Temporal Specificity of Extinction in Autoshaping

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Three experiments investigated the effects of varying the conditioned stimulus (CS) duration between training and extinction. Ring doves (*Streptopelia risoria*) were autoshaped on a fixed CS–unconditioned stimulus (US) interval and extinguished with CS presentations that were longer, shorter, or the same as the training duration. During a subsequent test session, the training CS duration was reintroduced. Results suggest that the cessation of responding during an extinction session is controlled by generalization of excitation between the training and extinction CSs and by the number of nonreinforced CS presentations. Transfer of extinction to the training CS is controlled by the similarity between the extinction and training CSs. Extinction learning is temporally specific.

Timing is integral to the acquisition and expression of associative learning. In many preparations, acquisition speed is inversely related to conditioned stimulus (CS) duration (Gibbon, Baldock, Locurto, Gold, & Terrace, 1977; Gibbon & Balsam, 1981; Lattal, 1999), except at very short interstimulus intervals (Smith, Coleman, & Gormezano, 1969), and is directly related to the time between trials (the trial-spacing effect; e.g., Humphreys, 1940; Gibbon et al., 1977; Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980). A particularly strong example of such time-dependency occurs in autoshaping, in which acquisition speed is related to the ratio of the intertrial interval (ITI; or the interreinforcement interval, C) to the CS–unconditioned stimulus (US) interval (T), across a broad range of C and T values (Gallistel & Gibbon, 2000; Gibbon & Balsam, 1981). Although recent studies of appetitive conditioning in rats indicate that the C/T ratio is not the sole determinant of acquisition speed (Bouton & Sunsay, 2003; Holland, 2000; Kirkpatrick & Church, 2000; Lattal, 1999), the research is in agreement that C and T exert robust effects on acquisition speed and response rate. Temporal parameters also affect the expression of conditioning. When animals are trained with a fixed delay between CS and US, the conditioned response often becomes linked to the CS–US interval, rather than to the onset of the CS. That is, the conditioned response (CR) is emitted to coincide with or anticipate the US (e.g., Smith, 1968). When the CS–US interval is long, this means that few CRs are emitted during the early portion of the CS. This phenomenon was originally described by Pavlov (1927) as “inhibition of delay” and has been observed in many conditioning preparations, including autoshaping (Gibbon, 1977; Ohyama, Gibbon, Deich, & Balsam, 1999), approach behavior in rats (Drew, Fairhurst, Malapani, Horvitz, & Balsam, 2003; Kirkpatrick & Church, 2000), aversive conditioning in goldfish (Behrend & Bitterman, 1964; Bitterman, 1964; Drew, Zupan, Cooke, Couvillon, & Balsam, in press), fear-potentiated startle in rats (Davis, Schlesinger, & Sorenson, 1989), and of course, salivary conditioning in dogs (Pavlov, 1927). The CS–US interval can also affect the form of the conditioned response (Holland, 1980; Silva & Timberlake, 1998). For instance, a noise CS signaling a 30-s delay to food elicits more magazine behavior than does a noise CS signaling a 10-s delay to food, but the latter CS elicits more startle than does the former (Holland, 1980).

Timing of conditioned responding has been studied directly by using the peak procedure (Bitterman, 1964; Roberts & Church, 1978). In the peak procedure, animals are trained on a fixed time or fixed interval schedule, and they are given interspersed peak trials on which the CS is presented for an extended period of time and no reinforcer is delivered. The average distribution of responding on peak trials is approximately Gaussian, with the location and spread of the peak related to the CS–US interval (e.g., Gibbon, 1977; see also Church, Meck, & Gibbon, 1994). Balsam, Drew, & Yang (2002) have shown that in aversive conditioning with goldfish, timing of the CR remains very stable during acquisition. In fact, the only significant change that occurs with training is an increase in the peak rate of responding; the peak location and spread do not change significantly. Timing is learned very rapidly in other aversive paradigms as well (Davis et al., 1989; Ohyama & Mauk, 2001). Similarly, in appetitive paradigms such as autoshaping and magazine approach, even the very first CRs emitted appear...
to be timed to anticipate the US (Balsam et al., 2002; Kirkpatrick & Church, 2000). In extinction, even though responding becomes less likely, when responding occurs it tends to occur around the expected time of reward (Ohyama et al., 1999).

All of the data are consistent with the notion that during conditioning with fixed delays, subjects develop specific temporal expectations about when the US will occur. One way to explain the timing of CR output is to assume that the CS is a compound cue containing information about the sensory characteristics of the CS (visual, auditory, spatial, etc.) and about the temporal relation between the CS and US (Pavlov, 1927, p. 103). Pavlov (1927, p. 104) hypothesized that these temporal–sensory cue compounds were possible because the sensory representation of the CS changes over time, and as a result, the nominal CS is effectively composed of multiple successive cues that can independently acquire associations with the US. The same idea is included in the class of real-time models of conditioning known as “componential trace” models (e.g., Brandon, Vogel, & Wagner, 2002, p. 263; Desmond & Moore, 1988). If, as these models suggest, the CS includes both sensory and temporal features, then nonreinforced exposure to both the sensory and temporal features may be necessary for extinction to occur. So, if one were to train subjects with a cue of a given duration but extinguish them with a briefer CS, one might find very little extinction when subjects were tested with the training duration because subjects had never received nonreinforced exposure to both the sensory and temporal features of the CS. On the other hand, lengthening the CS between training and extinction should have little effect on extinction because nonreinforced presentations of the trained CS duration will still occur.

Other theories of extinction do not require nonreinforcement of the specific training duration. Most notably, rate expectancy theory (Gallistel & Gibbon, 2000) predicts that the cumulative duration of nonreinforced CS presentations is the crucial determinant of extinction. The model specifies that the decision to stop responding is based on a ratio comparison between the cumulative amount of nonreinforced exposure to the CS (since the last reinforcer) and the previously experienced average waiting time in the CS for a reinforcer (T); the number of extinction trials is irrelevant. As the cumulative amount of nonreinforced exposure increases, the ratio of these variables approaches a criterion value at which responding ceases.

A third possibility is that changes in CS duration between training and extinction will have little effect on the loss of responding. Some models (e.g., Grossberg & Schmajuk, 1989) include the idea that the CS–US interval is timed from the onset of the CS. Subjects learn that the US comes T seconds after the onset of the CS but do not necessarily encode information about the timing of the offset of the CS. If CS onset is the primary temporal landmark, then changing the time of CS offset should have little effect on extinction. When the CS duration is shortened or lengthened between training and extinction, subjects will still receive in extinction presentations of the trained interval between CS onset and the US. As a result, varying the CS duration between training and extinction should have little effect on extinction. The cessation of responding in extinction will occur as a function of the number of trials received, irrespective of the duration of each trial. A similar prediction can be drawn from trial-based models (e.g., Bush & Mosteller, 1951; Rescorla & Wagner, 1972), which at least in their original formulation (cf. Brandon et al., 2002), did not explicitly include a role for time. In these models, mere presentation of the CS can initiate a process that increases or decreases associative value, and there is no mechanism whereby changes in CS duration would affect that process.

