Number and Time in Acquisition, Extinction and Recovery

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Abstract

We measured rate of acquisition, trials to extinction, cumulative responses in extinction, and the spontaneous recovery of anticipatory hopper poking, in a Pavlovian protocol with mouse subjects. We varied by factors of 4 number of sessions, trials per session, intersession interval, and span of training (number of days over which training extended). We find that different variables affect each measure: Rate of acquisition [1/(trials to acquisition)] is faster when there are fewer trials per session. Trials to extinction and amount of responding during extinction are unaffected by these variables. The number of training trials has no effect on recovery in a 4-trial probe session 21 days after extinction. However, recovery is greater when the span of training is greater. We discuss the theoretical and clinical implications of these and related findings.

Most accounts of spontaneous recovery view the time after extinction as a period during which changes in the strengths of excitatory and inhibitory associations occur. McConnell and Miller (2014) point out that the treatment of extinction in major associative theories of learning fall into two categories, those that posit the degradation of the excitatory association and those that posit the development of inhibitory associations. Perhaps the most common assumption regarding spontaneous recovery is that the inhibitory associations that develop during extinction fade faster than the excitatory associations (Hull, 1943; Pavlov, 1927; Rescorla, 1979, 1993a; Wagner, 1981).

Other theories focus not on the postulated effects of the passage of time on associations but rather on its effect on the behavioral expression of the associations. For example, with time after extinction the activation threshold for the extinction memory rises making it harder to retrieve (Kraemer & Spear, 1993), or the temporal context of extinction changes, which in turn results in the renewal of the acquisition memory at the time of testing (M. E. Bouton, 1991; M.E. Bouton, 1993), or the conditioned and unconditioned CS elements are redistributed, so that the unconditioned elements from extinction are outnumbered by the conditioned elements formed from the lengthier acquisition training (Estes, 1955b; Estes & Burke, 1953).

These explanations for recovery have two things in common. First, time and number are treated as the *media* for alterations in associative processes, not as crucial parts of the *content* of what is learned (Savastano & Miller, 1998). In these theories, the subject does not remember the durations and numerosities—the intertrial intervals, the intersession intervals, the span of days within which training occurs, the numbers of trials in a session, the numbers of reinforced trials in a session, the numbers of sessions, etc. Second, the effects of time and number are assumed to be mediated by their effects on processes of association formation and expression. Crucially, associations do not encode experiential facts (trials durations, session durations, numbers of trials, numbers of reinforced trials, etc.). Associations, both historically in philosophy and psychology, and currently in their neuroscientific interpretation, are conductive pathways whose strength (aka conductance) varies depending on the temporal pairing of events. Because associative and synaptic plasticity accounts of associative learning do not assume that associations encode facts, they do not specify a code (C.R. Gallistel, 2017).

Experimental work over the last several decades has led to two conclusions that are at variance with these assumptions: In associative conditioning, time and number are *messages*, not *media*. The numerosities of the events in the protocol and the intervals between the events are among the contents of memory.

*Time in conditioning.* Subjects in associative learning experiments make a temporal map of the conditioning experience (P.D. Balsam & Gallistel, 2009; Honig, 1981; Taylora, Joseph, Zhaoc, & Balsam, 2014). The temporal information in the map affects every aspect of Pavlovian and operantly conditioned behavior (Arcediano & Miller, 2002; Barnet, Grahame, & Miller, 1993; Barnet & Miller, 1996; Blaisdell, Denniston, & Miller, 1998; Burger, Denniston, & Miller, 2001; Cole, Barnet, & Miller, 1995; Cunningham & Shahan, 2018; J. C. Denniston, Blaisdell, & Miller, 2004; J.C. Denniston, Blaidsdell, & Miller, 1998; C. R. Gallistel, Craig, A., Shahan, T.A., 2019; Shahan & Cunningham, 2015; Theunissen & Miller, 1995).

