The Effect of Retention Interval on Temporal Control of Responding in Rats on the Peak Interval Procedure

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THE EFFECT OF RETENTION INTERVAL ON TEMPORAL CONTROL OF RESPONDING IN RATS ON THE PEAK INTERVAL PROCEDURE

by

CATHERINE TSIRIS

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

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Abstract

THE EFFECT OF RETENTION INTERVAL ON TEMPORAL CONTROL OF RESPONDING
IN RATS ON THE PEAK INTERVAL PROCEDURE

by

Catherine Tsiris

A considerable body of literature on time perception has investigated the effects of short intratrial retention intervals (RI) in the seconds range, on temporal performance, however there is a dearth of research examining the effects of long, intersession RIs (hours to days range) on timing. The present study examined the effect of two different RI durations (2 days and 70 days) on time estimates in subjects on the peak interval procedure. Twenty-four rats, eight per group, were exposed to one of three conditions. The experimental group was trained at 5 months and tested after a 70-day RI. One control group was trained at the same chronological age as the experimental group and tested after a 2-day RI. The second control group was trained at 7 months and tested at the same chronological age as the experimental group. Following the RI, peak time decreased in testing when compared with peak time during training for the group exposed to the 70-day RI, but not for the two groups exposed to a 2-day RI. An individual trials analysis showed that the decrement in peak time was accompanied by a decrease in stop time but no corresponding change in start time for the long RI group. Start and stop times at testing did not vary significantly from training for either of the control groups. Variability in middle, start and stop times was found to increase significantly for the experimental group but not the control groups. The current findings can be accounted for by an increase in variability of the
remembered time of reinforcement, or increased variability in start and stop thresholds of the
decision mechanism of the internal clock model posited by scalar timing theory.
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**Introduction**

Interval timing refers to the timing of durations in the seconds to minutes range and is a critical feature of associative learning and adaptive behavior in humans and animals (Balsam, 1984; Gibbon, 1977; Gibbon & Balsam, 1981; Miller & Barnett, 1993). The peak interval (PI) procedure has often been used to study interval timing in humans and animals. The PI procedure is a modification of the discrete-trial fixed interval (FI) procedure, wherein on each trial, the first response after a pre-specified interval has elapsed is reinforced. However, in the PI procedure, a number of the trials are non-reinforced probe trials, randomly interspersed with reinforced FI trials. On probe (test) trials, the trial signal (discriminative stimulus) is presented for a duration that is two to three times as long as the originally trained FI value. When response rate is averaged across probe trials for a well-trained subject, the mean response rate is found to increase as a function of elapsed time until it peaks at approximately the time that reinforcement is sometimes available, and to decline thereafter. Peak time, that is, the time of maximum responding, approximates the fixed interval value, and is used as a measure of the subject’s estimate of the FI duration (Roberts, 1981). The width of the function is a measure of the variability in responding and denotes precision of timing. The peak interval procedure has been an important tool in the study of the effects of different manipulations on timing behavior.

Scalar expectancy theory (SET) is currently the dominant model for studying and understanding human and animal interval timing as it relates to learning (Gibbon, 1977). SET is a mathematical model that describes performance of organisms on various interval timing tasks. SET was later developed as an information-processing model based on the internal clock model first proposed by Triesman (1963). The information-processing model henceforth referred to as scalar timing theory, describes psychological processes in operation in animal and human
temporal perception and during timing tasks (Church, 1984; Gibbon & Church, 1990; Gibbon, Church, & Meck, 1984).

According to the internal clock model, temporal processing occurs within three interrelated components: clock, memory, and decision modules (see Figure 1). The clock module consists of a pacemaker that emits pulses that are gated by a switch, to an accumulator. The memory module consists of a working memory component that represents the temporary contents of the accumulator, and an enduring, reference memory component that stores values of previously reinforced durations. The third component of the model is the decision module, which consists of a comparator that is continuously making comparisons between the contents of working memory during a trial and a sample of the expected time of reinforcement drawn from a distribution of values in reference memory. A response is generated according to a decision rule when the perceived elapsed time in working memory approximates the remembered value in reference memory.

Scalar timing theory accounts for temporal performance on the peak interval procedure. Following the onset of a visual or auditory timing signal, the switch, which is under stimulus control of the timing signal, closes, permitting pulses generated by the pacemaker to be transmitted to the accumulator. The number of pulses in the accumulator represent subjective elapsed time and are thought to increase linearly as a function of objective time (Allan, 1998). At trial onset, a value is also retrieved from a distribution in reference memory, the contents of which are based on prior experience of reinforcement times. As time elapses in the trial, the current time in the accumulator, or working memory, is continuously compared with the sample from reference memory. When the current perceived time resembles the sample from reference memory according to a ratio of elapsed time to remembered time of reinforcement generated by
the decision mechanism, the organism switches from a low rate of responding to a high rate. The
time of the transition is referred to as start time. On FI trials, the value of reinforced time is
assumed to be transferred to reference memory. Performance on probe trials is identical except
that reinforcement is not forthcoming, thus, when the elapsed time eventually exceeds the
duration sampled from reference memory the decision mechanism generates a second switch in
response rate from a high rate to a low rate. The time of this transition is referred to as stop time,
for the remainder of the trial. When response rate as a function of elapsed trial duration is
averaged across trials, variability among trials in the pattern of responding typically generates a
Gaussian-shaped response rate function with the highest rate of responding at approximately the
programmed FI value.

A number of studies in the interval timing literature have provided evidence regarding the
properties of the working memory component of the memory module. Short retention intervals,
in the seconds to minutes range, have typically been used to study how they affect short-term
memory or working memory (Cabeza de Vaca, Brown, & Hemmes, 1994; Church, 1980; Grant,
1975; Kraemer, Mazmanian, & Roberts, 1985; Roberts & Grant, 1978; Spetch & Wilkie, 1983).
For example, the gap procedure, which is a modification of the PI procedure, has provided
evidence bearing on temporal working memory. In this preparation, three types of trials are
presented, FI, empty probe trials as described above, and gap trials. Gap trials are identical to
probe trials except that the timing signal is terminated for a brief period of time during the trial
creating a gap, in an otherwise continuous timing signal. Insertion of a gap has been found to
influence peak time (e.g., Cabeza de Vaca, Brown, & Hemmes, 1994; Roberts, 1981). Some
research with rats found that peak time shifted by the same amount of time as the gap duration
itself, and thus suggested that the accumulation of time stops during the gap and resumes when
the timing signal is reinstated (Roberts, 1981). Other research with pigeons found that the shift in
the peak function was larger than the duration of the gap, close to the duration of the gap plus the
stimulus duration that preceded the gap. This outcome has suggested that the gap induces a
resetting of the duration stored in the accumulator so that accumulation starts at zero after the
gap (Roberts, Cheng, & Cohen, 1989). There are therefore two alternative explanations to
account for the peak time shift that is found with the PI Gap procedure depending on outcomes:
stop and reset.

Cabeza de Vaca, Brown, and Hemmes (1994) suggested another explanation of the gap
effect. These authors manipulated gap location, gap duration or both, in two experiments using
the peak interval procedure (FI 30 s) with pigeons. In the first experiment, the authors found, as
previous researchers had, that the peak time function on gap trials shifted to the right, but did not
correspond in magnitude to either stop or reset modes, rather, the shift fell between those two
predictions. In Experiment 2 the authors conducted three parametric manipulations in which
parameters of the gap were manipulated within sessions. In the first manipulation, location of
gap onset was fixed (6 s from beginning of trial) but gap duration varied (3, 6, 9, 12, or 15 s).
The second manipulation kept the duration constant (6 s) but varied the location of gap onset (3,
6, 9, 12, or 15 s from start of trial). The third manipulation varied the duration of the gap but kept
the location of the end of the gap constant (21 s from start of trial). The authors found results that
were inconsistent with both the stop and the reset hypotheses; rather, their results indicated that
peak times fell in between the values predicted by those hypotheses. The functional relationships
between peak time shift and each of the parameters were consistent with a common process.
Cabeza de Vaca et al. argued that their findings were consistent with exponential decay of
working memory during the gap. The same decay explanation accounted for the results of the
gap location and duration manipulations. According to this account, the gap imposes a retention interval, during which remembered elapsed time decays in value. That interpretation is consistent with data (choose short effect) from delayed matching to sample studies of temporal discrimination that have been interpreted in terms of subjective shortening of remembered duration during the retention interval (Spetch & Wilkie, 1983; Spetch & Rusak, 1989).

Spetch and Wilkie (1983) employed a delayed symbolic matching to sample (DSMTS) task to examine pigeon’s memory for duration. Pigeons were trained to peck a red choice key after a short-duration sample key, and a green choice key after a long-duration sample key. After training, delays were introduced between the sample and comparison keys. The authors found that when the two choice stimuli (red and green keylights) were presented following a retention interval (delay) as opposed to immediately following the presentation of a sample, pigeons accurately selected the “short” stimulus when matching to a short sample, however errors increased when birds were presented with a “long” sample stimulus prior to the delay. That is, subjects tended to exhibit a “choose short” bias. To account for these results, the authors proposed that the choose-short effect might be due to a subjective shortening of the representation of the event duration following long retention intervals. That is, the remembered value of a sample stimulus decreases over time so that at long delays it may appear to approximate that of the short stimulus, hence, more "short" responses are likely to be made. This interpretation is a version of a memory decay account, and envisions decay as subjective shortening of remembered duration during the retention interval.

Buhusi (2003) proposed a "time-sharing" interpretation to account for the findings in the gap procedure by suggesting an attentional mechanism that regulates the rate of decay in working memory. That is, during gap trials, attention focused on the gap results in a
corresponding decrease in attention to the timing task. The time-sharing hypothesis proposes that working memory is shared among various inputs in a competitive manner, and therefore decay in working memory is proportional to the salience of the gap and any distractors, that is, the greater the salience or duration of the gap, the greater the number of pulses lost. Buhusi, Perera, and Meck (2005) expanded upon the ideas put forth by Buhusi (2003) at the same time extending the memory-decay hypothesis put forth by Cabeza de Vaca et al., (1994). In Experiment 3, they used a PI 40-s procedure with three different gap durations (5, 10 and 15 s), and two intensity levels of the visual to-be-timed stimulus, low and high. Buhusi et al. found that response rate functions shifted more with the high intensity stimulus, in line with a reset rule in which after the gap the animals begins timing the interval from the beginning. Low intensity stimulus trials resulted in shifts that were less than those for the high intensity trials, although significantly greater than the stop rule, in which animals stop timing during the gap and resume timing when the signal resumes, suggesting that shifts in timing on gap trials is directly related to stimulus intensity. The authors proposed that an active decay process is occurring during high salience trials and invoked an attentional-sharing model to interpret the findings.

More is known about short retention interval effects on temporal working memory than about the effects of long retention intervals on reference memory. Whereas studies investigating the working memory component of the internal clock have used short retention intervals within trials, studies of long-term memory insert delays between sessions. Only a few researchers have explored this domain and the results are inconsistent (Campbell & Haroutunian, 1981; El Massioui, Doyere, & Brown, 2006; Gleitman & Bernheim, 1963).