Shipley (1974) directly assessed whether extinction is controlled by the number of extinction trials or the cumulative duration of nonreinforced exposure to the CS. Rats received pairings of either a 25- or 100-s CS with shock and were extinguished with different numbers of nonreinforced presentations of either a 25- or 100-s CS. In a test session following extinction, responding to a 150-s probe trial was inversely related to the cumulative amount of nonreinforced CS exposure during extinction, regardless of the training or extinction CS duration. This finding is inconsistent with all of the previously described models. The dependence on cumulative exposure is inconsistent with trial-based models and the lack of dependence on training CS duration is inconsistent with time-based models. However, there are several procedural problems that complicate interpretation of Shipley’s results. First, because all subjects had the same ITI during training (and thus different C/T ratios), it is probable that the groups differed in their asymptotic levels of conditioned responding. Moreover, because extinction was conducted in a new context outside the experimental chamber, Shipley was not able to assess responding during the extinction phase. It is thus not clear whether the observed pattern of results reflects an effect of cumulative nonreinforced exposure on the initial cessation in responding (as predicted by Gallistel & Gibbon, 2000) or only on the amount of recovery seen during the test session.

A recent article by Haselgrove and Pearce (2003) suggests that changes in CS duration between training and extinction may in fact speed the cessation of responding during extinction. Their experiments used a Pavlovian approach paradigm in which rats were trained with a fixed tone–food interval and then extinguished with presentations of the CS that were longer, shorter, or the same as the training interval. The primary result was that a change in CS duration between training and extinction, regardless of whether it was an increase or decrease, speeded the loss of responding. One limitation of the Haselgrove and Pearce study is that the training CS duration was never reintroduced after extinction. As a result, it is unclear whether the changes in CS duration increased the efficacy of the extinction treatment or merely reduced generalization of excitation between training and extinction. The latter interpretation is given weight by the fact that the changes in CS duration were quite large (3- to 27-fold). One way to differentiate between these hypotheses would have been to reintroduce the training duration after extinction, to assess how the extinction treatment impacts responding to the trained CS duration.

Implicit in our discussion of Shipley (1974) and Haselgrove and Pearce (2003) is the idea that extinction may be composed of two independent processes: the cessation of responding within a session of extinction and the transfer of extinction to the training CS. In this view, the data from these two studies tap different extinction processes. The Haselgrove and Pearce data suggest that changes in CS duration between training and extinction speed the cessation of responding in extinction. However, it is not known what effect these changes in CS duration have on responding to the trained cue. Conversely, the Shipley experiment suggests that extinction of responding to the trained duration depends on cumulative nonreinforced exposure to the CS. However, that experiment
provides no information about whether cumulative nonreinforced exposure also drives cessation.

The experiments reported here were aimed at elucidating the effects of temporal parameters on both cessation and transfer. Similar to Haselgrove and Pearce (2003), our strategy was to train subjects on a single CS duration, then to extinguish them on CS durations that were longer, shorter, or the same as the training duration. However, our design incorporated a postextinction test session in which subjects received nonreinforced exposure to the training CS duration. The test session was used to examine the degree to which the extinction experience, in which some animals received nonreinforced exposure to a novel CS duration, would transfer to the training CS duration. The test session was important because any between-groups differences in response rate (cessation) seen during initial extinction might be attributable either to generalization decrement (between the training and extinction CSs) or to a direct effect of temporal parameters on the loss of associative value during extinction. Changing a component of the CS between training and extinction may also provide a discriminative cue for nonreinforcement, protecting the training cue from the detrimental effects of the extinction treatment (Rescorla, 2003). To the extent that changing the CS duration between training and extinction produces generalization decrement, those groups extinguished with CS durations different than the training duration will show fast cessation and a large amount of recovery during the test phase; the associative value of the training CS will effectively have been preserved because of the ability of the animals to discriminate between the reinforced training CS duration and the nonreinforced extinction CS duration. If, on the other hand, changes in CS duration enhance the overall efficacy of the extinction treatment (e.g., by increasing the loss of the training CS’s associative value), then this will be reflected in low levels of recovery during the test session.

In sum, our design allows for examination of two possibly distinct extinction processes: the cessation of responding that will occur within a session of nonreinforcement and the transfer of the extinction experience to the training CS in the test phase. In the discussions that follow, we refer to these processes, respectively, as “response cessation” and “transfer.”

Experiment 1

Experiment 1 tested whether response cessation and transfer of extinction are driven by number of nonreinforced presentations of the training duration, cumulative nonreinforced exposure to the CS, or the number of nonreinforced trials. Ring doves were housed individually in stainless steel cages, where they were given water ad libitum and kept on a 12-hr light–dark cycle. The subjects were maintained at 85% of their free feeding body weights throughout the experiment.

Apparatus. Four experimental chambers were used. Two of them were identical Lehigh Valley/BRS (Laurel, MD) models with internal dimensions of $30 \times 30 \times 26$ cm, flat black walls, and floors lined with iodized cardboard. The keylight was positioned 7.0 cm to the left of the midline of the front panel and 13.0 cm above the floor. A stimulus projector located behind the response key served to illuminate the key during trials. The minimum force required to activate this response key was 0.18 N. A feeder aperture (6.5 × 7.0 cm) was located in the center of the front panel, its bottom edge at floor level. During grain presentation, the hopper was illuminated by one 24 PSB bulb. At all other times during the session, one No. 1829 light on the top of the front panel served as a houselight. The other two chambers had internal dimensions of $33 \times 33 \times 27$ cm, with three white plastic walls, one metallic wall, and cardboard-lined floors. The keylight was positioned 8.3 cm to the left of the midline of the front panel and 15.2 cm above the floor. The keylights and hoppers were the same as those in the other chambers. A computer housed in the same room was used to control stimulus presentations and record data.

Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of sessions</th>
<th>No. of trials</th>
<th>Cumulative duration of nonreinforced exposure (s)</th>
<th>No. of nonreinforced presentations of the training duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>$4 \times 64$</td>
<td>4</td>
<td>64</td>
<td>256</td>
<td>0</td>
</tr>
<tr>
<td>$4 \times 128$</td>
<td>8</td>
<td>128</td>
<td>512</td>
<td>0</td>
</tr>
<tr>
<td>$8 \times 64$</td>
<td>4</td>
<td>64</td>
<td>512</td>
<td>64</td>
</tr>
<tr>
<td>$16 \times 64$</td>
<td>4</td>
<td>64</td>
<td>1,024</td>
<td>64</td>
</tr>
</tbody>
</table>
Subjects were placed on a restricted diet. After reaching 85% of their free-feeding body weight, subjects were hopper trained for a maximum of 3 days at the rate of two sessions per day. A small amount of grain was placed in front of the hopper at the beginning of each session until they were eating regularly from the feeder. In each 20-min session of hopper training, the grain-filled hopper was presented for 5 s on a variable interval–40-s schedule, for a total of 30 presentations. At each hopper presentation, the house lights were turned off and the small light above the hopper was illuminated. Once birds were successfully eating from the hopper, autoshaping commenced.

In each daily autoshaping session, subjects were given 16 trials. On each trial, a white keylight was illuminated for 8 s and was followed by 4 s of grain presentation. The ITI was variable with a mean of 64 s. During each ITI, the house lights were on, and they remained on during the CS presentations. During grain presentation, the houselight was turned off and the hopper light was turned on. Subjects were trained for 17 days.

On Day 18, the extinction treatment commenced. Subjects were assigned to four groups counterbalanced for response rate during the final two training sessions. Each extinction session consisted of 16 nonreinforced keylight presentations with the same ITI used in training. The duration of each nonreinforced keylight presentation was varied between groups. Group 4 × 64 received 4-s keylight presentations. Group 8 × 64 received 8-s presentations. Group 16 × 64 received 16-s presentations. Groups 4 × 64, 8 × 64, and 16 × 64 received 4 daily extinction sessions, for a total of 64 nonreinforced CS presentations. There was also a Group 4 × 128, which received 8 daily extinction sessions with the 4-s nonreinforced cue, resulting in the same cumulative exposure to the CS as in Group 8 × 64.

On the day immediately following the last extinction session, subjects received a test session. The test session consisted of 16 eight-second presentations of the CS, nonreinforced. The ITI was variable with a mean of 64 s, as in training. Pecks at the keylight were recorded throughout all sessions.

Results

During the last 2 days of training, the mean response rates (pecks per second) during the 8-s CS were 1.20 for the Group 4 × 64, 1.18 for Group 4 × 128, 1.17 for Group 8 × 64, and 1.09 for Group 16 × 64. These values did not differ significantly F(3, 32) = 0.05. In the following analyses of the cessation and transfer data, response rates are expressed as a proportion of the baseline response rate, which is mean response rate taken across the final two sessions of training. These relative response rates were used to reduce within-group variability, thereby increasing statistical power.

Figure 1A shows mean relative response rates during the extinction session plotted as a function of cumulative amount of nonreinforced exposure to the CS. Each point represents the mean response rate for 32 consecutive seconds of the CS, divided by the baseline response rate. For subjects extinguished with 4-s CS presentations, each point represents 8 trials. For subjects extinguished with 8- or 16-s presentations, each point represents four trials or two trials, respectively. Data for Groups 4 × 64 and 4 × 128 were pooled. The cessation data were subject to an 8 (block) × 3 (group) analysis of variance (ANOVA), with block as a repeated measure. There were significant effects of block, F(7, 231) = 8.39, p < .01; of group, F(2, 33) = 6.52, p < .01; and of the Block × Group interaction, F(14, 231) = 3.05, p < .01. To probe the interaction, we individually subjected each block of 32 s to a one-way ANOVA. The groups differed significantly in Blocks 3–6, F(2, 33) > 6, ps < .01. Thus equivalent amounts of cumulative exposure do not result in equivalent cessation of responding.

Figure 1B shows the extinction relative response rates plotted as a function of extinction trials. Each data point is a mean taken across 16 consecutive trials on a 8-s schedule, for a total of 64 presentations. At each hopper presentation, the house lights were turned off and the small light above the hopper was illuminated. Once birds were successfully eating from the hopper, autoshaping commenced.

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Figure 1B shows the extinction relative response rates plotted as a function of extinction trials. Each data point is a mean taken across 16 consecutive trials on a 8-s schedule, for a total of 64 presentations. At each hopper presentation, the house lights were turned off and the small light above the hopper was illuminated. Once birds were successfully eating from the hopper, autoshaping commenced.
received 256 s of exposure in a single session, but it took the extinction results in more rapid response loss than massed extinction across-session spacing effect. It is conceivable that spaced briefer CS durations during extinction necessarily received a given constant in Experiment 1 means that the subjects exposed to cessation. Our decision to hold the number of trials per session controlled response cessation.


dence (LSD) tests. The Group 4 × 64 response rate was significantly higher than that of each of the other three groups, ps < .02. Groups 4 × 128, 8 × 64, and 32 × 64 did not differ significantly from each other, ps > .50.

Discussion

In this experiment subjects were trained with an 8-s CS–US interval, then extinguished with 4-, 8-, or 16-s presentations of the CS. The experiment addressed two questions. First, would the cessation of responding be driven by the number of nonreinforced presentations of the training duration, the cumulative duration of nonreinforced exposure to the cue, or the number of nonreinforced trials? Second, which of these components of the extinction experience control transfer of extinction to the training duration? To address the first question, we examined the rate of decrease in responding during the initial extinction sessions. Groups 8 × 64 and 16 × 64 received an equal number of nonreinforced presentations of the training duration and responding declined at about the same per-trial rate. Groups 4 × 64 and 4 × 128 received no nonreinforced presentations of the training duration but declined at about the same rate as Groups 8 × 64 and 16 × 64. Nonreinforcement of the training duration is not necessary for cessation of the conditioned response. With respect to the cumulative exposure hypothesis, the results clearly show that cumulative exposure did not control the diminution of responding during the extinction phase. The groups exposed to the larger number of trials extinguished more quickly for a given amount of cumulative exposure. When reductions in responding were examined as a function of the number of trials regardless of CS duration, there was little evidence of a difference between groups, suggesting that the number of trials controlled response cessation.

However, one feature of this experiment indicates caution in accepting the conclusion that cumulative exposure does not affect cessation. Our decision to hold the number of trials per session constant in Experiment 1 means that the subjects exposed to briefer CS durations during extinction necessarily received a given total amount of exposure over a larger number of sessions. It may be that the effects of our extinction treatment were moderated by an across-session spacing effect. It is conceivable that spaced extinction results in more rapid response loss than massed extinction (Westbrook, Smith, & Charnock, 1985). Group 16 × 64 received 256 s of exposure in a single session, but it took the groups extinguished on 8 s and 4 s two and four extinction sessions to receive equivalent exposure. It is thus possible that a spacing effect contributed to the rate of response cessation.

