EFFECTS OF THE NUMBER AND SPACING OF CONDITIONING SESSIONS ON SPONTANEOUS RECOVERY FROM EXTINCTION

by

EFSTATHIOS B. PAPACHRISTOS

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ABSTRACT OF THE DISSERTATION

Effects of the number and spacing of conditioning sessions on spontaneous recovery from extinction

By EFSTATHIOS B. PAPACHRISTOS

Dissertation Director:

Dr. Charles R. Gallistel

Although it has been more than 80 years since Pavlov first observed spontaneous recovery from extinction, the mechanisms underlying this phenomenon remain a mystery. The majority of the proposed models attribute spontaneous recovery to a time-induced change in extinction-related processes. Recent findings, however, point also to the importance of elapsed time since acquisition, not just since extinction. These findings suggest that various temporal parameters of the learning episode may be explicitly represented in order to guide future choice on whether an animal should invest to a signal that has produced more than it has failed. In the present experiments, we used a conditioned magazine approach in the mouse to investigate whether and how various temporal parameters of acquisition affected spontaneous recovery. We found that prolonging the duration of acquisition, either by distributing the same number of

acquisition trials across more sessions, or by spacing the same conditioning sessions more widely, augmented spontaneous recovery. Further investigation of the former effect revealed that the session is an important unit of learning experience, while the number of trials within a session is not a primary determinant for spontaneous recovery. Finally, we quantitatively characterized extinction at the level of the individual subject and found it to be abrupt. It took a few trials to appear but became complete almost immediately.

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Introduction

Extinction is one of the most intensively studied and theoretically important phenomena in the field of learning. The term refers to the progressive decline and ultimate cessation of responding to a well-trained conditioned stimulus (CS) that is repeatedly presented in the absence of the reward or the unconditioned stimulus (US). The nature of the process that is responsible for the reduction of the conditioned response (CR) has been a subject of intense speculation and experimentation. Perhaps the most intuitive rationale that extinction reverses (erases) the learning that occurred in training was early dismissed by Pavlov (1927) due to the observation of spontaneous recovery, that is, the reappearance of the extinguished response with the passage of time.

With the exception of some theorists that viewed spontaneous recovery as a procedural artifact that was unrelated to extinction (e.g., Skinner, 1950), most proposed accounts of extinction provided an explanation for this phenomenon, which they viewed as shedding some light on the mechanisms operating in extinction (Robbins, 1990). In this introduction, I will first discuss some broad classes of extinction models and their explanation of spontaneous recovery, following the classifications made by Robbins (1990) and Rescorla (2004a). Subsequently, I will briefly present some characteristics of spontaneous recovery that place constraints on various theoretical interpretations. Finally, I will present the question that motivated my dissertation research.

Theoretical Interpretations of Spontaneous Recovery

Associative Loss

Several prominent trial-based models (e.g., Mackintosh, 1975; Rescorla & Wagner, 1972) interpreted extinction as weakening previously established associations. If conditioned responding is guided by the strength of an associative connection between the representations of the CS and the US that grew during acquisition, extinction causes a decline in responding simply because the same underlying association is weakened by the repeated presentations of the CS without the US. The observation of spontaneous recovery poses a major challenge to this conceptualization. If extinction erased the original association, then responding could never return during testing. Thus, the mere recovery argues for some preservation of the original learning.

As Rescorla (1979; 2004a) has repeatedly noted, spontaneous recovery by itself does not provide evidence that there has been no removal of the original learning; only that at least some learning survived extinction. Therefore, the associative-loss models can still anticipate recovery if, for example, extinction is incomplete. As will be discussed later, local performance effects, such as temporary fatigue from the repeated production of the CR, or emotional responses like frustration (Amsel, 1958) caused by the omission of an expected reinforcer, can have disruptive effects on performance of the CR that are in the same direction as the effect of extinction, thereby expediting the response decrement (Rescorla, 2004a). The influence of these performance factors can be expected to diminish with time and when testing occurs in a subsequent session, the remaining strength of the partially extinguished association is free to cause the recovery of the CR.

Perhaps the most famous attempt to reconcile spontaneous recovery with an erasure account of extinction was made by Skinner (1950). He proposed that handling, transportational, and other cues present at the start of a training session can cause spontaneous recovery when they reappear at the beginning of a test session. In effect, the close temporal proximity of the early session CSs to these memorable cues makes them distinctively different than the CSs presented later in the session (Burstein, 1967). The incomplete extinction of these initial stimuli makes them partially effective in eliciting a CR at the beginning of the next session, when memory of these CSs is still recent. Thus, spontaneous recovery is simply due to incomplete extinction of the CSs that occur early in the session.

There are mainly two lines of evidence in support of Skinner's interpretation. Burstein and Moeser (1971) found that presenting a distinctive CS on the first trial of every session augmented spontaneous recovery of an instrumental response in pigeons. Thus, it seems that early-session CSs, which signal a subsequent series of reinforced trials, could support greater responding than later-session CSs. The second line of evidence comes from studies that investigated the role of handling and transportational cues in spontaneous recovery. In one such study, Welker and McAuley (1978) trained rats to lever-press using a variable-interval schedule of reinforcement. Subsequently, rats were given five sessions of extinction. During extinction, some rats experienced different handling and transportational cues than the cues they had received during initial training. When the initial cues were reinstated on a test day, these rats showed greater recovery compared to rats that had never experienced a change in those cues. Therefore, it seems

that local cues that have been associated more strongly with reinforcement can acquire control over and reinstate an extinguished CR.

However, subsequent studies showed that at least in the case of local cues, their contribution to spontaneous recovery is contingent on explicit discrimination training. Thomas and Sherman (1986), for example, obtained no evidence to support the idea that handling is necessary for spontaneous recovery. Pigeons that spent the entire 24-h interval between the end of extinction and the start of the test session inside the experimental chamber –and thus were never handled prior to testing-- showed equally strong recovery with pigeons that were handled in the same way as during all previous conditioning and extinction sessions. Moreover, in a different experiment in the same study the authors found no evidence to support the idea that handling cues that are associated only with reinforcement can augment spontaneous recovery. To avoid pairing the usual handling cues with extinction, they preceded the start of the extinction procedure with 10 min of reinforced responding. Thus, when extinction started, the memory of the handling cues was presumably too remote to become associated with extinction. Despite their presumably purely reward-predicting properties, these cues did not enhance the magnitude of recovery on the subsequent test session.

Similar difficulties for Skinner's interpretation have arisen recently from a study by Robbins (1990, Experiment 2). In autoshaping experiments pigeons showed spontaneous recovery to an extinguished keylight even when testing occurred in the middle of the session, after recovery to another excitatory keylight had disappeared completely. The time of testing (half way through the session) was chosen so that memory of the handling and transportational cues should have faded. Nevertheless, the

keylight was still able to elicit spontaneous recovery. Yet the greatest problem with Skinner's account, as already noted by others (Mackintosh, 1974; Rescorla, 2004a; Thomas & Sherman, 1986), is that it cannot explain the main feature of spontaneous recovery that its magnitude increases systematically with the passage of time after extinction.

