truncated V. The chamber was 28 cm high, 21 cm wide at the top, and narrowed to 5.25 cm wide at the bottom. The ceiling was constructed of clear Plexiglas, the front and back walls were black Plexiglas, and the side walls were stainless steel. The floor consisted of two 25.5-cm long parallel metal plates, each 2 cm wide and separated by a 1.25-cm gap. A constant-current footshock could be delivered through the metal walls and floor of the chamber. Each of six copies of Chamber V were housed in separate sound- and light-attenuating environmental isolation chests. Chamber V was illuminated by a 7-W (nominal at 120 VAC) bulb driven at 60 VAC. The bulb was mounted on the inside wall of the environmental chest, approximately 30 cm from the center of the experimental chamber, with the light entering the chamber primarily by reflection from the ceiling of the environmental enclosure. The light intensities in the two chambers were approximately equal; that is, the difference in the opaqueness of the walls in Chambers R and V compensated for the difference in the size of the house lights.

Each chamber (R and V) could be equipped with a water-filled lick tube. When inserted, the lick tube extended 1 cm from the rear wall of a cylindrical drinking recess that was set into one of the narrow Plexiglas walls of the chamber. The recess was 4.5 cm in diameter (axis perpendicular to the rear wall) and 5 cm deep. An infrared photobeam was projected horizontally across the recess, 1 cm in front of the lick tube. To drink from the tube, a rat had to insert its head into the recess, thereby breaking the photobeam. Thus, we could monitor when the rats were accessing the lick tube. Three 45-Ω speakers mounted on the inside walls of each environmental chest could deliver either a high-frequency complex tone (3000 and 3200 Hz) 10 dB(C) above background, a white noise 10 dB(C) above background, or a 6/s click-train stimulus 4 dB(C) above background. All CSs were 10 s in duration. The US was a 0.5-s, 1.3-mA footshock that could be delivered through the chamber floors. Ventilation fans in each enclosure provided a constant 76-dB(C) re. SPL background noise. The tone and the noise served as CSs A and B (the overshadowing stimulus and alternate [i.e., control] stimulus, respectively) counterbalanced. The clicks served as CS X, the overshadowed stimulus.

Context 1 consisted of Enclosures R or V (counterbalanced within groups), illuminated by their house lights. Context 2 consisted of Enclosures V or R (for each subject the opposite context to that used as Context 1), with the house light turned off and the addition of a methyl salicylate odor cue (two drops applied to a block of wood inside each enclosure). Additionally, the water-filled lick tube was never present in Context 1, but was always present in Context 2.

Procedure

Table 1 summarizes the critical aspects of the procedure along with specific group names. All sessions except testing were 50 min in duration.

Acclimation

On Day 1, all animals were acclimated to Context 1 with no nominal stimulus presentations during the session. On Day 2, subjects were acclimated to Context 2 as they were to Context 1 on Day 1 except that lick tubes were present.

Phase 1 (overshadowing treatment)

On Days 3 and 4, all subjects received four daily CS–US pairings in Context 1, with a 5-s interval (i.e., temporal gap) between CS termination and US onset. For Groups Update.Same, Update.Diff, OV.Same, and OV.Diff, the CS was a compound of A and X with these cues having simultaneous onset and termination. Groups Acq.Same, Acq.Diff, GenDec.Same, and GenDec.Diff received the same treatment except that X alone served as the CS (see Table 1). During each session, these trials were pseudorandomly distributed with a mean (±range) intertrial interval of 12 ± 2 min.

Phase 2 (updating of A–US temporal relationship)

On Days 5 through 10, animals in Condition Same received four daily presentations of A followed by US onset 5 s after CS termination (i.e., a 5-s gap) in Context 1, for a total of 24 A–US trials. Animals in Condition Diff received the same number and distribution of A–US pairings, but A was followed by the US immediately on CS termination (i.e., a 0-s gap).

Reacclimation

On Days 11 and 12, subjects were allowed, in Context 2, to access the lick tubes during each daily session to reestablish baseline levels of drinking, which are typically disrupted by footshock-induced generalized fear. During these sessions, there were no nominal stimulus presentations.

Testing

On Day 13, all subjects were tested in Context 2. Testing was performed in a different context than training because differences between conditions in training may have resulted in the training context acquiring different amounts of associative strength, which in turn could have confounded the dependent variable if testing had occurred in the training context. Subjects in Conditions Acq, OV, and Update were tested for conditioned lick suppression to X (the overshadowed CS) by presenting the stimulus on completion of 5 cumulative s of drinking (as measured by the total amount of time the infrared photobeam was disrupted). Thus, all rats were drinking at the time of CS onset. Times to complete this initial 5 cumulative s of licking in the absence of the test CS and times to complete an additional 5 cumulative s of licking in the presence of the CS were recorded. Subjects in Condition GenDec were tested on the AX compound in a manner similar to that used for testing X.

In all of the experiments reported, conditioned lick-suppression data were transformed to log (base 10) scores to better fit the assumption of parametric statistics concerning normal distributions of scores within groups. An alpha level of .05 (two-tailed) was adopted for all tests of statistical significance. Data from one rat (from Group OV.Diff) were lost due to equipment failure.