One way to evaluate the contribution of a sessions effect is to examine performance during just the first session of extinction. By doing so, we can compare the groups after they have received a common amount of cumulative exposure spread over a common number of sessions (1). We compared the average response rate for the four groups over the first 64 s of nonreinforced exposure. For the 4-s groups, this included the entire first extinction session. For Groups 8 × 64 and 16 × 64, it included the first 8 or 4 trials of the first session. Unfortunately, none of the groups showed a significant decline from baseline rates at this point in extinction, nor did they differ significantly from each other. Because the animals showed no significant decline in response rate during the early part of extinction, the lack of differences among groups is not informative, and we cannot rule out the spacing hypothesis. We directly address this problem in the next experiment.

The test session data show a different pattern of results and provide a test of our hypothesis that extinction of the trained duration requires nonreinforcement of the trained CS duration. Although birds in Group 4 × 128 received no nonreinforced presentations of the 8-s cue, their level of responding to the 8-s cue during the postextinction test session was identical to that of the groups that did receive nonreinforced presentations of the 8-s cue. The only difference between groups in the test session was that Group 4 × 64, which received the smallest amount of cumulative nonreinforced exposure, responded at a higher rate than the others. The difference cannot be attributed to the number of nonreinforced trials because Groups 4 × 64, 8 × 64, and 16 × 64 received the same number of nonreinforced trials. The results are thus roughly consistent with the hypothesis that cumulative nonreinforced exposure to the CS is inversely related to the amount of responding in the test session. Although Group 16 × 64, which received the most exposure to the CS, did not respond at a lower rate than Groups 8 × 64 and 4 × 128, this may be a floor effect, as all three groups responded at a low rate during the test session (0.1 response per second).

It is not possible, however, to rule out the hypothesis that extinction of the trained CS duration requires nonreinforcement of the trained duration. The crucial evidence against this hypothesis is that Group 4 × 128, which received no nonreinforced presentations of the trained duration, responded at about the same rate in the test session as did the other groups that did receive nonreinforced presentations of the trained duration. But in addition to receiving twice as many extinction trials overall, Group 4 × 128 received twice as many extinction sessions as did the other groups. This confound is problematic because it is plausible that the number of extinction sessions independently affects the amount of spontaneous recovery exhibited. For instance, there is some evidence that spontaneous recovery is caused by the incomplete extinction of early-session cues (Skinner, 1950; Welker & McAuley, 1978). Cues associated with the beginning of the session (e.g., the transport cage) are only experienced once per session, and as a result, it may take multiple extinction sessions to extinguish them. The low level of recovery (transfer) seen in Group 4 × 128 could be due to the more complete extinction of early-session cues rather than to the doubling of cumulative nonreinforced exposure.
Experiment 2

The results of Experiment 1 provide some support for the hypotheses that the cessation of responding during nonreinforcement is driven by the number of nonreinforced exposures to the CS and that transfer of extinction is controlled by the cumulative duration of nonreinforced exposure. However, the strength of these conclusions is limited by the experimental design, which used multiple extinction sessions. It was unclear whether the cessation of responding to the extinction CS was due to the presentation of nonreinforced trials or to a combination of the effects of cumulative nonreinforced exposure to the CS and the spacing of extinction trials. Moreover, because the groups varied in the number of extinction sessions they received, and thus potentially in the degree to which early-session cues were extinguished, it was difficult to interpret the results of the test session.

Experiment 2 circumvents these problems by using one, rather than multiple, extinction sessions. A second modification was that the number of subjects was increased to allow for a finer-grained analysis of within-session changes in response rate. As in Experiment 1, subjects were trained on an 8-s CS–US interval and then extinguished via 4-, 8-, or 16-s nonreinforced presentations of the same CS. The extinction treatment comprised 128 nonreinforced presentations of the CS in one session, except for Group 4 × 256, which received 256 nonreinforced presentations in one session (see Table 2). On the day following the extinction session, there was a test session in which the 8-s training CS was reintroduced.

Method

Subjects. Sixty-four birds with experience in a previous autoshaping experiment were used. They were housed and cared for as in Experiment 1.

Procedure. The apparatus described in Experiment 1 were used. During training, subjects were autoshaped using the same procedure as in Experiment 1, with the exceptions that a red keylight was used (rather than a white) and that subjects were trained for 15 days (rather than 17).

On Day 16, a rolling extinction procedure began. Birds were assigned to four groups, counterbalanced for group membership from the previous experiment and for pecking rate during the final 2 days of training. The extinction treatment comprised 128 nonreinforced presentations of the keylight in one session, except for Group 4 × 256, which received 256 extinction trials in one session. As in Experiment 1, the duration of the keylight presentations was varied between groups. Groups 4 × 256 (n = 13) and 4 × 128 (n = 17) received 4-s presentations; Group 8 × 128 (n = 17) received 8-s presentations; and Group 16 × 128 (n = 17) received 16-s presentations. Because the duration of the extinction sessions was long (3–6 hr), we ran only one squad on extinction per day. Each day, 1 bird from each group was extinguished, and the remaining birds were either trained or tested. Birds were selected for extinction on the basis of response rate, group membership, and test chamber. Birds with the highest response rate were extinguished first, with the constraint that 1 bird from each group be extinguished each day. All birds were extinguished in the chamber in which they were trained.

Subjects were given a test session on the day following extinction. The test session consisted of 16 nonreinforced presentations of the 8-s CS as in Experiment 1.

Results

During the final two days of training, response rates (responses per second) during the CS for Groups 4 × 256, 4 × 128, 8 × 128, and 16 × 128 were 1.14, 1.29, 1.21, and 1.19, respectively. These values were not significantly different, F(3, 60) = 0.15. As in the previous experiment, the following analyses use relative response rates computed by dividing each datum by the baseline rate, which was a mean taken across the final two training sessions.

Figure 3A shows relative response rates in extinction expressed as a function of cumulative duration of nonreinforced exposure to the CS. Each data point is a mean for 32 consecutive seconds of the CS, divided by the overall baseline response rate. For the 4-s groups, which were collapsed for this analysis, each point is a mean taken across 8 consecutive extinction trials. For Group 8 × 128 each point is a mean taken across 4 trials. For the Group 16 × 128 each point is a mean taken across 2 trials. The data were subject to a 16 (block) × 3 (group) ANOVA, with block as a repeated measure. There were significant effects of block, F(15, 915) = 53.61, p < .01; of group, F(2, 61) = 21.10, p < .01; and of the Block × Group interaction, F(30, 915) = 5.79, p < .01. As in Experiment 1, equivalent amounts of cumulative exposure did not result in equivalent cessation.