Estes (1955b) offered a similar account of spontaneous recovery as being due to incomplete extinction, without resorting to the masking effects of performance factors or the distinctive nature of early-session cues. In his stimulus sampling theory he assumed that any stimulating situation (e.g., the presence of the CS) is represented by a random sample of stimulus elements drawn from a larger population of elements available to the organism. All the elements that are sampled on an experimental trial become fully conditioned if the CS is paired with the US. In a similarly all-or-none fashion, the sampled conditioned elements return to the unconditioned state if the CS is not followed by the US. Finally, the sampled elements are replaced over time by random sampling from the larger population. Based on these assumptions, spontaneous recovery can be explained as follows. Suppose that at the end of conditioning the whole population of elements is conditioned. The same will be true for the elements of the sample drawn when extinction starts. At the end of extinction, performance could be entirely lost because the sampled elements will have returned to the unconditioned state, although the pool of CS elements maintains a significant number of conditioned elements (assuming that the extinction duration is too small for a significant exchange of elements between the sample and the larger set to occur). As time passes after extinction, the probability that conditioned elements from the larger set will be sampled increases. Thus, at the time

of testing the sample consists of many more conditioned elements than unconditioned ones, and responding recovers.

The stimulus sampling theory correctly anticipates that the magnitude of spontaneous recovery is directly related to the amount of time between extinction and testing. Moreover, it predicts that spontaneous recovery should decline after extensive extinction training. However, the basic tenet of this theory, as well as all the associative-loss models, that extinction erases the original learning, has been difficult to confirm empirically. In fact, Rescorla (1996), using sensitivity to US devaluation to measure associative strength, found excellent preservation of the original excitatory association through extinction.

Generalization Decrement

Another class of models view extinction as a case of generalization decrement. These models were primarily developed to deal with the partial reinforcement extinction effect (Robbins, 1990). For example, Capaldi (1967), in his sequential analysis of learning, proposed that on a given trial the animal remembers whether the previous trial was rewarded or not. If the animal is trained with mixed trial types (rewarded and non-rewarded), it often experiences reward on trials that immediately follow a non-rewarded trial (e.g., NR sequence) and it eventually learns to respond to memory of no-reward. Therefore, during extinction the animal persists in responding in the absence of reward and response decrement occurs more slowly.

The central view in this model is that extinction occurs because of the dissimilarity of its conditions from the conditions of acquisition. Similarly, spontaneous

recovery occurs because of the ambiguous state of the context at the beginning of the testing session (Robbins, 1990). In the absence of any trials, the conditions of the context during testing are equally similar to the conditions of acquisition and extinction. After the first few trials of no reward, the conditions resemble those of extinction and the recovered responding dissipates fast. With a similar extension, these models predict that spontaneous recovery should not appear if testing occurs in the middle of the session, after repeated non-reinforced presentations of a previously trained excitor, because there should have been ample time for the animal to classify the context as a no-reward context. Yet, as already mentioned, the appearance of spontaneous recovery is not constrained to the beginning of the test session (Robbins, 1990, Experiment 2).

Interference

Spontaneous recovery attests to the animal's retention (at least partially) of the original acquisition memory. The dynamics of spontaneous recovery –it only lasts for the first few trials and dissipates very fast—provide evidence that what was learnt in extinction is also retained. It is the relative strength of these two competing memories that some accounts of spontaneous recovery have focused on.

Kraemer and Spear (1993), for example, proposed that acquisition and extinction are each represented by a distinct memory. For either memory to be retrieved and subsequently to guide behavior, it has to be activated above its threshold. With the passage of time, the activation thresholds for both memories would ideally converge. However, because of the distinct affective component of each memory, the biologically more important one (acquisition memory) gains an advantage over the less salient one

(extinction memory), and its activation threshold drops below that of the extinction memory. The latter is harder to be retrieved, and spontaneous recovery merely attests to this retrieval failure of the extinction memory.

The above account views spontaneous recovery as a form of forgetting. Presumably, omission of the US during extinction is of less salient affective significance than its presentation during acquisition (but see Amsel, 1958, 1962). The latter is biologically more important for the animal and the easier retrievability of its memory reflects perhaps an adaptive mechanism by which the animal remembers better what is more important (Kraemer & Spear, 1993).

Bouton (1991; 1993) similarly suggested an interference model, which however attributes the weakening of extinction memories to their greater context dependence. What is learned during extinction is at least partially specific to the particular context. Thus, if testing takes place in a context other than the extinction context, the extinguished response is restored, a phenomenon called renewal (reviewed in Bouton, Westbrook, Corcoran, & Maren, 2006). There are several types of manipulations that can create this effect, the most common being ABA renewal –training in context A, extinguishing in context B, and testing in context A. Together with ABC and AAB renewal these forms indicate that the critical feature responsible for renewal is that testing occurs outside the extinction context.

The mechanism with which context modulates extinction performance is now thought to be similar to the mechanism operating during occasion setting (Bouton, 1991). That is, extinction leaves the CS with two meanings; under one (acquisition) it signals reinforcement, while under the other (extinction) not. During testing, the context helps

the animal disambiguate the current meaning of the CS. As long as the current context is different than the extinction context, the extinction memory is suppressed and the animal shows renewal.

To explain spontaneous recovery, Bouton (1993) suggested that apart from a physical change in context, as necessary for renewal, time can also create a functional change in context. Shortly after extinction the 'temporal' context (e.g., the animal's internal state) is still very familiar, and extinction memory dominates performance. When the delay between extinction and testing increases, so does the perceived change in the temporal context. Thus, retrievability of the extinction memory is reduced in favor of the acquisition memory, which causes spontaneous recovery. Therefore, spontaneous recovery is just another example of renewal (Bouton et al., 2006), with the change in context being due to the simple passage of time.

There is evidence to support the view that spontaneous recovery and renewal share the same mechanism. Introducing a retrieval cue for extinction during testing reduces both spontaneous recovery and renewal (reviewed in Bouton et al., 2006), because presumably the cue attenuates the perceived change from the extinction context and enhances retrieval of the extinction memory. However, there are instances in which recovery in conditioned responding is observed despite the similarity between the conditions of testing and extinction. Robbins (1990, Experiment 3), for example, trained and extinguished pigeons with a keylight conditioned stimulus, while also administering a second stimulus whose reinforcement outcome during any of the two phases was opposite to that of the CS. Thus, he used a reversal design (acquisition: A+, B-; extinction: A-, B+), keeping reinforcement density and session duration equated across

the two phases. During recovery testing, two days after extinction, he first gave reinforced presentations of the most recently reinforced stimulus (B+) until the point in the session where extinction criterion had been met. When he then presented the recently extinguished stimulus (A-) the pigeons showed robust recovery. Therefore, the fact that testing conditions resembled those of extinction rather than acquisition (due to the reinforced warm-up trials of the same stimulus, B, that was reinforced during extinction), did not prevent spontaneous recovery.