Results

The central observation from Experiment 1 was that overshadowing of Stimulus X by A was observed in Group Update.Same, in which Stimulus A maintained in Phase 2 the same temporal relationship to the US that it had had in Phase 1, but was not observed in Group Update.Diff, in which the temporal relationship between Stimulus A and the US was shifted in Phase 2 from the A–US temporal relationship that had been established during Phase 1. Overshadowing was also observed in Groups OV.Same and OV.Diff in which B rather than A was paired with the US in Phase 2. Groups GenDec.Same and GenDec.Diff did not evidence overshadowing. These outcomes are illustrated in Figure 1.

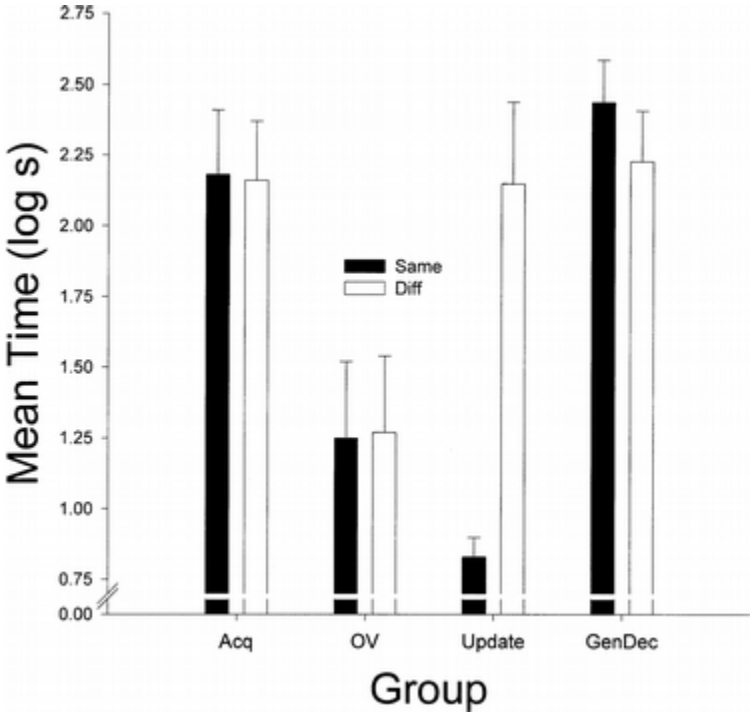

Figure 1. Experiment 1: Mean times (log s) to lick for 5 cumulative s in the presence of X (Conditions Acquisition [Acq], overshadowing [OV], and Update) or the AX compound (Condition Generalization Decrement [GenDec]). Error bars represent standard errors of means

A one-way analysis of variance (ANOVA) conducted on pre-CS time to complete 5 cumulative s of licking revealed no group differences, $F(7, 63) < 1.0$, $p > .60$. Moreover, no subject took longer than 60 s to complete these 5 cumulative s of licking.

A one-way ANOVA conducted on time to complete 5 cumulative s of licking in the presence of the test CS found an effect of treatment, $F(7, 63) = 9.79$, $p < .001$. A 2 × 2 ANOVA with Phase 1 treatment (AX or X) and Phase 2 treatment (0- or 5-s CS gap) as factors conducted on suppression scores for Groups Update.Same, Update.Diff, Acq.Same, and Acq.Diff revealed a main effect of Phase 1 treatment, $F(1, 36) = 14.01$, $p < .001$, a main effect of Phase 2 treatment, $F(1, 36) = 12.92$, $p < .001$, and an interaction between Phases 1 and 2 treatment, $F(1, 36) = 13.56$, $p < .001$. Planned comparisons were conducted, using the overall error term from the one-way ANOVA, to determine the source of these effects. Groups Acq.Diff and Acq.Same, Groups OV.Diff and OV.Same, and Groups GenDec.Diff and GenDec.Same did not differ from each other, Fs < 1.0, and thus the scores from the first two of these groups were pooled to create Group Acq ($n = 16$), the scores from the second two groups were pooled to create Group OV ($n = 15$), and scores from the last two groups were pooled to create Group GenDec, ($n = 16$) for the purpose of further analysis. A one-way ANOVA conducted on suppression scores of Groups Update.Same, Update.Diff, Acq, OV, and GenDec revealed an effect of treatment, $F(4, 66) = 17.66$, $p < .001$. Additional planned comparisons using the error term from this last one-way ANOVA found that Group OV suppressed less than did Group Acq, $F(1, 66) = 18.93$, $p < .001$, demonstrating overshadowing with our preparation. Group GenDec suppressed more to the AX compound than did Group OV, $F(1, 66) = 26.50$, $p < .001$, suggesting that the overshadowing effect in this situation was not due to stimulus generalization decrement. More important, Group Update.Diff suppressed more to X than did Group Update. Same, $F(1, 66) = 30.91$, $p < .001$, indicating that shifting A from being a trace excitor to being a delay excitor produced recovery from overshadowing. The apparent complete recovery from overshadowing observed in Group Update.Diff (compared to the Acq groups) was rather surprising to us. Surely, similarity of the X–US and A–US associations has dimensions in addition to their temporal relationships with the US on which they did not differ. Hence, we did not expect complete recovery from overshadowing. At this time, we merely note the completeness of recovery and have no comments to make concerning it.