Gleitman and Bernheim (1963) investigated performance on a fixed interval schedule in 6-month old rats after two different retention intervals (short and long). Animals were trained on
a FI 1 min schedule for three days after which one group was placed on a 24-hr retention interval whereas a second group was placed on a 24-day retention interval. Both groups were administered a single test session on an FI 1 min schedule the day following their prescribed retention interval. Examination of first-half ratios (a relative response rate measure defined as the number of responses during the first 30-s of the interval divided by the number of responses during the entire interval) revealed a significant effect of retention interval. The authors found that both groups showed similar patterns of responding on the last day of training. During testing, the animals exposed to the long retention interval of 24 days exhibited significantly higher first-half ratios when compared with the control group, indicating that the subjects in the 24-day group failed to suppress responding earlier in the interval compared to the 24 hr group. The authors attributed the early responding in the FI interval in the long retention interval group as forgetting, expressed as an increase in response strength early in the interval for the long retention interval group compared to the short retention interval group (Gleitman & Bernheim, 1963; Gleitman, Steinman, & Bernheim, 1965).

There are two possible interpretations of the foregoing results. They could signify an increase in variability of responding, as in the flattening of the function relating response rate to elapsed trial time. That is, Gleitman and Bernheim's data are consistent with a loss of temporal precision (increased variability) from training to testing for the 24-day group. Alternatively, the data may reflect an effect of retention interval on timing accuracy. The maximal rate of responding for the 24-day group may have shifted to an earlier time in the trial, suggesting a loss of accuracy in timing, and would have been demonstrated by an earlier peak in the response function. Neither of these interpretations can be supported without the use of the peak interval procedure. In order to ascertain if one interpretation fits the data better, or if both explanations
could account for the data, one could replicate Gleitman and Berheim using the peak interval procedure that would yield more complete information on the temporal pattern of responding, including a measure of peak time.

Campbell and Haroutunian (1981) trained 6, 12, and 26-month old rats on a FI 1 min schedule, after which the subjects were placed on a 16-day retention interval. Immediately following the retention interval, subjects were tested on the FI 1 min schedule for a single session. The authors reported mean percent of terminal rate as a function of elapsed trial time (i.e., the mean number of responses in each 10 s segment of the FI 1 min schedule divided by the mean number of responses during the final 10 s of the FI duration) on the last training day and the post-retention-interval test day. The results indicated a greater change in response pattern on the post retention interval session in older rats (26 months) as opposed to younger subjects (ages 6 and 12 months). Older subjects (26 months) responded earlier in the test trial compared to the 6 and 12 month old subjects. The authors proposed that the older animals responded earlier in the FI interval than 6 or 12 month old animals owing to forgetting of the temporal characteristics of the schedule.

As with Gleitman and Bernheim (1963), there are two ways to interpret Campbell and Haroutunian’s results. Campbell and Haroutunian’s interpretation focused on the increase in responding earlier in the trial, which could reflect an increase in the variability in responding. These authors’ interpretation is tantamount to a precision account. An alternative explanation is that for the older animals, remembered time of reinforcement decreased following the retention interval. The latter interpretation would suggest a decrement in timing accuracy. However, Campbell and Haroutunian’s study did not allow one to say whether or not the effects seen were attributable to changes in precision (variability) or accuracy (the time estimate) or both. In order
to determine this, one could use the PI procedure, where one can measure both the variability of the function and the time estimate—peak time.

In an unpublished study, El Massioui, Brown, & Doyère (2006), used a PI procedure employing a FI 30s criterion value and a tone signal, with a single group of 6 month old rats. Following training on the PI procedure, the subjects were placed on a 4 month retention interval after which they were returned to the experimental situation on the PI procedure. The authors found a significant reduction in peak time (time of maximum responding in the interval) in rats following the retention interval (mean peak time here), while peak rate (the rate of responding at peak time) was unaffected. These data are consistent with an accuracy interpretation—that is, a change in remembered duration of the FI value during the retention interval. Although data from only the first post retention interval session were reported, the authors also found that some recovery of peak time occurred across subsequent post-retention interval sessions toward peak times slightly shorter than pre-retention interval peak times (30 s) (personal communication).

It is worth noting that the studies investigating the effects of long retention intervals discussed above did not control for age at the time of testing. In order to rule out age effects, one must control for age at the time of training as well as at the time of testing. For example, El Massioui et al. found a decrease in peak time demonstrated by the subjects following a 4-month break that could conceivably be attributed to the aging of the animals as opposed to the retention interval. It is also worth noting that apart from El Massioui et al. (2006) no other study used the PI procedure. Nonetheless, El Massioui et al. restricted their examination of the data to a molar analysis. An investigation of the retention interval effect would profit from the use of additional, molecular, analyses of the data.
Examination of the data via a molar analysis alone does not permit distinguishing the mechanism that may underlie observed changes in the peak interval function post retention interval. The use of the peak interval procedure permits a number of measures of timing behavior. When plotted, the mean response rate averaged across probe trials (the peak function), normally exhibits a well behaved Gaussian distribution, with the mean of the function located at approximately the time of expected reinforcement. As indicated above, from this peak function one can derive peak time and peak rate. Response rate functions averaged across trials however do not permit examination of individual subject’s timing performance on single trials. An individual trials analysis provides information regarding start, stop, and middle times, in addition to spread, and can help interpret the effects obtained (e.g., Gibbon & Church, 1990; Church, Meck, & Gibbon, 1994; Cheng & Westwood, 1993; Gallistel, McDonald & King, 2004; Matell & Portugal, 2007; Taylor, Horvitz, & Balsam, 2007; Balci et al., 2009; MacDonald, Cheng, & Meck, 2012). It has been demonstrated that responding on the peak interval procedure on individual trials typically conforms to a three-state pattern of responding in which animals exhibit a low rate of responding early in the trial, before the previously trained FI value has elapsed, then switch to a high rate of responding around the previously trained FI value, followed by a transition to a low rate of responding when the FI period has elapsed. This low-high-low pattern of responding has been interpreted in terms of the threshold mechanism of the internal clock model in which a decision is to start responding (at a higher rate) is presumed to occur when perceived elapsed time is sufficiently similar to the remembered time of reinforcement, and responding stops (reduction in rate) when the current time is past the remembered time of reinforcement. The time of the transition from a low rate (or state) to a high rate of responding has been referred to as the start time, whereas stop time is the time of the transition back to a
lower rate of responding. These measures further generate a middle time (the mean of start and stop time), which is presumed to reflect a measure of the subject’s subject estimate of the time of reinforcement, while the spread of the function (the start time subtracted from the stop time) reflects temporal sensitivity to duration. Church, Meck and Gibbon (1994) reported a reliable positive correlation between start times and stop times in that when animals start responding later on a given trial, they likewise tend to stop responding later as well. The positive start-stop correlation reflects variability in reference memory. Additionally, a reliable pattern of negative correlations was revealed between start time and the spread indicating that animals tended to respond for a longer duration in the high state on trials in which they had started responding earlier. The negative start-spread correlation reflects variability in the decision threshold. These measures permit a distinction between alternative mechanisms that influence molar performance. For example, an effect on peak time may be due to changes in reference memory or to asymmetrical changes in start and stop thresholds. Further, an effect on variability may be due to changes in reference memory variability or to spread. The individual trial analysis can help determine which interpretation the data support.

The purpose of the present study was to investigate the effect of the duration of a between-session retention interval on timing performance on the peak interval procedure employing a similar procedure to El Massioui, Brown and Doyère (2006). For different groups of rats, the retention interval was either 2 days or 70 days. Furthermore, the current design controlled for age effects by employing a 2-day retention interval for a control group that was tested at the same time as the experimental group in addition to a 2-day retention interval for a control group (ES) that was trained at the same time as the experimental group. Following the retention interval, performance was examined on five successive test sessions. The goal of the
current research was to investigate whether or not a long retention interval would experimentally affect timing behavior, and if so, in what manner. That is, would temporal performance following the retention interval change in accuracy and/or precision. Furthermore, the study addressed the question of whether long retention intervals produced changes in temporal performance that implicated the reference memory component of the clock model, or whether other clock mechanisms were implicated. For this reason, both molar and molecular analyses of the data were conducted.
Method

Subjects

The subjects were 24 experimentally naïve, male, Long Evans rats approximately 150 days of age at the beginning of the experiment. The subjects were obtained from colonies maintained by the experimenter at the animal facilities of Queens College, City University of New York. The rats were housed individually in shoebox cages in a with a 12:12 hr dark/light cycle (lights on at 8:00 am) and all experimental sessions were conducted during the light-on portion of the cycle. Training and test sessions were conducted 7 days a week at approximately the same time. Subjects’ daily food allowance of Purina Rat Chow™ was gradually reduced over 2 weeks until they reach 80% of their free feeding body weight and were maintained at a weight adjusted for normal growth with daily supplemental feedings in their home cages after experimental sessions or at the post-session time on non-session days. Subjects were weighed daily and were maintained at approximately this 80% weight adjusted for normal growth for the duration of the experiment. Animals were provided with *ad libitum* access to water in their home cages. Manipulations were conducted in accordance with the guidelines approved by the Institutional Animal Care and Use Committee of the City University of New York.

Apparatus

Eight commercially constructed customized operant chambers (Gerbrands, Model 312) were used for the experimental sessions. The chambers measured approximately 31 cm wide x 33 cm deep x 30 cm high and were housed in sound attenuating, ventilated boxes (45.7 cm wide by 61 cm high and 91.4 cm deep). The chamber walls and ceiling were constructed of aluminum, except for the front door panel that was constructed of Plexiglas™, and the floor rods (spaced 20 mm apart) were constructed of stainless steel. A 28-V houselight was mounted in the center
of the ceiling. A 28-V houselight was mounted in the center of the ceiling on the back wall. Two non-retractable levers, each 5 cm wide and 11.5 cm apart from each other were located 5 cm above the grid floor. A force of 20 gm (.2 N) or greater was necessary to operate the levers. Only the right lever was used in the present experiment. Two sets of eight panel lights were located 4.5 cm above each lever. A single panel lamp over the right lever remained lit during experimental sessions to provide illumination. A 4.5 cm square opening was located midway between the two levers, and contained a feeder tray into which 45-mg Bio-Serv™ grain-based food pellets were delivered. A fan mounted on the back wall of each outer chamber provided ventilation and some white noise of approximately 70 dB throughout the sessions. Med associates multiple tone generators, model ENV-223, mounted on the back of each outer chamber generated the auditory timing signal tested at 85 dB and 5 KHz which was produced through standard 2 inch, 8-ohm speakers located at the center of the ceiling of each operant chamber. Experimental chambers were connected to Med Associates input and output cards. Experimental events were controlled and recorded via a PC computer running MED-PC IV for Microsoft Windows™ XP operating system located in an adjoining room.

**Experimental Design**

The twenty-four rats were randomly assigned to one of the eight chambers and to one of three groups of eight subjects each. Groups were labeled as follows: Group Early-Short (ES) was trained early at 5 months of age and tested after a short retention interval of 2 days. Group Early-Long (EL) was trained early at 5 months and tested after a long retention interval of 70 days. Group Late-Short (LS) was trained late at 7 months and was tested after a short retention interval of 2 days (see Table 1). Subjects in the two groups that received early training at 5
months were counterbalanced across two daily experimental sessions that were conducted approximately 2 hrs apart.