That subjects learn the distribution of wait times in conditioning experiments is revealed by the fact that their behavior during the conditioned stimulus (CS for short) varies depending on whether the delay of reinforcement during the CS is fixed or exponential. When it is fixed, the onset of responding is abrupt on any given trial. The distribution of onsets is centered approximately half way through a trial, regardless of CS duration, that is, the mean of the onset distribution scales with CS duration and the right edge is close to the anticipated reinforcement time (Church, Meck, & Gibbon, 1994; C.R. Gallistel, King, & McDonald, 2004; Gibbon, 1977). Moreover, in the peak procedure, where some fraction of the trials are not reinforced (and the CS continues for 3 or 4 times its duration on reinforced trials), subjects stop abruptly soon after the anticipated time of reinforcement has passed. The distribution of the stops is almost perfectly normal, and it is narrow (coefficient of variation is approximately 0.16, Balci et al., 2009; Balci et al., 2011; Church et al., 1994; C.R. Gallistel et al., 2004).

Subjects do computations with the wait times they have learned: We know this because the rate of learning [1/(trials to acquisition)] is a scalar function of the ratio of the basal average wait time for reinforcement in the training context and the average wait time for reinforcement following the onset of the conditioned stimulus (CS for short). The larger this ratio, the sooner the subject develops anticipatory behavior in response to CS onset (C.R. Gallistel & Gibbon, 2000; Gibbon & Balsam, 1981).

*Numerosity in conditioning.* There is also an extensive literature showing that the subjects in conditioning experiments learn the numerosities (Anobile, Cicchini, & Burr, 2015; Davison & Cowie, 2019; C. Gallistel & Gelman, 1990; C.R. Gallistel, 1990; Geary, Berch, & Koepke, 2015; Kutter et al., 2018). Strong evidence that subjects compute with the numerosities they have learned comes from studies of the partial reinforcement extinction effect. The probability of reinforcement during training has a dramatic effect on trials to extinction. If *on average* only 1 in *n* trials is reinforced during training, it takes *nk* trials to reach any given extinction criterion, where *k* is a constant that depends on the criterion (Chan & Harris, 2019; Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980).

The probability of reinforcement is the ratio of the number of reinforced trials to the total number of trials. Trials are widely spaced episodes (Crystal & Smith, 2014). When, on average, only 1 in 10 trials is reinforced, reinforced trials are even more widely spaced. This ratio of two numerosities is a crucial variable in those extinction protocols where there are experientially defined trials (episodes). This fact has posed a so-far unsolved problem for associative theories of associative learning for more than half a century (C.R. Gallistel, 2012).

*Content-based theories of learning.* The discovery of the rich mnemonic contents produced by conditioning protocols and the complex computations performed on those contents has stimulated the development of non-associative content-based theories of learning (C. R. Gallistel, Craig, A., Shahan, T.A., 2019; C. R. Gallistel & Wilkes, 2016; C.R. Gallistel, 1990, 2012; C.R. Gallistel & Gibbon, 2000; Gibbon, 1977; Wilkes & Gallistel, 2017). In these theories, learning has two components, the second of which presupposes the first. First, there is the encoding into memory of the sensory properties (e.g., texture and color) and nonsensory properties (e.g., duration and numerosity) of hierarchically structured episodes (C.R. Gallistel, 2017). Second comes the computation of stochastic models (C. R. Gallistel & Wilkes, 2016) based on these raw data. The stochastic models, which are themselves stored in memory, have two functions: 1) They enable more efficient coding of the data on which they are based. 2) They enable the predictions underlying the anticipation of future episodes.

Much of the second component—the computation of stochastic models—occurs off line. Consolidation and reconsolidation phenomena are plausibly considered manifestations of this second component, because stochastic model development leads to recoding memories so as to reduce the amount of memory required to preserve the same data (Dudai, 2012; Wang & Morris, 2010). The computation of stochastic models may also be the computational explanation for replay of episodes during sleep and quiet wakefulness (Foster & Wilson, 2006; Jafarpour, Fuentemilla, Horner, Penny, & Duze, 2014; Mattar & Daw, 2018; Ólafsdóttir, Bush, & Barry, 2018; Danielle Panoz-Brown et al., 2018; Zentall, 2019)

As suggested by the preceding brief and very incomplete review, most of the literature that has shown that subjects in conditioning experiments learn durations and numerosities has focused on the effects of trial parameters: trial duration, intertrial interval, number of trials and number of reinforced trials. In our experiments, we looked for effects at higher levels of the hierarchically structured episodes that conditioning protocols present to subjects. Trials are episodes, embedded within which are events such as the onset and offset of the CS and the reinforcements. The session is an episode, embedded within which are the trials. The days over which training occurs constitute a very lengthy “episode” (one might prefer the term *epoch*), embedded within which are the sessions. In these experiments, we ask whether the numerical and durational parameters of these higher-level chunks of experience affect acquisition, extinction and recovery.

