Title
Biasing Temporal Judgments in Rats, Pigeons, and Humans

Journal
International Journal of Comparative Psychology, 28(1)

ISSN
0889-3675

Authors
Daniels, Carter W
Fox, Adam E
Kyonka, Elizabeth G. E.
et al.

Publication Date
2015

DOI
10.46867/ijcp.2015.28.02.07

INTERNATIONAL JOURNAL OF COMPARATIVE PSYCHOLOGY

2015, 28
Fuat Balci
Special Issue Editor
Peer-reviewed

Biasing Temporal Judgments in Rats, Pigeons, and Humans

Carter W. Daniels
Arizona State University, U.S.A.

Adam E. Fox
St. Lawerence University, U.S.A.

Elizabeth G. E. Kyonka
West Virginia University, U.S.A.

Federico Sanabria
Arizona State University, U.S.A.

Models of interval timing typically include a response threshold to account for temporal production. The present study sought to evaluate the dependent concurrent fixed-interval fixed-interval schedule of reinforcement as a tool for selectively isolating the response threshold in rats, pigeons, and humans. In this task, reinforcement is available either at one location after a short delay or at another location at a longer delay. Because the reinforced location is not signaled, subjects normally respond on the first location and, if reinforcement is not delivered, then switch to the second location. The latency to switch between locations served as the primary dependent measure. After training rats, pigeons, and humans with equal reinforcement magnitudes in the short and long delays, the magnitude of reinforcement was increased threefold on the long-delay location. Consistent with model predictions, this biasing procedure decreased estimates of the response threshold of rats and pigeons, but also reduced temporal control in these species and increased response-threshold estimates in humans. Human and pigeon performance also suggested a magnitude-induced increase in the speed of the internal clock. Collectively, these results suggest that differences in reinforcement magnitude between response alternatives appear to modulate the response threshold, but not selectively, and may provide guidance for better isolating response-threshold effects in humans.

Interval timing is the ability of a wide range of species, including humans, to coordinate their behavior with periodic events, in the time-scale of seconds to minutes, on the basis of an endogenous clock-like mechanism (Boisvert & Sherry, 2006; Brunner, Kacelnik, & Gibbon, 1992; Henderson, Hurley, Bateson, & Healy, 2006; Higa & Simm, 2004; Lejeune & Wearden, 1991; Richelle & Lejeune, 1984; Yin, Lusk, & Meck, 2015). This ability is often studied using temporal production tasks (e.g., the fixed-interval, or FI, schedule of reinforcement; Guilhardi & Church, 2005; Lejeune & Wearden, 1991). The operation of the endogenous timing mechanism in production tasks is illustrated in the bottom panel of Figure 1 (also see Sanabria & Killeen, 2008 for a similar schematic). The onset of a stimulus signaling the to-be-timed interval t resets an accumulator and initiates the emission of pulses from a pacemaker to the accumulator, with an average inter-pulse interval of c (together, the pacemaker and accumulator comprise the internal clock). The number of accumulated pulses (A) is continuously compared with a

Please send correspondence to Dr. Federico Sanabria, Arizona State University (Email: Federico.Sanabria@asu.edu)
https://doi.org/10.46867/ijcp.2015.28.02.07

pulse count drawn from memory, m; when A reaches a proportion θ of m, an observable target response (e.g., reporting that 10 s have elapsed) is emitted. When the response is reinforced, the pulse count updates m.

In an animal well trained in a task in which the response does not significantly affect the timing of reinforcement (e.g., in FI schedules), memory for the to-be-timed interval is expected to be stable. Specifically, m (or the central tendency of m) will approximate the average number of pulses corresponding to the to-be-timed interval, i.e., $m \approx t/c$. The response criterion may thus be expressed as $\theta m = \theta t/c$. Because a Poisson process is assumed to generate inter-pulse intervals (Gibbon, 1992; Gibbon, 1977; Killeen & Fetterman, 1988; Simen, Rivest, Ludvig, Balci, & Killeen, 2013), these are expected to be exponentially distributed. Thus, target responses are expected to happen after a fixed number ($\theta t/c$) of exponentially-distributed intervals (with mean c), yielding a gamma distribution of target events (with shape parameter $\theta t/c$ and scale parameter c; the gamma distribution is the sum of exponential distributions). Consequently, the temporal distribution of target responses is expected to have a mean = θt, standard deviation (SD) = $\sqrt{(\theta tc)}$, and coefficient of variation (CV) = SD/mean = $\sqrt{[c/(\theta t)]}$ (Killeen & Fetterman, 1988).

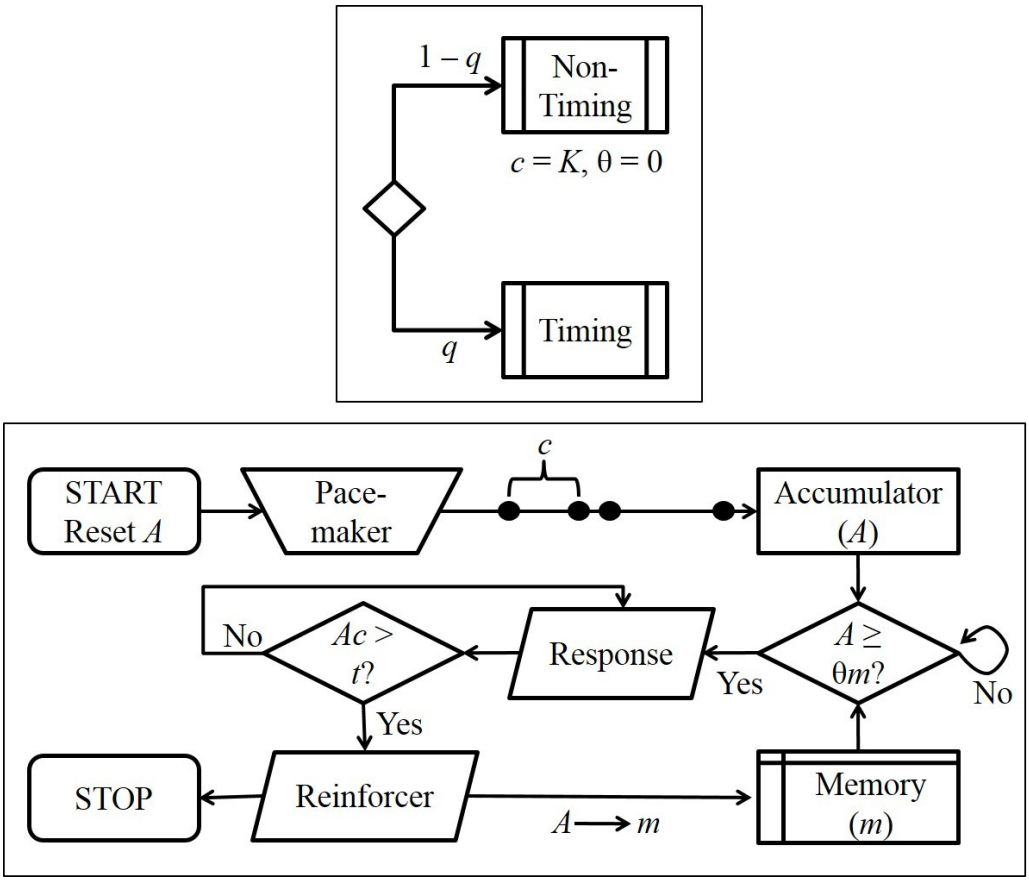

Figure 1. Schematic of the timing mechanism. See text for details.

This description of the clock mechanism implies that, when comparing performance in a production task across stable conditions (e.g., following extensive FI

training under a drug vs. in the absence of the drug), differences in the mean of the temporal distribution of target responses is due to changes in the response threshold, θ, and not due to changes in the speed of the clock, c. A faster clock implies larger pulse counts that induce a transient shortening of the mean, which return to baseline as memory updates with larger pulse counts. That is, after sufficient training, a faster clock (i.e., a smaller c) is expected to yield a smaller SD and CV, but the same mean of a slower clock. In contrast, a lower threshold θ is expected to yield a temporal distribution of target responses with a lower mean, smaller SD, but a larger CV.

The present study sought to evaluate an experimental paradigm, a dependent concurrent short FI long FI schedule of reinforcement, intended to isolate the response threshold. In this paradigm, animals are trained to respond for reinforcement at two locations, one associated with a short FI and one associated with a long FI. On any given trial, both locations are available but only one is active; as a result, the latency-to-switch to the long FI (LTS) is expected to be sensitive to the timing of reinforcement (Figure 2; Fetterman & Killeen, 1995; Stubbs & Pliskoff, 1969)[1]. The LTS thus serves as the target response in the FI FI schedule. This task (and others similar to it) has already proven useful in the study of temporal cognition in a variety of species (Balci, Papchristos, Gallistel, Brunner, Gibson, & Shuyatsky, 2008; Daniels et al., 2015; Maggi et al., 2014). However, few studies have examined how differences in the magnitude of reinforcement across FI components affect timing performance in this task (but see Daniels et al., 2015), or whether such an effect varies across species.