Figure 3B shows relative response rates in extinction expressed as a function of trials. Each point is a mean taken across 8 consecutive nonreinforced trials and divided by the baseline rate. The data were subject to a 16 (block) × 3 (group) ANOVA, with block as a repeated measure. There was a significant effect of block, F(15, 915) = 68.68, p < .01. There was no Block × Group interaction, F(30, 915) = 0.63, and no effect of group, F(2, 61) = 0.15. Again, the number of trials rather than the cumulative duration determined extinction speed.

Figure 4 shows relative response rates for the test session during which animals received nonreinforced presentations of the 8-s cue. The values are mean response rates taken across 8 trials and divided by the baseline response rate. The rates were subjected to a 2 (block) × 4 (group) ANOVA with block as a repeated measure. There were significant effects of block, F(1, 60) = 46.98, p < .01; of group, F(3, 60) = 4.54, p < .01; and of the Block × Group interaction, F(3, 60) = 3.08, p < .05. To probe the interaction, each 8-trial block was individually subject to a one-way ANOVA. The group response rates were significantly different in the first block of 8 trials, F(3, 60) = 3.93, p < .02, but not the second, F(3, 60) = 2.21, p = .10. Post hoc comparisons (LSD tests) revealed that Groups 4 × 256 and 4 × 128 both responded more than did Group 8 × 128 in the first block (p < .05). The 4-s groups did not differ from each other (p > .10), nor did Groups 8 × 128 and 16 × 128 (p > .10). Group 4 × 256 responded more than did Group 16 × 128 (p < .02), but Groups 4 × 128 and 8 × 128 did not differ (p = .08). As in Experiment 1, animals exposed to the 4-s

Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of sessions</th>
<th>No. of trials</th>
<th>Cumulative duration of nonreinforced exposure (s)</th>
<th>No. of nonreinforced presentations of the training duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 × 128</td>
<td>1</td>
<td>128</td>
<td>512</td>
<td>0</td>
</tr>
<tr>
<td>4 × 256</td>
<td>1</td>
<td>256</td>
<td>1,024</td>
<td>0</td>
</tr>
<tr>
<td>8 × 128</td>
<td>1</td>
<td>128</td>
<td>1,024</td>
<td>128</td>
</tr>
<tr>
<td>16 × 128</td>
<td>1</td>
<td>128</td>
<td>2,048</td>
<td>128</td>
</tr>
</tbody>
</table>
duration in extinction displayed the most recovery, but in this experiment additional nonreinforced exposure to the 4-s cue only extinguished the first 4 s of the trial and that the recovery rates primarily reflect responding during the second 4 s of each trial. Figure 5A shows the within-trial patterns of responding. In Figure 5A, the data are presented in 1-s bins, and each point is a mean response rate taken across the first 8 trials of the test session and divided by the baseline response rate. Because the maximum rates differ by group, it is difficult to assess whether the shape of within-trial patterns of responding differ. To more easily identify differences in the temporal distribution of responses, we normalized the response rates for each subject by dividing by that subject’s maximum response rate, yielding within-trial response distributions with a common asymptote of 1. Figure 5B shows the averaged normalized functions. (The 2 subjects in Group 4 × 128, the 7 subjects in Group 8 × 128, and the 4 subjects in Group 16 × 128 that failed to respond during the test session were not included in this analysis.) The normalized data were subjected to a 4 (group) × 8 (second) ANOVA, with second as a repeated measure. The only significant effect was that of second, \( F(7, 343) = 23.45, p < .01 \). The effects of group, \( F(3, 49) = 1.61, p < .15 \); and the Group × Second interaction, \( F(21, 343) = 0.50 \), were nonsignificant. This analysis indicates that the extinction CS duration did not affect the timing of responding in the test session.

Figure 3. Relative response rates during the extinction phase of Experiment 2. Data for Groups 4 × 128 and 4 × 256 are pooled. Response rates are expressed as a proportion of the baseline mean response rate. A: Response rates are expressed as a function of cumulative nonreinforced exposure to the conditioned stimulus (CS). Each point is a mean taken across 32 consecutive seconds of CS exposure. Each point represents 8 trials for Groups 4 × 128 and 4 × 256, 4 trials for Group 8 × 128, and 2 trials for Group 16 × 128. B: Response rates are expressed as a function of number of nonreinforced trials. Each point is a mean taken across 8 consecutive trials.

Figure 4. Relative response rates during the test session of Experiment 2. Each point is a mean response rate taken across 8 trials and expressed as a proportion of the baseline response rate.

Figure 5. Within-trial response patterns during the first 8 trials of the test session of Experiment 2. A: Response rates are expressed as a proportion of the mean baseline response rate. B: The same data are normalized by dividing by the maximum response rate, thus giving the groups a common asymptote of 1. CS = conditioned stimulus.
Discussion

The results of Experiment 2 confirm that the decrease in responding that occurs during nonreinforcement is driven by nonreinforced trials rather than by cumulative nonreinforced exposure to the cue. Subjects were trained on an 8-s CS–US interval and extinguished with presentations of the cue that were 4, 8, or 16 s in duration. Response rate decreased as a function of trials, and the rate of decrease was invariant across CS durations and different amounts of cumulative nonreinforced exposure.

It might be argued that the extinction functions in Figure 3B obscure an effect of CS duration because whole-trial response rates are used, giving the groups different opportunities for responding. For instance, the 4-s groups might be at a disadvantage because the 4-s CS included only the early portion of the training duration, during which inhibition of delay might be occurring. To address this concern, we reexamined the extinction data, using only the first 4 s of each trial in all groups. Figure 6 shows relative response rate as a function of trials, with only responses during the first 4 s of each trial in all groups. Figure 6 shows relative response rates during the extinction phase of Experiment 2 as a function of number of nonreinforced trials. Each point is a mean taken across 8 consecutive trials, but only the first 4 s of each trial are included.

To durations longer than T, although they include the trained temporal–sensory compound detract from the effectiveness of the extinction treatment. We return to this issue in Experiment 3.

The fact that doubling the number of nonreinforced exposures to the 4-s cue failed to substantially reduce responding in the test session is at variance with the pattern observed in Experiment 1. In Experiment 1, doubling the amount of nonreinforced exposure to the 4-s duration significantly reduced the amount of recovery exhibited in the test session. The discrepancy may be attributed to the fact that in Experiment 1, exposure was increased by doubling the number of extinction sessions, and the increase in the number of extinction sessions may have itself reduced the amount of spontaneous recovery shown in the test session (e.g., Skinner, 1950; Youtz, 1938).

