Temporal Learning in Random Control Procedures

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Experiments 1 and 2 delivered conditioned stimuli (CSs) at random times and unconditioned stimuli (USs) at either fixed (Experiment 1) or random (Experiment 2) intervals. In Experiment 3, CS duration was manipulated, and US deliveries occurred at random during the background. In all 3 experiments, the mean rate of responding (head entries into the food cup) in the background was determined by the mean US–US interval, and the mean rate during the CS was a linear combination of responding controlled by the mean US–US and mean CS onset–US intervals; the pattern of responding in time was determined by the interval distribution form (fixed or random). An event-based timing account, Packet theory, provided an explanation of the results.

In 1967, Robert Rescorla published the now classic article, “Pavlovian Conditioning and Its Proper Control Procedures,” based on somewhat earlier work on Sidman avoidance in dogs (Rescorla & LoLordo, 1965). In this article, Rescorla reviewed the then traditional control procedures for Pavlovian conditioning and concluded that some of the procedures were flawed because they introduced “nonassociative factors” while others produced inhibitory conditioning. He surmised that the best control would be one in which the conditioned stimulus (CS) and unconditioned stimulus (US) were presented randomly in the absence of any contingency and named this procedure the truly random control. In a random control procedure, CS and US deliveries are independent of one another. Thus, there are some CS–US pairings, but not a significantly different number than predicted by chance.

A few studies verified that the truly random control produced no discernible evidence of learning (Gamzu & Williams, 1971, 1973; Quinsey & Ayres, 1969; Rescorla, 1968, 1969). However, a number of investigations indicated that the truly random control resulted in significant acquisition of conditioned suppression in the conditioned emotional response paradigm in rats (Benedict & Ayres, 1972; Kremer, 1971; Kremer & Kamin, 1971; Quinsey, 1971), of autoshaped keypecks in pigeons (Durlach, 1982, 1983; Goddard & Jenkins, 1987), and most recently with a goal-tracking procedure in rats (Rescorla, 2000). In some instances, responding emerged and then waned with further training (e.g., Rescorla, 1972, 2000), but Rescorla (2000) demonstrated that even after overt goal-tracking behavior had disappeared, there were still discernible excitatory effects of the CS when it was imposed on an instrumental baseline. Thus, even in cases in which overt responses are not observed, the random control may result in some form of learning.

Most interpretations of the effects of random control procedures have been based on a division of the experimental session into mutually exclusive states. The analysis of contingency has relied on the comparison of occurrences of USs during the CS and background states. Because, in the random control procedure, the US is not contingent on the CS, it is important to define the term contingency precisely. Originally, a lack of contingency was defined as equality between two conditional probabilities, \( p(US|CS) \) and \( p(US|noCS) \), but the calculations were based on fixed time intervals of 2 min (Rescorla, 1968). The values that entered into the probability calculations were US delivery rates (number of USs per 2 min in the CS and the number of USs per 2 min in the background, or no CS period), not probabilities, which are restricted to be between 0 and 1. Thus, the original concept was that a critical determinant of performance is the relative rate of US occurrence in the presence and the absence of the CS, and a lack of contingency was defined as the equality of rates of US deliveries in the CS and background states.

Alternatively, a lack of contingency could be defined as equality of the mean time from an event (e.g., CS onset) until US delivery and the mean US–US interval. Taking this further, perhaps a lack of contingency should be defined in terms of the identity of the distribution forms and equality of all their parameters, not just the means. Most random control procedures fail to meet these alternative definitions of lack of contingency. For present purposes, any references to contingency refer to the original definition, but these alternative definitions are discussed in more detail later.

A different interpretation of the effects of random control procedures on responding may be based on the identification of a series of events in an experimental session. With a single CS, the differentiation of classes of theories into state-based and event-based is a relatively novel idea (see Church & Kirkpatrick, 2001) that provides an insightful global classification scheme for different types of theories. State-based theories assume that the key element of a procedure is the configuration of physical stimuli. Examples of state-based theories include the Rescorla–Wagner model (Rescorla & Wagner, 1972), rate
such as a tone, and a single US, such as delivery of a food pellet, the events (which are sometimes called time markers) are CS onset, CS termination, and US delivery. A critical determinant of performance may be the distribution of times from an event to the next US delivery; these are the intervals between CS onset, CS termination, and US until the next US (Kirkpatrick & Church, 1998, 2000a, 2000b).

Packet theory of timing proposes that (a) the mean response rate during an interval (e.g., US–US, CS onset–US) is determined by the mean interval duration; (b) the pattern of responding in time is determined by the interval distribution form; and (c) if two or more expectations (e.g., US–US and CS onset–US) are simultaneously active, the resulting rate of responding is determined by a combination of response rates supported by each conditional expected time function. Both the mean interval and interval distribution form are encoded by Packet theory through the conditional expected time to US, which is the mean expected time remaining in an interval until US delivery as a function of time since interval onset.

The top two panels of Figure 1 display the conditional expected time functions for fixed (left) and exponential random (right) intervals of different mean durations. The conditional expected time is the expected time remaining until US occurrence, given that the US has not already occurred (see Kirkpatrick, 2002; Kirkpatrick & Church, 2000a, 2003). It can be calculated using Equation 1, where \( f(x) \) is the density function of the variate \( x \) and \( S_t \) is the survival function at Time \( t \). For simple density functions, the conditional expected time can be calculated explicitly; or for any empirically generated density function it can be obtained by numerical integration with \( dx \) set at a short time unit.

\[
E_t = \int_{t-\infty}^{t} \frac{x f(x)}{S_t} dx - t. \tag{1}
\]

Each experimental event (e.g., US delivery, CS onset, CS termination) results in the formation of a conditional expected time function (and probability function).\(^2\) The conditional expected time function for an event is composed of the set of intervals between each occurrence of the event (e.g., CS onset) and the US. An expectation is updated in memory at the time of US occurrence. The conditional expected time function for a fixed interval starts at the value of the fixed interval and decreases linearly until the time of the US. The conditional expected time function for a random interval starts at the mean of the random interval and remains at that value. The conditional expected time function thus encodes both the mean interval \( E_t \) at \( t = 0 \) and the distribution form (shape of conditional expected time function), allowing for a prediction of mean response rate (which is inversely related to \( E_t \) at \( t = 0 \)) and pattern (timing) of responding (which is inversely related to the shape of the conditional expected time function).

The bottom row of Figure 1 displays the rate of packet generation (expected number of packets per second) in successive 1-s time bins, as determined by Packet theory (see Kirkpatrick, 2002; Kirkpatrick & Church, 2003). The rate of packet occurrence, \( r_p \), is equal to \( n_p \) where \( p_t \) is the probability of packet occurrence at Time \( t \) and \( n \) is a responsiveness parameter. Each function in Figure 1 was calculated in 1-s bins with \( n \) set to 2.0 so that each function sums to 2.0. As seen in the figure, the rate of packet occurrence (and hence response rate) is inversely related to the conditional expected time function. The packets of responding (small bouts) contain a mean of five responses; the interresponse time in a packet has a mean of 1.2 \( s \). The packet characteristics vary randomly from one instance to the next; these characteristics have been derived from the behavior of rats on basic time-based schedules of reinforcement (Kirkpatrick & Church, 2003). The model initiates a packet of responses if the momentary rate (in some small unit of time) exceeds a random threshold between 0 and 1. The packet-generating function, or decision vector, for a given event (e.g., CS onset) is reset to its initial value at the time of each occurrence of that event.

Because it is possible to determine a conditional expected time function for any distribution of events, Packet theory predicts that responding in random control procedures would be determined in the same way as for any other conditioning procedure. Specifically, the US–US interval would determine responding in the background. Because the US–US interval is always active (with a reset at each US delivery), responding during the CS would be determined by a combination of responding due to the US–US and CS onset–US decision vectors. The CS onset–US decision vector is active following CS onset up until food delivery. Thus, even if the CS onset–US and US–US intervals were the same mean expectancy theory (Gallistel & Gibbon, 2000, 2002), and contingency theory (Rescorla, 1968). There are also many models that fall into the event-based category. An event is a transition between states, so an event-based theory would focus on these transitions rather than on the states themselves. Many timing theories are event-based; these include the multiple oscillator model (Church & Broadbent, 1990), scalar timing theory (Gibbon & Church, 1984), and the learning to time theory (Machado, 1997). Finally, some theories contain both state- and event-based processes, such as many of the real-time models of conditioning (e.g., Sutton & Barto, 1981, 1990).
duration, one might expect a bigger response during the CS than during the background because during the CS there are two active decision vectors.