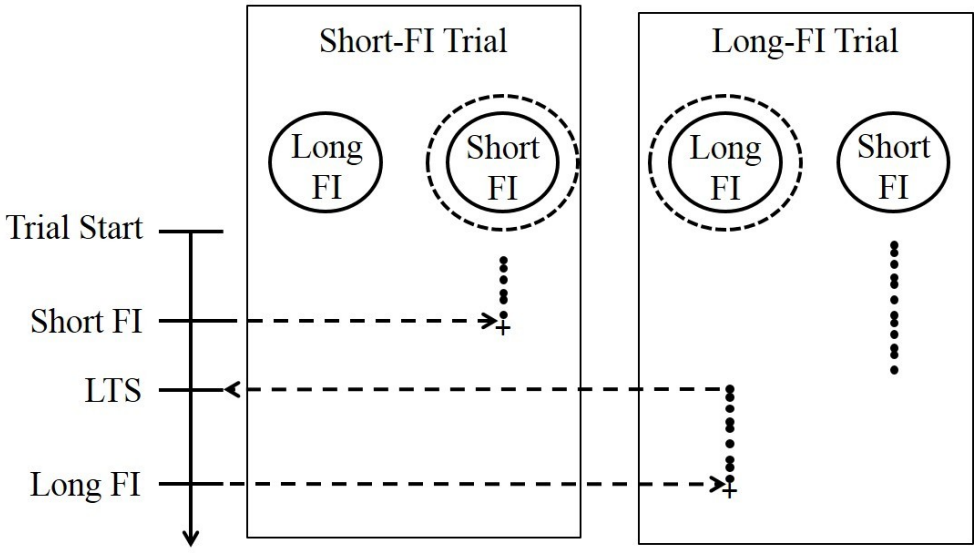

Figure 2. Schematic of the dependent concurrent FI FI schedule of reinforcement. Each panel corresponds to a trial type. The dashed circle indicates the active FI, not signaled to the subject. The dots illustrate the typical pattern of responding, first on the short FI and then, in long-FI trials, switching over to the long FI.

[1] Note that the latency-to-switch to the long FI (LTS) is one of several possible measures of interval timing; an alternative is the latency-to-depart from the short FI (LTD). In typical timing procedures, learning when to start responding (i.e., switch) is typically learned prior to learning when to stop responding (i.e., depart) and is relatively steady throughout training (MacDonald, Cheng, & Meck, 2012; Balci, Gallistel, Allen, Frank, Giibson, & Brunner, 2009). In well-trained subjects, however, these measures are expected to be highly positively correlated and thus yield the same information.

The “+” symbols indicate reinforced responses. Ticks in the vertical timeline indicate the LTS and when reinforcement on each FI is programmed. See text for further details.

The absolute magnitude of reinforcement appears to affect the speed of the clock (Fetterman & Killeen, 1991; Killeen, Hanson, & Osborne, 1978; but see Ludvig, Conover, & Shizgal, 2007 and MacEwen & Killeen, 1991), whereas recently the difference in the magnitude of reinforcement between response alternatives has been found to modulate the response threshold (Daniels et al., 2015; Sanabria, Thrailkill, & Killeen, 2009). Because in the proposed computational model of timing (Figure 1) θ represents the response threshold, a reduced θ is expected with larger reinforcement on the long FI, whereas larger θ is expected with larger reinforcement on the short FI.

In a recent test of this hypothesis, Daniels and colleagues (2015) trained rats on a dependent concurrent FI 8-s FI 16-s; some rats received the same magnitude of reinforcement on both FIs and some received a larger magnitude of reinforcement on the FI 16-s. The latter rats responded earlier than the former in a manner consistent with a decrease in θ. However, because differences in the magnitude of reinforcement were in place from the outset of training, it is possible that earlier responding was actually due to unequal reinforcement interfering with learning rather than a selective decrease in the response threshold. Thus, this study aimed at determining whether differences in reinforcement magnitude across concurrent dependent FI schedules, implemented after training with equal magnitudes of reinforcement, yield selective changes in θ. Specifically, it tested whether increasing the magnitude of the long-FI reinforcer causes a selective reduction of θ in rats, pigeons, and humans.

Meaningful comparisons of the performance of different species in a task are difficult, because tasks must be tailored to each species, potentially confounding species and task effects. Thus, the analysis of our results adopts the anagenetic approach proposed by Bitterman (1960, 1965; Greenberg, 1995; also see Horne & Lowe, 1993 and Madden & Perone, 1999 for a discussion of the anagenetic approach). This approach focuses not on comparing metrics of performance across species, but rather on how the effects of functionally analogous manipulations differ across species. To the extent that such effects are similar across species, a similarity in the underlying mechanism is inferred.

To estimate and manipulate θ, the FI FI schedule of reinforcement has some important advantages over the simple FI schedule. Sanabria et al. (2009) suggested that low responding early in FI trials may be explained by a competition between reinforcement of the target response and the hypothetical and constant rate of reinforcement of all alternative behaviors [similar to Herrnstein’s (1970) Ro, which is meant to quantify aggregate reinforcement of alternative behaviors, e.g. grooming]. Whereas the strength of FI-controlled behavior rises with reinforcer proximity, the strength of alternative behaviors is constant; θ indicates when the former rises to and above the level of the latter. This implies a particular difficulty in estimating θ from FI performance parameters, such as the distribution of latency-to-first response, because the strength of alternative behaviors is not controlled, is of unknown provenance, and it is likely to include the strength of magnitude-sensitive postprandial (post reinforcer consumption) behavior (Lowe, Davey, & Harzem, 1974). The FI FI schedule addresses these limitations by putting alternative behaviors under experimental control in the form of short-FI responses. Because short-FI and long-FI responses are both reinforced by food, the relative strength of these responses should vary only as a function of time

since trial onset, and thus provide more reliable estimates of θ, via the distribution of LTSs, and a more reliable means to manipulate it.

To isolate putative effects on θ, it is important to acknowledge that descriptive statistics do not always unequivocally support one inference over another. For example, shorter LTSs could be due to a reduced response threshold or an elevated speed of the clock. To select between these two explanations, measures of LTS dispersion are also needed. Furthermore, even after extensive training, timing tasks yield target responses that are not always sensitive to the passage of time (Daniels et al., 2015; Freestone, Balci, Simen, & Church, 2015; Lejeune & Wearden, 1991; Sanabria & Killeen, 2008). That is, some target responses appear to be emitted at random intervals. These random responses can confound the interpretation of measures of both precision and accuracy. Failure to partial out random responses can lead to inaccurate, misleading, or incomplete characterizations about psychological processes (Ludvig, Conover, & Shizgal, 2007; Sanabria & Killeen, 2008; Richards, Sabol, & Seiden, 1993). Thus, it is important to model data to provide a more clear and precise understanding of changes in interval timing.

To model interval timing contaminated with random responding, it is assumed that, at the beginning of every timing trial, animals either enter a timing state with probability q or enter a non-timing state with probability 1 – q (top panel of Figure 1). In the timing state, the internal clock produces gamma-distributed target responses according to the mechanism already described in the bottom panel of Figure 1. In the non-timing state, target responses are emitted at random times; their distribution over time is exponential. The non-timing state can be described as the operation of the timing mechanism with a mean inter-pulse interval K and a response threshold of zero (i.e., the first pulse causes emission of a target response). The non-timing state implies that the temporal distribution of target responses is expected to be a gamma-exponential mixture distribution,

$$p(\tau) = q\Gamma(\tau;\theta t/c,c) + (1-q)\exp(\tau;K), \quad (1)$$

where $1 \geq q \geq 0$, $\theta t > c$, $\langle c,K \rangle > 0$, τ is the time of a target response, q is the probability of entering a timing state in which pulses are emitted on average every c units of time, and a response is produced after θt/c pulses; K is the mean and standard deviation of non-timed intervals.

Rats, pigeons, and humans were trained and tested on a dependent concurrent short FI long FI schedule of reinforcement. After establishing a performance baseline, the magnitude of reinforcement on the long FI was increased threefold. First, an analysis of the empirical distribution of LTSs was conducted to characterize the observed effects. Second, it was determined whether the gamma-exponential mixture distribution (Eq. 1) provided an adequate fit to the baseline performance of all three species. Third, a model-comparison approach isolated potential mechanisms by which increasing the relative magnitude of reinforcement altered timing performance.

Method

Subjects

Pigeons. Twelve male pigeons (*Columba livia*) with previous experience on fixed-interval schedules of reinforcement served as subjects; they were housed individually in a room with a 12:12-hr day:night cycle with dawn at 0600 hr. All pigeons had free access to water and grit at their home cages. Pigeons' running weights were based on 80% of their free-feeding weight. Immediately prior to each experimental session each pigeon was weighed and was excluded from a session if its weight exceeded 8% of its running weight. When required, a supplemental feeding of ACE-HI pigeon pellets (Star Milling Co.) was given at the end of each day, at least 12 hr before experimental sessions were conducted. Supplementary feeding amounts were equal to 50% of the average amount fed over the last day, plus 50% of the deviation in weight from the last day, plus 50% of the current deviation from target running weight.