Discussion

We interpreted greater conditioned responding by Group Update.Diff relative to Group Update.Same as arising from the change in the temporal relationship between Overshadowing Stimulus A (X's putative comparator stimulus) and the US during Phase 2 such that, at test the A–US temporal relationship was different than the X–US temporal relationship. Blaisdell, Denniston, and Miller (1998) and Barnet et al. (1993) have previously demonstrated that a competing (i.e., comparator) stimulus that is initially trained with a different temporal relationship with the US than the target CS causes little attenuation of responding to the target CS, whereas an otherwise equivalent competing stimulus initially trained with the same temporal relationship with the US as the target CS does attenuate responding to the target CS. Thus, consistent with the temporal coding hypothesis, associative information appears to include the temporal relationship between associates; and, consistent with the comparator hypothesis, cue competition appears to be maximal between cues that predict outcomes that are the same in all of their attributes (including temporal relationships). Notably, the comparator hypothesis without the temporal coding hypothesis would view the revaluing of A from a trace excitor to a delay excitor as a form of association inflation of the comparator (i.e., competing) stimulus for CS X. Consequently, a decrease in responding to X would be anticipated. But, when the requirement that an effective comparator stimulus must have the same relationship to the US as the target CS is considered, and temporal relationships are recognized as part of every association, then the presently observed increase in responding to X becomes explicable.

An alternative explanation of the greater responding observed in Group Update.Diff relative to Group Update. Same in Experiment 1 is that, although X may have been successfully overshadowed by A in both groups, the amount of suppression that was observed could have been due to second-order conditioning. Suppression to X was possibly mediated by associations between X and A (due to Phase 1 compounding of A and X) and between A and the US, the latter of which might have been differentially strengthened during Phase 2, because delay conditioning is more effective in controlling behavior than is trace conditioning. This second-order conditioning interpretation can, therefore, explain the difference in responding between Groups Update.Same and Update.Diff. During Phase 1, A's associative strength may have approached a common asymptote. However, in Phase 2 A's associative strength may have asymptoted at a smaller value in Group Update.Same than in Update.Diff due to A being a trace excitor in the former group and a delay excitor in the latter group. As a result, responding to X mediated by its association with A would be expected to be greater in Group Update.Diff, in which A had greater associative strength, than in Group Update.Same.

Furthermore, a third explanation for the outcome of Experiment 1 that is closely related to the second-order conditioning explanation can be built on the acquisition models proposed by Holland (Holland, 1981, 1983; Holland & Forbes, 1982) and more recently investigated by Hall (1996) to account for some instances of retrospective revaluation. (Retrospective revaluation refers to posttraining changes in a CS's associative value on trials during which the CS is not presented.) In these models, active representations of absent stimuli have positive associability. Applied to our situation, these researchers might argue that presentations of CS A during Phase 2 activated the representation of the X stimulus. In this framework, the activated representation of X during the A–US pairings might have strengthened the X–US association. For Group Update.Diff, not only should A have become a delay excitor, but so should have the X representation. Delay conditioning is known to be more effective than trace conditioning (Pavlov, 1927); thus, stronger responding to X would be expected in Group Update.Diff than in Group Update.Same. Group Update. Same received trace conditioning of A in Phase 2, which should have resulted in a relatively weak X–US representation-mediated association (see Holland, 1981, Experiment 1 for evidence that representation-mediated conditioned responding is sensitive to temporal parameters). However, in Holland's representation-mediated effects as well as most sensory preconditioning and second-order conditioning preparations, the conditioned stimuli were presented serially, whereas in the present study they were presented simultaneously. Furthermore, in conventional sensory preconditioning and second-order conditioning preparations, the CSs are paired in the absence of the US. In the present study both stimuli were paired with the US prior to the A–US pairings. Thus, there are a number of differences between Holland and Hall's designs and our design that raise questions concerning the applicability of their conclusions

to Experiment 1. Nevertheless, retrospective revaluation in which active representations of absent stimuli have positive associability appears to provide a viable account of the outcome of Experiment 1.

In sum, there are at least three explanations for the greater responding in Group Update.Diff relative to Group Update. Same that was observed in Experiment 1: (a) the one based conjointly on the comparator hypothesis and temporal coding hypothesis, (b) second-order conditioning, and (c) retrospective revaluation. Experiment 2 was designed to differentiate between these three interpretations.

Experiment 2

Experiment 2 used the same design as Experiment 1 except for two changes. First, during Phase-1 training, all cues were trained with a delay instead of trace (5-s gap) excitatory conditioning procedure. That is, a 0-s gap between CS termination and US onset (AX $\xrightarrow{0}$US) was used. Phase 2 training remained as it was in Experiment 1(i.e., A $\xrightarrow{5}$US or A $\xrightarrow{0}$US). Second, Groups GenDec (Same and Diff) were not included because the failure of generalization decrement as a viable account of overshadowing in our preparation was demonstrated in Experiment 1.