The combination rule is key in predicting performance in the truly random control. Table 1 lists some possible combination rules, which were developed from a general linear model; the rules determine the means of combining decision vectors that are derived from the conditional expected time function. The summation (Kirkpatrick & Church, 2000a), linear combination (Meck & Church, 1984), and winner-take-all (Meck & Church, 1984) rules have proved effective in explaining responding under simultaneous timing arrangements (in which two or more intervals are simultaneously timed). The linear weighting rule is included for sake of completeness. The table contains a brief description of each rule, along with the parameter settings that would be applied to the general formulation. The linear combination rule is the most flexible, with two parameters estimated from the data. The linear weighting rule and the winner-take-all rules have one free parameter, and the summation rule has no free parameters. The linear weighting rule predicts performance that is intermediate between the two decision vectors; the winner-take-all rule predicts performance that is equal to one of the two decision vectors; the summation rule predicts performance that is equal to the sum of the two decision vectors. The linear combination rule is unique among the four rules because it incorporates the product of the two decision vectors; this rule predicts that the first decision vector (the US–US vector) will modulate any additional effect of the second decision vector (the CS onset–US vector).

In contrast to Packet theory, the traditional contingency analysis (relative rates of reinforcement in CS and background states) ignores any influence of CS duration, the time from one CS termination to the next CS onset, or the time between US deliveries as important contributors to learning. Contrary to this view, one factor that was recognized early on as a determinant of the magnitude of conditioning in the truly random control procedure is the duration of the CS. Kremmer and Kamin (1971) delivered alternating CS and background states that were each a fixed duration of 120 s or 900 s. The time between US deliveries was random with a mean of 360 s in both states, thus creating a zero contingency. Because the CS and background states were fixed, the CS onset–US interval was affected by CS duration. In the 120-s group, the CS onset–US interval could range from 0 to 240 s, with a mean of 120 s, whereas in the 360-s group, the CS onset–US interval could range from 0 to 1,800 s, with a mean of 360 s. It is interesting to note that there was much stronger conditioned suppression when the CS duration was shorter than when it was longer, even though there were more CS–US pairings in the longer

Table 1

<table>
<thead>
<tr>
<th>Combination rulea</th>
<th>Parameter settings</th>
<th>Description</th>
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<tbody>
<tr>
<td>Linear combination</td>
<td>$0 \leq a \leq 1$, $b = 0$, $0 \leq c \leq 1$</td>
<td>Linear combination of decision vectors</td>
</tr>
<tr>
<td>Linear weighting</td>
<td>$0 &lt; a &lt; 1$, $b = 1 - a$, $c = 0$</td>
<td>The weighted mean of decision vectors</td>
</tr>
<tr>
<td>Summation</td>
<td>$a = 1$, $b = 1$, $c = 0$</td>
<td>The sum of decision vectors</td>
</tr>
<tr>
<td>Winner-take-all</td>
<td>$a = 0$, $b = 1$, $c = 0$ or $a = 1$, $b = 0$, $c = 0$</td>
<td>Exclusive choice of one decision vector</td>
</tr>
</tbody>
</table>

* The present formulation is developed for responding during a conditioned stimulus (CS), with responding due to the combination of unconditioned stimulus (US)-US and CS onset-US decision vectors. A general linear model can be applied in all occasions but with different parameter settings: $r_t = a r_{tUS} + b r_{tCS} + c r_{tUS} r_{tUS}$, where $a$, $b$, and $c$ are weights that may vary from 0 to 1, $r_t$ is the predicted rate of packet generation (which is functionally equivalent to rate of response) during the CS, $r_{tUS}$ is the US-US decision vector, and $r_{tCS}$ is the CS onset-US decision vector. The values of $r_{tUS}$ and $r_{tCS}$ are determined from the conditional expected time function (see Footnote 2).
CS condition. This finding was subsequently replicated and extended (Kremer, 1971; Quinsey, 1971).

According to a contingency account, CS duration should have no effect on responding because the rate of reinforcement would be the same in the CS and background states in both conditions. However, according to Packet theory, there should be more responding during the short CS because the mean CS onset–US interval would be shorter, and shorter intervals are known to produce higher rates of responding. It is, however, possible that the effect of CS duration was due to the CS filling a smaller percentage of the session when it was short, thereby resulting in that CS becoming more salient. Thus, the present set of experiments sought to investigate whether the response rate was controlled by the relative reinforcement rate during the CS and background states, or whether it was controlled by the conditional expected time to reinforcement from CS onset and the previous US delivery. Measures of both the mean response rate and the timing of responding were examined to shed light on the role of temporal learning in these procedures.

Experiment 1

Experiment 1 sought to determine the contribution of state- and event-based learning to responding in a random control procedure, with emphasis on comparing the predictions of contingency theory and Packet theory. Rats received US deliveries at fixed intervals (45, 90, or 180 s) and random CS durations that occurred at random times in the session. There were two different interval manipulations that occurred across groups. First, the mean US–US interval duration was varied while holding the duration of the CS and the interval between one CS termination and the next CS onset constant. This manipulation did not change the CS–US contingency but did change the mean interval between CS onset and US delivery. Second, the duration of the CS and the background periods were manipulated while holding the mean US–US interval constant (this is the p vs. c manipulation in Figure 2). This manipulation affected the mean number of CSs per US–US interval but did not change the mean CS onset–US interval, the mean US–US interval, the probability of a CS–US pairing, or the CS–US contingency. The total CS exposure time per session was equated across all six conditions, so any effect of this variable would be factored out. The mean CS onset–US interval was not affected by the manipulation of CS duration in the present procedure (compared with earlier procedures such as the one used by Kremer and Kamin, 1971) because a different structure of events was used. In the procedures used in Experiment 1, the US–US interval was fixed and the CS duration varied. In all groups, the CS onset–US interval ranged between 0 and the US–US duration, with a mean equal to half the US–US interval duration.

Contingency theory predicts an absence of learning in all of the procedures because the rate of US delivery in the CS and background states is the same in all groups. In contrast, Packet theory predicts that (a) mean response rates in the background should be negatively related to mean US–US interval; (b) mean response rates in the CS should be negatively related to the mean CS–US interval—in the present procedures this was always equal to half the mean US–US interval; (c) responding should be higher during the CS than in the background because the mean CS–US interval is shorter than the mean US–US interval in all groups and also because during the CS both the CS onset and US expectations are active and response rates would be due to a combination of responding supported by the two individual expectations; and (d) responding during the CS should not be affected by the manipulation of CS and background durations because this manipulation had no effect on the mean US–US or CS onset–US intervals.

Method

Animals

Twenty-four male Sprague-Dawley rats (Rattus norvegicus; Taconic Laboratories, Germantown, NY) were housed individually in a colony room on a reversed 12-hr light–dark cycle (lights off at 8:45 a.m.). Dim red lights provided illumination in the colony room and the testing room. The rats were fed a daily ration of 15 g of FormulaLab 5008 food given in the home cage shortly after the daily sessions plus 45-mg Noyes pellets (Improved Formula A) that were delivered during the experimental sessions. Water was available ad libitum in both the home cages and experimental chambers. The rats arrived in the colony at 35 days of age and were

| Group | Procedure | Mean time to US from:
|-------|-----------|---------------------
| CS | no CS | CS onset | US |
| 45c | 45 s | 1.33 | 1.33 | 22.5 s | 45 s |
| 90c | 90 s | .67 | .67 | 45 s | 90 s |
| 180c | 180 s | .33 | .33 | 90 s | 180 s |
| 45p | 45 s | 1.33 | 1.33 | 22.5 s | 45 s |
| 90p | 90 s | .67 | .67 | 45 s | 90 s |
| 180p | 180 s | .33 | .33 | 90 s | 180 s |

Figure 2. Diagram of the procedure used in Experiment 1 for constant (45c, 90c, and 180c) and proportional (45p, 90p, and 180p) conditioned stimulus (CS) deliveries. Striped bars represent the CS, and filled triangles represent food unconditioned stimulus (US) deliveries. The time between successive food deliveries was fixed at 45, 90, or 180 s. The duration of the CS is marked above the CS, and the time between CSs (termination to onset) is marked in between successive CSs. Exponentially distributed random intervals are denoted by a tilde (−).
handled daily until the onset of the experiments. Training began when they were 48 days old.