**Procedure**

All experimental sessions (excluding magazine training) began and ended with an intertrial interval (ITI) with a mean duration of 60 s. Intertrial interval durations were 30, 60 and 90 s and were randomly selected without replacement from an array in which each duration was represented twice.

**Magazine training.** On day 1 all animals were provided with a 1 hr session in which a single pellet was delivered into the food cup on a fixed time (FT) 60 s schedule, independent of the subjects’ behavior. Visual inspection of all subjects during the session through a peephole in the sound attenuating enclosure was conducted on some trials throughout the session to confirm the animal’s approach and consumption of the food pellets upon delivery.

**Response training.** On days 2 through 4, all animals were exposed to a conjoint FT 60 s fixed ratio (FR) 1 schedule in which a single pellet was delivered into the food trough every 60 s in the absence of a response. In addition, each lever press response resulted in the immediate presentation of a food pellet. The session ended when 60 reinforcers were delivered or 2 hrs had passed, whichever came first.

**Training on a fixed interval procedure.** On days 5 through 10 (6 sessions) all animals were trained on a standard discrete-trial fixed interval (FI) 30 s procedure, in which a trial was signaled by the onset of the tone (auditory cue). The first lever press response after 30 s from cue onset was reinforced with a food pellet, and terminated the trial. Trials ended 60 s after trial onset in the event that a response did not occur. Each session consisted of 60 trials.
**Peak interval procedure.** On days 11-26 (16 sessions). All subjects continued training on a peak interval (PI) procedure that was identical to the previous FI training except that 25% of the trials were non-reinforced probe trials, randomly interspersed with FI trials, in which the auditory timing cue was presented at the start of the trial and remained on for 90 s. Response rate functions on probe trials were monitored daily to ascertain stable performance.

**Retention interval.** Following their respective PI training, subjects in Groups E-Short and L-Short were placed on a 2-day (48-hr) retention interval in their home cages. Subjects in Group E-Long were exposed to a 70-day retention interval in their home cages. Animals were maintained at 80% of their free feeding body weight during the retention interval and were provided with supplemental feedings in their home cages, to adjust for normal growth.

**Testing and reacquisition.** Following a 2-day retention interval, subjects in Groups E-Short and L-Short were returned to the experimental preparation on the PI procedure for five sessions. Group E-Long subjects were administered the five peak interval sessions starting on the first day following a 70-day retention interval.

**Data Analysis**

**Response rate.** Pre- and post-retention interval performance was assessed by analyzing performance during non-reinforced probe trials (15 probe trials per session). The number of responses on probe trials were collected in 1 s bins for analysis of the peak interval function, and response rate functions of elapsed time across trials were produced for each subject and for each session, from which peak time was ascertained.

Additional analysis of the response functions and peak times were conducted employing a smoothing procedure (Balci, et al., 2009). Individual subject's response rates averaged across trials in a session were smoothed in 19-s sliding blocks in 1-s increments. The length of the
sliding block was gradually increased across bins 1-10, to 19 s, and gradually decreased for bins 81-90.

Peak time was defined as the bin representing the center of the sliding block containing the maximum smoothed response rate. Owing to some early bursts of responding to the onset of the timing signal and resurgence observed at the end of the interval on some trials, a limitation was applied in which peak time was not permitted to occur in the first or last 5 seconds of a trial (Balci et al.,). In the event that peak time occurred within the first or last 5 seconds of a trial, the initial value was discarded and the next maximum smoothed rate was identified as the peak time.

**Individual trials.** In order to assess changes in post retention interval PI performance in both accuracy and precision, individual trial analyses were conducted (Cabeza de Vaca, Brown, & Hemmes, 1994; Church, Meck & Gibbon, 1994; Balci et al., 2009; Swearingen, & Buhusi, 2010). Responding on individual probe trials on the peak interval procedure has been found to conform to a three-state pattern of responding (also described in the literature as a break-run-break pattern) in which animals respond at a low rate ($r_1$) early in the trial, transition to a higher rate($r_2$) of responding prior to the FI value, and subsequently switch to a low rate ($r_3$) of responding when the FI duration has passed (Balci et al., 2009; Cheng & Westwood, 1993; Gibbon & Church, 1990). Start and stop times were obtained only on trials that conformed to the low-high-low state model. A three-state regression analysis was used to best fit performance on each trial to a three state model of low-high-low. Start time was defined as the time bin in which the rate of responding transitioned from a low rate state, in which the subject emitted few to no responses) to a high rate state ($r_1 < r_2$) in which the subject responded at a rate that was greater than in the low rate state, whereas stop time was defined as the time bin in which the rate of responding transitioned from a high rate state to a low rate state ($r_2 > r_3$). A regression analysis
was used with an iterative procedure that searched all possible start and stop times to identify the best fit of the data to the three-state model on each trial. Minimum durations for the three states (r1=6 s, r2=4 s, r3=6 s) were established to reduce the effect of brief bursts of high-rate responding not due to timing. The individual trial analysis provides an assessment of trial-to-trial variations in timing performance on the peak interval procedure and can provide information about variability in the different measures. For example, peak time and spread were analyzed to assess the variability and thus precision in timing of the PI function. Spread was obtained by subtracting stop time from start time on individual trials, whereas middle time (time of maximal responding on each trial) was calculated by dividing the sum of start time and stop time by 2 on individual trials. Group mean median times for start, stop, middle and spread for each session were taken as a measure of central tendency for each measure, as the median is less affected by outliers than is the mean. The median of each of the aforementioned measures was obtained for each subject on each session. Group means of the medians were then calculated. All statistical tests employed a significance criterion of $p < 0.05$. 
Results

The analysis of the data was confined to the last five sessions of peak interval (PI) training and the five post-retention interval test sessions for each group. Furthermore, only probe trials were used for the analysis (15 probe trials per session).

Response Rate

Responses on each trial were recorded in 1 s bins and averaged across each session. The last 5 days of PI training were averaged to represent the training data. Hereafter, training refers to the last five training sessions.

The top panel of Figure 2 depicts 90 s group mean response rates as a function of elapsed time in the trial for all three groups, averaged over the last five training sessions (sessions 12-16). To assess stability of performance, a 3 (group) x 5 (session) x 90 (bin) analysis of variance (ANOVA) of response rate was conducted. As projected, no significant differences were revealed between groups, \( F(2,21) = 1.70, p = .206 \), or between sessions, \( F(4,84) = 1.32, p = .268 \), and no significant interactions were found for group x session, \( F(8,84) = 0.82, p => .583 \), group x bin, \( F(178,1869) = 1.19, p = .054 \), session x bin, \( F(356,7476) = 1.03, p = .330 \), or group x session x bin, \( F(712,7476) = 0.79, p =1.000 \). As expected however, a significant effect for bin was found, \( F(89,1869) = 25.95, p <0.0001 \), as subjects’ response rates on trials changed as a function of elapsed time in the trial, initially increasing as the duration approached the FI training value and thereafter decreasing as the time of reinforcement elapsed. Owing to some resurgent responding occurring toward the end of the 90 s response rate functions during both training and test sessions, the response rate functions used in subsequent analyses were restricted to only the first 60 bins (refer to Appendix for all 90 s functions). Figure 2 depicts what appears to be a difference in response levels between both control groups (ES and LS) and the
experimental group (EL). Examination of response rate functions for each subject revealed that 2-3 subjects in each of the two control groups exhibited high maximum rates (2.5-3.5 responses per second) on some sessions, whereas all the animals in the experimental group exhibited maximum rates of 1.0-1.5 responses per second on most sessions, as reflected in the greater apparent variability of rates in the two control groups. Subjects were randomly assigned to condition and operant chamber, thus there was no procedural systematic basis for differences among groups. As noted above, these differences in level among groups were not found to be significant. The bottom panel of Figure 2 depicts truncated 60 s group response rates averaged over the last five days of training on the peak interval procedure (sessions 12-16) as a function of elapsed time on probe trials. A 3 (group) x 5 (session) x 60 (bin) analysis of variance of response rate was conducted to test for differences between the last five training sessions (12-16) on PI. Results were consistent with those on the 90-s functions. No significant differences were found between groups, $F(2,21) = 1.55, p > .05$, or between sessions, $F(4, 84) = 0.98, p > .05$, and no significant interaction was found for group x session $F(8, 84) = 0.78, p > .05$, group x bin, $F(118, 1239) = 1.19, p > .05$, session x bin $F(236, 4956) = 1.01, p > .05$, or group x session x bin, $F(472, 4956) = 0.82, p > .05$. As expected however, a significant effect for bin was found, $F(59,1239) = 18.67, p < 0.0001$. Henceforth, training refers to the averaged functions of the last 5 training sessions (12-16) on the PI procedure.

Figure 3 depicts response rate functions for training (last five sessions [12-16] averaged) compared with each of the five test days for all groups. Groups are presented in columns and sessions are presented in rows. The experimental group, trained early with a long retention interval, (Early-Long, EL) is depicted in the column on the left, the early-trained control group (Early-Short, ES) in the middle column, and the late trained control group (Late-Short, LS) is
shown in the column on the right. The upper left panel shows training with test day 1 for the experimental group (EL). On the test day, it appears that there was an increase in response rate as a function of elapsed time similar to that in training, but response rate declined earlier in the trial compared to training, yielding a distribution of responding that peaked earlier in testing than in training. In test session 2 (left column, second row) the peak of the function appears to have migrated to the right, similar in location to that in training. By test sessions 4 and 5 there are no evident differences in response functions between training and test sessions. In contrast to group Early-Long, there was no apparent shift in the location of response rate functions during testing compared to training for either of the control groups (ES and LS). The middle column portrays response rate functions for training and each test session for group Early-Short (ES). Although response rate appears to be slightly higher on test day 1 following a short retention interval when compared to training the location of the peaks of the functions do not appear to differ. The slightly higher response rate in testing when compared with training appears to continue across test sessions however there does not appear to be a shift in the peak of the test functions (test days 2-5). The right hand column shows training and test functions for group Late-Short (LS). This group demonstrated consistent performance between training and post retention interval test day 1 (see top right panel of Figure 3). The two functions appear to nearly superimpose. On test day 2 there appears to be more variability in the function, however, performance seems to have recovered by test session 3 and 4. The bottom right panel illustrates that although response rate seems to be higher on test day 5 than in training for group LS, there does not appear to be any migration in the peak of the function.

In order to evaluate performance differences, a 3 (group) x 6 (session Train, Test 1-5) x 60 (bin) ANOVA of response rate was conducted. The ANOVA did not yield a significant effect
for group $F(2, 21) = 2.91, p > 0.05$, nor for session $F(5, 105) = 2.19, p > 0.05$, however a significant effect for bin was seen, $F(59,1239) = 22.69, p < 0.0001$, indicating that response rates differed as a function of time in trial. A group x bin interaction, $F(118, 1239) = 1.49, p < 0.001$, signified that temporal patterns of response rate differed between groups, and a session x bin interaction, $F(295, 6195) = 1.56, p < 0.0001$, indicated that temporal patterns of response rate varied as a function of session. More importantly, a significant group x session x bin interaction was obtained, $F(590, 6195) = 1.17, p < 0.01$, indicating that differences in response rate patterns between groups varied as a function of session. Follow-up tests were conducted to examine that significant interaction further.