In Experiment 2, the comparator hypothesis and the temporal coding hypothesis conjointly predicted greater responding to X when in Phase 2 A was made a trace excitor than when A was maintained as a delay excitor. This prediction was based on the fact that in the Phase 2 trace condition (i.e., Group Update.Diff) the X–US and A–US temporal relationships were different at the time of testing (i.e., A and X predicted the US at different times). However, if responding to X by Group Update.Diff of Experiment 1 reflected second-order conditioning to A or representation-mediated conditioning to X, then we would expect the opposite outcome. In either of these frameworks, X should reflect changes in the A–US relationship. Thus, X should also become a delay excitor in Group Update.Same and a trace excitor in Group Update.Diff, due to the within-compound association which presumably underlies second-order conditioning or representation-mediated conditioning. Delay excitors are known to support stronger first- and second-order conditioned responding than are trace excitors (e.g., Ellison, 1964; Kamin, 1954, 1965; Pavlov, 1927; but see Cole, Barnet, & Miller, 1995b). Consequently, greater responding to X when A was maintained as a delay excitor than when A was made a trace excitor would be expected.

Method

Subjects and Apparatus

Twenty-four male (210–335 g) and 24 female (170–225 g) experimentally naive, Sprague–Dawley descended rats (*Rattus norvegicus*), bred in our colony from Holtzman stock, served as subjects. Animals were randomly assigned to one of six groups (*n*s = 12 for Groups Update.Diff and Update.Same; *n*s = 6 for Groups Acq.Diff, Acq.Same, OV.Diff, and OV.Same), counterbalanced for sex. The animals 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 procedure along with specific group names. All training sessions were 50 min in duration.

Table 2
Design of Experiment 2

Group	Phase 1	Phase 2	Test
Acq.Same	$X \xrightarrow{0} US$	$A \xrightarrow{0} US$	X
Acq.Diff	$X \xrightarrow{0} US$	$A \xrightarrow{5} US$	X
OV.Same	$AX \xrightarrow{0} US$	$B \xrightarrow{0} US$	X
OV.Diff	$AX \xrightarrow{0} US$	$B \xrightarrow{5} US$	X
Update.Same	$AX \xrightarrow{0} US$	$A \xrightarrow{0} US$	X
Update.Diff	$AX \xrightarrow{0} US$	$A \xrightarrow{5} US$	X

Note. Acq = acquisition control treatment; OV = overshadowing treatment with an alternate stimulus updated in Phase 2; Update = overshadowing treatment with the overshadowing stimulus updated in Phase 2; Same = temporal relationship between A and unconditioned stimulus (US) during Phase 2 was the same as that of Phase 1; Diff = temporal relationship between A and US during Phase 2 was different than that of Phase 1; A = overshadowing stimulus; X = overshadowed stimulus; B = alternate stimulus. Superscripts denote duration of gap (in seconds) between conditioned stimulus (CS) termination and US onset.

Design of Experiment 2

Acclimation

On Days 1 and 2, subjects were acclimated to Contexts 1 and 2 as in Experiment 1.

Phase 1

On Days 3 and 4, subjects received the same overshadowing treatment as in Experiment 1 except that the interval between termination of the CSs and onset of the US was 0 s (i.e., delay conditioning).

Phase 2, reacclimation, and testing

Phase 2 (updating of the A–US temporal relationship), reacclimation, and testing were conducted as in Experiment 1. Note that *Same* and *Different* refer to the CS–US temporal interval during Phase 2 relative to Phase 1 (see Table 2).

Results

The central observation from Experiment 2 was that overshadowing of Stimulus X by A was observed in Group Update.Same, but not in Group Update.Diff. Thus, posttraining updating of the A–US temporal relationship appears to have recovered responding to X even under conditions that precluded interpretation in terms of second-order conditioning or retrospective revaluation. Figure 2 depicts the test data from this experiment.

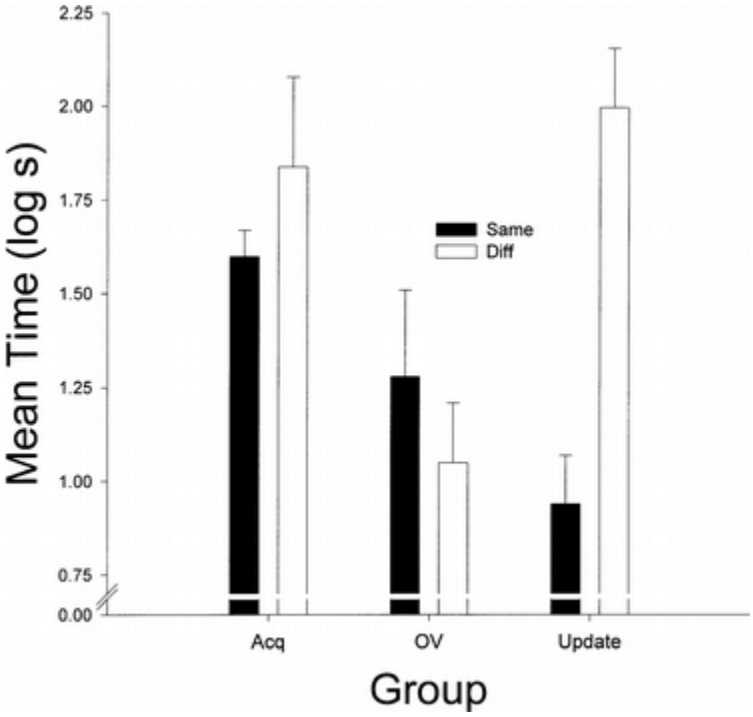

Figure 2. Experiment 2: Mean times (log s) to lick for 5 cumulative s in the presence of X. Error bars represent standard errors of means. Acq = acquisition; OV = overshadowing