**Apparatus**

Each of the 12 chambers (25 × 30 × 30 cm) was located inside a ventilated, noise-attenuating box (74 × 38 × 60 cm). A chamber was equipped with a food cup, a water bottle, and a speaker. A magazine pellet dispenser (Model ENV-203) delivered 45-mg Noyes (Improved Formula A) pellets into the food cup. Each head entry into the food cup was transduced by an LED photocell. A water bottle was mounted outside the chamber; water was available through a tube that protruded through a hole in the back wall of the chamber. The speaker for delivering the 70-dB white noise CS was situated above and to the right of the water tube. Two Gateway 486 DX2/66 computers running the Med-PC Medstate Notation Version 2.0 (Tatham & Zur, 1989) controlled experimental events and recorded the time at which each event occurred with 10-ms resolution.

**Procedure**

The rats were randomly assigned to one of six training groups: 45c, 90c, 180c, 45p, 90p, and 180p, resulting in 4 rats per condition. The groups are labeled according to the duration of the US–US interval and the duration of the white noise CS (see description below). Each group received 15 sessions of training with the random control procedure. Each session ended after the delivery of 160 food reinforcements. To equate the number of reinforcements in groups with different US–US interval durations, the training sessions were 2, 4, or 8 hr for groups that received 45, 90, or 180 s US–US intervals, respectively.

The procedure for each group is diagrammed in Figure 2 (column 2). The times between events are labeled as fixed (e.g., 90 s) or random (e.g., ~75 s). A tilde (~) is used to denote exponentially distributed random intervals. All groups of rats received food pellets that were delivered on a fixed time schedule, onset and termination of a white noise CS at random times, and independent food and CS deliveries.

There were three groups with the same schedule of CS deliveries: 45c, 90c, and 180c. All three groups received a white noise whose duration was an exponential random interval with a mean of 15 s, and an interval between the termination of one CS and the onset of the next CS that was an exponential random interval with a mean of 75 s—the minimum possible duration was 0.1 s for both intervals. Thus, there was a 90-s random interval between successive CS onsets (or terminations). The only difference among the constant groups was the interval between US deliveries, which was 45, 90, or 180 s. There were also three proportional groups: 45p, 90p, and 180p, each with a schedule of CS delivery that was proportional to the US–US interval. The 45p group received a random 7.5-s CS duration, with a random 37.5-s duration between the termination of one CS and the onset of the next CS, so that the time between successive CS onsets (and terminations) was random 45 s. The 90p group received the same procedure as the 90c group, with a random 15-s CS duration and a random 75-s interval between CS deliveries, so that the time between successive CS onsets (and terminations) was 90 s. Finally, the 180p group received a random 30-s CS duration, with a random 150 s between successive CS deliveries so that the time between successive CS onsets (and terminations) was 180 s.

The expected number of CSs per food–food interval was 1.0 in all three proportional groups, but the expected (mean) number of CSs per US–US interval was 0.5, 1.0, and 2.0 for groups 45c, 90c, and 180c, respectively. The probability of a CS–US pairing was 0.167 in all six groups.

Columns 3 and 4 of Figure 2 display USs per minute during the CS and the background (no CS), whereas columns 5 and 6 display the mean time to US from CS onset and the prior US delivery, respectively. The rate of reinforcement in both the CS and background periods was inversely related to the US–US interval, and the rates of reinforcement were the same during the CS as in the background. The interval between CS onset and US delivery was variable with a mean equal to half the US–US interval duration.

**Data Analysis**

The time of each CS onset or termination, each US delivery, and each head entry into the food cup (each time the photobeam was interrupted) was recorded with 10-ms accuracy. All analyses omitted the first 20 s of the US–US interval, when head entry rates were elevated because of consumption of the previously delivered food pellet (e.g., see Figure 3).

**Local response rate.** Local response rates during the US–US interval and during the CS were based on the number of responses (N) and the number of opportunities (O) to respond in each 5-s bin following food; local response rates during the CS–US interval were based on the number of responses and opportunities to respond in each 5-s bin following CS onset. Calculations of the number of responses in each 5-s bin were conducted by summing the total number of responses during each second over all intervals in the analysis. When the intervals were fixed, the number of seconds of opportunities to respond in each 5-s bin of a single interval was equal to the total number of intervals included in the analysis. When the intervals contained a random component, then the number of seconds of opportunities to respond differed from bin to bin. For example, if an interval was 25 s in duration, then each bin between 0 and 25 s would be filled with one opportunity to respond. Local rate, expressed as responses per minute, was then defined in each bin as 60 × (N/O)/B, where B was the width of the bins in seconds. For some analyses, 2-s bin sizes were used instead; these analyses were conducted in the same manner.

**Elevation score.** Elevation scores were defined as the difference between the mean response rate during the presence of the CS and the response rate during empty US–US intervals. Both rates excluded responding during the first 20 s after US delivery to avoid contamination by reactions to the US; the rats were usually consuming the prior food pellet during this time (an unconditioned response). If a US delivery occurred during the CS, then response rates were calculated from CS onset until the time of US delivery. Otherwise, responding was examined up until the time of CS termination.

**Baseline rates relative to a pseudostimulus.** For the analysis of response rates as a function of time since CS onset up until the time of CS termination or US delivery, whichever occurred first, a baseline comparison rate was calculated relative to a pseudostimulus. The pseudostimulus was not a real physical stimulus but rather was an event marker that was used for analysis purposes. The pseudostimulus onsets were randomly distributed in the empty US–US intervals (in which no CsS occurred). Response rates were calculated from pseudostimulus onset up until the time of a pseudostimulus termination or US delivery. The response rates relative to CS onset and pseudostimulus onset were determined in 5-s bins (see Local response rate calculation, above). The pseudostimulus duration was determined separately for each rat to match the CS duration. If the CS duration was, for example, random 15 s, then the time between pseudo-CS onset and termination was a random 15 s. Thus, the distribution of pseudostimulus onsets and terminations was engineered to closely approximate the distribution of CS onsets and terminations. Pseudostimuli only occurred in empty US–US intervals; if US delivery was scheduled during a pseudostimulus, then responding was calculated only up until the time of US delivery.

**CS responding as a function of time in the US–US interval.** The times of responses during the CS were determined for each CS onset until US delivery or CS termination, whichever occurred first. These response times were then placed in 5-s bins, according to their time of occurrence in the

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3 Because the interval between successive CS onsets is the sum of two exponential random intervals, the distribution of times between onsets is a generalized twofold gamma (McGill & Gibbon, 1965), with a mean of 90 s and a standard deviation that is equal to the square root of the sum of the variances of the two exponentials, which is 76.5 s.
US–US interval. The response rate in each bin in the US–US interval when the CS was present was determined using the equation for local response rate (see above). Similar response rate calculations were made for empty intervals, giving the local rate of responding as a function of time since US delivery on empty intervals.

Statistical analyses. Nonparametric tests were used for all analyses, except for the analyses on slopes of responding. The main reason for the use of nonparametric statistics is that many measures of responding suffered from violations of homogeneity of variance assumptions. In particular, shorter interval durations produced greater variation among rats in response rates. The hypothesized effects of interval duration are that shorter intervals should produce higher levels of responding than longer intervals, and therefore nonparametric methods that test for order relations are an appropriate alternative.