The groups showed similar within-trial patterns of responding during the test session. There was a somewhat steady increase in response rate over the first 5 s of the trial and asymptotic responding during the final 3 s. The generality of this pattern is surprising considering that the groups had different extinction experiences. Groups 8 × 128 and 16 × 128 received nonreinforced presentations of the full 8-s training duration, but the 4-s groups did not. One might expect that in the test session the 4-s groups would show a relatively high rate of responding during the second 4-s of the CS, the portion that was never extinguished. Although it is true that the 4-s groups responded more than the other groups overall, the temporal distribution of responses did not differ among groups, indicating the extinction experience did not affect the temporal organization of responses to the trained cue. Subjects may discriminate between the extinction duration and the training–test durations, such that a different temporal organization of responding is controlled by each situation. Alternatively, it may be that nonreinforcement does not affect the timing of responding, as others have shown (Ohyama et al., 1999). To further examine whether response timing changed between training and test, we compared the within-trial response distributions from the final two training sessions (preextinction) and the first 8 trials of the test session (postextinction). To mitigate the large difference in mean response rate between training and test, we discarded subjects that made fewer than 30 total responses during the first 8 trials of the test session. The individual subject data were normalized as above and subject to a 2 (phase: training vs. test) × 4 (group) × 8 (second) ANOVA, with phase and second as repeated measures. The analysis confirmed that CR timing does not differ among groups. The effects of group, F(3, 22) = 0.21, the Group × Phase interaction, F(3, 22) = 0.18, the Group × Second interaction, F(7, 21) = 0.51, and the three-way interaction, F(21, 154) = 0.56, all failed to reach significance. There was no main effect of phase, F(1, 22) = 2.77, p = .11. There were significant effects of second, F(7, 154) = 45.67, p < .01, and it is important to note, of the Phase × Second interaction, F(7, 154) = 3.28, p < .01. The significant Phase × Second interaction indicates that the temporal distribution of responses changed between training and test. The change in CR timing is apparent in Figure 7, which shows the normalized pre- and postextinction distributions of responding (because there are

![Figure 6](image_url)

**Figure 6.** Relative response rates during the extinction phase of Experiment 2 as a function of number of nonreinforced trials. Each point is a mean taken across 8 consecutive trials, but only the first 4 s of each trial are included.
no between-groups differences in CR timing, the data are collapsed across groups. It appears that the postextinction (test) distribution is slightly sharper than the preextinction (training) distribution. This observation is difficult to reconcile with earlier data (Ohyama et al., 1999) indicating that nonreinforcement does not affect the temporal distribution of responding. Nevertheless, the analysis confirms the more important point with respect to the current experiments, which is that CR timing did not differ among Groups, and thus differences in response timing cannot explain the overall between-groups differences in responding observed in the test session.

Experiment 3

The results of Experiment 2 support the view that extinction is temporally specific. In order for responding to an 8-s CS to extinguish, animals had to receive nonreinforced presentations of the CS that were at least 8 s in duration. Although nonreinforced presentations of a shorter duration caused an initial cessation of responding, the loss did not fully transfer to the longer trained duration. The data are consistent with the view that extinction of a trained duration requires extinction trials that are at least as long as the trained duration.

In Experiment 3 we further examine the role of time in extinction, considering the effects of a larger increase in CS duration between training and extinction. Until now we have assumed that nonreinforcement of any duration longer than T constitutes a nonreinforced exposure to the trained duration. However, if duration is a defining feature of stimuli, then large changes in CS duration between training, extinction, and test should reveal generalization decrement effects and reduce the efficacy of an extinction treatment in terms of its ability to reduce responding to the trained duration. Significant changes in duration between training and extinction may also speed the cessation of responding because the extinction CS will elicit less generalized responding, as suggested by Haselgrove and Pearce (2003).

Experiment 3 tested whether a large change in the CS duration between training and extinction affects the efficacy of the extinction treatment. Subjects were trained on an 8-s CS–US interval and then were extinguished on 4-, 8-, or 36-s presentations of the CS (see Table 3). After a single extinction session there was a test session in which the training duration was reintroduced.

Method

Subjects. The subjects were 36 birds, housed and cared for as in Experiments 1 and 2. Twenty-seven of the birds were naive; the remaining birds had experience in one previous, unrelated autoshaping experiment. Groups were counterbalanced for previous experimental experience.

Procedure. The apparatus and training were the same as in Experiment 2, with the exception that a white keylight was used rather than a red.

On Day 16, a rolling extinction procedure commenced. Birds were assigned to three groups, counterbalanced for pecking rate during the final 2 days of training. Group 4 × 64 received 64 nonreinforced 4-s presentations in the extinction session. Group 8 × 64 received 64 nonreinforced 8-s presentations. Group 32 × 64 received 64 nonreinforced 32-s presentations. On each extinction day, 4 birds were extinguished; the remaining birds were trained or tested. Of the birds extinguished on a given day, 2 were drawn from one group and one was drawn from each of the other two groups. The identity of the twice-represented group rotated with each extinction session.

On the day following the extinction session, birds received a test session consisting of 16 nonreinforced presentations of the 8-s cue.

Results

The response rates during the final two sessions of training were 1.09, 1.14, and 1.14 responses per second for Groups 4 × 64, 8 × 64, and 32 × 64, respectively. These values did not differ significantly, F(2, 36) = 0.03. As in the previous experiments, relative response rates were calculated by dividing by the mean response rate taken over the final two sessions of training.

Figure 8A shows the relative response rates in the extinction session as a function of cumulative nonreinforced exposure to the CS. Each point is a mean relative response rate taken across 32 consecutive seconds of CS exposure, computed as in the previous experiments. The data were subject to an 8 (block) × 3 (group) ANOVA, with block as a repeated measure. There were significant effects of block, F(7, 231) = 11.74, p < .01; of group, F(2, 33) = 3.59, p < .05; but not of the Block × Group interaction, F(14, 231) = 0.96. As in the previous experiments, equivalent amounts of cumulative nonreinforced exposure did not produce equivalent amounts of cessation.

Figure 8B shows the relative response rates in extinction as a function of trials. Each point is a mean relative response rate taken across 8 consecutive trials. The data were subject to an 8 (block) × 3 (group) ANOVA, with block as a repeated measure. There were significant effects of block, F(7, 231) = 30.61, p < .01; of group,
Figure 8. Relative response rates during the extinction phase of Experiment 3. A: Response rates are expressed as a function of cumulative nonreinforced exposure to the conditioned stimulus (CS). Each point is an average taken across 32 consecutive seconds of CS exposure. Each point represents 8 trials for Group 4 × 64, 4 trials for Group 8 × 64, and 1 trial for Group 32 × 64. B: Rates are expressed as a function of number of nonreinforced trials. Each point is a mean taken across 8 consecutive trials. C: Each point is a mean taken across 8 consecutive trials, but only the first 4 s of each extinction trial was used in computing response rates.

$F(2, 33) = 4.07, p < .05$; and of the Group × Block interaction, $F(14, 231) = 1.94, p < .05$.