A one-way ANOVA conducted on pre-CS time to complete 5 cumulative s of licking revealed no group differences, $F(5, 42) < 1.0$, $p > .80$. Moreover, no subject took longer than 60 s to complete these 5 cumulative s of licking.

A one-way ANOVA conducted on time to complete 5 cumulative s of licking in the presence of the test CS revealed an effect of treatment, $F(5, 42) = 7.72$, $p < .001$. A 2 × 2 ANOVA with Phase 1 treatment (AX or X) and Phase 2 treatment (0- or 5-s CS gap) as factors conducted on suppression scores for Groups Update.Same, Update.Diff, Acq.Same, and Acq.Diff revealed a main effect of Phase 2 treatment, $F(1, 32) = 14.88$, $p < .001$, and an interaction between Phases 1 and 2 treatment, $F(1, 32) = 5.84$, $p < .05$. The main effect of Phase 1 treatment was not significant, $F(1, 32) = 2.10$, $p > .15$. Planned comparisons were conducted, using the overall error term from the one-way ANOVA, to determine the source of these effects. Groups Acq.Diff and Acq.Same, and Groups OV.Diff and OV.Same did not differ from each other, Fs < 1.0, and thus the scores from the first two of these groups were pooled to create Group Acq and the scores from the latter two groups were pooled to create Group OV, (ns = 12) for the purpose of further analysis. A one-way ANOVA conducted on suppression scores of Groups Update.Diff, Update.Same, Acq, and OV revealed an effect of treatment, $F(3, 44) = 12.54$, $p < .001$. Additional planned comparisons using the error term from this last one-way ANOVA found that Group OV suppressed less than did Group Acq, $F(1, 44) = 7.93$, $p < .01$, thereby demonstrating overshadowing with our preparation. Group Update.Diff differed from Group Update.Same, indicating that shifting A from a delay excitor to a trace excitor produced recovery from overshadowing, $F(1, 44) = 29.23$, $p < .001$.

Discussion

In Experiment 2, the overshadowing deficit was attenuated (i.e., more responding to X was observed) when A was updated with a trace relationship to the US (i.e., Group Update.Diff). In both the second-order

conditioning and the retrospective revaluation frameworks, the amount of A-mediated responding to X observed in Group Update.Diff of Experiment 2 would be expected to decrease relative to A-mediated responding to X by the group in which A retained a delay association with the US (i.e., Group Update.Same). However, exactly the opposite outcome was observed. As can be seen in Figure 2, Group Update.Diff suppressed as strongly to X as did Group Acq.Diff, whereas Group Update.Same suppressed less than did Group Acq.Same. These outcomes argue against the second-order mediated excitation and the retrospective revaluation (i.e., representation-mediated conditioning) interpretations of Experiment 1.

Attenuation of the overshadowing effect resulted from changing the A–US temporal relationship so that it differed from the X–US temporal relationship. This observation is consistent with the view of the comparator hypothesis that comparator (i.e., competing) stimuli are maximally effective when they predict a US with all the same attributes as the US predicted by the target CS. Moreover, this finding is also consistent with the temporal coding hypothesis that interevent temporal relationships are part of what is encoded in an association. After delay overshadowing training, updating A so that it became a trace excitor disrupted its ability to overshadow the delay X stimulus. This account of the present results is predicated on the fourth tenet of the temporal coding hypothesis, namely that temporal information provided by different cues that were present during training will be integrated at test to determine the form and timing of the associative-based response to the target CS.