Results

Figure 3 contains the local response rates as a function of time in the empty US–US intervals. The response rate is shown in 5-s bins, collapsed over Sessions 6–15 of training. The last 10 sessions were analyzed because timing emerged over the first 5 sessions. The response rate was initially high, probably due to consumption of the previous food pellet. Thereafter, the response rate functions decreased and then increased again, reaching a maximum near the end of the interval. The response rate functions were affected by the mean US–US interval duration, with shorter intervals resulting in higher overall response rates, as measured by a Kruskal–Wallis test conducted on the mean response rates over 20 seconds to the end of the US–US interval, $\chi^2(2) = 13.4, p < .01$ (for all chi-squares in Experiment 1, $N = 24$). There was no effect of the constant versus proportional manipulation on the overall response rates, $\chi^2(1) = 0.01, ns$. The shape of the response rate functions is consistent with timing of the fixed US–US interval.

Figure 4 displays the elevation score (responses/min) to the CS as a function of three-session blocks of training. The elevation score was the rate during the CS minus the baseline rate of responding during empty intervals (see Data Analysis section above). This calculation was conducted for individual rats and then averaged across rats in a group.

The elevation scores were higher for groups that received shorter intervals. A Kruskal–Wallis test indicated a significant effect of interval duration during all blocks of training: smallest $\chi^2(2) = 7.1, p < .05$, except Block 4, $\chi^2(2) = 4.7, p = .09$. On the other hand, the number of CSs per interval, that is, the manipulation of constant versus proportional CS deliveries, did not affect the elevation scores, largest $\chi^2(1) = 1.9$ on Block 4.

Early pairings (in which the US occurs during a large percentage of CS presentations early in training) have been previously reported to affect the degree of conditioning in random control procedures (Benedict & Ayres, 1972). To determine whether this had an influence in the present experiment, the number of CS occurrences during which US was delivered (a CS–US “pairing”) after 1 or 10 CS presentations was used to create two groups of rats. Rats that received a pairing on Trial 1 were compared with rats that did not receive a pairing. In addition, rats that received two or more pairings in the first 10 trials were compared with rats that received no pairings or only a single pairing. Although the groups that received a US during the first CS presentation had higher elevation scores on the first session than groups that did not receive a pairing, this was not statistically significant, $\chi^2(1) = 2.0, p = .16$. Similarly, rats that received two or more pairings in the first 10 trials tended to have higher Session 1 elevation scores than rats that received zero or one pairing, but this was not statistically significant, $\chi^2(1) = 2.7, p = .10$. Moreover, early pairings had no
effect on the asymptotic elevation scores: Trial 1, $\chi^2(1) = 0.7$; Trial 10, $\chi^2(1) = 1.0$. One possible reason for the lack of a robust effect of early pairings was their relative infrequency. Only 6 of the 24 rats received food during the first noise presentation, and only 10 rats received two or more pairings in the first 10 noise presentations with a maximum of four pairings that was experienced by a single rat.

To assess whether the effect of the CS changed as a function of time in the CS, we determined the local rate of responding as a function of time since CS onset up until the time of CS termination or US delivery (see Figure 5). Baseline response rates were calculated using a pseudostimulus, which was a randomly distributed onset and termination marker in empty US–US intervals. Baseline rates were calculated from the time of pseudostimulus onset until pseudostimulus termination or US delivery (see Data Analysis above). The constant and proportional groups were combined because this manipulation did not have an effect on any measures of responding: largest $\chi^2(1) = 2.1$, ns. Responding is shown only for the first 15 s after event occurrence in the 45-s groups, 30 s in the 90-s groups, and 60 s in the 180-s groups. The upper bound of CS duration was a consequence of the infrequency of longer CS durations that were not truncated by food delivery, particularly in the 45-s groups.

Baseline rates of responding increased as a function of time, were higher for shorter intervals, and increased with a steeper slope for shorter intervals. The baseline rates differ somewhat from the functions in Figure 3 because the baseline rate calculations during the pseudo-CS began and ended at random times in the empty US–US intervals, whereas the data in Figure 3 were aligned from the prior food delivery. Presentation of the CS resulted in an increase in response rate that was maintained over time. The degree of increase produced by the CS was greater for shorter US–US intervals, which is consistent with the elevation scores in Figure 4.

The mean baseline and CS response rates were determined for successive thirds of the interval. An elevation score was also determined by subtracting the baseline rate from the CS rate during the corresponding third of the interval. The baseline rates, CS rates, and elevation scores were entered into a nonparametric repeated measures analysis to determine whether there were any changes in responding over time. The baseline rates increased as a function of time, $\chi^2(2) = 12.7$, $p < .01$, as did the CS rates, $\chi^2(2) = 14.6$, $p < .01$. However, the elevation scores did not change substantially over time; although there was a trend toward higher initial elevation scores this did not reach statistical significance: $45 \text{s}, \chi^2(2) = 1.8$, ns; $90 \text{s}, \chi^2(2) = 3.3$, ns; $180 \text{s}, \chi^2(2) = 4.8$, ns. Given the theoretical importance of this last analysis, it is important to examine further whether the elevation scores changed over time. Accordingly, linear functions were fit to the elevation scores as a function of time for each rat. The slopes of these functions were then tested against zero, with separate tests conducted for the different interval durations. These analyses did not reveal any significant departures from a zero slope: $45 \text{s}, t(7) = 1.9$, ns; $90 \text{s}, t(7) = 0.6$, ns; $180 \text{s}, t(7) = -0.2$, ns. Thus, the response to the CS did not appear to increase or decrease, relative to baseline rates, as a function of time since CS onset.

It is possible that the effect of the CS varied depending on the time in the US-US interval when it occurred. Figure 6 displays the baseline rates during empty intervals and rates of responding during the CS as a function of time since the previous food delivery (see Data Analysis above). Response rates were not calculated during the first 20 s following US delivery because the rats were most likely engaged in eating the food (see Figure 3), and this may have affected the response to the CS. Response rates were collapsed across the constant versus proportional manipulation because this had no effect on performance, $\chi^2(1) = 0.08$, ns.

As seen in Figure 6, both the baseline and CS response increased over the course of the US-US interval, but the magnitude of response to the CS increased at a higher rate than the baseline response. Thus, the CS was relatively more effective in invoking responding when it occurred later in the US-US interval. Elevation scores (CS rate – baseline rate) were calculated in successive thirds of the interval between US deliveries. A Kruskal–Wallis analysis revealed a significant effect of time on the elevation scores: $45 \text{s}, \chi^2(2) = 14.3$, $p < .001$; $90 \text{s}, \chi^2(2) = 14.3$, $p < .001$; $180 \text{s}, \chi^2(2) = 9.3$, $p < .01$. Pairwise Wilcoxon tests revealed that responding to the CS increased steadily as a function of time in the US-US interval (middle vs. early: $z = 4.3$, $p < .001$; late vs. middle: $z = 3.4$, $p < .01$). To verify the increase in elevation score as a function of time, we fitted linear functions to the elevation scores for each rat and then tested these against zero. The one-sample $t$ tests revealed significant positive slopes for all three interval durations: $45 \text{s}, t(7) = 6.5$, $p < .001$; $90 \text{s}, t(7) = 5.2$, $p < .001$; $180 \text{s}, t(7) = 3.0$, $p < .025$.

**Discussion**

There were four major findings of Experiment 1. First, there was evidence of responding to a CS that was presented at random with respect to the US (Figure 4). Second, the mean US-US interval was negatively related to the mean rate of responding during the background (Figure 3), and the mean CS onset-US interval was

![Figure 5](image-url)
negatively related to the mean rate of responding during the CS (Figure 5), so that the magnitude of conditioned responding was greater when the CS onset–US interval was shorter (Figure 4). These results are consistent with earlier research in which shorter CS–US intervals produced more robust conditioning in the truly random control (Kremer, 1971; Kremer & Kamin, 1971; Quinsey, 1971), but those studies had confounded mean CS–US interval and total exposure time to the CS. In the present study, the constant versus proportional manipulation had no effect on the magnitude of conditioning, indicating that the number of CSs and percentage of the session filled by the CS did not play a role in determining responding.