Rats. Sixteen experienced male Wistar rats (Charles River Laboratories, Hollister, CA) served as subjects. Rats arrived on post-natal day (PND) 60 and were immediately pair-housed upon arrival. Prior to PND 90, when the present experiment began, half of the rats were exposed to twice-daily injections of nicotine (0.6 mg/kg) for 12 days followed by 12 sessions of a Pavlovian conditioned approach task (no significant effects of nicotine exposure on performance were observed, see Results section). Rats were housed on a 12:12 hr light cycle, with dawn at 1900 hr; all behavioral training was conducted during the dark phase of the light cycle. Food restriction protocols were implemented shortly after arrival. Access to food was reduced daily from 24, to 18, 12, and finally 1 hr/d. During behavioral training, food was provided 30 min after the end of each training session, such that at the beginning of the next session weights were on average 75% of mean ad libitum weights estimated from growth charts provided by the breeder.

Humans. Eleven undergraduate students (2 male, 9 female) between 18-19 years old participated in the experiment. Students were recruited from an introductory psychology course at St. Lawrence University and earned course credit for their participation. Prior to the experiment, participants were asked about having taken any drugs within the past 72 hours, alcohol within the past 12 hours, and nicotine or caffeine within the past 2 hours. All participants responded no to all these questions, except for one participant who had consumed caffeine and another who had consumed an over-the-counter cough medicine.

Apparatus

Pigeons. Experimental sessions were conducted in five Med Associates modular test chambers. The sidewalls and ceiling of the experimental chambers were clear plastic. The floor consisted of thin metal bars above a catch pan. The test panel contained three plastic transparent response keys (25 mm in diameter; MED Associates, ENV-123AM), located 70 mm from the ceiling and arranged horizontally. Each key could be illuminated by green, white, or red light emitted from diodes behind the keys. A square opening 77 mm across was located 20 mm above the floor on the front panel and could provide access to milo grain when the food hopper (Coulbourne Instruments, part H14-10R) was activated. A house light was mounted 12 mm from the ceiling on the back wall. The ventilation fan on the rear wall of the enclosing chamber provided masking noise of approximately 60 dB. Experimental events were arranged and recorded via a MED PC® interface connected to a PC controlled by MED-PC IV® software.

Rats. Experimental sessions were conducted in 16 MED associates (St. Albans, VT, USA) modular test chambers (3 chambers were 305 mm long, 241 mm wide, and 210 mm high; 13 chambers were 305 mm long, 241 mm wide, and 292 mm high), each enclosed in a sound- and light-attenuating box equipped with a ventilating fan. The front and back walls and the ceiling of test chambers were made of Plexiglas; the front wall was hinged and served as a door to the chamber. One of the two aluminum side panels served as a test panel. The floor consisted of thin metal bars positioned above a catch pan. The reinforcer receptacle was a square opening (51-mm sides) located 15 mm above the floor and centered on the test panel. The receptacle provided access to a dipper (MED Associates, ENV-202M-S) fitted with a cup (MED Associates, ENV-202C) that could hold 0.01 cc of a liquid reinforcer (33 % sweetened condensed milk diluted in tap water, Great Value brand, Walmart, Bentonville, AK). The receptacle was furnished with a head entry detector (ENV-254-CV). A multiple tone generator (MED Associates, ENV-223) was used to produce a 15-kHz tone at approximately 75 dB through a speaker (MED Associates, ENV-224 AM) centered on the top of the wall opposite the test panel and 240 mm above the floor of the chamber. Two retractable levers (ENV-112CM) flanked the reinforcer receptacle. A house light located behind the wall opposite to the test panel could dimly illuminate the test chambers. A ventilation fan provided masking noise of approximately 60 dB. Experimental events were arranged via a MED PC® interface connected to a PC controlled by MED-PC IV® software.

Humans. Experimental sessions were conducted in isolation in a 3.7 m x 1.8 m cubicle furnished with an HP Compaq Elite 8300 desktop computer. The computer was equipped with a standard keyboard and mouse. For this experiment, the keyboard was set aside and participants used the mouse to respond in the task. The task was programmed in Microsoft Visual Basic ®.

Procedure

Following apparatus acclimation and manipulanda training, all subjects were trained on dependent concurrent FI FI schedules of reinforcement. In this procedure, two manipulanda were presented on every trial, each associated with a different FI schedule (rats and pigeons: 8 s vs. 16 s; humans: 4 s vs. 8 s). On any given trial, only one FI was effective but was not signaled. Subjects sensitive to the timing of reinforcement were expected to respond first on the short-FI manipulandum and later switch over to the long-FI manipulandum. The time to the first response to the long-FI manipulandum was labeled “latency to switch” (or LTS); it served as a measure of temporal judgment. Following the LTS, responses on the short-FI manipulandum were not prevented, nor did they cancel the trial or exclude it from analysis. It was verified, nonetheless, that nearly all trials contained only long-FI responses past the LTS.

Condition 1: Baseline Training.

Pigeons. Each session began with a warm-up period in which the houselight was illuminated for 135 s, followed by the first training trial. At the onset of each trial, the houselight was turned off and the left and right keys were illuminated red and green, respectively. Reinforcement was then programmed according to one of two schedules: FI 8-s programmed on the left/red key or FI 16-s programmed on the right/green key. Schedules were selected with equal probability by random sampling without replacement from a list of 6 items (“FI 8-s”, “FI 8-s”, “FI 8-s”, “FI 16-s”, “FI 16-s”, and “FI 16-s”); selected schedules were not signaled to the pigeons. Following completion of the active schedule, the keys were turned off and the food hopper was activated for 2 s. Reinforcement was followed by a 135-s ITI. Each session continued for 90 minutes or until the completion of 24 trials. Sixteen sessions were conducted; visual inspection of the data suggested behavior was stable over the last twelve sessions.

Rats. Each session began with a 180-s warm up period during which the house-light was illuminated. After the warm-up period each trial began with offset of the house-light, extension of the left and right levers and illumination of the yellow light above each lever. Reinforcement was programmed according to one of two schedules: FI 8-s assigned to the left lever and FI 16-s assigned to the right lever for half of the rats, and vice versa for the other rats. Schedules were selected with equal probability by random sampling without replacement from a list of 6 items; selected schedules were not signaled to the rats. Following completion of the active schedule, levers were retracted, yellow lights were turned off, a 15-kHz tone was turned on, and the liquid dipper was activated for 2.5 s. Following reinforcement, the tone was turned off and the house-light was illuminated for 5 s before the following trial started. Each session lasted 75 minutes. Training continued for a minimum of 10 sessions until all rats demonstrated stable temporal control. Stability was determined by a non-significant regression of the median and inter-quartile range (IQR) of LTSs over four consecutive sessions.

Humans. At the beginning of the experiment, participants sat in front of the computer with the following instructions presented on the screen and read aloud by the experimenter:

> You can respond on the computer screen by clicking the mouse or tapping the screen (you may be directed to do one or the other, but not both). Only tap or click one of the boxes at a time. You may respond as many times as you want. On some trials, points will be available for a response on the left box ("A") after a SHORT delay. On other trials points will be available on the right box ("B") after a LONG delay. Your task is to learn the delays and maximize the points you earn. Please do not count in an attempt to keep track of time. Just try to learn the delays "naturally." Occasionally, you will be provided breaks. You may exit this room during breaks. Your breaks can be as long or as short as you'd like. Feel free to use the restroom or get a drink during your break. When you are finished, just get up and leave and inform the experimenter. Please do not attempt to exit the "you are finished" screen. Do you have any questions? Please begin when you are ready by clicking the "Start" button.

After clicking the start button each trial began with the presentation of two rectangles on the center-left and center-right of the computer screen labeled “A” and “B,” respectively. Reinforcement was programmed according to one of two schedules: FI 4-s assigned to the A rectangle and FI 8-s assigned to

the B rectangle. Schedules were selected pseudo randomly from a 12-item list such that each occurred six times out of every 12 trials, and neither schedule was selected more than 12 consecutive times. Following the first response after completion of the active schedule a point was delivered. If no clicks occurred during a 1-s limited hold on the active schedule in a trial, a 3-s blackout occurred during which the options were covered with a black rectangle in the center of which the words "Missed Point(s)" were displayed. The next trial started immediately following feedback. This training was in effect until 30 points were earned on the long FI schedule, except for two participants for whom conditions continued until 40 points were earned. Points were hypothetical and not exchangeable.

Following completion of the experiment (Condition 1-2), the following survey was administered to determine if any strategies were used to help time the intervals.

1. What was the best way to earn points?
2. Please describe any strategies you used over the course of the experiment.
3. If you were required to time certain intervals or estimate durations, did you count (overtly or covertly) to assist yourself in timing those intervals/durations? Please explain.

Condition 2: Bias Training.

All experimental parameters were the same as in Baseline (Condition 1), except that the magnitude of reinforcement on the long-FI schedule was increased. For pigeons, hopper activation was increased from 2 s to 6 s. For rats, the number of dipper arm activations was increased from 1 to 3, with 1-s intervals between deliveries. For humans, the number of points delivered increased from 1 to 3. This condition was in effect for all species using the same criterion as in Baseline. Rats and pigeons experienced each condition once. Humans experienced a third condition in which the number of points delivered in the long-FI schedule remained at 1, but increased to 3 in the short-FI schedule (data not shown). Humans experienced all three conditions twice in counterbalanced order within each of two blocks, except that Baseline always occurred first in the first block.