An alternative account of the results of Experiment 2 is provided by acquisition models that allow retrospective revaluation and in which active representations of absent cues have negative associability (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994). If a delay US has a higher value of λ (maximum associative value supportable by the US) than does the same US in a trace relationship to the CS, then A $\xrightarrow{5}$ US trials during Phase 2 may have actually decreased the associative strength of A in Group Update.Diff, but not in Group Update.Same. Furthermore, in the terminology of the Van Hamme and Wasserman (1994) model, during Phase 2 the presentation of A $\xrightarrow{5}$ US to Group Update.Diff might have activated a representation of X (with a negative associability because X was not physically present) and a value of $(\lambda - V_A)$, which is negative because λ is a low positive number based on the trace US of Phase 2 and V_A is a larger positive number based on the pairings of A with a delay US in Phase 1. As the product of two negative numbers is positive, the Van Hamme and Wasserman model (as well as that of Dickinson & Burke, 1996) predicts an increase in the associative strength of X for Group Update. Diff. This is not the case for Group Update.Same, for which $(\lambda - V_A)$ during Phase 2 should be a positive number because λ is a large positive number based on the delay US of Phase 2. Thus, acquisition models that allow retrospective revaluation with active representations of physically absent cues having negative associability can explain the results of Experiment 2. However, the results of Experiment 1, in which the trace overshadowing stimulus was in Phase 2 transformed into a delay CS to produce recovery from overshadowing, cannot be explained by acquisition models of retrospective revaluation such as that of Van Hamme and Wasserman. According to these models, the delay conditioning of A in Group Update.Diff during Phase 2 of Experiment 1 should have strengthened the A–US association and weakened the X–US association, which is contrary to the recovery from overshadowing of X that was observed in that group. In contrast, the other family of acquisition models that were designed to explain retrospective revaluation effects in which the absent CS is assumed to have positive associability (Hall, 1996; Holland, 1981, 1983) can explain the results of Experiment 1, but cannot account for the results of Experiment 2.

Let us consider the possibility that an integration of the mediated conditioning hypothesis (Hall, 1996; Holland, 1981, 1983) with the temporal coding hypothesis could account for both sets of results. According to this modification of the mediated conditioning hypothesis, presentations of A during Phase 2 A–US trials would retrieve the representation of the X, thus allowing it to enter into an association with the US with A's new temporal location with respect to the US. Through this mechanism, the X–US temporal relationship in Condition Diff but not in Condition Same was possibly shifted during Phase 2. However, on closer inspection, we see that such an integration of the mediated conditioning process and the temporal coding process actually predicts no recovery of conditioned responding to X. If the X–US temporal relationship is updated as a result of Phase 2 A–US temporal shifting, X and A would still share the same temporal relationship with the US. Two cues sharing the same temporal relationship with the US presumably should still compete. Thus, mediated updating of the

X–US temporal relationship so that it matches the A–US temporal relationship predicts no recovery from overshadowing.

An explanation of the recovered responding observed to CS X in Experiment 2 based on a decrease in the effective strength of the A–US association is also congruent with the comparator hypothesis without recourse to the temporal coding hypothesis. The comparator hypothesis explicitly predicts that posttraining weakening of the effective A–US association will enhance responding to X (Kaufman & Bolles, 1981; Matzel et al., 1985, 1987). However, the assumption that shifting Group Update.Diff from A $\xrightarrow{0}$ US in Phase 1 to A $\xrightarrow{5}$ US in Phase 2 weakened the A–US association is inconsistent with the temporal coding hypothesis, in that the temporal coding hypothesis suggests that the small A–US gap used in the trace conditioning of Group Update.Diff during Phase 2 acted more to alter the learned A–US temporal relationship than to change the strength of the A–US association. Cole, Barnet, and Miller (1995b) presented data supportive of this latter view for the present parameters (i.e., 0 vs. 5 s). Of course, with a sufficiently long trace interval, surely the A–US association would be weakened, but that was likely not the case in the present research. Contrasting the results of Experiments 1 and 2 allows us to empirically evaluate this possibility. In Experiment 1, A was made a delay excitor in Phase 2 after trace conditioning in Phase 1. If the associative strength of CS A had increased as a result of Phase 2 (A $\xrightarrow{0}$ US) training, then A would have been expected to better compete with X for control of responding, with the result of stronger overshadowing (i.e., less responding to X). However, greater responding to X was observed, thereby indicating that the potential of A to compete with X decreased as a result of Phase 2 updating of A.

To recapitulate, the outcome of Experiment 1 is consistent with either the comparator hypothesis–temporal coding hypothesis view, a second-order mediated responding account, or a Holland (1981)–Hall (1996) retrospective revaluation account, but not with a Van Hamme andWasserman (1994)retrospective revaluation of A interpretation. The outcome of Experiment 2 is consistent with either the conjoint comparator hypothesis–temporal coding hypothesis account or a Van Hamme and Wasserman retrospective revaluation account, but not with a second-order mediated responding account or a Holland or Hall retrospective revaluation account. Thus, the conjoint comparator hypothesis–temporal coding hypothesis account uniquely provides an account of the effects of updating the A–US temporal relationship on responding to X in both Experiments 1 and 2.