We examined the effect on the post-extinction recovery of conditioned nose-poking in the mouse of 3 numerical and temporal training variables: total number of training trials, number of training sessions, and the span in days over which these sessions occurred. The variations in these parameters of acquisition training are shown in Table 1. The intertrial intervals in all 4 Experiments were drawn from an exponential distribution with a mean of 180s, to which a 10s interval was added, so that there was no intertrial interval shorter than 10s. Thus, the average intertrial interval was 190s, and it did not vary between experimental groups.

As may be seen in Table 1, the number of trials in a session varied from as few as 2 to as many as 40. Because the average intertrial interval was the same for every group, but the number of trials in a session varied, session duration scaled with trials-per-session.

Reinforcement was the delivery of a food pellet at the termination of the 10s white noise CS. Our index of conditioned responding was the *elevation score*, the difference in the number of pokes during the 10s CS and the number during the 10s interval immediately preceding the onset of the CS.

Extinction occurred during a single session the day after the last session of the acquisition training. Our first measure of performance during extinction was the traditional trials to extinction; 5 CS presentations without a response, terminated the session after 5 additional trials, thereby yielding the trials to extinction measure. Our second measure was the cumulative elevation score during the extinction session. If, as sometimes happened, the mouse made more pokes during the 10s pre-CS intervals than during the CS intervals, this measure could be negative.

A pilot experiment showed negligible recovery of the elevation score in a 4-trial session on the 7th day post-extinction, but substantial recovery on a second 4-trial session on the 21st day post extinction. Therefore, each of the groups had a 4-trial recovery session with no reinforcement at a “short” post-extinction lapse and again at a “long” lapse. For the 7 groups in the first three experiments, the short probe for recovery was on Day 7 post extinction and the long probe on Day 21. For the groups in Experiment 4, the probe days were Day 3 and Day 18. The short probes always yielded negligible recovery, replicating the pilot result. Therefore, we focus our analysis on the results from the long probes, which always yielded significant recovery.

Table 1: *Training Parameters*

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Group | # Sessions | Trials/Session | Span (days) | Total Trials |
| Experiment 1 |  |  |  |  |
| Group 1.1 (*n*=6) | 7 | 40 | 7 | 280 |
| Group 1.2 (*n*=6) | 28 | 10 | 28 | 280 |
| Group 1.3 (*n*=6) | 7 | 40 | 28 | 280 |
| Experiment 2 |  |  |  |  |
| Group 2.1 (n=6) | 24 | 10 | 24 | 240 |
| Group 2.2 (n=6) | 6 | 40 | 6 | 240 |
| Experiment 3 |  |  |  |  |
| Group 3.1 (n=6) | 8 | 10 | 8 | 80 |
| Group 3.2 (n=6) | 8 | 40 | 8 | 320 |
| Experiment 4 |  |  |  |  |
| Group 4.1 (n=6) | 28 | 2 or 3, mean=2.5 | 28 | 70 |
| Group 4.2 (n=6) | 7 | 40 | 7 | 280 |

**Results**

*Acquisition*

Figure 1 plots the cumulative records (cumsum) of the trial-by-trial elevation scores (number of responses during the CS minus number during 10 s pre-CS interval) throughout the entire procedure (acquisition, extinction, and recovery) for the subjects in Experiment 4. The top 6 panels are for the subjects that received either 2 or 3 trials (on average 2.5 trials per session) for 28 daily sessions of acquisition training (Group 4.1 in Table 1). The bottom 6 panels are for the subjects that received 40 trials per session for 7 daily sessions during acquisition training (Group 4.2). Thus, there was a 4-fold difference in the number of training sessions and in the number of training trials, but in opposite directions; the group with the 28 training sessions had 4-fold fewer total training trials than the group with only 7 sessions.