The first row of Figures 3 shows training functions for each group respectively compared with test day 1. In line with the principal experimental question investigating the effect of retention interval on performance on the peak interval procedure, a 3 (group) x 2 (session) x 60 (bin) ANOVA of response rate was conducted in which training was compared with test session one. The analysis yielded no significant main effect for group, $F(2, 21) = 2.81, p > 0.05$, or for session, $F(1, 21) = 0.90, p > 0.05$; a bin effect was obtained $F(59, 1239) = 17.21, p < 0.0001$ as expected. A significant group x session interaction was found, $F(10, 21) = 3.89, p < 0.05$ signifying that overall rate differences among groups varied as a function of session. Additionally, a significant group x bin interaction, $F(118, 1239) = 1.35, p < .01$, suggests that response rate functions differed for the 3 groups in sessions training and test day 1. No session x bin interaction was found $F(59, 118) = 0.70, p > 0.05$. More importantly, the group x session x bin interaction was significant, $F(118, 1239) = 1.78, p < .0001$, in line with a difference in response patterns between training and test session 1 for group EL, but not for the control groups.
ES and LS as evidenced in the panels depicting train and test session one functions for each group (Figure 3).

In order to parse the foregoing three-way interaction, follow up two-way tests were conducted for each group. A 2 (session) x 60 (bin) ANOVA examining training and test session 1 yielded a significant effect for bin for groups EL, $F(59, 413) = 5.12, p < .0001$, ES, $F(59, 413) = 8.42, p < .0001$, and LS, $F(59, 413) = 5.80, p < .0001$. A significant effect for session was revealed for group ES, $F(1, 7) = 6.97, p < 0.05$, but not for groups EL, $F(1, 7) = 1.68, p > 0.05$, or LS, $F(1, 7) = 0.29, p > 0.05$. The critical effect for parsing the three-way interaction for the comparison of training and test session 1 is the session x bin interaction for each group. That interaction was significant for the experimental group EL, $F(59, 413) = 2.10, p < .0001$, and the early-trained control group (ES), $F(59, 413) = 1.76, p > .001$, but not for the late-trained control group (LS), $F(59, 413) = 0.64, p > .05$, indicating that temporal patterns of response rate functions varied between sessions for groups EL and ES but not for group LS. The between session change in pattern of responding for the experimental group (EL) is consistent with a decrease in peak time between sessions (Figure 3, left column first row panel). While control group ES also exhibited the interaction (middle column, first row), it does not appear to be associated with a shift in peak time. This finding will be addressed in the analysis of normalized response rate below.

In order to determine whether there were any differences in temporal performance between training and test sessions 2 through 5, a 3 (group) x 5 (sessions) x 60 (bins) ANOVA was conducted on these sessions. The test revealed a significant main effect of bin, $F(59, 1239)=23.45, p < .0001$, and no effects of group, $F(2,21)=2.68, p > 0.05$, or session, $F(4, 84)=2.38, p >0.05$. In addition, although no session x group interaction was revealed, $F(4,
indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was obtained, $F(118, 1239)=1.44, p < .01$, indicating that there were group differences in response rate patterns, and a session x bin interaction was 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Moreover a group x session x bin interaction was found, $F(472, 4956)=1.12, p < .05$, indicating that response function patterns varied as function of session and group. In order to parse the latter interaction, a 5 (session) x 60 (bin) ANOVA was conducted to test sessions training and tests 2 through 5 for each group. The statistical test yielded the anticipated bin effect for all three groups, however no other effects for groups EL or ES. On the other hand, a session x bin interaction was obtained for group LS, suggesting that for the late-trained group, response rate pattern varied across sessions. As seen in Figure 3 (right column, last row), the response function for test session 5 appears to be higher than the training function in the region of the FI value for group LS. Thus the session x bin interaction could be accounted for by a selective difference in response rate in the region of the FI value rather than by a difference in temporal pattern of performance on test session 5. This question will be addressed in the next section with the statistical analysis of normalized response rate functions.

**Normalized Response Rate**

The session x bin interactions revealed for individual groups by previous analyses could represent differences in levels of responding as opposed to differences in the temporal location of the two functions between training and test sessions. To address that issue, the mean response rate functions from the last five training sessions and each of the five test sessions for each subject were normalized using the mean of each 60-s function. In order to eliminate differences in response rate levels between sessions each point on the response rate function was divided by the mean of the function. Normalized training functions were obtained by first averaging...
response rate functions from the last five training days for each subject, then normalizing the average function to obtain a single function for training from each rat. As a result of normalizing, each subject contributed equally to the shape of the group function. The training and test functions were then subjected to additional analyses.

Figure 4 shows the group mean normalized response rate functions for each group from the training phase. A 3 (group) x 60 (bin) ANOVA of normalized response rate yielded a significant bin effect, $F(59, 1239) = 25.67, p < .0001$, however, no group x bin interaction was obtained, indicating no evidence of differences in temporal patterns of responding among groups prior to treatment implementation.

Figure 5 portrays normalized 60 s functions for training (average of last five sessions on PI) and each test session for all three groups. The left hand column displays the experimental group, EL, the middle column represents the control group, ES, and the column on the right depicts the control group, LS. Successive rows represent each of the five test sessions. The normalized functions for the control groups ES and LS do not exhibit differences in response rate patterns between training and test sessions 1-5. On the other hand, the experimental group (EL) exhibited a difference in response rate pattern between training and test session 1. The first test-session function for group EL is shifted toward the left and the function decreases earlier in the trial than in training. Test sessions 2-5 do not appear to show differences in temporal patterns of responding. In line with the previous analyses of the response rate data, a 3 (group) x 6 (session) x 60 (bin) ANOVA of normalized response rate was conducted. It yielded the customary bin effect, $F(59, 1239) = 40.46, p < .0001$, a session x bin interaction, $F(295, 6195) = 1.41, p < .0001$, indicating that response patterns varied as a function of session, and a group x session x bin interaction, $F(590, 6195) = 1.20, p < .01$ indicating that response rate pattern varied as a
function of group and session. In order to examine the latter interaction in more detail, follow up tests were conducted on training and testing data.

The first row of Figure 5 depicts normalized response rate functions for all groups on training and test session 1. It appears that group EL responded earlier in the trial and responding declined earlier in the trial on the first test session when compared to training. The two control groups ES and LS do not appear to differ in their pattern of responding between training and test session 1. A 3 (group) x 2 (session) x 60 (bin) ANOVA was conducted on normalized response rate for training and test session 1. A significant effect for bin was found, $F(59, 1239) = 22.54, p < .0001$. Neither the group x bin interaction, $F(118, 1239) = 0.99, p > .05$, nor the session x bin interaction was significant, $F(59, 1239) = 1.30, p < .05$, however, a significant group x session x bin interaction was obtained, $F(118, 1239) = 2.05, p < .0001$ indicating that the difference in temporal performance between training and test session 1 varied as a function of group. In order to parse that interaction, follow up two-way 2 (session) x 60 (bin) ANOVAs were conducted for each group. A significant bin effect was obtained for group EL, $F(59, 413) = 4.08, p < .0001$, and a session x bin interaction, $F(59, 413) = 2.63, p < .0001$, supporting the response pattern change as a function of session for this group. This is consistent with the evident change in performance indicated on the top left panel of Figure 5 suggesting a leftward shift in the peak of the function. The expected bin effect was obtained for groups ES, $F(59, 413) = 12.47, p < .0001$, and LS, $F(59, 413) = 8.99, p < .0001$, but no session x bin interaction was obtained for either control group, $F(59, 413) = 0.87, p > 0.05$, and $F(59, 413) = 0.89, p > 0.05$, respectively. Follow up one-way ANOVAs were conducted to test response rate differences bin by bin for group EL. The results of the tests yielded a significant difference between sessions for bins 3, $F(1, 14) = 7.27, p < .0.05$, 11, $F(1, 14) = 7.45, p < .0.05$, and 16, $F(1, 14) = 6.23, p < .0.05$, with reversals on bins 34,
This pattern of response rate differences is consistent with a temporal pattern difference between training and test functions, with a shift from greater relative response rate early in the trial on the test session compared to training, and a reversal of the response rate difference later in the trial. Rows 2 through 5 on Figure 5 represent data on training compared with subsequent test days 2-5. A 3 (group) x 5 (session) x 60 (bin) ANOVA was conducted on training and test sessions 2-5. The expected bin effect was obtained, $F(59, 1239) = 41.47, p < .0001$, as well as a significant session x bin interaction, $F(236, 4956) = 1.28, p < .01$, indicating that temporal patterns of responding varied as a function of session. No significant group x bin interaction, $F(118, 1239) = 1.13, p > .05$, or group x session x bin interaction was obtained, $F(472, 4956) = 1.00, p > .05$.

Performance between groups on training and test session 1 was also compared by between-groups analyses for individual sessions. Figure 6 depicts normalized response rate functions plotted for the three groups, on training (average of last 5 sessions on PI), and on each of the test days, permitting a comparison among groups on different sessions. The top left panel of Figure 6 portrays normalized training functions for the three groups, reproducing data from Figure 5. As indicated above (p. 25), there was no evidence of a difference in temporal pattern of performance among groups during training. The top right panel of Figure 6 compares normalized response rates for all three groups on the first test session following the retention interval. It appears that group EL responded earlier in the trial and responding declined earlier in the trial than either of the two control groups ES and LS on this session. The two control groups did not appear to differ from each other in their pattern of responding on test session 1. A 3 (group) x 60 (bin) ANOVA on normalized rate for test session 1 yielded a significant effect for
bin $F(59, 1239) = 10.74$, $p < .0001$, and a significant group x bin interaction $F(59, 1239) = 1.48$, $p < .001$, indicating that performance varied among groups as a function of time in trial. In order to examine whether the aforementioned interaction was specific to the experimental group (EL) a 2 (group) x 60 (bin) ANOVA was conducted on normalized response rates for the two control groups (ES and LS) on test session 1. The test yielded only an effect for bin, $F(59, 826) = 12.14$, $p < .0001$, but no group x bin interaction, $F(59, 826) = .854$, $p = .7759$, indicating that temporal patterns of responding did not differ between the two control groups. The 2 (group) x 60 (bin) ANOVA was also conducted for group EL and ES, yielding a significant bin effect, $F(59, 826) = 6.70$, $p < .0001$, and a significant group x bin interaction, $F(59, 826) = 2.16$, $p < .0001$. The same 2 (group) x 60 (bin) ANOVA between groups EL and LS found the anticipated bin effect, $F(59, 826) = .511$, $p < .0001$, and a marginally non-significant group x bin interaction was obtained, $F(59, 826) = 1.33$, $p = .0553$. The change in performance was most evident between the experimental group (EL) and the early trained control group (ES), with a similar trend, though not significant between EL and the late trained control group (LS). Rows 2-3 of Figure 6 depict performance on test sessions 2-5. A 3 (group) x 4 (test sessions 2-5) x 60 (bin) ANOVA yielded a significant effect for bin, $F(59, 1239) = 36.84$, $p < .0001$, response patterns varied as a function of time in the trial, and a significant session x bin interaction, $F(118, 3717) = 1.32$, $p = .0037$. No group x bin interaction, $F(118, 3717) = 1.08$, $p = .2792$, or group x session x bin interaction, $F(354, 3717) = 1.03$, $p = .3583$ was obtained however.