Data Analysis

The last twelve (for pigeons) and four (for rats) sessions of Baseline (Conditions 1) and Bias (Condition 2) were analyzed; for humans, all data from both determinations of Conditions 1 and 2 were analyzed. On every trial in which the long-FI schedule was active, all responses were recorded with the time between trial onset and the last response on the short FI constituting the latency to depart (LTD), and the time between trial onset and the first response on the long FI constituting the latency to switch (LTS). Prior to the analysis of interval timing, the assumption that subjects started on the short FI and remained on the long FI after switching was confirmed by inspecting the proportion of trials in which subjects started on the short FI and the proportion of trials in which subjects remained on the long FI after switching. Additionally, the correlation between the distribution of LTSs and LTDs was inspected to confirm the extent to which these two measures provide the same information. If the distributions were both highly and positively correlated, then only LTSs were analyzed; otherwise, both LTSs and LTDs were subjected to the same analysis.

For humans, trials in which a LTS did not occur (7.5% of all trials) were omitted. In a first analysis, the parameters of the empirical distribution were compared across conditions (Baseline vs. Bias) within each species using dependent t-tests with $\alpha = .05$. In a second analysis, Eq. 1 was validated for each species using data only from baseline sessions; then, maximally-likely estimates of the parameters of Eq. 1 (Myung, 2003) were obtained for each individual subject and compared across conditions using the corrected Akaike Information Criterion (AICc; Burnham, & Anderson, 2002). AICc is a model selection criterion that favors models that balance high likelihood with low complexity (see Appendix A for details). AICc is asymptotically equivalent to leave-one-out cross-validation (Fang, 2011; Stone, 1977). The size of all reported effects is reported as Cohen's d. If the distribution of LTS and LTDs were highly positively correlated or yielded the same effects in the first analysis then only LTSs were subjected to quantitative modeling.

Results

Empirical Distribution of LTSs

Figure 3 shows the mean, standard deviation (SD), and coefficient of variation (CV = SD / mean) of LTSs under both Baseline and Bias conditions for each species. Prior to analysis, independent *t*-tests revealed no significant difference in measures of temporal performance between rats exposed to nicotine and rats exposed to saline in a prior experiment (all *p*s > 0.05 and all *t*s < 2.5; see Subjects section for details). Additionally, the assumption that subjects began on the short FI and then after the LTS responded primarily the long-FI manipulandum was verified on baseline performance. All species started on the short FI in most of their trials, with humans doing so in 98% of trials, pigeons in 95%, and rats in 75% of their trials. Furthermore, humans and pigeons switched back to the short FI in only 13% and 6% of their trials, respectively, but rats switched back in 51% of their trials. Although rats switched back to the short FI often, they start on the short FI more often than chance indicating they had learned the task. Consistent with these results, the LTSs and LTDs were highly and positively correlated for pigeons, $r = 0.98$, and humans, $r = 0.93$, but not rats, $r = 0.32$.

Dependent t-tests revealed that, for rats, increasing the magnitude of reinforcement on the long-FI schedule reduced the mean of LTSs, $t(15) = 5.92$ $p < 0.001$, d = 1.48, and LTDs, $t(15) = 5.13$, $p < 0.001$, d = 1.28, the SD of LTSs, $t(15) = 4.17$, $p < 0.001$, d = 1.04, and LTDs, $t(15) = 2.74$, $p = .02$, d= 0.68, and the CV of LTSs, $t(15) = 2.29$, $p = .036$, d =0.57, but not of LTDs, $t(15) = 0.56$, $p = 0.59$, d = 0.15. This pattern of effects suggests that, for rats, the Biasing manipulation had a similar effect on both LTSs and LTDs.

For pigeons, increasing the magnitude of reinforcement only reduced the mean of LTSs, $t(11) = 2.55$, $p = 0.027$, d = 0.73; no significant effect was observed on their SD, $t(11) = 0.02$, $p = 0.98$, d < 0.001, or CV, $t(11) = 0.52$, $p = 0.67$, d = 0.19. For humans, increasing the magnitude of reinforcement increased the mean of LTSs, $t(10) = 2.56$, $p = 0.0268$, d = 0.77, reduced their SD, $t(10) = 2.99$, $p = 0.014$, d = 0.90, and reduced their CV, $t(10) = 3.14$, $p = 0.019$, d = 0.95.

Quantitative Modeling of LTSs

The presence of timed and non-timed LTSs was determined for each species under baseline by comparing the likelihood of Eq. 1, corrected for the number of free parameters, against two nested models, all fit to individual empirical distributions of baseline LTSs. One nested model fixed q at 1 and allowed θ and c to vary freely; this model assumed that all LTSs are timed. The second nested model fixed q at zero and allowed K to vary freely; this model assumed that all LTSs are non-timed. AICc selected the full model, that is, the model that assumed a mixture of timed and non-timed LTSs in experimental performance (see Appendix B, Table B1). After correcting for free parameters, the full model was at least e^{217} times more likely, for all three species, than the model that assumed only timed LTSs (i.e., q = 1, the second best model for every species, according to AICc).

Preliminary estimates of the parameters of Eq. 1 suggest that increasing the magnitude of reinforcement in the long FI schedule had a complex array of effects. For

rats and pigeons, mean estimates of all parameters (c, θ, q, and K) declined between Baseline and Bias conditions. For humans, mean estimates of c also declined, but estimates of θ, q, and K increased. To determine the reliability of these effects, models that excluded some or none of the purported effects were compared within each species using AICc and protocols established by previous research (See Appendix A; Avila et al., 2009; Daniels et al., 2015).

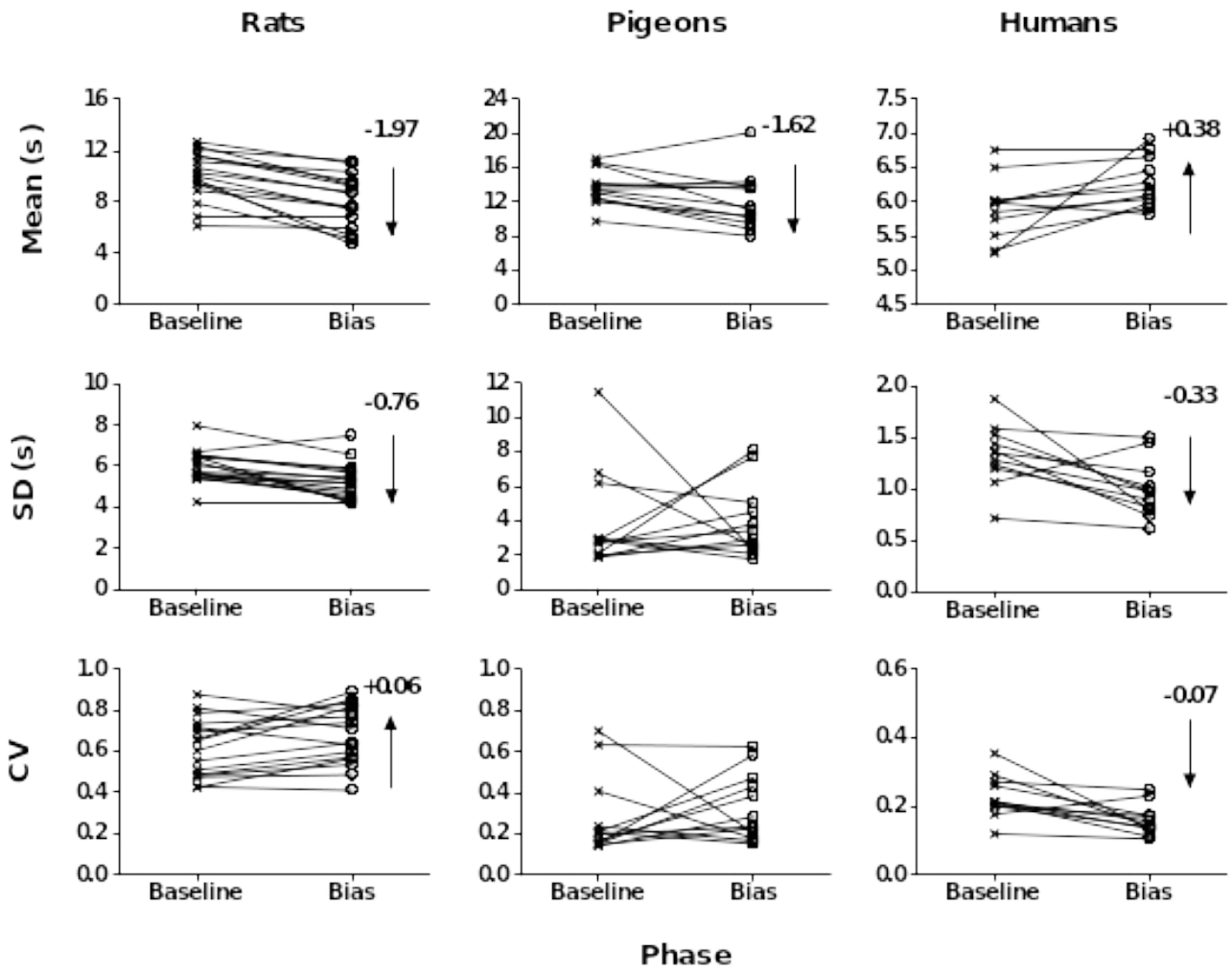

Figure 3. Empirical mean, standard deviation (SD), and coefficient of variation (CV = SD / mean) of the distribution of LTSs of each of three species performing in a concurrent dependent fixed-interval (FI) FI schedule of reinforcement. The FI schedules were 8 vs. 16 s for rats and pigeons and 4 vs. 8 s for humans. Symbols represent data from individual subjects and are connected by solid lines to indicate the direction of change. Reinforcement across FI schedules was equal in Baseline condition ("x" symbols) but larger in the longer FI in Bias condition (circles). The direction of significant differences between baseline and bias conditions ($p < 0.05$) is indicated by black arrows with the average difference between conditions indicated above the arrow.