The relatively few training trials for the top group explains why their cumulative records only attain an asymptotic cumulative difference of between 200 and 300 responses (y axis), and also why these cumulative records terminate at fewer than 150 trials (x axis). The trials from the extinction session are included in these records. Their inclusion explains why the records abruptly level off. The trials from the two recovery sessions are also included. They give rise to the small upturn at the end of each record.

By contrast, the cumulative elevation score for the lower group rise, in 5 out of 6 cases, to a cumulative difference above 1000 (and to above 600 for the 6th subject). They, too, level off abruptly during extinction. Because of the 4- or 5-fold differences in the y-axis scales, the small upturns at their conclusion, which are due to the two recovery sessions, are not perceptible in the bottom 6 plots. The imperceptibility of these recovery upturns foreshadows the fact, presented later, that a 4-fold increase in the number of training trials—from 70 or 80 total trials to 240 or 280—has no effect on the cumulative elevation score in the recovery sessions.



**Figure 1.** *Cumulative records of the elevation scores for subjects in Experiment 4 (cumulative elevation score versus cumulative number of trials). Thin vertical lines mark trial on which consistently elevated nose poking during the CS is estimated to have first appeared. Prior to that trial, the slope of the cumulative record is flat or negative. This occurs when pokes in the 10s interval prior to the CS were as frequent or more frequent than during the CS.*

The thin vertical lines in Figure 1 mark trials to acquisition, as estimated by a simple algorithm applied to the cumulative records (see Methods). The distributions of the loci of the vertical lines in the two sets of panels do not overlap; every subject in the top group acquired in fewer than 50 trials; every subject in the bottom group acquired after more than 50 trials. For this comparison, we have a 2-tailed *t*(10) = 5.16, with *p* <<.001 and a bi-directional Bayes Factor (BF) of almost 1,000:1 against the null hypothesis that the means of these two distributions do not differ. (For details of the BF calculation, see Methods.)

In sum, our first result is that the efficiency with which repeated trials promote the appearance of elevated responding during the CS is increased by having short sessions with very few trials. It takes more few-trial sessions to reach a given cumulate number of trials; therefore, elevated responding appears only after a longer span of training. This suggests that the span of training (number of days over which sessions are distributed) may itself be an important variable. From a content-theoretic perspective, this suggests that before a subject commits to making anticipatory responses during a CS, it waits to see that the predictive power of the CS is not here today and gone tomorrow. This would explain why trial efficiency is reduced in multi-trial sessions. In content-based theory of conditioning, the span of training could only affect behavior in a subject that kept in memory an ongoing count of the days since training began.

Figure 2 plots trials to acquisition as a function of the span of training. One sees that the green points in this scatter plot are outliers. They are for the upper group in Figure 1. If we exclude this group, there appears to be no effect on trials to acquisition of the other combinations of span and number of trials per session. The two large circles connected by a thin almost horizontal line are the means of all the points to the left of a span of 9 days and all the points to the right of a span of 23 days (excluding the green points). The difference between these two means does not approach statistical significance whether assessed by a 2-tailed t test or by a Bayes Factor. The other comparisons required to justify pooling the data that went into the circle for the left cluster and pooling the data that went into the right cluster also did not yield p values or BFs that approached conventional alpha levels (.05 for *p*’s, 3 for BFs). (See Methods for details of the statistical comparisons and their results.)