**Peak Time**

Figure 7 portrays mean peak time for all groups during training and each of the five test sessions. In comparison with mean peak time during training, there was a decrease for the experimental group (EL) only on the first test session following the retention interval, whereas
there was no evident decrease for the control groups ES and LS. Mean peak times for test session 1 were 18.00 s (2.71 s), 30.86 s (2.16 s), and 28.50 s (2.14 s) for groups EL, ES, and LS, respectively. In line with the analysis conducted for response rate, mean peak time for the last five training sessions (Sessions 12–16) was tested for differences. A 3 (group) x 5 (training session) ANOVA yielded no significant differences among groups, $F(2, 21) = 1.73, p = .202$, or sessions, $F(4, 84) = 0.09, p = .986$, nor a group x session interaction $F(8, 84) = 0.62, p = .760$. Therefore, peak times were averaged across sessions for each subject for this phase, hereafter referred to as “training” for all subsequent analyses of peak time. Group mean peak times (SEM) for training were 33.56 s (1.73 s), 32.86 s (1.29 s), and 28.8 s (1.53 s) for groups EL, ES, and LS, respectively. A two-tailed one sample $t$ test found that mean peak time pooled across groups time did not differ significantly from 30 s $t(23) = 1.48, p > .05$.

In order to test the effects of retention interval, analyses similar to those for response rate were conducted on peak time, comparing the three groups across the training and five test sessions. A 3 (group) x 6 (session) ANOVA revealed no effect for group, $F(2, 21) = 1.09, p = .354$, however a session effect was obtained, $F(5, 10) = 3.25, p = .0091$, as well as a significant group x session interaction, $F(10,105) = 2.79, p = .0042$. A one-way ANOVA was conducted for each group to examine differences in mean peak time among sessions. A simple session of effect was found only for the group EL, $F(5, 35) = 4.16, p = .005$. Neither of the control groups demonstrated differences in mean peak times across sessions, $F(5, 35) = 0.26, p = .935$ for group ES and, $F(5, 35) = 1.58, p = .190$ for group LS. In order to parse the session effect for the experimental group (EL), pairwise comparisons were conducted using the LSD test. Training and test session 1 were found to differ significantly, $p = .015$, in line with a decrease in peak time from training to test session 1. Mean peak time for group EL diminished considerably following
the long retention interval when compared to training. Test session 1 was also found to differ significantly from test session 3, \( p = .003 \), test session 4, \( p = .030 \), and test session 5, \( p = .010 \), reflecting an increase in peak time with continued testing on the PI procedure. No other pairs of sessions were found to differ significantly, minimum \( p = .059 \).

One-way ANOVAs were next conducted to test for differences among groups at each session. The ANOVA for training yielded no simple effect of group, \( F(2, 21) = 1.71, p = .205 \). Test session 1 however yielded a significant group effect, \( F(2, 21) = 7.43, p = .0036 \), whereas there was no group effect for test sessions 2, \( F(2, 21) = 0.22, p = .804 \), session 3, \( F(2, 21) = .403, p = .674 \), or session 4, \( F(2, 21) = .398, p = .677 \). A significant group effect was found on test session 5 however, \( F(2, 21) = 5.30, p = .0137 \). This difference between groups is suggested on Figure 7 in which it appears that peak time for the control group LS decreased on test sessions 2-5 with the lowest level appearing to be during test session 5. Pairwise comparisons using the LSD test were conducted on test sessions 1 and 5. On test session 1, peak time for EL was significantly less than peak time for ES, \( p = .002 \), or LS, \( p = .008 \). Groups ES and LS did not differ significantly, \( p = .511 \), Peak time did not differ on test session 5 for groups EL and ES, \( p = .345 \), however peak time for group LS was significantly less than peak time for either group EL, \( p = .005 \), and ES, \( p = .038 \).

**Peak Rate**

Figure 8 depicts mean peak rate for all groups, across all sessions (train and test days 1-5). Mean peak rates for each group on training and test sessions are provided in Table 3. As in previous analyses, mean peak rate across the last five training sessions (Sessions 12-16) was tested for differences. A 3 (group) x 5 (training session) ANOVA yielded no significant differences between groups, \( F (2, 21) = 1.45, p > 0.05 \), or sessions, \( F (4, 84) = 1.05, p > 0.05 \), nor
a group x session interaction $F(8, 84) = 0.96, p > 0.05$. Therefore, peak rates were averaged across sessions for each subject for this phase, and are henceforth referred to as “training” for all subsequent analyses of peak rate. Mean peak rate for the experimental group (EL) appears to be lower than those of the control groups ES and LS overall, but the group effect was not reliable. A 3 (group) x 6 (session [training and 5 test sessions]) ANOVA was conducted on peak rate. Although no group effect was revealed, $F(2, 21) = 2.57, p = .101$, a session effect was obtained, $F(5, 105) = 3.74, p = .004$, indicating that peak rate differed across sessions, and the interaction between group and session was marginally non-significant, $F(10, 105) = 1.88, p = 0.057$.

Peak rate decreased from training and test session 1 to test session 2, then increased steadily until test session 5. Pairwise comparisons using the LSD test ($\alpha = .05$) revealed significant differences between training (M=1.23) and test session 5 (M=1.43), test sessions 1 (M=1.29) and 2 (M=1.13), test sessions 2 and 4 (M=1.30), 2 and 5, ($p = .001$), and 3 (M=1.19) and 5.

**Individual Trials**

Individual trials analyses were performed to examine start and stop times, middle times, and spread time on training and test sessions (see Method section). In addition, the response rate in the high-rate state, designated here as R2, was analyzed.

Figure 9 depicts group mean middle time as a function of group and session. A 3 (group) x 6 (session) ANOVA of middle time yielded no group or session effects, $F(2, 21) = 1.73, p = 0.2025$, and $F(5,105) = 2.22, p =0.0575$ respectively. Nor was a group x session interaction found, $F(10,105) = 1.28, p = 0.2513$.

Figure 10 portrays group mean start times for the three groups across training and test sessions 1-5. Start times were found not to vary significantly among groups, $F(2, 21) = 2.41, p$
or across sessions, $F(5, 105) = 1.10$, $p = 0.3671$, nor was there a group x session interaction, $F(10,105) = 1.56$, $p = 0.1291$. No changes in start time from training values were observed among groups across sessions.

Figure 11 portrays group mean stop times for all groups as a function of session. It appears that stop times decreased for the experimental group (EL) from training to test session 1, but not for the two control groups ES and LS. In addition, it appears that stop time for group EL increased following test session 1, suggesting a recovery of performance for this group. The group (3) x session (6) analysis of stop times yielded no main effect of group, $F(2, 21) = 0.32$, $p = 0.7322$. However, a significant session effect was obtained $F(5, 105) = 2.56$, $p = 0.0316$ and a group x session interaction was found, $F(10, 105) = 2.05$, $p = 0.0349$, indicating that the change in stop times across sessions differed among groups.

A one-way ANOVA was conducted for each group to analyze differences in stop time among sessions (training and 5 test sessions). A simple effect of session was obtained only for the experimental group EL, $F(5, 35) = 2.93$, $p = 0.0259$. Neither of the control groups exhibited changes in stop time across sessions, $F(5, 35) = 0.66$, $p = 0.6551$ for ES, and $F(5, 35) = 2.04$, $p = 0.0966$ for LS. In order to parse the session effect for group EL, pairwise comparisons were conducted using the LSD test. Mean stop time in training (50.86) was found to differ significantly from test session 1 (42.19) ($p < .05$). Mean stop time for group EL decreased significantly following the long retention interval. Test session 1 was also found to be significantly lower in mean stop time from test session 3 (54.56) ($p < .05$). Test session 3 had a significantly higher mean stop time than session 4 (47.56) ($p < .05$). No other pairs were found to differ significantly.
One-way ANOVAs were conducted to test for differences among groups at each session. A significant group effect was obtained on session 1, $F(2,21) = .462, p < .05$, but not on training or any of the other test sessions, max $F(2,21)=1.78, p > .05$. Pairwise comparisons for session 1 using the LSD revealed that group EL was significantly different from groups ES, ($p = .021$), and LS, ($p = .012$), however groups ES and LS did not vary significantly from each other, ($p = .795$).

Figure 12 depicts median spread time as a function of session for all groups. There was no group effect seen for spread $F(2, 21) = 2.78, p = 0.8460$, nor was a session effect obtained, $F(5, 105) = 0.86, p = 0.5113$. The data suggest a tendency toward lower spread time on initial test sessions for group EL compared to the other groups, but the group x session interaction failed to meet a conventional level of significance, $F(10, 105) = 1.89, p = 0.0540$.

Figure 13 represents $R^2$, the mean response rate during the high-rate state. No group effect was obtained, $F(2, 21) = 1.80, p = 0.1900$, and there was no group x session interaction, $F(1,105) = 1.09, p = 0.3760$. A significant session effect, $F (5, 105) = 3.01, p = 0.0141$, indicated that response rate varied across sessions. LSD pairwise comparisons revealed that mean $R^2$ values in training and test session 2 were significantly lower than on test session 5 ($p = 0.018$ and .004, respectively), and $R^2$ on test session 3 was significantly lower than on both test session 4, ($p = .043$) and test session 5, ($p = .001$).

The Pearson $r$ was calculated for each subject for start-stop and start-spread values for the last 5 training sessions (12-16), the average of the last 5 training sessions, and test session 1. Start and stop times were positively correlated for all above noted sessions conforming to the predictions of scalar timing theory (Gibbon & Church, 1990; Church, Meck, & Gibbon, 1994), indicating trial by trial variability in memory of the time of reinforcement. (Group mean $r$ for last
five training sessions = .53, significantly different from zero, \( t(23)=11.78, p < .0001 \). A 3 (group) x 2 (session) ANOVA was conducted on start and stop correlations for the last training session (session 16) and test session 1. No differences were found among groups, \( F(2,21), .190, p = .829 \), or between sessions, \( F(1,21), .110, p = .744 \), and no group x session interaction was obtained, \( F(2,21), .510, p = .608 \). A 3 (group) x 2 (session) ANOVA was also conducted on the average correlations of the last five training days (12-16) and test day 1. No significant group effects were revealed, \( F(2,21), .528, p = .0597 \), nor session effects, \( F(1,21), .004, p = .949 \), and no group x session interaction, \( F(2,21), .174, p = .842 \).