Third, the shape of the response rate functions was determined by the distribution of intervals: The fixed US–US interval resulted in increasing response rates (Figure 3), and the random CS onset–US interval resulted in a constant increase in response rates (Figure 5). A constant increase in rate of responding is the predicted pattern if the rats had learned the variable nature of the CS onset–US interval duration (Catania & Reynolds, 1968; Church & Lacourse, 2001; Kirkpatrick & Church, 2000a, 2003; LaBarbera & Church, 1974; Libby & Church, 1975; Lund, 1976).

Finally, the magnitude of response to the CS was temporally dependent. The rats demonstrated a systematic increase in the response to the CS as a function of the time that the CS occurred in the US–US interval (Figure 6). Thus, combining the results of Figures 5 and 6, it appeared that when the CS occurred later in the US–US interval, it elicited a larger increase in response rate (Figure 6), but the response rate was then maintained at a relatively constant higher level throughout the duration of the CS (Figure 5).

State-based accounts (as described in the general introduction) do not predict any differential responding during the CS versus the background because the rate of reinforcement in the two states was the same. One variation of contingency theory has been to assume that chance early pairings, in which the US occurs during the CS early in training, may result in a misperception that the rate of reinforcement in the presence of the CS is higher than the rate in the absence of the CS (Benedict & Ayres, 1972). However, there was no robust effect of early pairings on CS responding in the present experiment. It remains plausible that early pairings affect response rate early in training but that other factors are involved after more substantial training.

On the other hand, the results are consistent with predictions of an event-based account (e.g., Packet theory—see Figure 1). Analyses of the temporal pattern of responding were consistent with tracking of the expected time functions. For example, shorter intervals resulted in higher rates of responding; this was expected because the rate of packet generation (r) would be higher for shorter intervals (see bottom row of Figure 1). Moreover, the pattern of responding in time was consistent with the shape of the packet-generating functions. This was perhaps best demonstrated in Figure 5, which displayed a constant increase in the response rate following CS onset (compared with baseline) as a function of time in the CS.

An examination of the response to the CS as a function of time in the US–US interval revealed that the CS produced a larger response when it occurred late in the US–US interval (Figure 6). The pattern of response suggests that a linear combination rule (see Table 1) would best apply to the present data. Meck and Church (1984) found a similar pattern in their study of simultaneous timing. They trained rats on an overall fixed interval that was 1 min in duration, with a segment stimulus that was turned on every 20 s and lasted for 10 s. The response to the segment stimulus (relative to baseline) was greater when the stimulus occurred later in the overall fixed interval. They implemented a linear combination rule in which the response during the segment stimulus was due to a weighted sum of the response to the overall signal and the response to both signals. The results in Figure 6 could be fit in the same manner. Because the response during the CS is the result of responding due to the US–US decision vector (the packet-generating function) plus the product of the response due to the US–US and CS onset–US decision vectors, the resulting rate of response will be strongly influenced by the US–US decision vector. In the present experiment, the US–US interval was fixed, so the effect of the US–US decision vector on the response during the CS will increase over time in the US–US interval. This combination rule also predicts greater responding during the CS (e.g., Figure 3) in the shorter interval conditions, if weights a and c are held constant across groups, because the CS onset–US decision vector will be determined by the mean interval duration and interval distribution form (see Figure 1).

The present results indicate that learning of times between events may be a major contributor to random control performance. However, it is possible that a contingency approach could apply if one were to consider an alternative definition of contingency. Although the rate of reinforcement in the CS and background states was the same in all groups, the mean time from CS onset to US was not the same as the mean time from US to US (Figure 2), nor was the distribution of times the same. Thus, Experiment 2 implemented a truly random control in which these alternative definitions of lack of contingency were satisfied, as well as the original definition.
Experiment 2

In Experiment 2, we examined the effect of CS onset–US interval duration on conditioning in a conventional truly random control with a random US–US interval and CS presentations at random times. If the manipulation of the mean CS onset–US interval affects responding in a similar manner to Experiment 1, then this would lend further support to an event-based analysis (e.g., Packet theory) compared with a state-based analysis (e.g., contingency). This is because the random control used in Experiment 2 passes the definition of a lack of contingency in terms of relative rates of reinforcement, mean intervals to reinforcement, and distributions of times to reinforcement.

Based on Packet theory, combined with the results of Experiment 1, we predicted that mean interval duration (US–US or CS onset–US) would be negatively related to mean response rate (in the US–US or CS onset–US intervals) and that there would be more responding during the CS than in the background because of linear combination of responding from the US–US and CS onset–US decision vectors. In other words, the results of Experiment 2 are expected to closely mirror those of Experiment 1 in the analysis of mean rates. However, it was expected that responding during the empty US–US intervals in Experiment 2 should be relatively constant over time because the conditional expected time to US for a random US–US interval is constant over time (see Figure 1). The change in the US–US interval from fixed to random will also allow for a better assessment of the linear combination rule applied to the results in Figure 6. If the response to the CS is affected by the local expected time to US (via a linear combination rule), then the magnitude of CS responding should not change as a function of time in a random US–US interval. Again, this is because the expected time to US is constant over time for random intervals, and thus there will be no differential effect of the US–US expectation over time on the response to the CS.

Method

Animals

Twelve male Sprague-Dawley rats (Taconic Laboratories, Germantown, NY) arrived in the colony at 35 days of age and were handled daily until the onset of the experiments. Training began when they were 48 days old. All conditions of their housing and treatment were the same as in Experiment 1.

Apparatus

The apparatus was the same as in Experiment 1.

Procedure

The rats were randomly assigned to one of two training groups: 45 s or 90 s, with 6 rats per condition. The groups were labeled according to the duration of the US–US interval. There was no constant versus proportional manipulation because this had no hint of an effect in Experiment 1. Each group received 15 sessions of training with the random control procedure. Each session resulted in the delivery of 160 CS deliveries. Because the different groups received different US–US interval durations, the training sessions were 2 or 4 hr for groups that received 45 or 90 s US–US intervals, respectively.

The procedure received by each group is diagrammed in Figure 7. The US–US interval was an exponential random interval with a mean of either 45 or 90 s. Both groups received a white-noise CS duration that was an exponential random duration (~) with a mean of 15 s and a 75-s exponential random interval between the termination of one CS and the onset of the next CS. Thus, there was a 90-s random interval between successive CS onset (or terminations). The mean time between CS onset and US delivery was random 45 s in Group 45 and random 90 s in Group 90. The mean rate of US delivery was 1.33 and 0.67 reinforcers per minute during both the CS and background periods for Groups 45 and 90, respectively (see columns 3 and 4). The mean interval between CS onset and US delivery was equal to the mean US–US interval in each group (see columns 5 and 6).

Figure 8 displays the response rate as a function of time since food during empty US–US intervals in which there were no CS deliveries, collapsed over Sessions 6–15 of training. Following an initial high response rate, probably due to consumption of the food pellet, the response rates during the random US–US interval were relatively constant, albeit with some modest increase in Group 45. The mean response rates did not differ between the groups, $\chi^2(1) = 0.03$, ns (for all chi-squares in Experiment 2, $N = 12$).

Figure 9 displays the elevation score (responses/min) to the CS over the course of training. The elevation scores were calculated as in Experiment 1 (see Data Analysis section). The elevation scores were higher for Group 45 compared with Group 90, indicating that shorter intervals resulted in higher levels of CS responding.

The elevation scores were collapsed into three-session blocks. A Kruskal–Wallis test indicated a group effect for Blocks 3–5, smallest $\chi^2(1) = 4.3$, $p < .05$, but not during the first two blocks of training: Block 1, $\chi^2(1) = 2.1$, ns; Block 2, $\chi^2(1) = 3.1$, ns.

An analysis of the effect of early pairings on the elevation scores was conducted as in Experiment 1. Only 1 rat received a pairing on the first trial, so the effect of a pairing on the first CS presentation could not be determined. The rats were grouped according to whether they received zero to one pairings versus two to four pairings in the first 10 trials. A Kruskal–Wallis test indicated that there were no significant group differences on Session 1 perfor-
There was a hint of difference between the two groups in their asymptotic performance (Sessions 6–15) that did not quite achieve significance, \( \chi^2(1) = 3.5, \text{ns.} \) This difference was in the opposite direction to that predicted: The rats that received more pairings early on were performing worse at asymptote.