**Figure 2.** *Trials to acquisition for all 54 subjects broken down by the 4 possible combinations of few vs many trials per session and few vs many sessions.* *The data from the 6 subjects in the upper panels of Figure 1, the ones with 28 2- or 3-trial sessions, are the green cluster at lower right. The data for the other 6 subjects in that experiment are among those in the red cluster at left. The thin line with large circles at either end connects the mean of all the subjects with fewer than 9 daily sessions and the mean of all the subjects whose training spanned more than 23 days with 10- or 40-trial sessions (the blue data). The green points come from training that spanned 28 days, but they have been offset one day less in this plot to separate them from the set of blue points at 28 days.*

Besides the training of Group 4.1 (green points in Figure 2), the training of three other groups spanned more than 23 sessions: Group 1.2 had 28 daily 10-trial sessions. Group 2.1 had 24 daily 10-trial sessions. Group 1.3 had 7 40-trial sessions. The intervals between these Group 1.3’s sessions ranged between 1 and 7 days, with an average intersession interval of 4 days. Our intent in including this group was to determine whether a long span of training induced stronger recovery even when that span contained relatively few sessions. We adjusted the number of trials per session to equate total training trials. Our focus in designing these first three experiments was on recovery, not trials to acquisition. However, as already shown, our first result was that having many trials in a session reduces the efficiency of the trials in promoting the emergence of conditioned anticipatory responding during the CS. Moreover, it appears from our results that “many” means more than a very few (2 or 3), because, when it comes to trials to acquisition the groups with 28 10-trial sessions or 7 40-trial sessions fall in with all the groups for which the span of training was less than 9 days with daily sessions containing 10 to 40 trials. In the Discussion, we predict the training parameters that should produce acquisition in a very few sessions with a very few trials (widely spaced long sessions with a few widely spaced trials) and what one might expect the recovery results to be after these training conditions.

*Extinction and Recovery*

Figure 3 plots the results from the extinction and recovery phases of the experiment. The recovery 7 days post extinction was weak or non-existent in every group, replicating the pilot finding, so we consider only the recovery at 21 days.

In the top left panel of Figure 3, we see that the total number of training trials has a strong effect on the rate of responding at the end of conditioning. Rate of responding is a common measure of associative strength. However, we see in the top right panel that total training trials has no effect on the cumulative elevation score during an extinction session. And, we see in the middle left panel that it also has no effect on trials to extinction. Therefore, if rate of responding measures associative strength, then associative strength at the conclusion of training has no effect during extinction.

In the middle-right panel, we see that the span of days over which training occurs has no effect on the amount of responding observed during extinction. And, in the bottom left panel, we see that it also has no effect on trials to extinction.

Finally, in the bottom right panel, we see that the span of training has a strong effect on the cumulative responses during a 4-trial probe for recovery 21 days post extinction. The total number of training trials has no effect, because each cluster contains points from a group with a relatively few total training trials (70-80) and from groups with a great many total training trials (240-320). A comparison within each cluster of the data from the few total training trial group to the data from the groups with many total training trials does not yield p values or Bayes Factors that approach conventional levels of significance (see Methods).

**Discussion**

*Theoretical Implications*

We believe these results challenge the associative theories of conditioning known to us. The challenge arises from the fact that associative theories are not theories about the experiential content of memory, nor about computations conducted on that content. The mnemonic elements that associative learning theories are concerned with are the associations. Associations do not encode anything. The content of memory resides in the gnostic units, or stimulus traces, or microstimuli, or stimulus elements that enter into associative processes. Associative theories are not about those content containing elements. They are about the associations and the processes that strengthen them or weaken them. Their focus is, therefore, on the trials, the episodes within which occur the temporal pairing of events that engages the associative process.

Here is not the place to elaborate on those challenges nor on how they might be met. Here, we are concerned with what our results suggest for content-based theories of associative learning, theories in which the concept of an association plays no role.



**Figure 3.** *Results from the Extinction and Recovery phases.* *The thin lines connect the means of the clusters at the left and right of each panel. Also shown are the results from a 2-tailed t test comparing the two clusters and from a bidirection Bayes Factor computation.*

Content-based theories of learning take it for granted that the brain is a powerful computer, which, like all known powerful computers, has a random-access symbolic memory. This memory architecture solves the problem of variable binding, and it enables the unbounded composition of functions. Content-based theories also take it for granted that brains have at their disposal the basic computational operations, such as concatenation and the operations whose axiomatization establishes the system of arithmetic (Knopp, 1952).