Pearson's \( r \) was also computed for start and spread times for each subject on the last five training sessions, the average of the last five training sessions, and the first test session. As anticipated, start-spread correlations were negatively correlated, in line with predictions of scalar timing theory noted by Gibbon and Church (1990), Church et al., (1994) and others, indicating variability in decision thresholds to start and stop responding. (Group mean \( r \) for last five training sessions = -.46, \( t(23) = -13.76, p < .0001 \)). Start-spread correlations were subjected to the same analyses as start-stop. A 3 (group) x 2 (session) ANOVA on the last training session (16) and the first test session found no differences among groups, \( F(2,21), .246, p = .784 \), session, \( F(1,21), .711, p = .409 \), and no group x session interaction, \( F(2,21), .510, p = .607 \). A 3 (group) x 2 (session) ANOVA on the average correlations of the last 5 training sessions with the first test session failed to reveal any group effects, \( F(2,21), .462, p = .636 \), session effects, \( F(1,21), .022, p = .885 \), or group x session interaction, \( F(2,21), .506, p = .610 \).

The interquartile range for middle time was computed for each subject to examine variability of time judgments as a function of retention interval. Figure 14 displays the group mean interquartile range for middle times for all groups as a function of session. The figure
indicates that the interquartile range on initial test sessions was higher for the experimental group (EL) as compared with training, but that no differences were observed for the two control groups ES and LS. A 3 (group) x 6 (session) ANOVA yielded a significant group effect for the interquartile range, \(F(2,10),7.44, p = 0.004\), no session effect, \(F(5,105),1.10, p = 0.362\), and a group x session interaction, \(F(5,105),2.88, p = 0.003\). For the experimental group (EL), the mean interquartile range of middle time increased from training (M=14.11, SD=5.61) to test session 1 (M=29.66, SD=19.75), and decreased across subsequent test sessions. Groups ES and LS did not exhibit a comparable change in the variability of middle time from training, ES (M=13.09, SD=3.52), LS (M=19.84, SD=9.67) to test session 1, ES (M=10.38, SD=3.17), LS (M=14.81, SD=7.76). A one-way ANOVA, parsed by group, was conducted to test differences in interquartile range of middle times between training and test session 1. The test found no significant differences between training and test session 1 for groups EL, \(F(1, 7) = 4.82, p = 0.064\), ES, \(F(1, 7) = 3.02, p = 0.125\), or LS, \(F(1, 7) = 1.39, p = 0.276\).

In order to parse the aforementioned group x session interaction, a between groups ANOVA was conducted by session. No differences were found between groups for mean middle interquartile range on training \(F(2,21) = 1.47, p > .124\), however a significant group effect was found on test session 1, \(F(2,21) = 5.32, p = 0.014\), test session 2, \(F(2,21) = 12.39, p = .0001\), and test session 3, \(F(2,21) = 8.08, p = .003\). No significant group effect was found on either test session 4, \(F(2,21) = 1.93, p = .170\), or test session 5, \(F(2,21) = 336, p = .718\). In conjunction with the functions depicted on Figure 14, these results indicate that differences in variability between groups were minimal in training, however variability increased significantly in testing for group EL exposed to the long retention interval. On the other hand, variability did not change significantly for either of the control groups exposed to short retention intervals. A decline in
variability for the experimental group (EL) was seen across sessions in line with the expectation that performance for this group would recover as a function of subsequent training on the PI procedure. A follow up LSD test performed on mean middle-time interquartile range found that group EL was significantly different from group ES ($p = .005$) and group LS ($p = .026$) on test session 1, however ES and LS did not differ significantly ($p = .482$). Group EL also differed significantly in test session 2 from groups ES ($p = .0001$) and LS ($p = .001$) but ES and LS did not differ from each other ($p = .512$). Likewise in test session 3 EL differed from ES ($p = .001$) and LS ($p = .007$), but ES and LS were not found to differ significantly, ($p = .416$).

The interquartile range was analyzed for start and stop times to examine variability in these measures. Figure 15 displays the interquartile range for start times for all groups as a function of session. It appears that the experimental group EL exhibited greater variability during the initial test sessions compared with training. In contrast, the control groups ES and LS do not appear to exhibit a similar change in variability from training to testing. A 3 (group) x 6 (session) ANOVA yielded a significant group effect for the interquartile range for start times, $F(2,10) = 6.46, p < 0.05$, no significant session effect, $F(5,105) = 1.60, p > 0.05$, but a group x session interaction, $F(5,105) = 2.95, p < 0.05$ was obtained. A follow up ANOVA was conducted to parse the group x session interaction, examining group differences in mean interquartile start times by each session, training and test sessions 1-5. No significant effect for group was found for training, $F(2,21) = 3.36, p = .054$, however a significant effect of group was obtained for test session 1, $F(2,21) = 6.493, p = .006$, test session 2, $F(2,21) = 5.80, p = .010$, and test session 3, $F(2,21) = 8.84, p = .002$, but not in sessions 4 $F(2,21) = 3.05, p = .069$, and 5, $F(2,21) = .813, p = .457$, indicating that groups differed in variability in start times on initial test sessions. Pairwise comparisons using the LSD test were conducted for sessions 1-3 to examine
where the differences in mean interquartile start times were. On test session 1 group EL differed significantly from group ES ($p = .002$), and from group LS, ($p = .043$), but ES and LS did not vary significantly from each other, ($p = .170$). Group EL also differed significantly from ES ($p = .003$), and LS ($p = .044$), on test session 2, and test session 3 ES ($p = .0001$), and LS ($p = .017$) respectively. ES and LS were not different from each other on either test session 2, ($p = .233$), or on test session 3, ($p = .132$). In sum, Group EL exhibited greater variability in mean start times than either of the control groups in early test sessions.

Figure 16 depicts the interquartile range for stop times for each group across sessions. There appears to be an increase in variability from training to initial test sessions for the experimental group (EL), however this increase is not evident in the control groups (ES and LS). The two-way ANOVA of interquartile range for stop times yielded a significant effects of group, $F(2, 10) = 4.07, p < .05$, and session, $F(5, 105) = 2.54, p < .05$, and a significant group x session interaction, $F(5, 105), 2.55, p < .05$, suggesting that the difference in variability of stop times among groups varied as a function of session. Consistent with the previous analyses conducted above, a between groups ANOVA was conducted on mean interquartile stop times for sessions training and tests 1-5. The results yielded a marginally non-significant group effect for training, $F(2,21) = 3.45, p = .051$, significant group effects for test session 1, $F(2,21) = 4.86$, $p = .018$, test session 2, $F(2,21) = 3.89, p = .036$, and test session 3, $F(2,21) = 3.62, p = .045$, but no group effect for test session 4, $F(2,21) = 2.618, p = .097$, or test session 5, $F(2,21) = 195, p = .824$. Post–hoc LSD tests were conducted to assess the locus of group differences on each session. Groups ES and LS significantly differed in training, ($p = .021$), however EL did not differ significantly from either ES, ($p = .613$), or LS, ($p = .062$) in training. In test session 1, EL differed significantly from ES, ($p = .005$), but not from LS, ($p = .075$); ES and LS did not differ
significantly from each other, \((p = .236)\). On test session 2 group EL differed from ES, \((p = .011)\), but not from LS, \((p = .136)\), nor did the control groups differ from each other, \((p = .230)\). A similar pattern was observed on test session 3 in which EL differed from ES, \((p = .016)\), but not from LS, \((p = .414)\), nor did the control groups differ from each other, \((p = .086)\). As illustrated in Figure 16, group EL demonstrated a trend toward greater variability in mean stop times following the long retention interval than either of the control groups, however, group EL differed significantly from ES but not from LS. Groups ES and LS however did not differ from each other.

In summary, the long retention interval produced greater variability in middle times for the experimental group EL during the earlier test sessions than did the short retention interval for the control groups ES and LS. Similar trends were observed for variability in start and stop times, with more robust effects found for start times.
Discussion

The effect of retention interval (2 days and 70 days) on timing performance was investigated using the peak interval procedure. In general, experimental effects of retention interval were restricted to the experimental group (EL) on test session 1 following the 70-day retention interval, with performance recovering on test sessions 2-5.

The temporal pattern of responding during training on the peak interval procedure was compared with performance on post retention interval test sessions. An analysis of response rate as a function of elapsed trial time for training compared with the post-retention interval test sessions was conducted. The mean response rate function for the experimental group EL peaked earlier in test session 1 than in training. Responding in this group declined earlier in the trial as can be seen in both the raw mean response rate functions (Figure 3) and in the rate-normalized functions (Figure 5). The control groups ES and LS exposed to a 2-day retention interval did not exhibit a similar change in the pattern of the peak interval function from training to testing. Later test sessions did not differ from training for the experimental or control groups. Although a significant session x bin interaction of response rate functions on training and test session 1 was obtained for control group Early-Short (ES) (Figure 3), the interaction disappeared when rates were normalized (Figure 5) implying changes in local rate of responding between sessions that was unrelated to timing. In addition, the experimental group (EL) exposed to the long retention interval exhibited an earlier peak time for the first test session compared with peak time during training, in contrast to either of the control groups exposed to a short retention interval, which did not exhibit changes in peak time from training to test session 1. On subsequent test sessions, the experimental group (EL) exhibited peak times similar to group mean peak times during training. Peak rate was not found to vary among sessions for any of the groups. The earlier peak
time seen in the group exposed to the long retention interval in addition to the unchanged group peak rate was consistent with the findings of El Massiou et al., (2006).

A molecular analysis of performance on individual trials revealed no changes in middle or start times between training and test sessions or between groups. In contrast, stop time decreased from training to test session 1 for the experimental group (EL) but not for either control group ES or LS. Stop time for the experimental group was earlier than that for either of the control groups on test session 1, but not during training, or on test sessions 2-5. The data suggested a decrease in spread of the function for the experimental group EL (Figure 12) but not for either of the control groups ES and LS, however the effect was not significant. Further, R2, the rate of responding in the high state of the 3-state model, was lower in training, and test sessions 1-3 for the experimental group, but increased in test sessions 4 and 5. The control groups did not demonstrate similar changes across sessions in R2. Correlations between start and stop times for the last 5 training sessions and the first test session following the retention interval were overall positive, in line with the predictions of scalar timing theory, indicating trial by trial variance accounted for by variability in the remembered time of reinforcement. Similarly, correlations between start and spreads times were in general negative, suggesting variability in the decision thresholds from trial to trial, according to the predictions of scalar expectancy theory (Gibbon & Church, 1990; Church, Meck, & Gibbon, 1994).

Interquartile range corresponding to middle, start and stop times was examined. Mean interquartile range for middle times in group EL increased marginally, although not significant, from training to the first test session and thereafter decreased gradually as a function of test sessions, whereas interquartile range of middle times did not vary for either of the control groups from training to testing. A similar pattern of results were observed for interquartile range of start
times for the experimental group EL, and the control groups. The analysis of interquartile range of stop times indicated increased variability in stop times for the experimental group on initial test sessions. This pattern of greater variability in mean stop times exhibited by the experimental group persisted until test session 4. The variability in stop times for group EL was found to be significantly greater than for group ES, however not significantly different from group LS. The control groups ES and LS did not differ from each other.