Interference models have also been challenged by observations of spontaneous recovery in spatial mapping tasks, which are thought to be resistant to interference (Devenport, 1998; Lattal, Mullen, & Abel, 2003). For example, Devenport (1998) exposed rats to two patches, one of which was baited, while the other was empty. Subsequently, the role of each patch (baited vs. empty) was reversed. For some rats the overall quality of the two patches (after the two stages of training) was largely different (e.g., Patch A > Patch B), while for the rest it was equal. Testing occurred at different delays and in the absence of any contextual cues that could cause a preferential retrieval of the memory from a particular stage of training. While shortly after the second stage of training all rats preferred almost exclusively the most recently baited patch, longer delays (48 h) caused the appearance of spontaneous recovery, namely an increase in choice for the patch that had been baited only during the first stage of training. However, the most impressive finding was that the magnitude of recovery depended on the average quality of the two patches. Rats for whom the two patches were of equal average value showed equal preference, while rats for whom A>B, showed an exclusive preference for Patch A. To calculate the average value of each patch, the animals needed to maintain and retrieve

memories from both stages of training. Thus, these findings are hard to accommodate under the main interference model assumption of selective memory retrieval.

Evidence that animals retrieve memories of reward probabilities from more than just the most recent phase has also been obtained in matching experiments. For example, Mazur (1995) trained pigeons to peck two response keys that delivered reward on a probabilistic basis under a variable-interval schedule. The probability of reinforcement under any key was constant for a set number of sessions before it changed to a different value. Although pigeons adjusted to the new reinforcement schedules within a session, they reverted to the pre-change schedules at the start of subsequent sessions. Based on the magnitude of these spontaneous recoveries, Mazur (1995) concluded that these results could be predicted only if the pigeons employed some sort of weighted averaging of the reinforcement schedules that were in effect during the past several sessions. Similar reversals to the response ratios (and reward ratios) of earlier sessions have been observed in matching experiments in the mouse (Gallistel et al., 2007b), and the rat (Gallistel, Mark, King, & Latham, 2001; Mark & Gallistel, 1994).

Associative Inhibition

S-S inhibition. Several formal and comprehensive theories of classical conditioning (e.g., Pearce & Hall, 1980; Wagner, 1981) offer a view of extinction as resulting in new learning. However, they are clear in specifying that the type of learning that results during extinction is inhibitory in nature. Influenced by Konorski's (1967) conceptualization of conditioned inhibition, Pearce and Hall's model suggests that extinction results in the formation of a new association between representations of CS

and no-US. Activation of the no-US representation inhibits that of the US representation and leads to a reduction in the strength of the conditioned response. Similarly, Wagner's "Sometimes Opponent Process" (SOP) model allows the CS to acquire inhibitory tendencies during extinction, which cancel out the existing excitatory tendencies from acquisition, and as extinction training continues, the net associative strength of the CS declines to zero. The important feature of those accounts is that the inhibitory association or tendencies that develop during extinction do not destroy the original excitatory association. In other words, contrary to the associative-loss models' view, extinction does not erase the original learning, but rather results in a new one, inhibitory in nature, that counteracts the effects of the former excitatory association on the CR. To explain spontaneous recovery, these models make the assumption that inhibition fades with time, so that during testing, the original excitatory association is left without competition to control responding.

There is some empirical support of the idea that the power of inhibition may diminish with time (e.g., Hendersen, 1978). However, the assumption that extinction establishes associative inhibition between the CS and the US is not well founded. In fact, there are more reports to refute it (Hendry, 1982; Reberg, 1972; Rescorla, 1969; Robbins, 1990) than to support it (Calton, Mitchell, & Schachtman, 1996). Robbins (1990, Experiment 4), for example, used a negative patterning procedure in which pigeons received the following types of trials: A+, B+, X+, A→X-, B→X-. This training endowed the three stimuli with excitatory properties when presented alone, while it also established A and B as inhibitors on the sequential compound trials with the target X. One of the inhibitors was then extinguished, and subsequently both of them were tested

for their effect on X. If extinction produced inhibitory links between the CS and US centers, then the extinguished stimulus should enhance its ability to suppress key-pecking elicited by X on compound trials. Instead, the results showed that the extinguished stimulus had lost both its excitatory (when presented alone) and inhibitory (on compound trials with X) properties. Moreover, after a 48-h delay, both properties recovered. Thus, not only did extinction not enhance a conditioned inhibitor's properties, but also the inhibition recovered, as opposed to faded, with the passage of time.

S-R inhibition. Recently, Rescorla (1993b; 1997), explored the hypothesis that the response decrement observed during extinction is caused by the development of an inhibitory association between the CS and the CR. In other words, during extinction the animal learns not to respond to the CS anymore. Evidence for this comes from experiments (Rescorla, 1997, Experiment 2) in which during extinction Rescorla manipulated the relative likelihood of two responses in the presence of a Pavlovian CS, and subsequently observed that the same CS evoked the response that had been least preferred (during extinction), although the CS had shared outcomes with both of the responses. The fact that the CS favored the production of one of the responses during extinction undermined its ability to evoke that particular response between two otherwise equivalent alternatives. Since the CS maintained its ability to evoke a response that was trained with the same outcome but was not extinguished in its presence, these results could be explained only in terms of an inhibitory S-R association. In support of this hypothesis, extinction can be enhanced by the concurrent presence of an excitatory CS (Rescorla, 2000), or conversely, a stimulus is protected from extinction when it is accompanied by an inhibitory CS (Rescorla, 2003).

Non-associative Inhibition

Finally, the last class of models attributes extinction to inhibition that develops on separate elements of the association. Despite the disagreement with regards to the locus of inhibition, all these models share the postulation that neither the original association is affected, nor a new association results from the extinction regime.

CS-based Inhibition. Pavlov (1927) was the first to suggest a non-associative account of extinction, according to which extinction resulted in the loss of CS processing (Robbins, 1990). Although he considered extinction a form of internal inhibition, he was clear that the center of inhibition is confined on the neuronal cells that represent the CS. In specific, he wrote "when a positive conditioned stimulus repeatedly remains unreinforced, it acquires inhibitory properties, i.e. the corresponding cortical cells enter under its influence into a state of inhibition" (Pavlov, 1927, p. 234). Moreover, at a later lecture in his book, he explicitly postulated that extinction did not interfere with the original association – "the inhibitory process arises in the nerve cells themselves and not in the connecting path between those cells excited by the conditioned stimulus and those excited by the special unconditioned stimulus employed" (Pavlov, 1927, p. 391). In Pavlov's view, repeated presentations of the CS in the absence of reward created an "exhaustion" of the neuronal population that represented the CS. He likened the inhibition that resulted from extinction to "a scattered sleep, sleep of separate groups of cellular structures [the ones excited by the CS]" (Pavlov, 1927, p. 253). Therefore, according to this view, spontaneous recovery occurs because with the passage of time and in the absence of any stimulation of the CS centers, the latter recover from their functional exhaustion.

Robbins (1990) equated Pavlov's view with the idea that extinction results in a loss of attention to the CS. In other words, extinction makes a CS an ineffective stimulus. In fact, this hypothesis accords with the results of his previously mentioned experiment in which he observed that extinction temporarily attenuated both excitatory and inhibitory properties of a stimulus, and that these properties recovered with the passage of time.