Figure 10 displays the response rate as a function of time since either CS onset or the randomly distributed pseudo-CS during empty US–US intervals (see Experiment 1 Data Analysis). Response rates were relatively constant during both the baseline (pseudo-CS) and CS periods. The two functions did not appear to converge or diverge over time. This was verified by repeated measures Friedman tests conducted on successive thirds of the interval for the baseline, largest \( \chi^2(2) = 0.7, \text{ns;} \) CS, largest \( \chi^2(2) = 2.0, \text{ns;} \) and an elevation score that was the baseline rate subtracted from the CS rate in each third of the interval: 45 s, \( \chi^2(2) = 1.0, \text{ns;} \) 90 s, \( \chi^2(2) = 0.3, \text{ns.} \) Additional analyses were conducted on the baseline rates, which appeared to differ between the two groups. Consistent with similar analyses reported in conjunction with Figure 8, there was no difference in the baseline scores during any of the three time periods (early, middle, and late) in the interval: largest \( \chi^2(1) = 0.9, \text{ns.} \) Additional analyses of the elevation scores were conducted as in Experiment 1 by testing the slopes against zero. There were no significant differences from a zero slope: 45 s, \( t(5) = -0.6, \text{ns;} \) and 90 s, \( t(5) = 0.0, \text{ns,} \) indicating that the CS increased responding by a constant amount as a function of time since CS onset.

The effect of the CS as a function of time of its occurrence in the US–US interval is displayed in Figure 11. The elevation scores added to the US–US interval from the time of the prior US delivery (these are the same data as in Figure 8).
were not calculated during the first 20 s following US delivery because the rats were engaged in consuming the prior food delivery, and this may have affected their response to the CS. There was a modest increase in responding early in the US–US interval, but thereafter the elevation scores were relatively constant. Repeated measures nonparametric tests conducted on the elevation scores in progressive thirds of the US–US interval did not reveal any significant effect of time in the interval: 45 s, $\chi^2(2) = 4.3$, ns; 90 s, $\chi^2(2) = 1.3$, ns. Analyses conducted on the slope of the elevation score as a function of time also revealed no significant departures from a zero slope: 45 s, $t(5) = 2.2$, ns; 90 s, $t(5) = -0.5$, ns. Thus, in contrast to Experiment 1 in which the CS was more effective when it occurred later in the US–US interval, with random intervals the effect of the CS was relatively constant over time.

**Discussion**

Changing from a fixed to a random US–US interval changed the pattern of responding during the US–US interval (Figure 8). In contrast to the fixed interval (Figure 3), the random interval resulted in relatively constant responding over time. This is the expected pattern based on previous research with random intervals (Catania & Reynolds, 1968; Church & Lacourse, 2001; Kirkpatrick & Church, 2000a, 2003; LaBarbera & Church, 1974; Libby & Church, 1975; Lund, 1976) and is consistent with tracking of the conditional expected time to food. However, it is puzzling that the 45-s random US–US interval did not result in greater responding than the 90-s interval. Typically, halving the US–US interval duration would result in a substantial increase in responding as in Experiment 1 and many other studies of interval schedules of reinforcement (e.g., de Villiers & Herrnstein, 1976), but perhaps not always (Kirkpatrick & Church, 2003).

As in Experiment 1, there was evidence of conditioning to the CS in that the rate of response during the CS was higher than in the background (Figure 9). In addition, the mean CS onset–US interval was related to the magnitude of conditioned responding, indicating that this result was not specific to fixed US–US intervals.

Given that the truly random control is considered to lack any contingency between the delivery of the CS and US, there is no reason to suppose that learning should occur from a contingency perspective. This lends support for an event-based approach, such as Packet theory.

It is interesting that the magnitude of responding to the CS as a function of its time of occurrence in the US–US interval (Figure 11) was different for the random US–US interval (compared with Figure 6 in Experiment 1). The elevation of responding during the CS was more or less constant as a function of time in the US–US interval. This further suggests that the response to the CS may be influenced by the conditional expected time until US, relative to the prior US. The linear combination rule proposed in Experiment 1 predicts this pattern of results. Because the US–US decision vector (bottom row of Figure 1) is constant over time, there would be no differential effect over time in the US–US interval on the combined response during the CS.

Although there is considerable support for the notion that learning in the random control is determined by the temporal intervals that make up the procedure, there are alternative explanations that may apply to at least a subset of the results. Because the manipulation of CS onset–US interval occurred through variations in mean US–US interval, it is possible that arousal produced by the density of US delivery could have contributed to the observed responding to the CS. The greater magnitude response to the CS exhibited in the shorter US–US interval groups may be due to a generalization of arousal from the denser schedule of US delivery. The groups that demonstrated the greatest CS response (45-s groups in Experiments 1 and 2) were the most responsive overall. Although an arousal interpretation could possibly explain the effects of temporal variables on the magnitude of responding, it cannot easily explain the pattern of responding as a function of time since food or CS onset because general arousal would not be expected to vary systematically as a function of interval distribution form. Thus, at a minimum, temporal information contributed by shaping the form of responding in time.

**Experiment 3**

Experiment 3 sought to investigate the effect of CS onset–US interval on conditioning in a random control procedure by manipulating CS duration. The main purpose was to determine whether the effects of CS onset–US interval on conditioning in Experiments 1 and 2 would occur in situations in which the density of reinforcement was held relatively constant. For this reason, the present experiment used a random control procedure that was developed by Bennett, Maldonado, and Mackintosh (1995).

The procedure involved food deliveries occurring in the background, with a constant probability of food per second of background exposure. Food deliveries were presented at the time of CS termination with the same probability as at any other time in the background. Thus, CS–US pairings occurred at chance, but the US never occurred during the CS, only at CS termination.

Different groups of rats were given this procedure, but with different duration CSs (10, 30, or 60 s). It was hypothesized that the rats should (a) demonstrate more responding during the CS compared with the background, as in Experiments 1 and 2; (b) show weaker responding when the CS is longer because of the increase in mean CS onset–US interval; and (c) demonstrate an increasing response rate over the course of the CS because of its fixed duration but demonstrate a constant rate of responding during the background period because of the randomly distributed food deliveries.

**Method**

**Animals**

Twelve male Sprague-Dawley rats (Harlan, UK) arrived in the colony at 35 days of age and were handled daily until the onset of the experiments. Training began when they were 55 days old. The rats were housed in pairs in a colony room with a 12-hr light–dark cycle (lights on at 8 a.m.). All other conditions of their housing and maintenance were as in Experiment 1.

**Apparatus**

The apparatus was the same as in Experiment 1, except that the boxes were controlled by two Viglen Pentium III computers running Med-PC for Windows (Version 1.23) with 2-ms resolution for recording and delivering events.
Procedure

The rats were randomly assigned to one of three training groups: 10 s, 30 s, or 60 s with 4 rats per condition. The groups were labeled according to the duration of the houselight CS. Each group received 15 sessions of training with a random control procedure. Each session resulted in the delivery of 20 reinforcements, with sessions lasting for approximately 1 hr.

The procedure received by each group is diagrammed in Figure 12; it is a fairly direct replication of Bennett et al. (1995). The procedure received by the three groups was the same in all respects except for the duration of the CS (10, 30, or 60 s fixed). The duration of the background period (when the CS was off) was a uniformly distributed variable interval ranging from 60 to 180 s, with a mean of 120 s plus the pre-CS period, which was 60 s in all groups. The variable interval for the background duration was randomly selected without replacement from the following list of 10 interval durations: 60.0, 73.3, 86.7, 100.0, 113.4, 126.7, 140.1, 153.4, 166.8, and 180.0 s. The time of US onset was determined by dividing the background period (excluding the pre-CS period) into 10 equally spaced bins. US deliveries occurred with probability 0.1 at the start of a bin, so the probability of the CS–US pairing was 0.1 (on 10% of the CS presentations, the US occurred at the time of CS termination). Trials were delivered in two blocks of 10 trials each that contained (a) 1 empty background trial, (b) 1 trial in which the US occurred at the time of CS termination, (c) 1 trial in which there were two US deliveries at random times within the background, and (d) 7 trials in which there was one US delivery at a random time within the background. US deliveries never occurred during the pre-CS period. Training was conducted over the course of 15 daily sessions.