The models most favored by AIC are shown in Table 1 (an analysis of all models is in Appendix B, Table B2). AICc selected models q,θ,K for rats, q,θ,c for pigeons, and θ,c and q,θ for humans. Figure 4 shows mean fits of the selected models to data under both Baseline and Bias conditions (for humans, the fit of θ,c is shown). This figure

confirms that the selected models adequately describe the data and how the Bias condition shifted the distribution of LTSs for each species. Estimates of the parameters of these models and some key derived statistics are presented in Table 2.

For rats, the biasing manipulation decreased model-based estimates of the probability of a timed response q by .10 (d = 1.19), of the response-threshold, θ, by 0.08 (d = 2.02), and of the mean non-timed LTS, K, by 1 s (d = 1.06). For pigeons, the biasing manipulation decreased estimates of q by .09 (d = 0.77), of θ by 0.08 (d = 1.20), and of the mean inter-pulse interval, c, by 0.12 s (d = 0.50). In contrast, for humans, two models were selected. In model θ,c the biasing manipulation increased estimates of θ by 0.03 (d = 0.61), and decreased estimates of c by 0.03 s (d = 0.73). In model q,θ the biasing manipulation increased estimates of θ also by 0.03 (d = 0.61) and q by .06 (d = 0.89). Importantly, in all three species, the biasing manipulation influenced estimates of θ, but only in the expected direction in rats and pigeons.

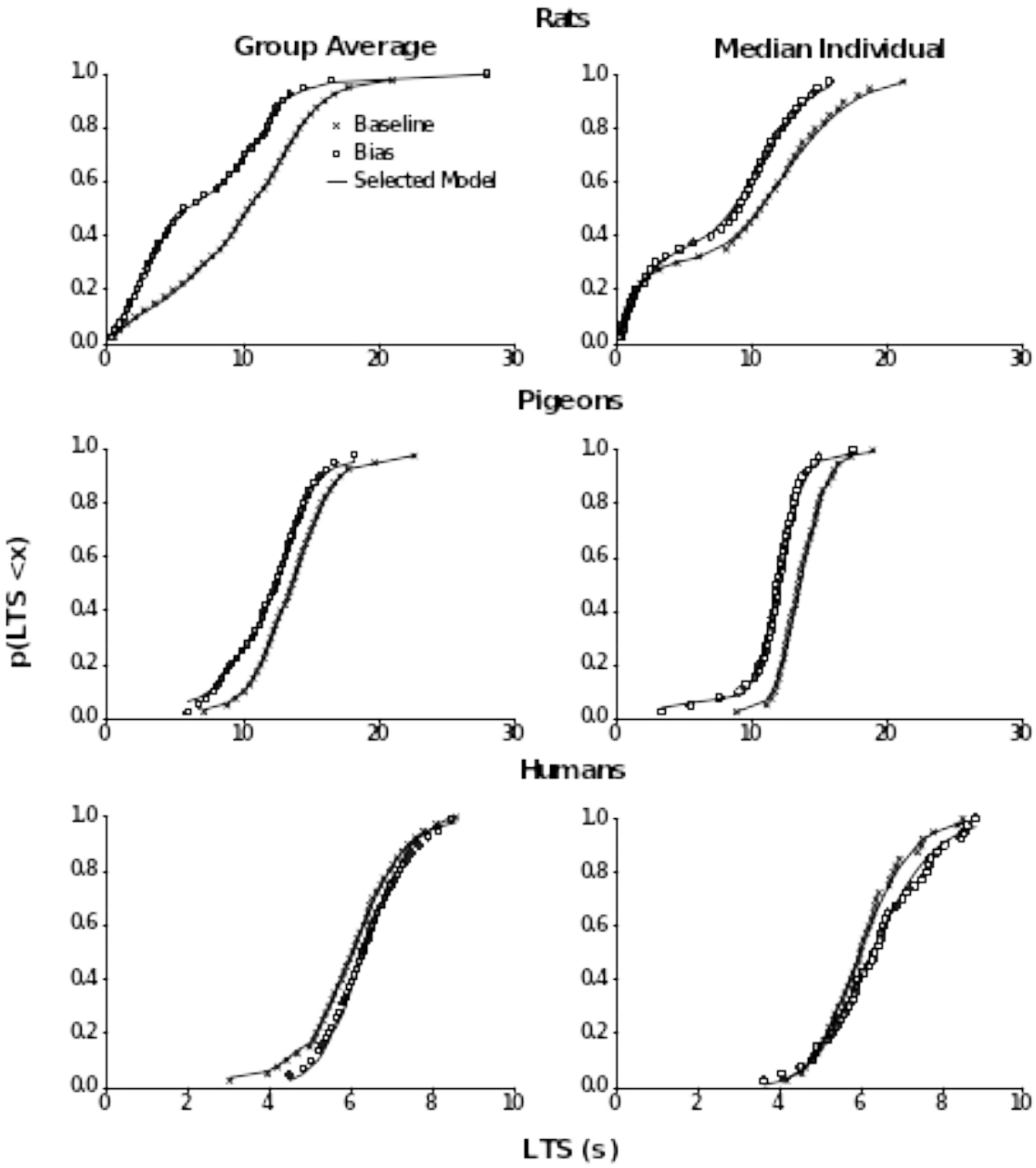

Figure 4. Empirical cumulative distribution of baseline LTSs (symbols) and mean fitted trace of Eq. 1 for rats (top row), pigeons (middle row), and humans (bottom row). The mean empirical cumulative distribution is show in the left panel of each row; and an empirical cumulative distribution of a representative subject is shown in the right panel of each row. Representative subjects were chosen as those with the median change in mean LTS between the Baseline and Bias conditions. Individual LTSs are organized in 40 bins, each containing 1/40th of the LTSs.

Table 1
Summary of selection among models of biased timing

	Rats		Pigeons		Humans	
Full Model	$c_{baseline} > c_{bias}$, $\theta_{baseline} > \theta_{bias}$, $q_{baseline} > q_{bias}$, $K_{baseline} > K_{bias}$		$c_{baseline} > c_{bias}$, $\theta_{baseline} > \theta_{bias}$, $q_{baseline} > q_{bias}$, $K_{baseline} > K_{bias}$		$c_{baseline} > c_{bias}$, $\theta_{baseline} < \theta_{bias}$, $q_{baseline} < q_{bias}$, $K_{baseline} < K_{bias}$	
Nested Models	MLE	ΔAICc	MLE	ΔAICc	MLE	ΔAICc
θ	-36340.71	278.57	-8410.46	150.99	-2112.63	4.66
q,θ	-36207.01	43.62	-8340.24	35.49	-2100.15	<u>3.72</u>
θ,*c*	-36324.64	278.87	-8382.07	119.16	-2098.3	<u>0</u>
q,θ,*c*	-36172.59	7.29	-8309.93	<u>0</u>	-2091.85	11.50
q,θ,*K*	-36168.94	<u>0</u>	-8328.09	36.33	-2101.84	31.49
q,θ,*c*,*K*	-36154.21	3.14	-8298.68	2.81	-2089.59	31.77

Note. Tested models are labeled with the free parameters that were allowed to vary between baseline and bias conditions, in the direction indicated by the full model. The number of free parameters for each tested model is equal to the number of free parameters allowed to vary multiplied by the number of subjects. Each model was fit to 12875 data points for rats, 3456 for pigeons, and 1433 for humans under both Baseline and Bias conditions. All data are provided to calculate ΔAICc. The selected models are underlined. Only the models with one of the three lowest ΔAICc in at least one species are included; the full list of models is reported in Appendix B, Table B2.

Bias-induced changes in estimates of the parameters of Eq. 1 were reflected in the derived statistics in a manner consistent with the descriptive statistics reported in Figure 3. Reductions in estimates of θ in rats and pigeons, and of c in pigeons, are reflected in shorter and less dispersed timed LTSs in these species. Because estimates of c in rats were robust to the biasing manipulation, the CVs of timed LTSs increased slightly but noticeably for this species (d = 1.69), whereas the mean CV decreased slightly for pigeons (d = 0.47). These patterns of change, combined with a bias-induced increase in the proportion of relatively short estimates of non-timed LTSs, yielded shorter mean LTSs in non-human species under bias conditions.