US-based Inhibition. Similarly to the previous account, Rescorla (1979; Rescorla & Cunningham, 1978) offered another non-associative view of extinction, one that emphasized its role in attenuating the processing of the US. According to this view, the CS always evokes the representation of the US, but the absence of the latter during extinction attenuates its representation. This reduced US representation can be restored with time, and so can the extinguished CS increase its ability to elicit responding, leading to the observation of spontaneous recovery. In support of this idea, Rescorla and Cunningham (1978) observed that spontaneous recovery to an extinguished CS was considerably diminished if testing had been preceded by non-reinforced presentations of another excitatory CS. The latter could erase the spontaneously recovered response only if it had been trained as an excitor. Therefore, since a neutral CS did not possess the same erasing properties, it seemed that the mechanism of action was that the non-reinforced presentations of the excitor depressed the US representation that had been recovered with time after extinction, leading to a fairly reduced observation of spontaneous recovery.

The reduced US-processing hypothesis fails to explain Robbins' (1990, Experiment 2) findings of robust spontaneous recovery to a stimulus tested in the middle of a session, when spontaneous recovery to another extinguished CS had dissipated completely. The latter stimulus was presented without reward, and therefore ought to

have suppressed the US representation. This in turn should have attenuated spontaneous recovery to the stimulus tested in the middle of the session, a prediction opposite to what was observed.

Response-based Inhibition. The third and final proposed locus of non-associative inhibition is the conditioned response itself. Hull's (1943) theory attributed part of the reduction in performance during extinction to a non-associative reactive inhibition. The latter term denotes a state of the organism which is produced by the conditioned response and is motivationally negatively charged, so that it inhibits the very same conditioned response that caused it. Although he drew a parallel between this state and the state of fatigue, Hull wrote that it should "be understood in the present context as denoting a decrement in action evocation potentiality, rather than an exhaustion of the energy available to the reacting organ" (Hull, 1943, p. 278). In other words, the tendency to perform the conditioned response declines progressively, as long as the CR is not followed by sufficient reinforcement. This build-up of reactive inhibition dissipates with time –the physical basis for this state, presumably a substance residing in the effector organ responsible for the response, is removed by the blood stream- and so does its negative influence on the effective reaction potential, the potential available for the conditioned response. Thus, spontaneous recovery is observed.

From Hull's proposition one can derive the prediction that the magnitude of spontaneous recovery should be negatively correlated to the effortfulness of the CR. There is evidence for (e.g., Mowrer & Jones, 1943) but also against (e.g., Mackintosh, 1974) this prediction. Moreover, Robbins' finding of robust spontaneous recovery in the middle of the session, after extensive warm-up trials with high rates of responding,

argues against a reactive inhibition (fatigue) account (Robbins, 1990). Finally, the mere assumption that the actual performance of a response is necessary for extinction has been challenged by the observation of "latent extinction" (Gleitman, Nachmias, & Neisser, 1954).

Temporal Weighting Rule

Devenport (1998) offered a different account of spontaneous recovery based on the Temporal Weighting Rule model (TWR) (Devenport & Devenport, 1993; Devenport & Devenport, 1994; Devenport, Hill, Wilson, & Ogden, 1997). According to TWR, an animal can increase foraging efficiency when it follows two strategies. It should stick with very recent information because it is more accurate –soon after a visit in a patch, the probability that the patch quality as a food resource has changed is low. However, the bigger the interval since the feeding episode, the higher the chances that the specific patch will be barren of food because, for example, during a long interval there are more opportunities for other foragers to drain its resources. Thus, in the absence of very recent information, the animal's best policy is to rely on the average quality of the patch, as calculated by considering all the instances of successful and unsuccessful foraging there (Devenport & Devenport, 1993).

Although the model was primarily developed as a foraging account, it has readily been applied to explain spontaneous recovery. In this case, based on its experiences with a stimulus, the animal obtains an estimate of the value of that stimulus as a predictor of reward (i.e., food). More recent experiences are weighted heavier, but their privileged weight is discounted in a hyperbolic fashion with the passage of time. Therefore, while

soon after extinction, the extinction experience carries a heavier weight and responding is still depressed, after a longer delay, the internal estimate regresses to the true (unweighted) mean, thereby causing the appearance of spontaneous recovery. Thus, according to TWR, spontaneous recovery merely reflects the animal's decision to invest to a CS, because on average it has produced more than it has failed.

In its mathematical formulation the model specifies that the value of a stimulus as a signal of reward is a weighted average (V_w) calculated as the sum of all experiences (Q_i) with the stimulus, each weighted by its recency (inverse of time, T, since that experience), divided by the sum of all recencies (to obtain the weighted average):

$$V_w = \sum_{i=1}^{n} (Q_i \times \frac{1}{T_i}) \Big/ \sum_{i=1}^{n} (\frac{1}{T_i}) . \quad (1)$$

To clarify how the model works, assume that an animal is trained on appetitive Pavlovian conditioning with constant reinforcement for five days. On the 6th day it receives an extinction session. If testing occurs next day, the weighted value of the CS as a predictor of food is calculated as follows: V_w = [1*(1/6) + 1*(1/5) + 1*(1/4) + 1*(1/3) + 1*(1/2) + 0*(1/1)] / [(1/6 + 1/5 + 1/4 + 1/3 + 1/2 + 1/1)] = 0.59. The heavier weighted extinction session has a negative impact on the weighted average and responding should be weak. If, alternatively, testing occurred one week after extinction, the weight of extinction would have been discounted so much that the weighted average (in this scenario, V_w = 0.78) would approximate the true average (= 0.83), and recovery should be observed.

Equation 1 requires that memories of both stages of training be simultaneously accessible at the time of testing. In other words, after a history of contradictory experiences with a stimulus, and in the absence of any retrieval cues, choice is not

determined randomly but by implementing computations on retrievable memories of all past experiences with that stimulus (Devenport et al., 1997). From this perspective, spontaneous recovery merely reflects an animal's current probabilistic estimate of the CS's reward-signaling properties.

Along with Estes' model, TWR specifies both the time course and the magnitude of spontaneous recovery (Devenport et al., 1997). However, because it is a relatively new model, its predictions on several aspects of spontaneous recovery have yet to be empirically tested. One such interesting prediction is that one should observe regression of responding if acquisition follows a period of no-reinforcement (Rescorla, 2004a). Note that according to TWR the relative advantage of recent experiences is not constrained by their nature (rewarding or not rewarding). Thus, as spontaneous recovery reflects a CS's value that is not dominated any more by the extinction experience (the second of two contradicting experiences), in the same way, regression should be observed when enough time has passed so that acquisition does not bare a significantly heavier weight than the first non-reinforced training regime. To put it differently, TWR anticipates that there should be "spontaneous recovery" of latent inhibition. In support of this prediction, Spear, Hill, and O'Sullivan (1965) observed a decline in runway speed at the first trial of each training day (after reward was introduced) in groups which, prior to this rewarded training, had received placement in the runway without any food. This regression towards poor performance was significantly worse than that observed in animals with no such prior non-reinforced history. Contrary to this result, however, there is evidence that the effect of latent inhibition in the conditioned emotional response can be released by inserting a longer delay between conditioning and testing (Kraemer, Randall, & Carbary,

1991). Additionally, Rescorla (2007) did not observe recovery to a stimulus with a history of reinforcement following an initial period of no reinforcement. Moreover, Rescorla (2005) failed to find evidence of spontaneous recovery of conditioned inhibition (but see Robbins, 1990, Experiment 4). If the order with which an organism experienced the two conflicting properties of the CS was irrelevant, then recovery of the first experienced property (in this case, conditioned inhibition) should have been observed. Thus, this result casts some more doubts to TWR's assumption that, other things being equal, the nature of the two contradicting experiences and the order with which they are obtained are two variables that do not interact in the production of spontaneous recovery.