The molar analysis provided data that are consistent with a change in reference memory during the long retention interval compared to the short interval. One interpretation of the observed decrease in peak time after a long retention interval is that temporal information regarding the FI value in reference memory has changed during the retention interval. The basis for that change would be different from that proposed for the effect of a within-trial retention interval (gap) as engendering a decrease in the representation of subjective time in working memory (Buhusi & Meck, 2006, 2005, 2002; Cabeza de Vaca, Brown, & Hemmes, 1994). The memory decay interpretation in working memory was supported by the findings of Cabeza de Vaca, Brown, & Hemmes (1994) who studied the effect of within trial short retention intervals using the PI procedure with gaps. In their study, they found shifts in peak time on gap trials consistent with a shortening of the accumulated subjective time in working memory as time elapsed during the gap period. The notion of decay in timing of duration however has different implications for working memory as opposed to reference memory. For working memory, decay of duration is regarded as a loss of accumulated pulses which results in a subjective shortening of the interval. In contrast, in reference memory, decay is more plausibly envisioned as increased uncertainty about remembered time of reinforcement, which would be evidenced by increased variability (decreased precision) in responding during the interval. Although the molar analysis
of the present data revealed peak time shifts from the pre-retention interval training session to post retention interval test session 1 that appear to be consistent with a decrease of the remembered times to reinforcement over the duration of the 70-day retention interval, a molecular analysis at the level of individual trials calls that interpretation into question and provides support for a precision account.

Scalar timing theory (Gibbon & Church, 1990; Gibbon, Church, & Meck, 1984), predicts that changes in reference memory would produce correlated changes in start and stop times. That is, if the observed shift in peak time was due to a shift in the remembered time of reinforcement, in which values in the reference memory distribution associated with the expected time of reinforcement would have been expected to decrease by some amount, proportional changes in start and stop times would have been expected as well, reflecting the scalar property in accordance with the decision mechanism of the internal clock model (Gibbon & Church, 1990; Church, Meck & Gibbon, 1994; Gibbon, Church, & Meck, 1984). That is, an earlier peak time would be accompanied by earlier start and stop times.

The data obtained in the current study indicate that for the experimental group (EL) a decrease from training to testing occurred in stop times but no related change was found to occur in start times. In contrast, the control groups ES and LS did not demonstrate changes in either start or stop times from training to testing. Moreover, no change was found in middle times from training to testing for any of the groups. Therefore the current results are inconsistent with the possibility that a differential change among groups occurred in the content of reference memory during the long retention interval.

An alternative account for the change in the temporal performance seen in the experimental group but not the control groups is the decision mechanism of the internal clock.
The decision rule to start responding on a given trial is based on three elements, involving a ratio comparison between the 1) current, or perceived elapsed time in working memory and 2) a value sampled from reference memory, and 3) a decision threshold. Moreover, the same three elements govern the organism’s decision to stop responding on a probe trial when the elapsed time in the trial has exceeded the remembered value in reference memory. Figure 17 depicts the operation of the decision mechanism in the information processing model (Church, Meck & Gibbon, 1994; Gibbon & Church, 1990). Elapsed time in the trial is represented on the abscissa, \( S^* \) represents a value of remembered time of reinforcement sampled from reference memory and \( \beta \) represents the start and stop response thresholds. Two independent thresholds are depicted for start and stop respectively. The Gaussian shaped distributions represent the variability in both the sample from reference memory and in thresholds on individual trials. As time in the trial elapses, the difference in subjective elapsed time and the value of \( S^* \), sampled from reference memory decreases, depicted by the downward slope of the "V". When the duration in working memory is perceived to be similar enough to the remembered time of reinforcement, the subject initiates an increase in response rate (start time). On non-reinforced trials, when the subjective elapsed time is judged to exceed \( S^* \), response rate on the trial decreases (stop time). This pattern of responding results in the 3 state model of low-high-low observed on individual trials.

Although initially, a single threshold governing the start and stop of responding on a given trial was proposed by the developers of the internal clock model (Church, 1994; Gibbon et al., 1984, Gibbon & Church, 1990) subsequent research supported the notion of two independent thresholds governing start and stop times respectively (for example, see: Agostino, Cheng, Williams, West & Meck, 2013; Balci et al., 2009; Church et al., 1994; MacDonald, Cheng & Meck, 2012; Matell & Portugal, 2007; Matell, Bateson & Meck, 2006; Taylor et al., 2007).
A number of these researchers have conducted manipulations that produced an effect on either start time or stop time. For example, Taylor, Horvitz and Balsam (2007) demonstrated that amphetamine administration in rats on the PI procedure produced a decrease in peak time when compared to rats administered saline. This outcome appears to be in line with an increase in clock speed, which according to scalar timing theory, would be expected to produce both earlier start times as well as earlier stop times. However, an individual trials analysis of the data revealed that these measures did not change in the expected correlated manner, but instead, the molecular analysis yielded earlier start times but no significant change in stop times, a result that is inconsistent with a faster clock account and supports the notion of independent decision thresholds. Matell, Bateson and Meck (2006) reported leftward shifts in peak interval functions in rats exposed to varying doses of methamphetamine. A molecular analysis of the data on PI trials indicated that the leftward shifts in peak time appeared to be due to earlier stop times with no corresponding change in start times. In a series of experiments using administration of anisomycin, a protein synthesis inhibitor, MacDonald, Cheng, and Meck (2012) found that start and stop times in rats were selectively impaired during acquisition of responding on the PI procedure. Disrupting normal protein synthesis in the dorsal striatum resulted in earlier start times whereas disruption of protein synthesis in the ventral striatum resulted in later stop times. The implication was that threshold modifications were responsible for the observed effects.

These studies are part of a growing body of literature representing psychopharmacological manipulations that may selectively influence one threshold and not the other, providing support for the idea of independent decision thresholds. Fewer studies have investigated the notion of dissociated decision thresholds with non-psychopharmacological manipulations (Jacobs, 2015; Balci et al., 2009; Matell & Portugal, 2007). Matell and Portugal
Matell and Portugal (2007) exposed one group of rats to extended nosepoke training on the peak interval procedure, while a second group of animals was placed on a behaviorally dependent variable interval schedule (bdVIPI) incorporating a second nosepoke aperture. In the latter procedure, reinforcement became available for a nosepoke only during intervals in which an animal had its snout in the nosepoke opening. The authors found that stop time decreased for the animals trained on the extended PI procedure, whereas start times were not affected for this group. On the other hand, for the rats trained on the bdVIPI schedule both starts and stops changed. Start times increased while stop times decreased. Matell and Portugal thus demonstrated that there are circumstances in which both start and stops change with extensive training, and other situations in which only one threshold changes. These studies and others employing the peak interval procedure have found selective changes produced in either starts or stops that suggest two independent thresholds. As the reference memory module of the internal clock model cannot account for the observed changes in stop time with no corresponding change in start time as observed in the current study, it is reasonable to presume that the results of the present study represent an example of a threshold modification produced by a long retention interval. That is, the long retention interval may selectively decrease the stop threshold for the experimental group, but have no effect on the start threshold.

The objective of the current study was to investigate the effect of a between-sessions retention interval using a procedure similar to that used by El Massioui, Brown, & Doyère, (2006). The results of the current study are consistent with those found by El Massioui, et al. Similar to the present study, those authors obtained a leftward shift in peak time with no change in peak rate, following a 4 month retention interval. A molecular analysis of their data was not conducted, however. Therefore it is not known whether the replication of their findings extends
to the selective effect on stop time obtained in the present study or the increased variability in middle, start and stop times following the 4-month retention interval. Further, El Massiouï et al. did not control for age effects. The current study controlled for age at training and testing with the addition of two control groups, one trained at the same age as the experimental group and the other tested at the same age as the experimental group. Neither control group exhibited changes in temporal patterns of responding from training to testing, hence ruling out the effect of age on performance in the experimental group exposed to the 70-day retention interval.

The present findings are consistent with the results of Gleitman and Bernheim (1963). Those authors investigated performance on a FI 60s schedule of reinforcement in 6-month old rats after either a short (24-hr) or long (24-day) retention interval measuring the proportion of responses that were made during each second of the FI interval expressed as first-half ratios, that is, the number of responses during the first 30 s of the interval divided by the number of responses during the entire interval. The authors found a significant effect of retention interval. First-half ratios at testing were significantly higher for the 24-day group than for the 24-hr group suggesting that the long retention interval resulted in earlier responding in the interval compared to the short retention interval. The authors proposed that the animals in the long retention interval group forgot the temporal parameters of the FI schedule, resulting in an increase in responding in the first half of the interval.

A possible explanation of Gleitman and Bernheim’s results might be that the animals exposed to the 24-day retention interval exhibited a leftward shift in their response rate function as a function of the retention interval that could be interpreted as a possible decrease in the remembered time of reinforcement, implicating reference memory. Alternatively, the effect of the longer retention interval may have been to increase variability in temporal performance,
which would account for Gleitman and Bernheim’s finding. Increased variability in temporal performance may presumably result from an increase in variability of remembered time of reinforcement, or from an increase in variability of start and stop thresholds. In order to compare the present results with those of Gleitman and Bernheim, the present data were analyzed similarly to theirs. First-half ratios were calculated from probe trials on the last training session and first test session following the retention interval. Consistent with Gleitman and Bernheim, first half ratios among the three groups were similar in training, for EL (M=.38, SD=.11), ES, (M=.36, SD=.07) and LS (M=.35, SD=.07), however the experimental group exhibited a higher first half ratio on test session 1 (M=.45, SD=.11) than either of the control groups (M=.36, SD=.10) for ES and (M=.36, SD=.11) for LS. The difference in first half ratios between training and testing for group EL were statistically significant, but not those for groups ES and LS. Although no molecular analysis was provided by Gleitman and Bernheim, the similarity of the present findings with theirs suggests that similar mechanisms may underlie the results of both studies. The 24-day retention interval may have resulted in an increase in variability in start and stop times in that group, comparable to the increase in variability seen in the experimental group in the present study.

The molecular analysis in the present study revealed substantial increases in variability in start, stop, middle and spreads times for the experimental group. As reported above (p.35-36) the experimental group demonstrated increased variability in middle times on test sessions 1-3 when compared with training whereas the control groups did not exhibit similar increases in interquartile range of middle times. The increased variability in responding in the experimental group in the initial post retention interval test sessions when compared with training was also demonstrated in start times and stop times suggesting that the long retention interval affected
response precision. This outcome is consistent with findings examining the effects of delayed testing on the generalization gradient. For example, Thomas and Lopez (1962) employed three durations of retention interval between training and testing (immediate, 1-day, & 1-week) in a stimulus generalization procedure with pigeons. The study found that animals tested 1-day and 1-week after training, exhibited similar flattening of their generalization gradients, compared to the animals tested immediately after training, reflecting a loss of stimulus control in the subjects exposed to a retention interval between training and testing, consistent with the results of Gleitman and Bernheim, and with the results of the current study showing a loss of stimulus control after a long retention interval. Although the current study did not find increased variability in middle, start or stop times in the control groups exposed to a 2-day retention interval, this difference may be attributed to the different task and species used in the current study. Nevertheless, the effect of retention interval on temporal precision in the present study is comparable to the effect of retention interval in a non-temporal preparation.