The bias-induced elevation of the estimates of θ in both models chosen for humans is reflected in a small increase of 0.27 s (d = 0.61) in timed LTSs. According to model θ,c the small size of this effect is due to a bias-induced reduction of estimates of c in humans, and is reflected in a reduced dispersion (d = 0.73) and CV (d = 0.78) of human LTSs. Alternatively, according to model q,θ the bias-induced reduction in dispersion could be due to an increase in estimates of q in humans, suggesting that the bias increased the probability of emitting timed responses.

Table 2

Mean parameter estimates and derived statistics of the distribution of LTSs

	Rats		Pigeons		Humans			
Selected Model	$q_{baseline} > q_{bias}$, $\theta_{baseline} > \theta_{bias}$, $K_{baseline} > K_{bias}$		$q_{baseline} > q_{bias}$, $\theta_{baseline} > \theta_{bias}$, $c_{baseline} > c_{bias}$		$\theta_{baseline} < \theta_{bias,}$ $c_{baseline} > c_{bias}$		$q_{baseline} < q_{bias}$, $\theta_{baseline} < \theta_{bias}$	
Parameter	*Baseline*	*Bias*	*Baseline*	*Bias*	*Baseline*	*Bias*	*Baseline*	*Bias*
q	0.59 (0.04)	0.47 (0.04)	0.92 (0.04)	0.83 (0.05)	0.97 (0.01)		0.94 (0.02)	0.99 (0.01)
θ	0.79 (0.01)	0.71 (0.03)	0.88 (0.03)	0.80 (0.04)	0.76 (0.01)	0.79 (0.01)	0.76 (0.01)	0.79 (0.01)
c	0.51 (0.06)		0.43 (0.09)	0.31 (0.05)	0.19 (0.03)	0.13 (0.02)	0.16 (0.02)	
K	6.59 (0.68)	5.46 (0.63)	13.97 (3.49)		2.37 (0.43)		3.17 (0.42)	
Derived Statistics								
Mean of timed LTSs (s)	12.73 (0.18)	11.28 (0.21)	14.09 (0.49)	12.81 (0.59)	6.07 (0.11)	6.34 (0.10)	6.07 (0.11)	6.34 (0.10)
SD of timed LTSs (s)	2.48 (0.14)	2.33 (0.14)	2.37 (0.27)	1.93 (0.19)	1.04 (0.07)	0.91 (0.06)	0.95 (0.06)	0.97 (0.06)
CV of timed LTSs	0.19 (0.01)	0.21 (0.01)	0.16 (0.01)	0.15 (0.01)	0.17 (0.02)	0.14 (0.01)	0.17 (0.01)	0.15 (0.01)
Mean LTS (s)	10.09 (0.47)	8.03 (0.52)	13.69 (0.66)	12.18 (0.87)	5.96 (0.12)	6.22 (0.11)	5.87 (0.13)	6.31 (0.11)

Note. Values in parentheses are SEM of parameter estimates. Models are described according to the free parameters that were allowed to vary across the baseline and bias condition. Derived statistics were obtained as follows: mean of timed LTSs = θt, where t is the length of the long FI requirement; SD of timed LTSs = $\sqrt{(\theta ct)}$; CV of timed LTSs = $\sqrt{(c/\theta t)}$; mean LTS = $q\theta t + (1 - q)K$.

Discussion

The present study tested, in three species, whether increasing the magnitude of reinforcers available at a later time and in a different location induced a sustained, earlier transition to that location. More specifically, a model of interval timing predicted that such manipulation selectively reduces a threshold to respond in the rich location without affecting the internal clock. Increasing the magnitude of reinforcement in the longer of two dependent concurrent FI FI schedules yielded earlier LTSs in rats and pigeons, but later LTSs in humans; it reduced the dispersion of LTSs in both rats and humans, but did not affect dispersion of LTSs in pigeons. This pattern of effects suggests that the biasing manipulation reduces the response threshold for rats. However, the pattern observed for pigeons does not unequivocally support any particular hypothesis, because no effect was observed on the LTS dispersion; the pattern observed for humans is inconsistent with changes in the response threshold or the speed of the clock. Estimates of the parameters of a model of LTSs (Eq. 1) were consistent with inferences on bias-induced changes in response threshold in rats and suggested the operation of a similar mechanism in pigeons. Furthermore, it provided potential explanations for the lack of an effect on LTS dispersion and inconsistent effects on LTS dispersion for pigeons and rats, respectively. Specifically, the model selection procedure suggested that the biasing manipulation induced (a) a reduction in the threshold to respond in the rich location (in rats and pigeons), (b) a compensatory speeding up of the internal clock (in pigeons and potentially humans), and (c) other effects on non-timing parameters of performance (particularly in non-human animals and possibly in humans).

A bias-induced increase in clock speed in pigeons, expressed as a reduction in c, provides a potential explanation for the non-significant effect of the biasing manipulation on the dispersion of their LTSs. Such an effect may also have been present for humans (according to model θ, c). It is possible that the bias-induced increase in c resulted from a confound between relative and average reinforcement magnitude: when the difference in reinforcement magnitude between FI schedules was introduced, the overall amount of reinforcers obtained per unit of time increased. It appears that the speed of the clock is a function of the general arousal level of the animal (Killeen & Fetterman 1988; MacEwen & Killeen, 1991; Machado, 1997), which is modulated by the rate of reinforcement (Beam, Killeen, Bizo, & Fetterman, 1998; Bizo & White, 1994; Bizo & White, 1995; Bizo & White, 1997; Killeen & Fetterman, 1988) and, potentially, by the magnitude of reinforcement (Killeen, Hanson, & Osborne, 1978). However, some studies have failed to find evidence that increasing the magnitude of reinforcement increases the speed of the clock (Bizo & White, 1994; Fetterman & Killeen, 1991; Galtress & Kirkpatrick, 2009; Ludvig, Conover, & Shizgal, 2007; McEwen & Killeen, 1991). The present experiment failed to find such effect in rats. Although the conditions under which increased reinforcement speeds up the clock are unclear, it appears that the experimental conditions implemented in the present study induced such effect in pigeons, potentially in humans, but not in rats.

Differences in reinforcement magnitude between schedules also covaried with the order in which training conditions were presented: baseline conditions were generally presented before bias conditions. Therefore, some of the effects reported may be due to learning rather than to changes in the response threshold. The acquisition of FI performance is associated with longer and less dispersed post-

reinforcement pauses in rats (Taylor, Crofton, & MacPhail, 2002) and pigeons (Berry, Kangas, & Branch, 2012), so learning effects are expected to be expressed as later and less dispersed LTSs. The biasing manipulation induced earlier and more dispersed (higher CV) LTSs in rats and pigeons, but later and less dispersed (lower CV) LTSs in humans. Furthermore, two models were selected for humans; both suggested a bias-induced elevation in the response threshold, with one also suggesting an increase in the probability of a timed response and the other also suggesting a reduction in c. Both of these models suggest the LTSs became longer due to an elevation of the response threshold and that dispersion was reduced because either humans were engaged in timing more often or because the speed of the clock increased. Taken together, these data suggest that the effects observed in rats and pigeons are unlikely to reflect the acquisition of FI FI performance, but it cannot be ruled out in humans.

The ostensible bias-induced elevation of the response threshold in humans is particularly perplexing. This effect is not only inconsistent with theoretical expectations, but is not readily explainable by any alternative theory. It is important to keep in mind, however, that this effect was substantially weaker than the opposite effect in non-human species. The weakness and direction of this effect may be related to peculiarities in the way humans track time (Allan, 1998), to practice effects wherein responding becomes more efficient (Kangas & Branch, 2012; Nagarajan, Blake, Wright, Byl, & Merzenich, 1998; Rammsayer, 1994), to the free-operant variant of the task on which humans were trained and tested (Ludvig, Conover, & Shizgal, 2007), to the organization of conditions, to the nature of the reinforcers (Kollins, Newland, & Critchfield, 1997), or to the reinforcers serving as discriminative stimuli (Bonem & Crossman, 1988). Additionally, inspection of post-experimental survey answers revealed that 7 of the 11 participants engaged in some form of counting behavior (e.g., counting mouse clicks or foot-taps). This occurred even though participants were explicitly instructed not to count (previous research indicates that such instruction is a sufficient and simple way to avoid counting; Rattat & Droit-Volet, 2012; Wearden & Lejeune, 2008). This counting behavior could have contributed to effects observed in human behavior. Future research may explore which of these possibilities better explains when human timing behavior deviates from expected theoretical outcomes.

It is interesting that the biasing manipulation appears to reduce overall temporal control on behavior, as indexed by q, the probability of emitting a timed-response, in rats and pigeons. Such an effect replicates previous findings in rats (Daniels et al., 2015) and suggests that adding a dimension on which two response alternatives vary (magnitude of reinforcement added to location and time of reinforcement) reduces overall temporal control. It appears that variable dimensions related to reinforced behavior sometimes compete for control of such behavior, at least in non-human animals (but see Rice, Grace, & Kyonka, 2014). Further research may integrate this competition for control to theories of timing to build more comprehensive accounts of animal behavior.