The abundance of proposed theoretical accounts of spontaneous recovery reveals the complexity of this phenomenon. Although there is experimental evidence in support of virtually all the above ideas, none of them can give a complete account of the empirical properties of spontaneous recovery (Rescorla, 2004a). A brief discussion of some of these properties follows.

Behavioral Characteristics of Spontaneous Recovery

Spontaneous Recovery Increases with the Passage of Time. Perhaps the most striking feature of spontaneous recovery is that its magnitude increases with time. In specific, the strength of recovery is an inverse exponential function of the interval since extinction (Ellson, 1938; Quirk, 2002; Robbins, 1990). Although by means of different accounts, most of the proposed models can explain why the strength of spontaneous recovery is positively related to the resting interval between extinction and testing. Time since extinction increases the probability that conditioned elements from the larger available

population will be sampled (Estes, 1955b), raises the activation threshold for the extinction memory (Kraemer & Spear, 1993), changes the current temporal context from the extinction context (Bouton, 1991; 1993), or decreases the weight of extinction to essentially similar levels to the acquisition weights (Devenport, 1998). Similarly, the associative and non-associative inhibition accounts can explain this feature because they inherently assume that inhibition fades with time. Although they don't specify the time-course of the fading process, so many biological processes obey first order kinetics that this assumption is hardly surprising (Rescorla, 2004a). Perhaps the only account that fails to predict the time course of spontaneous recovery is Skinner's hypothesis. Indeed, without further assumptions, it is hard to imagine why the passage of time should enhance the excitatory properties of an inextinguished early-session CS.

Spontaneous Recovery is Incomplete and Short-lasting. It is widely accepted that spontaneous recovery is almost never complete and dissipates very rapidly (Rescorla, 2004a; Robbins, 1990). However, there is reason to suspect that spontaneous recovery may be complete in the first trial or two. Unfortunately, the vast majority of the published data show performance averaged across blocks of trials or even a whole session, making it hard to establish the exact course of spontaneous recovery. Nevertheless, the rapidity with which the CR declines reveals that there is a good maintenance of the extinction memory. In this aspect, models that emphasize the ambiguity with regard to the similarity between the conditions of testing and extinction as the main cause of spontaneous recovery (e.g., Bouton's view of spontaneous recovery due to temporal-context renewal)

seem to have an advantage. The first couple of non-rewarded trials during testing render the current conditions similar to extinction and responding declines rapidly.

Spontaneous Recovery Is Negatively Affected by the Acquisition-Extinction Interval. A recent finding about spontaneous recovery is that the acquisition-extinction interval inversely affects its magnitude (Rescorla, 2004b). Rescorla trained rats in a magazine approach procedure with two stimuli that differed only in the interval between the acquisition and extinction training periods. For one of the stimuli the two periods were separated by an interval of 8 days, while for the other that interval lasted only 1 day. Both stimuli were allowed the same 2-day resting period after extinction. Despite an identical extinction curve for both stimuli, the stimulus with the shorter interval between acquisition and extinction showed greater spontaneous recovery during testing. Thus, this result reveals that not only the extinction-testing interval but also the acquisition-extinction interval is an important temporal parameter that an account of spontaneous recovery should explain.

Similar findings that the interval between two antithetical learning episodes affects subsequent performance have been obtained in interference paradigms other than extinction. In verbal learning studies with humans, for example, Underwood and Freund (1968) observed that subjects that learned two lists in succession had a lower recall rate of the most recent list, than subjects for which the two learning episodes were separated by 3 d. In an animal study of counter-conditioning, Gordon and Spear (1973) trained rats first on passive avoidance and then on conflicting active avoidance. After a short retention interval, performance on the latter task was shown to be a positive function of

the intertask interval. Finally, similar findings have been obtained in matching (Mazur, 1996). In this study, pigeons first received several sessions with two keys delivering equal number of rewards. In the second phase, the proportion of rewards between the two keys shifted so that one key delivered the majority of the rewards. At the start of the second transition session pigeons exhibited spontaneous recovery, that is, they reverted to the pre-transition reinforcement schedule (50%). Interestingly, the magnitude of this recovery was decreased when a 3-day interval was inserted between the two phases.

The above finding proves troublesome for the majority of the discussed accounts of spontaneous recovery. In specific, none of the inhibition models have anything to say about this effect, because all of them assume that the inhibition, regardless of whether associative or non-associative in nature, fades with time and causes the appearance of spontaneous recovery. Time does not impact the acquisition memory, and so the interval between acquisition and extinction should be irrelevant for spontaneous recovery.

Interference models encounter similar difficulties accommodating this finding. For example, Bouton's model is based on the assumption that only the extinction memory is context dependent. Therefore, it is not immediately obvious how a temporal change of the acquisition context, induced by prolonging the acquisition-extinction interval, could hurt the chances of retrieval of the context-independent acquisition memory. Similarly, the Kraemer and Spear model claims that the asymmetrical affective nature of the two learning episodes causes an increase of the activation threshold of the less salient extinction memory over the retention interval. Again, there is no rule that governs any changes in the retrievability of the acquisition memory before extinction starts. Thus, the delay of the conflicting experience (extinction) has no place in this model. Indeed, the

observation of identical extinction of both stimuli by Rescorla (2004b) attests to a perfect retrieval of the acquisition memory at the start of extinction. Therefore, the Kraemer and Spear account, too, offers no explanation of this effect.

Estes' stimulus sampling theory cannot predict this finding either. There is no tenet in his theory that requires the conditioned elements in the larger population to spontaneously become unconditioned with the passage of time. That would amount to a time-induced memory loss during the 8-day interval between the end of conditioning and the onset of extinction, a claim that would immediately be dismissed. In the absence of such spontaneous unconditioning, it is not clear how a long acquisition-extinction interval should affect spontaneous recovery.

Devenport's TWR is the only model, to my opinion, that provides an explanation of why the training-test interval can affect spontaneous recovery. What is important in TWR is the relative distance (in time) of the two conflicting experiences (acquisition and extinction) from the time of test. Inserting an interval between acquisition and extinction makes extinction significantly more recent than acquisition. Its relative proximity endows it with a bigger weight (relative to the acquisition weights), which dominates the value of the stimulus as a predictor of reward. Thus, the more recent extinction memory inhibits the appearance of spontaneous recovery.