The last four columns of Figure 12 contain the specification of the reinforcers/min during the CS and background (no CS) and the times between CS onset and prior US and the following US occurrence. The CS termination–US interval was random with a mean of 54 s in all groups. The mean duration of the background period was 120 s, with USs occurring on average halfway into the background period, but only occurring at the start of a bin so that the mean CS termination–US interval was equal to one half the mean duration of the background minus the mean bin width: (120 – 12)/2. The mean CS onset–US interval was equal to the CS duration plus the mean CS termination–US interval. The mean US–US interval was equal to the CS duration (10, 30, or 60 s) + the mean background duration (120 s) + the pre-CS duration (60 s). Although the probability of pairing was the same across groups, the reinforcers per minute during the CS varied as a function of CS duration, as did the mean CS onset–US interval. The rate of reinforcement during the background, however, was 0.3 reinforcers per minute in all groups.

Results

Figure 13 displays the response rate during empty intervals from CS termination to the next CS onset as a function of time since CS termination, collapsed over Sessions 6–15 of training. Response rates were initially high, due to elevated responding during the CS (Figure 14). Within about 10 s responding had dropped to a relatively constant level. The overall response rates were highly similar in the three groups, χ²(2) = 0.3, ns (for all chi-squares in Experiment 3, N = 12), which would be expected because the mean CS termination–US interval is the same in all groups, and the mean US–US interval was similarly long in the three groups.4

Figure 14 displays the elevation score (responses/min) to the CS over the course of training. The response rate during the pre-CS period was subtracted from the response rate during the CS to form the elevation score. The pre-CS period used in the analysis was the same duration as the CS (e.g., the last 10 s of the pre-CS period was used for the group with a 10-s CS).

The elevation scores were ordered according to the duration of the CS, with the highest responding in the 10-s group. A Kruskal–Wallis test was conducted on the elevation scores in three-session blocks. There was a significant group effect during Blocks 3 to 5, smallest χ²(2) = 6.6, p < .05, but not during Blocks 1 or 2, biggest χ²(2) = 1.1, ns.

Figure 15 displays the response rate as a function of time relative to CS (light) onset. Times prior to CS onset (pre-CS period) are negative, and times after CS onset are positive. Response rates increased following CS onset, with the greatest increase in the group that received the 10-s CS. A Friedman statistic run on the mean response rates in successive thirds of the CS revealed a significant effect of time since CS onset on response rate, χ²(2) = 9.2, p < .05. The increase in response rate is

<table>
<thead>
<tr>
<th>Group</th>
<th>Procedure</th>
<th>Reinforcers per min during</th>
<th>Mean time to US from CS onset</th>
<th>US</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 s</td>
<td>10 s</td>
<td>6</td>
<td>190 s</td>
<td></td>
</tr>
<tr>
<td>30 s</td>
<td>30 s</td>
<td>.2</td>
<td>210 s</td>
<td></td>
</tr>
<tr>
<td>60 s</td>
<td>60 s</td>
<td>.1</td>
<td>240 s</td>
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</tbody>
</table>

4 Although the variations in CS duration increased the US–US interval, the mean US–US intervals in all three groups were sufficiently long that one would not expect a significant contribution to responding. The relation between interval duration and mean rate has been demonstrated to be a decreasing nonlinear function with a slope of approximately −1 in a variety of conditioning paradigms (Kirkpatrick, 2002; Kirkpatrick & Church, 2003). Thus, doubling interval duration approximately halves response rate. In the range of US–US intervals used in Experiment 3, there are two factors that would lead one to predict little or no effect of the mean interval variations on performance. First, the mean intervals were all lengthy and should support low rates of responding. Second, the percentage change in the mean intervals was negligible, which will lead to the prediction of only small variations in mean response rates in the US–US intervals.
consistent with anticipation of the upcoming opportunity for US delivery in the background period.

There was no analysis of the effect of the CS as a function of time in the US–US interval because of the structure of the events in the procedure. The CS never occurred late in the US–US interval (because of the 60-s pre-CS period), and thus the opportunity to observe any variation in CS effectiveness was restricted.

Discussion

The predictions, based on Packet theory, were confirmed. First, conditioned responding was acquired during the CS, and lengthening the CS duration (and hence the CS onset–US interval) resulted in a reduction of responding to the CS. This result confirms the effect of CS onset–US interval on conditioning to the CS when CS duration is manipulated. Although there were some unavoidable variations in the US–US interval across conditions (increasing CS duration necessarily increases the time between US deliveries because there is one US delivery per CS/background cycle on average), the CS termination–US interval and the rate of US delivery in the background were the same in all groups. Moreover, the baseline rates of responding in the background were low and did not differ among groups (see Footnote 4), further implying that the differences in responding were not due to generalized arousal.

In addition, the response rate increased as a function of time since CS onset during the fixed-duration CS, indicating that the rats anticipated the opportunity to receive upcoming US deliveries. The response rate during the background was relatively constant as expected because of the random US deliveries during the background.

General Discussion

Summary of Results

Across three experiments that implemented variations of the random control procedure, the rats demonstrated a substantial impact of temporal learning on their performance both during the CS onset–US and US–US intervals. In all three experiments CS onset–US interval was related to the magnitude of conditioned responding during the CS, with shorter intervals resulting in higher rates of responding. This result is consistent with earlier research with the random control (Kremmer, 1971; Kremmer & Kamin, 1971; Quinsey, 1971) as well as research on interval duration effects on performance in other conditioning paradigms (e.g., Holland, 2000; Kirkpatrick, 2002; Kirkpatrick & Church, 2000a, 2003; Lattal, 1999). Although CS onset–US interval manipulations were confounded with US–US interval changes in Experiments 1 and 2, Experiment 3 indicated that the CS onset–US interval was the primary contributor to the effects on performance.

In addition, there was an inverse relationship between mean US–US interval duration and mean rate of responding in the background. This was seen most clearly in Experiment 1, in which the US–US interval was fixed in duration and was manipulated over a fairly wide range. This finding is consistent with previous observations in temporal conditioning paradigms, in which the
variation of mean US–US interval was a strong predictor of mean response rate regardless of whether the interval was fixed or random (Kirkpatrick & Church, 2003). This relationship is closely related to Herrnstein’s hyperbolic rule, which has been applied to predict the relationship between reinforcement density (which is the inverse of US–US interval duration) and mean response rate in operant schedules of reinforcement (de Villiers & Herrnstein, 1976; Herrnstein, 1970).

Additional evidence of temporal learning was apparent in measures of the pattern of responding as a function of time in all three experiments. When the rats received fixed intervals, they demonstrated an increasing response rate as a function of time in the interval (Figures 3 and 15). When the rats received random intervals, however, response rates were relatively constant over time (Figures 5, 8, 10, and 13).

State-Based Accounts

There are a number of different ways of characterizing the random control procedure. The most popular approach has been to focus on the rates of reinforcement during the CS and background states, as in contingency theory (Rescorla, 1968), the comparator hypothesis (Miller & Maltzel, 1988), and rate estimation theory (Gallistel & Gibbon, 2000, 2002). If the rates of reinforcement in the CS and background states are the same, then no learning should occur, which is clearly at odds with the present results as well as prior studies of the random control (Benedict & Ayres, 1972; Durlach, 1982, 1983; Goddard & Jenkins, 1987; Kremer, 1971; Kremer & Kamin, 1971; Quinsey, 1971; Rescorla, 2000). The comparator hypothesis can produce some conditioning to the CS, but it is not apparent how this model could accommodate the effect of CS onset–US interval duration on the magnitude of conditioning.