Further research is necessary to more effectively isolate bias-induced changes on the mechanisms underlying timing performance. For instance, control of overall rate of reinforcement and potential learning factors may aid in maintaining non-threshold parameters invariant. Further research is also necessary to better adapt the FI FI schedule to human participants. For example, simply telling participants to not count was not as effective as has been previously suggested (Rattat & Droit-Volet, 2011;

Wearden & Lejeune, 2008). The congruity of non-human performance in this task with theoretical expectations drawn from a simple pacemaker-accumulator model, suggest that the standard analysis of performance in standard timing paradigms is highly vulnerable to confounds between timing and non-timing processes. Nevertheless, the present study suggests that (a) differences in the magnitude of reinforcement across response alternatives modulate the response threshold (Daniels et al. 2015; Galtress & Kirkpatrick, 2009; Ludvig, Balci, & Spetch, 2011; Ludvig, Conover, & Shizgal, 2007), (b) that this modulation is very similar in rats and pigeons, and (c) that further refinements in the application of the dependent concurrent FI FI schedule of reinforcement may isolate response-threshold effects, regardless of species.

Acknowledgments

We thank Paula Overby, Christine Herrera, Jesse St. Amand, Sanjana Khana, Raul Garcia, Alexander Spitzer, Samantha Auty, Brady Cooper, Molly Haskell, Aerial Ramey, and Depika Singha for their invaluable help with data collection.

References

Avila, I., Reilly, M. P., Sanabria, F., Posadas-Sánchez, D., Chavez, C. L., Banerjee, N., Killeen, P., & Castañeda, E. (2009). Modeling operant behavior in the Parkinsonian rat. *Behavioural Brain Research*, *198*, 298-305.

Balci, F., Papachristos, E. B., Gallistel, C. R., Brunner, D., Gibson, J., & Shumyatsky, G. P. (2008). Interval timing in genetically modified mice: a simple paradigm. *Genes, Brain and Behavior*, *7*, 373-384.

Balci, F., Gallistel, C. R., Allen, B. D., Frank, K. M., Gibson, J. M., & Brunner, D. (2009). Acquisition of peak responding: what is learned?. *Behavioural Processes*, *80*, 67-75.

Beam, J. J., Killeen, P. R., Bizo, L. A., & Fetterman, J. G. (1998). How reinforcement context affects temporal production and categorization. *Animal Learning & Behavior*, *26*, 388-396.

Berry, M. S., Kangas, B. D., & Branch, M. N. (2012). Development of key-pecking, pause, and ambulation during extended exposure to a fixed-interval schedule of reinforcement. *Journal of the Experimental Analysis of Behavior, 97*, 333-346.

Bizo, L. A., & White, K. G. (1997). Training with controlled reinforcer density: Implications for models of timing. *Journal of Experimental Psychology: Animal Behavior Processes*, *23*, 44.

Bizo, L. A., & White, K. G. (1995). Reinforcement context and pacemaker rate in the behavioral theory of timing. *Animal Learning & Behavior*, *23*, 376-382.

Bizo, L. A., & White, K. G. (1994). The behavioral theory of timing: Reinforcer rate determines pacemaker rate. *Journal of the Experimental Analysis of Behavior*, *61*, 19-33.

Boisvert, M. J., & Sherry, D. F. (2006). Interval timing by an invertebrate, the bumble bee *Bombus impatiens*. *Current Biology*, *16*, 1636-1640.

Bonem, M., & Crossman, E. K. (1988). Elucidating the effects of reinforcement magnitude. *Psychological Bulletin, 104*, 348.

Burnham, K. P., & Anderson, D. R. (2002). *Model selection and multimodel inference: a practical information-theoretic approach*. New York, New York: Springer Science & Business Media.

Bitterman, M. E. (1965). Phyletic differences in learning. *American Psychologist*, *20*, 396-410.

Bitterman, M. E. (1960). Toward a comparative psychology of learning. *American Psychologist*, *15*, 704-712.

Brunner, D., Kacelnik, A., & Gibbon, J. (1992). Optimal foraging and timing processes in the starling, *Sturnus vulgaris*: Effect of inter-capture interval. *Animal Behaviour*, *44*, 597-613.
Daniels, C. W., Watterson, E., Garcia, R., Mazur, G. J., Brackney, R. J., & Sanabria, F. (2015). Revisiting the effect of nicotine on interval timing. *Behavioural Brain Research*, *283*, 238-250.
Fang, Y. (2011). Asymptotic equivalence between cross-validations and Akaike Information Criteria in mixed-effects models. *Journal of Data Science, 9*, 15-21.
Fetterman, J. G., & Killeen, P. R. (1991). Adjusting the pacemaker. *Learning and Motivation*, *22*, 226-252.
Fetterman, J. G., & Killeen, P. R. (1995). Categorical scaling of time: implications for clock-counter models. *Journal of Experimental Psychology: Animal Behavior Processes, 21*, 43.
Freestone, D. M., Balcı, F., Simen, P., & Church, R. M. (2015). Optimal response rates in humans and rats. *Journal of Experimental Psychology: Animal Learning and Cognition*, *41*, 39-52.
Galtress, T., & Kirkpatrick, K. (2009). Reward value effects on timing in the peak procedure. *Learning and Motivation*, *40*, 109-131.
Gibbon, J. (1992). Ubiquity of scalar timing with a Poisson clock. *Journal of Mathematical Psychology*, *36*, 283-293.
Gibbon, J. (1977). Scalar expectancy theory and Weber's law in animal timing.*Psychological Review*, *84*, 279.
Greenberg, G. (1995). Anangenetic Theory in Comparative Psychology. *International Journal of Comparative Psychology*, *8*, 31-41.
Guilhardi, P., & Church, R. M. (2005). Dynamics of temporal discrimination.*Learning & Behavior*, *33*, 399-416.
Henderson, J., Hurly, T. A., Bateson, M., & Healy, S. D. (2006). Timing in free-living rufous hummingbirds, *Selasphorus rufus*. *Current Biology*, *16*, 512-515.
Herrnstein, R. J. (1970). On the law of effect. *Journal of the Experimental Analysis of Behavior, 13*, 243-266.
Horne, P. J., & Lowe, C. F. (1993). Determinants of human performance on concurrent schedules. *Journal of the Experimental Analysis of Behavior, 59*, 29-60.
Higa, J. J., & Simm, L. A. (2004). Interval timing in Siamese fighting fish (*Betta splendens*). *Behavioural Processes*, *67*, 501-509.
Killeen, P. R., & Fetterman, J. G. (1988). A behavioral theory of timing. *Psychological Review*, *95*, 274.
Killeen, P. R., Hanson, S. J., & Osborne, S. R. (1978). Arousal: Its genesis and manifestation as response rate. *Psychological Review*, *85*, 571.
Kollins, S. H., Newland, M. C., & Critchfield, T. S. (1997). Human sensitivity to reinforcement in operant choice: How much do consequences matter?. *Psychonomic Bulletin & Review, 4*, 208-220.
Lejeune, H., & Wearden, J. H. (1991). The comparative psychology of fixed-interval responding: some quantitative analyses. *Learning and Motivation*, *22*, 84-111.
Lowe, C. F., Davey, G. C. L., & Harzem, P. (1974). Effects of reinforcement magnitude on interval and ratio schedules. *Journal of the Experimental Analysis of Behavior, 22*, 553-560.
Ludvig, E. A., Balci, F., & Spetch, M. L. (2011). Reward magnitude and timing in pigeons. *Behavioural Processes*, *86*, 359-363.
Ludvig, E. A., Conover, K., & Shizgal, P. (2007). The effects of reinforcer magnitude on timing in rats. *Journal of the Experimental Analysis of Behavior*, *87*, 201-218.
Madden, G. J., & Perone, M. (1999). Human sensitivity to concurrent schedules of reinforcement: effects of observing schedule-correlated stimuli. *Journal of the Experimental Analysis of Behavior, 71*, 303-318.
Maggi, S., Garbugino, L., Heise, I., Nieus, T., Balcı, F., Wells, S., Tocchini-Valentini, G.P., Mandillo, S., Nolan, P.M., & Tucci, V. (2014). A cross-laboratory investigation of timing endophenotypes in mouse behavior. *Timing & Time Perception*, 2, 35-50.
MacDonald, C. J., Cheng, R. K., & Meck, W. H. (2012). Acquisition of “Start” and “Stop” response thresholds in peak-interval timing is differentially sensitive to protein synthesis inhibition in the dorsal and ventral striatum. *Frontiers in Integrative Neuroscience, 6, 1-16*.