The outcomes of the current experiments can be compared with the results of studies of posttraining associative deflation. In the framework of the comparator hypothesis (Miller & Matzel, 1988; Miller & Schachtman, 1985), the comparator (i.e., competing) stimulus effectively competes with the target CS for controlling the conditioned response, and posttraining devaluation of the comparator stimulus–US association prior to testing reduces such cue competition. Posttraining deflation consists of extinguishing the effective comparator stimulus–US association after the target CS and comparator stimulus are compounded and followed by the US. The effect of massive posttraining devaluation treatment of the comparator stimulus is to enhance responding to the target CS, which would otherwise evoke little responding due to competition with the comparator stimulus (e.g., Blaisdell, Gunther, & Miller, in press; Cole et al., 1995a; Matzel et al., 1985). These devaluation effects have been cited as evidence that an effective target CS–US association is readily established during training in cue competition paradigms (e.g., overshadowing), though the target CS does not ordinarily evoke a conditioned response at test. A posttraining change in the temporal relationship between the comparator stimulus and the US also attenuates the effectiveness of the comparator stimulus in reducing responding to the target CS. However, it does so, not by degrading the comparator stimulus–US association, but by disqualifying the comparator stimulus–US association from the comparator process as a result of the comparator stimulus coming to predict a US that differs in some attribute (temporal here) from the US that is predicted by the target CS. Thus, the present study provides further evidence that overshadowing results from a failure to express the target CS–US association, and not from a failure to encode a target CS–US association. As does posttraining deflation, posttraining shifting of the overshadowing stimulus–US interval alleviates the overshadowing deficit. In the conjoint framework of the comparator hypothesis and the temporal coding hypothesis, this outcome is due to the reduced ability of the temporally shifted overshadowing stimulus to compete with the overshadowed CS for controlling the conditioned response. Thus, assuming temporal coding, the comparator hypothesis can explain the recovered responding to the target CS as a result of the effects

both of posttraining devaluation and of posttraining temporal shifting of the comparator stimulus–US relationship.

General Conclusion

The present series of experiments investigated the prediction, made by the comparator hypothesis in conjunction with the temporal coding hypothesis, that overshadowing would be attenuated if the overshadowing stimulus–US temporal relationship was changed such that it no longer matched the overshadowed stimulus–US temporal relationship. This prediction held true (a) when both CSs were trained in a trace relationship with the US in Phase 1 with subsequent updating of A as a delay excitor in Phase 2 (Experiment 1), and (b) when both stimuli were trained in a delay relationship with the US in Phase 1 with subsequent updating of A as a trace excitor in Phase 2 (Experiment 2). Further evidence that this effect was dependent on the change in temporal attributes of the A–US association arises from the fact that Phase 2 training in which A had the same temporal relationship with the US as it had in Phase 1 failed to reduce the overshadowing effect (i.e., Group Update.Same).

What we believe is required to explain the outcomes of Experiments 1 and 2 is a model of how associations are integrated, such as the temporal coding hypothesis, along with a model of how responses based on those associations are generated, such as the comparator hypothesis. Although it is less parsimonious to attempt to explain conditioning phenomena with an integration of these two models rather than with a single model, this loss of parsimony appears to be offset by the ability of the conjoint comparator–temporal encoding hypothesis to account for the present results.

References

Barnet, R. C., Arnold, M. A., & Miller, R. R. (1991). Simultaneous conditioning demonstrated in second-order conditioning: Evidence for similar associative structure in forward and simultaneous conditioning*Learning and Motivation*, *22*, 253–268.

Barnet, R. C., Grahame, N. J., & Miller, R. R. (1993). Temporal encoding as a determinant of blocking. *Journal of Experimental Psychology: Animal Behavior Processes*, *19*, 327–341.

Blaisdell, A. P., Denniston, J. C., & Miller, R. R. (1997). Unblocking with qualitative change of unconditioned stimulus. *Learning and Motivation*, *28*, 268–279.

Blaisdell, A. P., Denniston, J. C., & Miller, R. R. (1998). Temporal encoding as a determinant of overshadowing. *Journal of Experimental Psychology: Animal Behavior Processes*, *24*, 72–83.

Blaisdell, A. P., Gunther, L. S., & Miller, R. R. (in press). Recovery from blocking achieved by extinguishing the blocking CS. *Animal Learning & Behavior*.

Church, R. M. (1984). Properties of the internal clock. In J. Gibbon & L. G. Allen (Eds.), Timing and time perception. *Annals of the New York Academy of Sciences*, *423*, 566–582.

Cole, R. P., Barnet, R. C., & Miller, R. R. (1995a). Effect of relative stimulus validity: Learning or performance deficit?*Journal of Experimental Psychology: Animal Behavior Processes*, *21*, 293–303.

Cole, R. P., Barnet, R. C., & Miller, R. R. (1995b). Temporal encoding in trace conditioning. *Animal Learning & Behavior*, *23*, 144–153.

Denniston, J. C., Blaisdell, A. P., & Miller, R. R. (1998). Temporal coding affects transfer of serial and simultaneous inhibitors*Animal Learning & Behavior*, *26*, 336–350.

Denniston, J. C., Miller, R. R., & Matute, H. (1996). Biological significance as a determinant of cue competition. *Psychological Science*, *7*, 325–331.

Desmond, J. E., & Moore, J. W. (1988). Adaptive timing in neural networks: The conditioned response*Biological Cybernetics*, *58*, 405–415.

Dickinson, A., & Burke, J. (1996). Within-compound associations mediate the retrospective revaluation of causality judgements*Quarterly Journal of Experimental Psychology*, *49B*, 60–80.

Ellison, G. D. (1964). Differential salivary conditioning to traces. *Journal of Comparative and Physiological Psychology*, *57*, 373–380.

Gibbon, J. (1977). Scalar expectancy theory and Weber's law in animal timing. *Psychological Review*, *84*, 279–335.