An alternative state-based approach is to presume that the CS and context accrue strength independently. The Rescorla and Wagner (1972) model is an example of such an approach; both the CS and the context accrue strength, each at a different rate depending on salience. During the CS, the resulting response rate is a function of the sum of the strengths of the CS and the context because the context is assumed to always be present. During the background, the context alone would control responding. According to the Rescorla–Wagner model, a CS can evoke more responding than the background early in training in a random control procedure if the CS salience is higher than the salience of the context. However, with continued training the context will eventually take over and the CS will lose associative strength. This is due to the fact that the CS is paired with the US on only a small percentage of occasions, whereas the context receives credit for all reinforcements. The present results are problematic for the Rescorla–Wagner model in that conditioning to the CS emerged and was maintained over many hundreds of presentations. Moreover, as noted in the introduction, Rescorla (2000) found that even though responding during the CS waned with further training, it still facilitated lever pressing, indicating that some learning about the CS–US relationship was still intact.

Perhaps the most serious problem with state-based accounts is that they overlook the temporal learning that was consistently observed during both the US–US and CS onset–US intervals. The present results indicate that these intervals controlled both the mean rate of responding and the form of responding in time. Moreover, in Experiment 1, the response to the CS was influenced by the passage of time in the US–US interval (see Figure 6). This result would seem difficult to predict in the absence of a representation of time since the prior US delivery. Thus, an event-based approach may prove more fruitful in explaining the full range of results.

Event-Based Approach

The effects of mean interval duration on response rate and interval distribution form (e.g., fixed, random, fixed + random) on the pattern of responding over time have been modeled within a single framework by using the conditional expected time function (see Figure 1). The conditional expected time function at Time 0 (interval onset) is equal to the mean interval. Interval distribution form determines the shape of the expected time function. It has been observed previously in delay (Kirkpatrick & Church, 2000a), trace (Kirkpatrick, 2002), and temporal conditioning procedures (Kirkpatrick & Church, 2003) that mean response rates are inversely related to the expected time at interval onset (mean interval duration) and that the form of responding in time is inversely related to the shape of the conditional expected time function. The present results indicate that the conditional expected time function can predict the effect of interval duration and distribution form on responding, even in procedures that are not supposed to result in any learning.

The conditional expected time function was related to both the form of responding and the mean rate of responding in all three experiments. Moreover, the modulation of the response to the CS as a function of time of its occurrence in the US–US interval can be explained by presuming that the momentary probability of response to the CS is due to a linear combination of the US–US and CS onset–US decision vectors.

The linear combination rule is related to the combination rule used by Meck and Church (1984), therefore indicating that linear combination may occur in at least some simultaneous timing arrangements. Alternatively, some prior studies of simultaneous temporal processes have yielded data consistent with summation (Kirkpatrick & Church, 2000a), whereas others have indicated that a winner-take-all rule is more appropriate (Meck & Church, 1984). As demonstrated in Table 1, these rules can all be obtained from the same general formulation, but with different settings of the three weights. Further research is needed to determine the procedural aspects that may encourage one rule in favor of another. One factor that may prove important is whether the two intervals are trained together or separately; Meck and Church (1984) found evidence of linear combination when two differently signaled intervals were trained together in the same phase, whereas winner-take-all was obtained when the two signaled intervals were trained separately and then were later compounded.

In addition to accounting for the present data, an event-based approach can explain the effect of some other manipulations that modulate the magnitude of CS responding in random control paradigms. One effect that impairs CS responding is the presence of additional unsigned USs in the background (Ayres, Benedict, & Witcher, 1975; Keller, Ayres, & Mahoney, 1977). To reiterate the fundamental assumptions of Packet theory (Kirkpatrick, 2002; Kirkpatrick & Church, 2003), the mean US–US interval is the arithmetic mean of all intervals between successive USs. The mean CS onset (or termination)–US interval is the mean of all intervals
between US occurrence and the most recent CS onset, regardless of whether that US occurs during the CS or during the following background period. These assumptions lead to the prediction of an enhancement of the CS response following the introduction of extra background US deliveries because this manipulation decreases the mean CS onset–US interval, which will increase CS responding. Another established effect in random control procedures is an increase in CS responding if backward CS–US pairings are removed from the procedure (Wagner & Larew, 1985). This manipulation will increase the mean US–US interval and thus will decrease the background response so that the relative response to the CS (measured by an elevation score) will increase.

As noted in the introduction, there are many instances in which the random control procedure has resulted in learning, but there are also instances in which the random control has resulted in no discernible evidence of learning (Gamzu & Williams, 1971, 1973; Quinsey & Ayres, 1969; Rescorla, 1968, 1969). According to Packet theory, one would expect conditioning to occur in all instances; the rate during the CS will always be higher than the rate in the background because the CS rate is a combination of rates controlled by the US–US and CS onset–US intervals. However, the extent to which there will be a noticeable increase in rate during the CS will be determined by the US–US and CS onset–US intervals. As these intervals are lengthened, the impact on responding will diminish because response rates will decrease toward zero (see Footnote 4). As a result, it is expected that longer CS onset–US interval durations would yield only a small increase in response rate during the CS, perhaps too small to discern statistically. Although the intervals that comprise the procedures are not reported in many of the previous failures to observe conditioning, many of these experiments have been conducted with conditional emotional response paradigms, which typically involve quite long interval durations. Thus, these prior failures to observe conditioning may be due to the diminished effect of long CS onset–US intervals. In fact, in the present experiment there was little evidence of conditioning in the longest interval arrangements (e.g., the 180-s conditions in Experiment 1 and the 60-s condition in Experiment 3), yet there was fairly robust conditioning with the shortest intervals (e.g., the 45-s conditions in Experiments 1 and 2 and the 10-s condition in Experiment 3). These results indicate that within the same experiment it is possible to observe varying levels of success in producing learning in the random control by varying the interval durations.

Although the present article has focused on Packet theory, there are other event-based theories that may be able to accommodate at least some of the present results. For example, the temporal difference model of conditioning (Moore & Choi, 1997) presumes that there is a cascade of activation following both CS onset and CS termination events and that responding is determined by the sum of the strengths of the two cascades. Although this model would have difficulty predicting the effect of the time since the prior US on responding to the CS (Figure 6), it could account for many of the present results. In fact, any event-based model that incorporates multiple interval timing in some fashion would be able to account for at least a subset of the present results.

**Conclusion**

The random control procedure was developed to control for nonassociative factors that may influence conditioned responding. Given that learning does occur in the random control procedure, one must question the fundamental assumption underlying its development. In fact, Rescorla (2000) stated that the presence of learning in the random control procedure does “little to increase the attractiveness of a random procedure as a control condition for Pavlovian conditioning” (p. 338). Rescorla went on to indicate that the learning that he observed in the random control must “discourage the view that identification of an appropriate control procedure can be made on purely operational grounds” (p. 338). There has been a fairly rich history of argumentation over the selection of a proper control for simple conditioning, and the proposals have included US alone, CS alone, explicitly unpaired CS–US presentations, and uncorrelated CS–US presentations. All of these procedures have been demonstrated to result in learning. Most classical conditioning procedures are composed of events that occur in time. Animals learn about the relationships among these events, and they most certainly learn about the time course of events. Control procedures are no different: For example, unpaired conditioning has been reported to result in inhibitory learning because the animal learns that the CS predicts a period of time during which the US cannot occur. Perhaps a better way forward would be to determine the common or unique factors involved in learning and performance under different arrangements of experimental events, regardless of the formal names that are given to the procedures that deliver the events.

The present results argue against a contingency account of random control performance. We are not unique in proposing that the concept of contingency has little merit in understanding the nature of learning in classical conditioning experiments. Based in part on the observation of learning in the truly random control, Papini and Bitterman (1990) concluded that “The evidence suggests that CS–US contingency is neither necessary nor sufficient for conditioning and that the concept has long outlived any usefulness it may once have had in the analysis of conditioning” (p. 396). It is clear that the concept of contingency in its traditional sense is not useful in explaining the present results. Perhaps part of the problem is that the same contingency can be created with vastly different CS onset–US and US–US intervals. As shown in the present set of experiments, both of these intervals have a profound impact on responding. If one were to abandon the notion of contingency as suggested by Papini and Bitterman (1990), then a focus on the intervals that compose a procedure would be one possible, much-improved alternative.

**References**


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