To probe the interaction, we subject each block to a one-way ANOVA. There were significant differences among groups in Blocks 3, 4, and 5 (Trials 17–40), $F$s(2, 33) > 3.3, $p$s < .05. LSD post hoc tests confirmed that in those blocks, Group 32 × 64 responded significantly less than each of Groups 4 × 64 and 8 × 64, $p$s < .05. Groups 4 × 64 and 8 × 64 did not differ. Group 32 × 64 ceased responding faster than the other groups, but as in the other experiments, Groups extinguished on 4 s or 8 s extinguished at the same per-trial rate.

As in Experiment 2, we reanalyzed the extinction session data using only the first 4 s of each extinction trial (see Figure 8C). Each point was an average response rate taken across 8 consecutive trials including only responses during the first 4 s of each trial. For each subject, the resulting values were divided by the subject’s mean response rate for the first 4 s of trials during the last 2 days of training. The resulting data were subjected to an 8 (block) × 3 (group) ANOVA. There was a significant effect of block, $F(7, 231) = 20.46, p < .001$. Although Group 32 × 64 appears to be extinguishing faster than the other groups, the Block × Group interaction did not reach significance, $F(14, 231) = 1.46, p = .13$; nor did the effect of group, $F(2, 33) = 2.18, p = .13$.

Figure 9 shows the relative response rates for the test session, in which all subjects received nonreinforced presentations of the 8-s duration. The values are means taken across 8 trials. Because of our prediction of greater recovery in Groups 4 × 64 and 32 × 64 relative to Group 8 × 64, the data for each block of 8 trials were subject to planned comparisons (t tests). For the first block of 8 trials, Groups 4 × 64 and 32 × 64 exhibited significantly more recovery than did Group 8 × 64, $t$(22) = 2.20, $p$ < .05. Groups 4 × 64 and 32 × 64 did not differ from each other, $t$(22) = 0.17. None of the groups differed in the second block of 8 trials $t$(22) < 1.8, $p$s > .09.

As in Experiment 2, we examined the temporal distribution of responses during the test session. Figure 10A shows the within-trial response patterns, and the response rates are expressed as a proportion of the baseline response rate. To test whether the shape of the functions differed, we normalized the data, as in Experiment 2, expressing each subject’s data as a proportion of that subject’s maximum response rate during the test session. The resulting normalized functions are shown in Figure 10B. The normalized data were subject to a 3 (group) × 8 (second) ANOVA with second as a repeated measure. Only those subjects that responded during the test session were included in this analysis. The only significant effect was of second, $F(7, 168) = 20.62, p < .01$. Neither the group effect, $F(3, 24) = 3.00, p = .07$; nor the Second × Group interaction, $F(14, 168) = 0.79$, reached significance. The groups exhibited the same temporal distribution of responses during the test session.

Discussion

Experiment 3 tested the effects of a large increase in the CS duration between training and extinction. Three groups of birds were trained on an 8-s CS–US interval and then extinguished with
pattern controverts the cumulative exposure hypothesis; although

There are two main conclusions that can be drawn. First, the

group 32 irrespective of the duration of each trial. The performance of

sation is driven by the number of nonreinforced trials received,

ation must be made to the earlier hypothesis that response ces-

long CS is still apparent. The speed, although the trend toward faster cessation with the very

are no statistically reliable differences among groups in cessation
testing at about the same rate. Group 32 × 64, however, ceased

responding faster than the other groups, confirming the observa-
tion of Haselgrove and Pearce (2003) that a large change in CS

duration facilitates response cessation. The lower response rates in
groups extinguished with the very long CS may be, in part, due to
using whole-trial response rates. If responding peaks around the
expected time of reinforcement (Ohyama et al., 1999), then long
CSs include a period of time during which subjects show little
responding, as compared with CSs of shorter duration. When we
analyze only the first 4 s of each trial in the extinction phase, there
are no statistically reliable differences among groups in cessation
speed, although the trend toward faster cessation with the very
long CS is still apparent.

The results of the extinction session indicate that some modifi-
cation must be made to the earlier hypothesis that response ces-
sation is driven by the number of nonreinforced trials received,
irrespective of the duration of each trial. The performance of
Group 32 × 64 suggests that when the CS duration is changed
significantly between training and extinction, the responding de-
clines more quickly. The most parsimonious account of the data is
that a very large change in CS duration results in some general-
ization decrement between training and extinction.

In the test session following extinction, both the 4 × 64 and the
32 × 64 Groups responded at higher rates than did Group 8 × 64.
There are two main conclusions that can be drawn. First, the
pattern controverts the cumulative exposure hypothesis; although
Group 32 × 64 received eight times the exposure of Group 4 × 64,
the two groups showed identical recovery in the test session.
Furthermore, Group 32 × 64 received four times as much exposure
as Group 8 × 64 yet showed much more responding during
the transfer test. The second conclusion is that significant changes
in CS duration between training and extinction detract from the
effectiveness of the extinction treatment in reducing responding to
the trained duration. Compared with extinction with the training
duration, extinction with a CS that is either longer or shorter than
the training CS produces less of a decrement to the training CS.
Recovery depends on the similarity between the training–test and
extinction cues. Extinction learning is temporally specific.

General Discussion

The experiments reported here addressed two aspects of extinc-
tion: the cessation of responding during nonreinforcement and the
transfer of extinction to the trained CS duration. These processes
appear to be independent and are considered in turn.

The cessation of responding during extinction was controlled
largely by the number of nonreinforced trials received. Doubling
or halving the CS duration between training and extinction had no
effect on response cessation. In the groups in which the CS
duration was doubled, halved, or unchanged between training and
extinction, there was a steady decrease in responding as a function
of trials, and the extinction functions superimposed. This pattern
directly contradicts rate expectancy theory (Gallistel & Gibbon,
2000), which predicts that the loss of responding will occur as
function of the cumulative duration of nonreinforced exposure to
the CS. According to this time-based model, doubling or halving
the CS duration between training and extinction should have the
effect of halving or doubling, respectively, the number of trials
needed to reach an arbitrary extinction criterion.