We take our results and many others (Bratch et al., 2016; Crystal & Smith, 2014; D. Panoz-Brown et al., 2016; Danielle Panoz-Brown et al., 2018; Wilson, Mattell, & Crystal, 2015; Zhou, G. Hohmann, & Crystal1, 2012) to imply that this computational machinery allows brains to build hierarchically structured records of a subject’s conditioning experience. The levels of this hierarchy are dictated by the many different time spans at which structured experiences unfold—from point events (lighting flashes) to epochs (days, maybe months, maybe years when the CS predicted reinforcement, days, maybe…, when it did not).

*The construal of experience*. Complex, highly structured representations allow the same experiences to be *construed* in many different ways (many different representations of the same experience). These different construals of the training experience permit different policies. Minimalist construals allow only a few policies. A construal formulated entirely in terms of the trials in our protocols would not allow for session-level properties (trial spacing, number of trials per session, session duration, the probability of reinforcement) to affect the conditioned behavior. A construal formulated entirely at the session level would allow neither trial-level properties (e.g., trial duration, the distribution of CS-US intervals) nor span level properties (e.g., the duration of the span, the number of sessions within the span, the distribution of inter-span intervals) to affect the behavior.

Our results suggest that mice construe their conditioning experience at many different levels, and apply different policies to these different construals in the different phases of the experiment. The policy that determines how rapidly they respond during a CS takes the reinforced trials so far accumulated as one of its inputs. The policy that determines how many responses they will generate before not responding at all for 5 successive trials does not take reinforcements so far accumulated into account (our current result). When executing the policy that it adopts when confronted with evidence that the probability of a reinforced trials has declined, however, it does take into account the probability that a trial during the training epoch was reinforced (M.E. Bouton, Woods, & Todd, 2014; J. A. Harris, 2019; J. A. Harris, Kwok, & Gottlieb, 2019; J.A. Harris & Andrew, 2017) . On the other hand, the policy it adopts when confronted with context extinction—placement in a context where reinforcements were previously provided gratis, but where now no more reinforcements occur—must be based on elapsed time without reinforcement. The only possible definition of a (observable!) trial in context conditioning is the session. However, Mustaca et al (1991, Figure 4) show context conditioning extinguishing during a single session, followed by diminishing spontaneous recovery in subsequent sessions, each recovery followed by within-session re-extinction. Elapsed time without reinforcement must drive the within-session progress of the extinguishing of context conditioning.

Our results further suggest that when it comes to the policy they pursue post extinction, the span of the epoch during which they experienced sessions in which there were reinforced trials is an important input. Our intuition is that the longer the span of time in which the CS was observed to have predictive power, the longer and more vigorously subjects explore the possibility that its predictive power has returned. We also assume that this prior knowledge will lead the subject to rapidly resume anticipatory responding when given renewed evidence of its predictive power (Napier, Macrae, & Kehoe, 1992; Ricker & Bouton, 1996). So far as we know, there is little quantitative work on the effect of span on recovery. The experiments here reported were exploratory. To get some idea of what variables mattered and what did not, given the large parameter space, we had to have many groups. For practical reasons, they had to have small *n*’s. Thus, the data in hand do not support the development of a computational model of recovery. We have, for example, almost no data on the time course of recovery under our conditions.

*A prediction.* The current state of our knowledge does, however, suggest a prediction. The trials required before the appearance of a conditioned response in appetitive Pavlovian conditioning (and in eyeblink conditioning) generally number close to half a hundred and often much more (see Figure 2, for example). We know, however, that this number depends strongly on the ratio of the *average* intertrial interval to the *average* wait for reinforcement following CS onset (C.R. Gallistel & Gibbon, 2000; Gibbon & Balsam, 1981; Gottlieb, 2008). Once one has chosen an average duration for the CS, the longer one makes the average interval between trials (hence, the smaller the duty cycle), the more informative CS onset becomes. The more informative the onset of the CS, the fewer the trials required for the conditioned anticipatory response to appear (P. D. Balsam, Fairhurst, & Gallistel, 2006; Gottlieb, 2008; Ward, Gallistel, & Balsam, 2013; Ward et al., 2012).