Spontaneous recovery seems to be a complex phenomenon that can be determined by many factors (Rescorla, 2004a). The wealth of theoretical explanations attests to this conclusion. There is evidence for and against each one of the proposed accounts. However, the majority of them fail to provide an efficient explanation of what causes

spontaneous recovery, resorting to arbitrary ad-hoc assumptions to accommodate the empirical findings. To my opinion, TWR is unique in this respect; it provides a thorough explanation of spontaneous recovery from the perspective of foraging theory, and it is given quantitative expression that allows specific predictions about the magnitude and the time course of spontaneous recovery. Moreover, it is currently the only model that anticipates the finding that apart from the extinction-test interval, the acquisition-extinction interval is also important.

Time as Part of the Content of Learning

The observation that time since acquisition is as important for spontaneous recovery as time since extinction indicates that time is part of the content of what is learned. This idea is captured only in TWR. By contrast, the vast majority of the models view time as the medium where changes in psychological processes related to learning occur but not an aspect of past experience that is explicitly represented. For example, with the passage of time the activation threshold for extinction rises (Kraemer & Spear, 1993), the extinction temporal context changes (Bouton, 1991, 1993), the inhibition that occurred in extinction fades (Hull, 1943; Pavlov, 1927; Rescorla, 1979, 1993a; Wagner, 1981), or the conditioned and unconditioned elements reach an equilibrium (Estes, 1955b; Estes & Burke, 1953). In none of these interpretations is time treated as part of the learned content.

The absence of time in the theorizing of spontaneous recovery is quite surprising. The idea that time plays an important role in Pavlovian conditioning has long been recognized. Successful conditioning is considered to depend on the temporal duration of

stimuli as well as their temporal relation (Pavlov, 1927). Nonetheless, time has had virtually no place within traditional theories of associative learning (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; but see Gibbon, 1977). For these theories, temporal factors play only a facilitative role in the formation of associations, but the temporal information itself is not encoded in the association (Savastano & Miller, 1998). This assumption has been challenged (Barnet, Arnold, & Miller, 1991; Barnet, Cole, & Miller, 1997; Cole, Barnet, & Miller, 1995; Matzel, Held, & Miller, 1988; Miller & Barnet, 1993) and new theories like the Temporal Coding Hypothesis have emerged, according to which the temporal relationship between the experienced events is represented as part of the association. Gallistel (1990; Gallistel & Gibbon, 2000), in his Rate Estimation Theory, has taken a more extreme position in which he abandons the construct of association altogether and relies only on time to explain learning.

Memory for the temporal characteristics of the learning event is not just constrained on intervals on the second to minute scale, the scale that applies in conditioning experiments. Instead, it has been shown to include intervals lasting several days (Clayton & Dickinson, 1998, 1999; Clayton, Yu, & Dickinson, 2003). For example, Clayton et al (2001) first taught scrub jays that crickets remain fresh after 1 d, but perish after 4 d, whereas peanuts are non-perishable. Subsequently, they allowed the birds to cache peanuts in one side of a tray, and crickets in the other. During testing, the jays searched the tray for food. (All the food items had been removed from the tray.) When testing was conducted 1 day after caching, the birds showed preferential search for the crickets, whereas, if the retention interval was 4 days, they preferred looking for food at the peanut side. Thus, not only did birds learn the rates of perishing, but also remembered

how long ago they had cached the food. Subsequent work by the same group showed that the birds could apply new information about the rate of perishing of food items that they had already cached, even when this information was acquired during the retention interval (Clayton et al., 2003). This indicated that the scrub jays had already encoded in their memory the time of the caching episode and combined this memory with the newly acquired information in order to guide their behavior appropriately.

As already mentioned, among the theories of spontaneous recovery, only TWR treats time as an important feature of the learning episode. What the animals need to remember, according to the model, are two aspects of each encounter with the CS: (a) its quality (food-signaling quality), and (b) how long ago it occurred. One question of theoretical importance that arises is what counts as an experience with the CS. In other words, do trials or sessions constitute the unit of experience that enters into the averaging process? Put it otherwise, is it time since each trial or each session that is retained in memory?

There is reason to believe that the session may be an important unit of experience. Changes in conditioned responding usually occur at the beginning of a new session (Gallistel et al., 2007a; Papachristos & Gallistel, 2006). Furthermore, distributing the same number of trials across more sessions speeds up acquisition, an effect known as the *trials-per-session* effect (Kehoe & Macrae, 1994; Levinthal, 1973; Papini & Dudley, 1993; Papini & Overmier, 1985), which indicates that the number of sessions is an important parameter of learning. Moreover, as long as the contingencies do not change within a session, it would be computationally advantageous for memory load purposes if the animal maintained in memory the time since each session, as opposed to each trial.

The question of whether trials or sessions are the important units of experience for spontaneous recovery can be addressed experimentally because TWR makes opposite predictions regarding these two possibilities. If at the time of testing the animal calculates the quality and time since each session, then the number of sessions becomes an important parameter of spontaneous recovery, while the number of trials within a session is irrelevant. Therefore, increasing the number of sessions, while keeping overall number of trials constant, should enhance spontaneous recovery from extinction.

Increasing the number of sessions results in an increase of the duration of acquisition. Such an increase may positively affect spontaneous recovery by a different mechanism than the one proposed by TWR. For example, Papachristos and Gallistel (2006) offered an interpretation of spontaneous recovery as being due to the animal's uncertainty about the current status of the CS-US relation. This predictive relation used to hold for an extended period of time (acquisition) before it failed (extinction). A single failure of a relatively stable process may be just a transient event. Among other factors, this decision of transitivity could depend on how long the animal observed this relation to hold in acquisition. Thus, under their hypothesis, any manipulation that prolongs the acquisition regime should positively affect spontaneous recovery, because it increases the evidence for a temporally stationary CS-US relation.

Not surprisingly, the extent to which such parameters of the initial acquisition training affect spontaneous recovery is unknown. The most pertinent experiments that have addressed this issue have done so under the auspice of the effects of "trial-spacing" manipulations on spontaneous recovery. In one such study, Homme (1956) trained rats on free-operant bar pressing. Several groups received a single conditioning session, differing

on the number of reinforcements (ranging from 15 to 250). Subsequently, all groups received five extinction sessions spaced 24 hr apart. He observed that the number of reinforcements (or the number of trials within the single training session) positively affected the strength of responding during the first 3 min of the first extinction session, a result hardly surprising. Unfortunately, a similar comparison on subsequent extinction sessions was not reported. Instead, the author compared the groups on their mean totals across all five extinction sessions, and although the number of reinforcements tended to positively affect the strength of responding, the effect was not statistically significant. In the absence of comparisons on the performance of these groups at the beginning of extinction sessions past the first one, no meaningful evaluation of the effect of number of trials on spontaneous recovery can be made.

In the same study, the evaluation of the trials-per-session effect on spontaneous recovery was similarly unsuccessful. A group that received 250 trials distributed over 5 sessions of 50 trials each responded more during the first extinction session than its control group that received the same number of trials in a single session. But again, no statistics on initial performance of these groups on subsequent extinction sessions was made. Thus, the question of whether spreading out acquisition trials across more sessions affects spontaneous recovery remains unresolved.