Gibbon, J., & Balsam, P. (1981). Spreading association in time. In C. M.Locurto, H. S.Terrace, & J.Gibbon (Eds.), *Autoshaping and conditioning theory* (pp. 219–253). New York: Academic Press.

Grahame, N. J., Barnet, R. C., & Miller, R. R. (1992). Pavlovian conditioning in multiple contexts: Competition between contexts for comparator status*Animal Learning & Behavior*, *20*, 329–338.

Hall, G. (1996). Learning about associatively activated stimulus representations: Implications for acquired equivalence in perceptual learning*Animal Learning & Behavior*, *24*, 233–255.

Holland, P. C. (1981). Acquisition of representation-mediated conditioned food aversions*Learning and Motivation*, *12*, 1–18.

Holland, P. C. (1983). Representation-mediated overshadowing and potentiation of conditioned aversions*Journal of Experimental Psychology: Animal Behavior Processes*, *9*, 1–13.

Holland, P. C., & Forbes, D. T. (1982). Representation-mediated extinction of conditioned flavor aversions*Learning and Motivation*, *13*, 454–471.

Honig, W. K. (1981). Working memory and the temporal map. In N. E.Spear & R. R.Miller (Eds.), *Information processing in animals: Memory mechanisms* (pp. 167–197). Hillsdale, NJ: Erlbaum.

Kamin, L. J. (1954). Traumatic avoidance learning: The effects of CS–US interval with a trace conditioning procedure*Journal of Comparative & Physiological Psychology*, *47*, 65–72.

Kamin, L. J. (1965). Temporal and intensity characteristics of the conditioned stimulus. In W. F.Prokasy (Ed.), *Classical conditioning* (pp. 118–147). New York: Appleton-Century-Crofts.

Kaufman, M. A., & Bolles, R. C. (1981). A nonassociative aspect of overshadowing. *Bulletin of the Psychonomic Society*, *18*, 318–320.

Killeen, P. R., & Fetterman, J. G. (1988). A behavioral theory of timing. *Psychological Review*, *95*, 274–295.

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcements*Psychological Review*, *82*, 276–298.

Maleske, R. T., & Frey, P. W. (1979). Blocking in eyelid conditioning: Effect of changing the CS–US interval and introducing an intertrial stimulus*Animal Learning & Behavior*, *7*, 452–456.

Matzel, L. D., Held, F. P., & Miller, R. R. (1988). Information and expression of simultaneous and backward associations: Implications for contiguity theory*Learning and Motivation*, *19*, 317–344.

Matzel, L. D., Schachtman, T. R., & Miller, R. R. (1985). Recovery of an overshadowed association achieved by extinction of the overshadowing stimulus*Learning and Motivation*, *16*, 398–412.

Matzel, L. D., Shuster, K., & Miller, R. R. (1987). Covariation in conditioned response strength between elements trained in compound*Animal Learning & Behavior*, *15*, 439–447.

Miller, R. R., & Barnet, R. C. (1993). The role of time in elementary associations. *Current Directions in Psychological Science*, *2*, 106–111.

Miller, R. R., Esposito, J. E., & Grahame, N. J. (1992). Overshadowing-like effects between potential comparator stimuli: Covariation in comparator roles of context and punctate excitor used in inhibitory training as a function of excitor salience*Learning and Motivation*, *23*, 1–26.

Miller, R. R., & Matute, H. (1996). Biological significance in forward and backward blocking: Resolution of a discrepancy between animal conditioning and human causal judgment*Journal of Experimental Psychology: General*, *125*, 370–386.

Miller, R. R., & Matzel, L. D. (1988). The comparator hypothesis: A response rule for the expression of associations. In G. H.Bower (Ed.), *The psychology of learning and motivation, Vol. 22,* (pp. 51–92). San Diego, CA: Academic Press.

Miller, R. R., & Schachtman, T. R. (1985). Conditioning context as an associative baseline: Implications for response generation and the nature of conditioned inhibition. In R. R.Miller & N. E.Spear (Eds.), *Information processing in animals: Conditioned inhibition* (pp. 51–88). Hillsdale, NJ: Erlbaum.

Pavlov, I. P. (1927). *Conditioned reflexes*. London: Oxford University Press.

Pearce, J. M., & Hall, G. (1980). A model of Pavlovian learning: Variations in the effectiveness of conditioned but not of unconditioned stimuli*Psychological Review*, *87*, 532–552.

Rescorla, R. W., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A.Black & W. F.Prokasy (Eds.), *Classical conditioning II* (pp. 64–99). New York: Appleton-Century-Crofts.

Schreurs, B. G., & Westbrook, R. F. (1982). The effect of changes in the CS–US interval during compound conditioning upon an otherwise blocked element*Quarterly Journal of Experimental Psychology*, *34B*, 19–30.

Van Hamme, L. J., & Wasserman, E. A. (1994). Cue competition in causality judgments: The role of nonrepresentation of compound stimulus elements*Learning & Motivation*, *25*, 127–151.

Wagner, A. R. (1981). SOP: A model of automatic processing in animal behavior. In N. E.Spear & R. R.Miller (Eds.), *Information processing in animals: Memory mechanisms* (pp. 5–47). Hillsdale, NJ: Erlbaum.