Given our current results and those just cited, we assume that the policy that determines the appearance of consistent anticipatory responding in a Pavlovian delay conditioning protocol takes into account both the informativeness of the trials and the evidence that there is day-to-day stability in the predictive value of CS onset. In any given session, one or a very few reinforced trials establish for the subject that the predictive power of the CS remains in force. That is why adding more reinforced trials to the sessions does not affect the amount of responding seen in probes for recovery. By contrast, increasing total trials by adding more (few-trial) sessions does affect recovery. These tentative conclusions lead us to an interesting prediction: One should be able to get appetitive conditioned behavior in a Pavlovian delay protocol after a very few trials, provided one uses highly informative CSs (short with very long average intervals between them) and provided one has long average intersession intervals (mean of several days). Moreover, training that ceases soon after the conditioned response has appeared should produce strong responding during long-delayed post extinction recovery probes, despite the few total training trials.

*Clinical Implications*

If our conclusions hold up under further experimental tests, they suggest an explanation for the difficulty of permanently extinguishing maladaptive learned behavior. They suggest that the longer the underlying construal of the situation that elicits the behavior has lasted, the more difficult it will be for extinction experiences to persuade the brain that there is a vanishingly small probability of that construal becoming again worth entertaining at some point in the perhaps distant future. The only way to forestall this would be to repeat the extinction experience at least briefly at random intervals more or less indefinitely.

**Methods**

Subjects

The subjects were male C57Bl/6 mice obtained from Harlan (Indianapolis, IN). They were about 9-11 weeks old and weighed between 16.3 and 20.9 g when the experiments started. They were housed individually in plastic tubs, and maintained on a 12:12 hr photoperiod, with lights on at 22:00 hr. Behavioral testing occurred during the dark phase of the photoperiod. Water was available *ad lib* in both the home cage and the experimental chambers, while food was restricted to keep body weight at approximately 85% of free-feeding weight. Standard rodent chow was given at the end of each session. Mice remained on their deprivation schedule until the first spontaneous recovery test, after which they received unrestricted food until 4 days prior to the second test, when they returned on their deprivation schedule.

Apparatus

Experimental sessions took place in modular operant chambers (Med Associates, Georgia, VT, model # ENV307W) measuring 21.6 cm x 17.8 cm x 12.7 cm, housed in individual ventilated, sound-attenuating boxes. Each chamber was equipped with a pellet dispenser connected to a feeding station on the center of one side. The station was a cubic hopper, 24 mm on a side, equipped with an infrared (IR) beam that detected nose pokes and a 5-watt light that illuminated the hopper when turned on. Mounted on the opposite wall were a clicker generator (80 dB, 10 Hz), a white noise generator (80dB, flat 10-25,000 Hz), and a house light (28 V DC, 100 mA). At the end of the feeding latency (10 s) a 20-mg precision pellet (TestDiet, 5TUM 1811143) was delivered in the feeding station. The experiment was controlled by computer software (Med-PC IV, Med Associates) that also logged and time-stamped the events—the onsets and offsets of interruptions of the IR beams in the station, the onsets and offsets of white noise, and the delivery of food pellets. Event times were recorded with a resolution of 20 ms.

Procedure

Body weights were recorded right before the start of each session. The house light remained illuminated throughout the experiment.

*Acquisition.* Sessions started with an intertrial interval (ITI) drawn from an exponential distribution with a mean of 180 s. This ITI was followed by a fixed, unsignaled 10-s interval (pre-CS period), at the end of which a trial started. A 100-ms clicker signaled pellet delivery.

*Extinction*. The day after their last acquisition session all mice received a single extinction session. There were no pellets or clickers delivered at the end of the white noise. In every other aspect, the extinction session was identical to an acquisition session. After the first 20 trials, an extinction criterion was employed. A mouse should make no responses during the CS for five consecutive trials. The session ended five trials after this criterion was met.

*Spontaneous Recovery Tests*. All mice were tested for spontaneous recovery 1 and 3 weeks post extinction, except in Experiment 4, where they were tested at 3 and 18 days post extinction. Each test included four presentations of the white noise in the absence of a reward.