This dissertation was designed to explore these issues. Its goal was to investigate the effect of various temporal parameters of acquisition, such as the number of sessions, the spacing of sessions, and the number of trials within a session, on spontaneous recovery. Specifically, Experiment 1 asked whether an increase of acquisition duration would affect the magnitude of spontaneous recovery. The duration of acquisition was

increased in two ways: (a) by distributing the same number of trials across more sessions and (b) by spacing the same number of sessions more widely. Experiment 2 was a partial replication of Experiment 1 and aimed at establishing the effect of number of sessions during the first spontaneous recovery test after 1 week. Experiment 3 used a within-subjects design to establish which of the two manipulations had the strongest effect on spontaneous recovery. Experiment 4 evaluated whether the number of trials within an acquisition session affects spontaneous recovery. Experiment 5 provided a more direct test of whether the number of trials or the number of sessions is a more relevant parameter for spontaneous recovery.

A second goal of this dissertation was to characterize extinction quantitatively at the level of the individual subject, as well as investigate any effects of the above-mentioned acquisition manipulations on the rate of extinction. There is evidence that extinction occurs at a slower rate than acquisition (Rescorla, 2002) and an inspection of published extinction curves reveals that, like acquisition curves, they too are gradual (Amsel, 1962; Bouton & Garcia-Gutierrez, 2006; Cain, Blouin, & Barad, 2003; Corcoran & Maren, 2001; Kamprath & Wotjak, 2004; Lewis, 1956; Milad & Quirk, 2002; Moody, Sunsay, & Bouton, 2006; Quirk, Garcia, & Gonzalez-Lima, 2006; Rescorla, 2002, 2004a; Rescorla & Cunningham, 1978). The gradual increase of responding in acquisition curves seems to be an artifact of averaging across blocks of trials and subjects, because the individual curves entail a strikingly abrupt rise after a delay that varies greatly across subjects (Gallistel, Fairhurst, & Balsam, 2004; Papachristos & Gallistel, 2006). Thus, the seemingly smooth decrease in responding on the group-average extinction curve may not appropriately indicate how extinction proceeds at the individual subject. To my

knowledge, this is the first attempt for a quantitative analysis of the individual subject's extinction curve.

Experiment 1

This experiment investigated the relation between acquisition duration and spontaneous recovery. We manipulated duration in two ways: while keeping the overall number of conditioning trials constant, we either increased the number of sessions or increased the spacing of those sessions. Thus, we trained three groups of mice on a Pavlovian magazine approach procedure in which a 10-s white noise signaled food. The control group received 7 daily sessions (that is, one session per day for seven days) of 40 trials each. Another group received the exact same 7 sessions spaced on average 4 days apart. The third group received 28 daily sessions of 10 trials each. Thus, all groups were equated for total trial number (280 trials) and context exposure (they received identical ITI sequences). The day after the last acquisition session, all mice went through a single extinction session. Finally, all mice were tested for spontaneous recovery 1 and 3 weeks later.

The only two models that make specific predictions regarding the two manipulations are Estes' statistical theory (Estes, 1955b) and Devenport's TWR (Devenport, 1998). Estes' model predicts that both manipulations should increase the magnitude of spontaneous recovery because they both result in a wider distribution of conditioning trials. By inserting longer intervals between trials, more elements from the larger population have the opportunity to be sampled and become conditioned. Thus, at the time of testing, the proportion of conditioned elements in the sample will be greater for the two experimental groups with the prolonged acquisition duration compared to the control group.

TWR, on the other hand, makes a different prediction. Session spacing should, if anything, impair spontaneous recovery because it brings early acquisition experiences even farther away from the time of testing, thus diminishing their influence on the weighted average. Regarding the session number manipulation, TWR's predictions depend on whether trials or sessions constitute the unit of "experience" with the CS. Note that according to TWR the animal maintains in memory the time since all instances of experience with the CS. The question that arises is whether trials or sessions constitute the units of experience with the CS that enter into the averaging process. If at the time of testing the animal calculates the time since each session, then increasing the number of sessions should enhance spontaneous recovery. (By administering more conditioning sessions, the sum of their weights (recencies of those sessions) is added to both the numerator and denominator of Equation 1. Adding the same positive number to both terms of a cluster that is less than 1 brings the value of the cluster closer to 1. Therefore, increasing the number of sessions should augment spontaneous recovery from extinction.) The opposite is true if it is the time since each trial that enters into the computation. The rationale is similar to that applying to the session spacing manipulation; distributing the trials across more sessions moves many of them to a greater distance from the time of the test, thus diminishing their positive influence on the weighted average.

Method

Subjects. The subjects were 18 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 experiment 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. They were located 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.

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 trial consisted of 10-s presentation of white noise, which ended with the delivery of a pellet in the feeding station. A 100-ms clicker also signaled pellet delivery. The group given many daily trial-poor sessions received 28 sessions of 10 trials each, while the two groups given only a few trial-rich sessions received seven sessions of 40 trials each. One of the two latter groups got their few trial-rich sessions on successive days, while the other received a trial-rich session every 4 days on average (range 1-7 d). The house light remained illuminated throughout the experiment.

In this design, the total temporal extent of training was the same (28 days) for the group that got many trial-poor daily sessions and the group that got a few widely spaced trial-rich sessions. The interval between sessions was the same (1 day) for the group that got many trial-poor daily sessions and the group that got a few trial-rich daily sessions. All three groups got the same number of trials. And, all three groups got the same total amount of context exposure, because session length covaried with the number of trials in a session. For the group that got a few daily trial-rich sessions, all of its acquisition trials were concentrated relatively close to the beginning of extinction and, hence, to the beginning of the tests for spontaneous recovery. For the other two groups, a majority of all the trials occurred further from extinction, hence, also from the tests for spontaneous recovery.

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 response 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. Each test included four presentations of the white noise in the absence of any reward.

Analysis. Our index of conditioned responding was the elevation score, that is, the difference in the number of pokes during the CS and a 10-s period (pre-CS) immediately preceding the onset of the CS.

In our quantitative analysis of the extinction curve, we sought to determine mainly two parameters: (i) how fast extinction appeared, and (ii) how fast it became complete. Given the use of an extinction criterion in our experiments, the third parameter of the asymptote was *a-priori* forced to be close to 0. To determine those parameters, we used a modification of an algorithm recently developed by Gallistel, Mark, King, and Latham (2001) that finds changes in the slope of the cumulative record. The use of cumulative records to visualize extinction capitalizes on Skinner's insight that the slope of a cumulative record indicates the momentary rate of responding, because the slope is the rate of change (increase or decrease) of the behavioral measure per trial, when responding is measured on a trial-by-trial basis (Skinner, 1976). Thus, an abrupt extinction curve will be depicted on the cumulative record with a sudden decrease in the slope at the point where responding dropped.

The modified algorithm uses a Bayesian approach to determine whether a change in the slope has occurred. This approach has three stages. It first specifies alternative models. Then it computes the likelihood of the data under each model. Finally, it compares the likelihoods to determine how much more likely one model is relative to the other (Glover & Dixon, 2004; Kass & Raftery, 1995).

In our case, there are two alternative models. Under the no-change model, all the data in the segment of the cumulative record under consideration come from the same distribution; under the change model, a specific trial (to be determined by the algorithm) separates the segment into two distributions: the one comprising all the data before and including the trial of the change point, and the one comprising all the data of the trials after it.