Campbell and Haroutunian (1981) examined FI 60s performance in 6, 12 and 26-month old rats following a 16-day retention interval after training. The authors found that 26-month animals responded earlier in the trial during the post retention interval test when compared to the last training session, whereas the 6 and 12 month old rats exhibited no change in performance from training to test session. The authors suggested that the older animals may have forgotten the temporal parameters of the FI schedule and therefore respond earlier in the FI trial. Forgetting as invoked by the authors could mean either of two things, a subjective shortening of the interval to be timed, that is decrement in accuracy, and/or an increase in variability or decrement in precision of responding. The latter explanation is in line with the outcome of the present research and may conceivably account for Campbell and Haroutunian’s results. Campbell and
Haroutunian did not employ the peak interval procedure which would have enabled assessment of time estimates, in addition to permitting examination of start and stop times. Nevertheless, the normalized response rate functions analyzed in the current study are similar to Campbell and Haroutunian’s treatment of their data. In their study, they presented percentage of the terminal rate of responding, which was calculated for each animal by taking the mean number of lever press responses on each 10 s segment of the FI60 interval compared to lever presses on the last 10 s of the interval on the last day of training and the test session.

The older animals in Campbell and Haroutunian’s study appeared to respond earlier on tests trials compared to training, consistent with the change in normalized response rate seen in the experimental group of the present study (see Figure 5, first panel). This evident similarity in performance following a long retention interval between Campbell and Haroutunian’s study and the present study suggests a similar mechanism may be able to account for the change in temporal patterns of responding seen in both experiments. Campbell and Haroutunian’s results were restricted to the older, 26 month old rats, thus, their findings diverge from those of Gleitman and Bernheim (1963) and the current study, in which the effects were seen in younger animals. The shorter duration of the retention interval (16 days vs. 24 or 70 days) that Campbell and Haroutunian employed may account for the results found by these authors in the older animals, whereas younger animals (6 and 12 month old) were not affected. Older animals may have greater sensitivity to the effects of shorter retention intervals, whereas younger animals require longer retention intervals to disrupt temporal performance.

Conclusion

In summary, the rationale of the present study was to investigate the effect of retention interval on temporal patterns of performance in rats on the peak interval procedure. Although the
molar analysis of response rate functions was in line with a reference memory account of the observed shift in peak time found in the experimental group EL, the molecular analysis of start and stop times was not consistent with that account. The single trials analysis revealed that the observed shifts in the peak interval functions were related to a decrease in stop times only, and increases in variability in middle, start and stop times in the experimental group, thus, a molar analysis alone did not suffice to identify the mechanisms that produced the shift in the peak interval function. Taken together, the analysis of the present data suggest that the effects of retention interval are better understood in terms of decision mechanisms and decreased precision rather than reference memory.

The current study was not unique in producing a selective change in either start times or stop times as there is a growing body of literature investigating effects on these measures. The present results underscore the importance of conducting an individual trials analysis which may result in conclusions that are at variance with those reached by a molar analysis alone. Thus it contributes to the literature examining the effect of various manipulations on either start or stop times on timing performance on the peak interval procedure by providing evidence that long retention intervals selectively affect one response threshold but not the other, lending support to the notion of a dissociation between the response thresholds of the decision mechanism. Further, the long retention interval resulted in substantial increases in response variability on individual trials. An implication that can be drawn from the latter findings is that variance in the remembered time of reinforcement and response thresholds may alone or in combination contribute to retention interval effects. Further, the present study supports the notion that the effect of intratrial retention intervals is not equivalent to the effect of intersession retention intervals. The data suggest that intratrial retention intervals in timing preparations produce
changes in accuracy for working memory (shifts in peak time), whereas intersession retention intervals produce changes in precision (increased variability).
Table 1

*Time of events for each group.*

<table>
<thead>
<tr>
<th>Group</th>
<th>Training</th>
<th>Retention Interval</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early-Short</td>
<td>5 months</td>
<td>2 days</td>
<td>7 months</td>
</tr>
<tr>
<td>Early-Long</td>
<td>5 months</td>
<td>70 days</td>
<td>8 months</td>
</tr>
<tr>
<td>Late-Short</td>
<td>7 months</td>
<td>2 days</td>
<td>8 months</td>
</tr>
</tbody>
</table>

*Note.* Group Early-Short began training early at 5 months of age and was exposed to a short retention interval of 2 days. Group Early-Long was also trained early at 5 months and was exposed to a long retention interval of 70 days. Group Late-Short begins training late, at 7 months and was exposed to a 2-day retention interval. Testing occurred for each group the day after their assigned retention interval ended.
Table 2

*Mean peak time for each group on training and test sessions.*

<table>
<thead>
<tr>
<th>Group</th>
<th>Training (SEM)</th>
<th>Test 1 (SEM)</th>
<th>Test 2 (SEM)</th>
<th>Test 3 (SEM)</th>
<th>Test 4 (SEM)</th>
<th>Test 5 (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-Long</td>
<td>33.56 (2.68)</td>
<td>18.00 (2.90)</td>
<td>29.75 (3.90)</td>
<td>33.75 (3.24)</td>
<td>29.63 (2.91)</td>
<td>34.63 (2.66)</td>
</tr>
<tr>
<td>E-Short</td>
<td>32.86 (1.45)</td>
<td>30.88 (2.31)</td>
<td>31.50 (2.71)</td>
<td>32.63 (2.15)</td>
<td>31.63 (2.16)</td>
<td>31.75 (1.39)</td>
</tr>
<tr>
<td>L-Short</td>
<td>28.80 (1.53)</td>
<td>28.50 (2.28)</td>
<td>32.63 (2.42)</td>
<td>30.63 (1.88)</td>
<td>28.25 (2.93)</td>
<td>25.13 (2.11)</td>
</tr>
</tbody>
</table>

*Note.* Training consists of the average of the last 5 training sessions (12-16). Group E-Short was trained at the same time as group EL and exposed to a short retention interval of 2 days. Group E-Long was exposed to a long retention interval of 70 days. Group L-Short was exposed to a 2-day retention interval and was trained later than the experimental group but tested at the same time as Group EL. Testing occurred for each group the day after their assigned retention interval ended. SEM is standard error of the mean.
Table 3

*Mean peak rates for each group on training and test sessions.*

<table>
<thead>
<tr>
<th>Group</th>
<th>Training (SEM)</th>
<th>Test 1 (SEM)</th>
<th>Test2 (SEM)</th>
<th>Test 3 (SEM)</th>
<th>Test 4 (SEM)</th>
<th>Test 5 (SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-Long</td>
<td>.9032 (0.10)</td>
<td>0.6835 (0.13)</td>
<td>0.6418 (0.14)</td>
<td>0.7461 (0.08)</td>
<td>0.8733 (0.13)</td>
<td>1.0075 (0.12)</td>
</tr>
<tr>
<td>E-Short</td>
<td>1.3474 (0.23)</td>
<td>1.7180 (0.35)</td>
<td>1.4671 (0.30)</td>
<td>1.5123 (0.35)</td>
<td>1.5254 (0.32)</td>
<td>1.5057 (0.36)</td>
</tr>
<tr>
<td>L-Short</td>
<td>1.4365 (0.32)</td>
<td>1.4758 (0.32)</td>
<td>1.2719 (0.24)</td>
<td>1.4136 (0.28)</td>
<td>1.5026 (0.28)</td>
<td>1.7715 (0.40)</td>
</tr>
</tbody>
</table>

*Note.* Training consists of the average of the last 5 training sessions (12-16). Group E-Long (trained early with a long retention interval), Group E-Short (trained early and exposed to a short retention interval, and group L-Short (trained late and exposed to a short retention interval. SEM is standard error of the mean.
Figure 1. Diagram of the information processing clock model based on Church & Gibbon (1990).
Figure 2. Top panel shows 90 s group mean response rates averaged over the last five days of training on the peak interval procedure (sessions 12-16) as a function of elapsed time on probe trials. Bottom panel shows 60 s truncated group response rates averaged over the last five days of training on the peak interval procedure (sessions 12-16) as a function of elapsed time on probe trials. Error bars represent the standard error of the mean.
Mean responses per second

Group EL
Test Session 1

Group ES
Test Session 1

Group LS
Test Session 1

Test Session 2

Test Session 3

Test Session 4

Test Session 5

Test Session 2

Test Session 3

Test Session 4

Test Session 5

Elapsed time in trial (s)
Figure 3. Mean response rate as a function of elapsed time in trial (the first 60 bins) in training is compared with the mean response rate function for each of the 5 post-retention interval test sessions for each group. Group EL (early-trained, long retention interval) is in left hand column, group ES (early-trained, short retention interval) is depicted in middle column and group LS (late-trained, short retention interval) is shown on right hand column. Test sessions 1 through 5 are presented in rows 1-5 respectively.
Figure 4. Normalized response rate functions using the mean of the function for the average of the last five training days for groups EL (early trained, long retention interval), ES (early trained, short retention interval), and LS (trained late, short retention interval).
Figure 5. Normalized (using the mean of the function) 60 s response rate functions for training and all test sessions for groups EL (early trained, long retention interval) in the left column, ES (early trained, short retention interval) in the center column, and LS (trained late, short retention interval) in the right column.
Figure 6. Normalized response functions for all groups on training and test sessions 1-5.
Figure 7. Represents mean peak times obtained using the smoothing procedure for each group as a function of session (mean of the last five days of PI training and all five test sessions). Error bars represent the standard error of the mean.
Figure 8. Mean peak rates are displayed for each group as a function of session. The error bars represent the standard error of the mean.
Figure 9. Group mean median middle time (s) as a function of session for each group.
Figure 10. Group mean median start time (s) as a function of session for each group.
Figure 11. Group mean median stop time as a function of sessions for each group.
Figure 12. Group mean median spread time as a function of sessions for each group.
Figure 13. Group mean median R2 (rate of responding in the high state of the 3 state model) as a function of sessions for each group.
Figure 14. Interquartile range (upper and lower quartiles) for middle times for the three groups across training and test sessions. The error bars represent the standard error of the mean.
Figure 15. Interquartile range (upper and lower quartiles) for start times for the three groups across training and test sessions. The error bars represent the standard error of the mean.
Figure 16. Interquartile range (upper and lower quartiles) for stop times for the three groups across training and test sessions. The error bars represent the standard error of the mean.
Figure 17. The information processing model. On the abscissa, (t) represents elapsed time in trial and $S^*$ represents a value in reference memory. $\beta$ represents the decision threshold. Two independent thresholds are depicted for start and stop respectively. The Gaussian shaped distributions represent variability found in both the sample in reference memory and in thresholds. As time in the trial elapses, the relative difference between elapsed time and the value sampled from reference memory decreases. When the subject perceives time to be similar enough to the remembered time of reinforcement, responding is initiated and is represented in the figure by the arrow labeled start time. On non-reinforced trials, when the subject perceives relative elapsed time to be greater than $S^*$, responding is terminated, represented in the figure by the arrow labeled stop time. This pattern of responding results in the 3 state model of low-high-low observed on individual trials.
Appendix A

Mean responses per second

[Graphs showing mean responses per second for different test sessions and groups]
Appendix A. Mean response rate as a function of elapsed time in trial (90 s) in training is compared with the mean response rate function for each of the 5 post-retention interval test sessions for each group. Group EL (early-trained, long retention interval) is in left hand column, group ES (early-trained, short retention interval) is depicted in middle column and group LS (late-trained, short retention interval) is shown on right hand column. Test sessions 1 through 5 are presented in rows 1-5 respectively.
Bibliography