It would be hard to believe, however, that very large changes in
CS duration between training and extinction have no effect on the
loss of responding. Performance during the extinction phase must
depend at least partially on the degree to which responding gen-
eralizes from the trained duration to the extinction duration. Given
that duration appears to be a defining feature of stimuli, changes in
CS duration between training and extinction ought to diminish that
generalization. This account explains the performance of Group
32 × 64 of Experiment 3, which was trained on an 8-s CS duration
then extinguished via 32-s presentations. Group 32 × 64 extin-
guished faster than the groups extinguished on 4 or 8 s. One fact
that appears inconsistent with the generalization interpretation is
that Group 32 × 64 responded at about the same rate during the
first block of extinction trials (see Figure 8) as did the group
exposed to the 8-s CS. When generalization occurs along a tem-
poral dimension, however, it is conceivable that generalization
decrements will not manifest immediately. In the case of Group
32 × 64, subjects could not know whether the CS duration had
increased until after 8 s (T) had elapsed. Thus at the beginning of
trials, subjects were ignorant of the total trial duration. If gen-
eralization across different CS durations is to impact responding
across the whole trial, then subjects must learn that the onset of a
trial signals a new CS duration. Thus it might take several trials
before the subjects come to consistently expect that the cue pre-
vented during the extinction phase will be different from the
training cue. Variability in timing and temporal memory processes
(e.g., Gibbon, 1977) also makes it likely that multiple trials would
be necessary to detect a change in CS duration. Consistent with this view, recent data from Gallistel, Mark, King, and Latham (2001) show that rats require several trials to detect a change in reward timing when it is changed for the first time.

The extinction session results are consistent with generalization decrement models of extinction (e.g., Capaldi, 1967, 1971; Mowrer & Jones, 1945), which argue that the rate of decline in the responding during extinction is determined by the dissimilarity between the training and extinction contexts. These models predict that explicit changes in stimulus characteristics between training and extinction will accelerate loss of responding. We observed that a large change in CS duration between extinction and test expedited the cessation of responding in extinction. Changes in other features of the CS have a similar effect. The design of the current experiments is very similar to that of Dubin and Levis (1973), who studied the effect of varying the frequency of a tone CS between training and extinction. They found that such changes reduced the amount of responding during the extinction treatment. Similar decreases in extinction responding have been observed after changes in the size of a visual CS (Orenstein, Schumsky, Roth, & Brunner, 1972). The current finding of faster cessation after a large change in CS duration between training and extinction may thus be another instance of generalization decrement.

In sum, cessation of responding during extinction is affected by two factors. First, nonreinforced presentations of the CS cause a diminution of responding. Second, the similarity of the training cue duration to the extinction cue duration affects the rate at which responding declines. If the extinction cue is very different than the training cue. To explain our results, one only need posit that excitation has a broader generalization gradient than extinction, an idea that has been put forward elsewhere (e.g., Bouton, 1993; but see cf. Hall & Honey, 1989). Thus the high degree of generalization of excitation from the 8-s training cue to the test 4-s extinction cue generates comparable cessation functions in the groups extinguished with these two durations. However, the narrow extinction gradient results in more responding to the 8-s test cue when subjects were previously extinguished on a different duration.

These data are also generally consistent with memory models of extinction (e.g., Bouton, 1993; Bouton & Bolles, 1985) that argue that extinction involves the acquisition of new learning that coexists with, rather than replaces, the original learning about the CS–US contingency. Because memories of extinction and training coexist, postextinction performance depends on whether the test situation more readily recalls memories of training or of extinction. For this reason, presentation of stimuli uniquely associated with training can produce renewal of the conditioned response. Consistent with this view, test session responding was enhanced in groups receiving an extinction CS duration that was different from the CS duration received during training and testing.

One puzzling feature of our results is that they are from those described by Shipley (1974). In Shipley’s experiment, recovery depended only on cumulative nonreinforced exposure to the CS. It did not depend on the similarity between the extinction CS duration and the training CS duration. One possible explanation for the disparity between the two studies is that extinction of aversive conditioning differs from extinction of the appetitive conditioning used here. However, several other procedural issues might also contribute to the disparity. Shipley’s test session oc-
curred on the same day as the extinction session, whereas ours was 24 hr later. Shipley’s test session used a CS duration that was different from both the extinction and training CS durations, so the experimental groups differed with respect to the similarity of the test cue to both the original training CS and to the extinction CS. The level of responding during the test might then be a product of this complex interaction. Perhaps most important, because Shipley’s groups were trained with different CS–US intervals and a common ITI, it is likely that the groups differed in their final level of conditioned responding before extinction (shorter CS–US intervals tend to produce faster acquisition and may also produce higher asymptotic levels of responding; Gibbon et al., 1977).

Our data implicitly provide a test of the role of the response in extinction. The theory that extinction depends on nonreinforced responding is more closely associated with instrumental paradigms (e.g., reflex reserve; Skinner, 1938). But similar arguments have been advanced with respect to classical conditioning (Holland & Rescorla, 1975; Rescorla, 1997). Our data indicate quite clearly that the rate of loss of the CR does not correlate with the amount of responses emitted in extinction. Recall that in Experiment 2, the groups responded at virtually identical per-second response rates (relative to each other) in each block of extinction trials (see Figure 3B). Because the CS durations varied, this means that Group 16 × 128 emitted about four times as many responses per trial block as the 4-s groups and twice as many as Group 8 × 128. Cessation was not controlled by nonreinforced responding. Performance in the transfer test was also unrelated to responding in extinction. Group 16 × 128 emitted a mean of 312 responses during extinction, and Group 8 × 128 a mean of 120, yet Group 16 × 128 showed a modestly (but nonsignificantly) higher rate of responding in the test session.

It is important to point out that the transfer tests in the current experiments can be conceptualized in different ways. We have favored the view of transfer test. But the test session is also a spontaneous recovery test. In many conditioning paradigms, when there are successive sessions of nonreinforcement, conditioned responding tends to reappear at the beginning of sessions, despite the fact that responding had ceased by the end of the previous session (e.g., Robbins, 1990). The different conceptualizations of the test sessions illustrate that the recovery levels displayed in the test sessions could reflect the action and interaction of two processes: “transfer” of extinction and spontaneous recovery. Thus our results might be interpreted as showing an effect of extinction cue duration on spontaneous recovery rather than on transfer. However, an independent effect of extinction CS duration on spontaneous recovery is unlikely, given the results of Experiment 1. Because Experiment 1 kept the same CS duration in effect across multiple extinction sessions, the extinction phase data should reveal any effects of cue duration on spontaneous recovery. The fact that the group per-session response rates did not differ during the extinction phase (see Figure 1B) thus argues against an independent effect of extinction CS duration on spontaneous recovery, per se.

In summary, the process of extinction includes two distinct components: cessation and transfer. Cessation occurs as a function of the number of nonreinforced exposures to a CS, but it is also modulated by the degree to which excitation generalizes between the training and extinction CS. Our experiments show that significantly changing the CS duration between training and extinction can speed the cessation of responding. The capacity of the extinction experience to affect responding to the training CS is also a function of generalization. In this case, the similarity of the extinction cue to the original training cue modulates the extent to which responding to the original training cue is suppressed. More generally, the experiments suggest that, like the learning that takes place in acquisition, the learning that takes place in extinction is temporally specific.

References