*Statistical Analyses*

*Estimating the trial at which consistent anticipatory responding begins*. This was done by a custom Matlab™ function:

function t =Acq(V,c)

% computes an estimate of the trial at which conditioned responding began

% V is the cumsum of poke elevation scores (# pokes during CS minus

% # pokes during immediately pre-CS interval of same duration). c is the

% decision criterion (amount by which cum rec must enduringly rise above

% minimum in order for algorithm to decide that subject acquired

M = min(V); % low point in cumulative record

Im = find(V<=M,1,'last');

t = Im + find(V(Im:end)>M+c,1); % trial on which rise from last minimum

% meets criterion

The criterion (c) was 20.

*Statistical comparisons.* We did two tests for each comparison, a two-tailed t test, using Matlab’s™ ttest2 function, followed by a custom BF2 function (a Bayesian alternative). The Matlab™ code for the BF computation is provided in a publicly accessible [repository](https://github.com/CRGallistel/TimeNumberAcqExtRecov). In the Bayesian test, the normalized likelihood function for the mean of the "control" group is the null prior. It represents the hypothesis that the mean of the "experimental" does not differ from the mean of the "control" group. The computation of a Bayes Factor requires a second prior distribution, called the alternative prior. This distribution represents the hypothesis that the mean of the "experimental" group may differ from that of the "control" group. It specifies the range of deviations that might reasonably be expected. In the Bayesian equivalent to the *two-tailed* t test, the alternative prior distribution spreads out beyond both tails of the null prior. The more widely it spreads, the more the resulting BF will favor the null hypothesis. Thus, there is always the question what a reasonable spread is. If the data are numerous (say, more than 20) on both sides of a comparison and the distributions do not overlap, there is no point in doing a statistical test. Regardless of what the test one uses, the probability that the two distributions have the same mean will be infinitesimal. Therefore, the fact that one thinks it appropriate to do a statistical test implies that the data on at least one side of a comparison are not numerous (e.g., *n*=6) and/or the distributions overlap. In the light of these considerations, we chose as the prior for our alternative to the null a flat distribution that extended to 2σ to either side of the mean of the null prior. The value for sigma was the estimate of the pooled standard deviations delivered by the two-tailed t test that preceded the BF computation. Figure 4 shows the graphs of the null prior, alternative prior and likelihood function produced by the BF2 command applied to three of the comparisons. The question which group was the “control” and which the "experimental" was moot in these experiments, but the results from BF2.m are the same regardless of which group is assigned which role (C.R. Gallistel, 2009)*.*

The raw data and the Matlab™ code that analyzed them are in a publicly accessible repository (<https://github.com/CRGallistel/TimeNumberAcqExtRecov>). Included in the *DataFiles* folder in the repository are files *LongTable.mat* and *LongTable.csv*, containing a long table with the subject-by-subject summary statistics that entered into the plots and statistical comparisons in this report. The Matlab™ code that generated the figures and the statistical comparisons, using data in that long table, is in the file *CRGcode.m* in the *MatlabCode* folder in that repository. The Matlab™ commands for the statistical comparisons and the results they produced may be accessed in the *LT.Properties.UserData* of the *LT* table variable in the *LongTable.mat* file.

**Figure 4** *The computation of the Bayes Factor asks which prior hypothesis better predicts the likelihood function. The prior hypotheses are represented by prior distributions, one for the null hypothesis that the two means do not differ (the null prior, solid light blue) and one for the alternative to the null (dashed dark blue). These prior distributions are plotted against the left (blue) probability density axis. The question is which hypothesis puts more probability under the likelihood function (red), which describes the likelihood of different possible values for the mean of the “experimental” group given the data from that group. The likelihood function is plotted against the right (red) likelihood axis. These three examples come the comparisons made in the course of this data analysis. The width of the alternative prior was determined by the results of the t test. The alternative prior was the mean of the null prior +/- 2σ, where σ is the pooled estimate of the standard deviation of the distributions. The t test assumes the two distributions have equal variance. The t test was 2-tailed, meaning that there was no prior hypothesis about the direction of the possible difference in the means. Notice that the Bayes alternative prior extends to either side of the mean of the null prior. The null prior is the normalized likelihood function given the data from the “control” experiment.*

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