MacEwen, D., & Killeen, P. (1991). The effects of rate and amount of reinforcement on the speed of the pacemaker in pigeons' timing behavior.*Animal Learning & Behavior*, *19*, 164-170.
Machado, A. (1997). Learning the temporal dynamics of behavior.*Psychological Review*, *104*, 241-265.
Myung, I. J. (2003). Tutorial on maximum likelihood estimation. *Journal of Mathematical Psychology*, *47*, 90-100.
Nagarajan, S. S., Blake, D. T., Wright, B. A., Byl, N., & Merzenich, M. M. (1998). Practice-related improvements in somatosensory interval discrimination are temporally specific but generalize across skin location, hemisphere, and modality. *The Journal of Neuroscience, 18*, 1559-1570.
Rammsayer, T. H. (1994). Effects of practice and signal energy on duration discrimination of brief auditory intervals. *Perception & Psychophysics, 55*, 454-464.
Rattat, A. C., & Droit-Volet, S. (2012). What is the best and easiest method of preventing counting in different temporal tasks?. *Behavior Research Methods, 44*, 67-80.
Rice, N., Grace, R. C., & Kyonka, E. G. (2014). Pigeons learn signal-food intervals independently in a multiple peak procedure. *Journal of Experimental Psychology: Animal Learning and Cognition, 40*, 241-248.
Richards, J. B., Sabol, K. E., & Seiden, L. S. (1993). DRL interresponse-time distributions: Quantification by peak deviation analysis. *Journal of the Experimental Analysis of Behavior, 60*, 361-385.
Richelle, M., & Lejeune, H. (1984). Timing competence and timing performance: A cross-species approach. *Annals of the New York Academy of Sciences*, *423*, 254-268.
Sanabria, F., Thrailkill, E. A., & Killeen, P. R. (2009). Timing with opportunity cost: Concurrent schedules of reinforcement improve peak timing. *Learning & Behavior, 37*, 217-229.
Sanabria, F., & Killeen, P. R. (2008). Evidence for impulsivity in the Spontaneously Hypertensive Rat drawn from complementary response-withholding tasks. *Behavioral Brain Function*, *4*, 1-17.
Simen, P., Rivest, F., Ludvig, E. A., Balci, F., & Killeen, P. (2013). Timescale invariance in the pacemaker-accumulator family of timing models. *Timing & Time Perception*, *1*, 159-188.
Stone, M. (1977). An asymptotic equivalence of choice of model by cross-validation and Akaike's Criterion. *Journal of the Royal Statistical Society. Series B, 39*, 44-47.
Stubbs, D. A., & Pliskoff, S. S. (1969). Concurrent responding with fixed relative rate of reinforcement. *Journal of the Experimental Analysis of Behavior, 12*, 887-895.
Taylor, M. M., Crofton, K. M., & MacPhail, R. C. (2002). Schedule-controlled behavior in rats exposed perinatally to the PCB mixture Aroclor 1254. *Neurotoxicology and Teratology, 24*, 511-518.
Wearden, J. H., & Lejeune, H. (2008). Scalar properties in human timing: conformity and violations. *The Quarterly Journal of Experimental Psychology, 61*, 569-587.
Yin, B., Lusk, N. A., & Meck, W. H. (2015). Interval-timing protocols and their relevancy to the study of temporal cognition and neurobehavioral genetics. In V. Tucci (Ed.) *Neuro-phenome: Cuttingedge approaches and technologies in neurobehavioral genetics. Hoboken, New Jersey: Wiley-Blackwell*.

Appendix A

Eq. 1 describes a model that proposes LTSs are a mixture of two processes, a timing process and a non-timing process. Within Eq. 1 are other nested models that correspond to different hypotheses about the distribution underlying LTSs. For example, if q = 1, Eq. 1 reduces to a gamma distribution and corresponds to the hypothesis that all LTSs are sensitive to the passage of time. These nested models

were compared to Eq. 1 to determine whether the full complexity of Eq. 1 was necessary for each species.

Each nested model was fit to the data of each subject using the method of maximum likelihood estimation (MLE; Myung, 2003). It is important to note that, because each model was fit to each subject separately, between-subject variability is built into these fits and informs the MLE. The MLE of each model was used to compute the corrected Akaike Information Criterion (AICc; Burnham & Anderson, 2002). AICc is a model selection criterion that favors nested models that balance goodness-of-fit (higher MLE) against parsimony (fewer free parameters); lower AICc values are indicative of better balance. Given the AICc of each model, ΔAICc was calculated for each model i as the difference between the AICc of model i and the lowest AICc among all models. The model with the fewest free parameters among those with ΔAICc < 4 was selected as the model providing the best balance between fit and parsimony.

Following validation of Eq. 1, a model-comparison approach was implemented (for similar implementations see Avila et al., 2009 and Daniels et al., 2015) to isolate the mechanism by which increasing the magnitude of reinforcement alters timing performance. Eq. 1 was fit to data of each species from the Baseline and Bias conditions to determine the potential pattern of effects. For example, this might suggest a model in which $c_{baseline} > c_{bias}$, $\theta_{baseline} < \theta_{bias}$, $_{qbaseline} > q_{bias}$, $K_{baseline} < K_{bias}$. Within this model are nested models such as $c_{baseline} > c_{bias}$, $\theta_{baseline} < \theta_{bias}$, $q_{baseline} > q_{bias}$, $K_{baseline} = K_{bias}$, and $c_{baseline} = c_{bias}$, $\theta_{baseline} = \theta_{bias}$, $q_{baseline} = q_{bias}$, and $K_{baseline} = K_{bias}$; the latter corresponds to the null hypothesis. To determine whether the full complexity of the model for each species was necessary, we asked which combination of these parameters needed to vary between the Baseline and Bias conditions. For each species, this yielded 16 competing models. ΔAICc was used to select among these models.

Appendix B

Model Selection Outcomes

Table B1

Selection among models of timed and non-timed LTSs under baseline conditions

	Model	Free Parameters	MLE	AICc	ΔAICc
Rats	q, θ, c, K	64	-19844.4	39817.9	<u>0</u>
n = 16	q = 1, θ, c	32	-22261.4	44587.03	4769.1
	q = 0, K	16	-22917.9	45867.9	6049.9
Pigeons	q, θ, c, K	48	-4097.5	8291.6	<u>0</u>
n = 12	q = 1, θ, c	24	-4443.19	8934.6	642.9
	q = 0, K	12	-6172.9	12369.9	4078.4
Humans	q, θ, c, K	44	-1113.1	2320.1	<u>0</u>
n = 11	q = 1, θ, c	22	-1354.2	2753.8	433.8
	q = 0, K	11	-2017.5	4057.3	1737.2

Note. Tested models are labeled with the free parameters that were allowed to vary. The number of free parameters for each tested model is the number of free parameters allowed to vary multiplied by the number of subjects, n. Each model was fit to 6975 data points for rats, 1728 for pigeons, and 726 for humans under baseline conditions. All data are provided to calculate ΔAICc. The selected model is underlined.

Table B2

Selection among models of biased timing

	Rats		Pigeons		Humans	
Full Model	$c_{baseline} > c_{bias}$, $\theta_{baseline} > \theta_{bias}$, $q_{baseline} > q_{bias}$, $K_{baseline} > K_{bias}$		$c_{baseline} > c_{bias}$, $\theta_{baseline} > \theta_{bias}$, $q_{baseline} > q_{bias}$, $K_{baseline} > K_{bias}$		$c_{baseline} > c_{bias}$, $\theta_{baseline} < \theta_{bias}$, $q_{baseline} < q_{bias}$, $K_{baseline} < K_{bias}$	
Nested Models	MLE	ΔAICc	MLE	ΔAICc	MLE	ΔAICc
Null	-36642.91	850.61	-8603.89	513.07	-2139.75	35.29
q	-36408.17	413.48	-8528.37	386.79	-2121.89	23.18
θ	-36340.71	278.57	-8410.46	150.99	-2112.63	4.66
c	-36635.37	867.89	-8581.94	493.94	-2120.82	21.06
K	-36528.26	653.68	-8578.36	486.77	-2136.07	51.54
q,θ	-36207.01	43.62	-8340.24	35.49	-2100.15	<u>3.72</u>
q,c	-36389.05	407.69	-8511.55	378.11	-2113.85	31.11
q,K	-36380.76	391.12	-8515.64	386.30	-2121.19	45.78
θ,c	-36324.64	278.87	-8382.07	119.16	-2098.3	<u>0</u>
θ,K	-36267.37	164.34	-8388.72	132.46	-2109.25	21.91
K,c	-36418.27	466.13	-8557.53	470.08	-2116.79	36.98
q,θ,c	-36172.59	7.29	-8309.93	<u>0</u>	-2091.85	11.50
q,c,K	-36365.34	392.79	-8493.94	368.03	-2113.51	54.82
θ,c,K	-36253.25	168.63	-8361.65	103.44	-2094.49	16.78
q,θ,K	-36168.94	<u>0</u>	-8328.09	36.33	-2101.84	31.49
q,θ,c,K	-36154.21	3.14	-8298.68	2.81	-2089.59	31.77

Note. Details are found in Table 1.

Financial conflict of interest: This research was supported by the National Institutes of Health (MH094562) and a seed grant from the College of Liberal Arts and Sciences, Arizona State University.
Conflict of interest: No stated conflicts.

Submitted: *May 2nd, 2015*
Resubmitted: *July 17th, 2015*
Accepted: *July 29th, 2015*