To compute the likelihood of each model we first need to hypothesize the shape of the underlying distribution. Since our CR index is the elevation score, that is, the difference between two counts, the distribution of elevation scores is the Skellam distribution (Skellam, 1946). This is the discrete probability distribution of the difference of two Poisson-distributed variables with different expected values. It is a two-parameter distribution, the parameters being the rates of poking in the presence of the CS and during the pre-CS period. The Skellam probability density function that best fits the data provides for each elevation score a likelihood. In essence, this likelihood is the probability density at the location specified by the datum (elevation score). The overall likelihood of the data under a given model is the product of these likelihoods.

Once the overall likelihood for each of the two models has been calculated, one needs to take the ratio of the two likelihoods to determine the odds that favor one model

relative to the other. This is the Bayes Factor. Note that the Bayes Factor will always favor models with more free parameters. However, the use of additional parameters should not come without a cost. Schwarz (1978) proposed a procedure that corrects for model complexity. Under this correction, the Bayes Factor is calculated as follows:

$$Odds_{Model\ 1} = \exp[LL_{Model\ 1} - LL_{Model\ 2} - 1/2(d_1 - d_2)\log(n)]$$

where LL is the overall log likelihood* of a given model, d_1 and d_2 are the number of free parameters of models 1 and 2 respectively, and n is the sample size (number of likelihoods that entered into the calculation of the overall likelihood of any one model).

The modified change point algorithm steps in the cumulative record datum by datum computing whether an earlier change occurred. For clarity purposes, let's assume that the algorithm has now reached the 20^{th} trial. If there has been an earlier change in the slope, there are 19 trials at which that change could have occurred. Thus, there are 19 change models to be considered. For example, the model that places a change on Trial 5 stipulates that responding on Trials 1-5 is different than responding during Trials 6-20. In other words, the elevation scores of Trials 1-5 come from a Skellam distribution with different parameters from the Skellam distribution underlying the data from Trials 6-20. The overall log likelihood of the data under the Trial-5 change model is the sum of the log likelihoods of the elevation scores from Trials 1-5 (obtained from the Skellam probability density function that best fits these first five elevation scores) and the sum of the log likelihoods of the elevation scores from Trials 6-20 (obtained from the Skellam probability density function that best fits these 15 observations). (To compute the two

* Because usually likelihoods are lower than 1, their products tend to be so small that it has been customary to use log likelihoods instead. One can then take the anti-log of the difference between the overall log likelihoods of the models in order to estimate the odds favoring one model over the other.

parameters of each of the two underlying Skellam distributions the algorithm uses the maximum likelihood estimate function in Matlab.)

The algorithm computes the overall log likelihood for each of these 19 possibilities (change models) and selects the one with the highest log likelihood (e.g., Trial 12 change-model). This is the selected model that specifies that there was a change in responding after trial 12. Its alternative is a model that says that there is no change during these 20 trials. In other words, it says that there is one Skellam distribution that best fits all of the 20 observations. Thus, contrary to the change model that has four parameters (two for each Skellam distribution), the no-change model has only two free parameters. The algorithm computes the overall log likelihood of the no-change model and then it calculates the odds favoring the change model using the Schwarz criterion. If the odds exceed a user-specified criterion, then the algorithm accepts Trial 12 as a true change point. At this moment the algorithm truncates the data record taking the change point as its origin and the first datum after the change point (that is, Trial 13) as the first observation, and it begins anew.

For our analyses we used a Bayes Factor criterion of 100. In other words, the change model had to be at least 100 times more likely than the no-change model in order to be accepted. This criterion is considered as an indicator of strong to decisive evidence for the model it favors (Kass & Raftery, 1995).

Once the algorithm operates on the extinction data and detects changes in the rate of responding, it produces a step plot with as many steps as the change points it found. This graph is a plot of the average elevation score across all the trials contained between successive change points. The beauty of the algorithm is that instead of using an

arbitrary, fixed number of trials per block, it determines the size of successive blocks and the location of the boundaries between them based on the data. Performance during each step is stable and statistically different from performance during the previous or following step.

In quantifying extinction based on this approach we defined three measures of extinction learning: (a) onset latency is the number of trials up to and including the first downward change point. This is the number of trials it took a mouse to initiate extinction. (b) The extinction trial is the trial of the change point that led to a slope not significantly different from 0 (equal responding during the two periods) or a negative slope (that is more responding during the pre-CS than during the CS). This is the trial at which extinction became complete. (c) The dynamic interval is the range of trials between the onset latency and the extinction trial. This is a measure of the abruptness of extinction. That is, it measures how long it took extinction to become complete since it first appeared.

Results and Discussion

Extinction. Figure 1 shows the extinction step plots of 12 mice, chosen for illustrative purposes, as created by the change point algorithm. As can be seen in the plots, extinction in most cases appeared abruptly after only a few trials. Moreover, it became complete as soon as it appeared. Only in very few cases was there a more prolonged decline in responding.

The picture depicted in the step plots of Figure 1 can be generalized to the whole sample. The median onset latency for all subjects was only 4.5 trials. By Trial 13, 75% of

the subjects had initiated extinction. What is more impressive is that for 83% of the sample responding extinguished as soon as it first declined from its initial level. In other words, 15 out 18 mice exhibited no dynamic interval at all; their latency to the first decline was equal to the extinction trial.

To determine whether the acquisition-duration manipulations had an effect on extinction, we compared the groups on the three measures. Figure 2 shows the means of the three groups on onset latency, dynamic interval, and extinction trial. One-way Anovas performed on each measure revealed no differences between the groups (all ps>.07). Thus, a prolonged acquisition duration did not have a serious impact on the rate of extinction, at least when extinction took place in a single session.

To determine whether and how the extinction learning parameters relate to one another, we computed the correlations between the initial level of performance at the start of extinction (that is, the slope of the first step), the onset latency, the dynamic interval, and the extinction trial. The latter correlated positively with the onset latency (Pearson r=.61, p<.01) and the dynamic interval (r=.58, p=.01). In other words, a late onset or a long dynamic interval predicted a late extinction trial. These correlations are hardly surprising, since the extinction trial is the sum of the onset latency and the dynamic interval. What is more interesting is the absence of any correlation between these measures and the performance level at the start of extinction. One may expect that the number of trials required for conditioned responding to extinguish depends largely on its initial level. At least this is what associative models that utilize the delta rule to specify the kinetics of associative change during extinction would expect. If the rate of extinction is constant, a curve that starts at a higher level will require more trials to reach its

asymptote compared to a curve that starts from a lower point. Nonetheless, even if such an effect exists but we failed to detect it due to low statistical power, it is probably small.

Spontaneous Recovery. Figure 3 shows the performance of all three groups during the first and last two-trial blocks of extinction, as well as the course of the two spontaneous recovery tests. Surprisingly, the groups did not respond at similar rates at the start of extinction. The mean elevation score for the first two extinction trials was 0.9 for the group that got many daily sessions, 4.6 for the group that got only a few daily sessions, and 7.6 for the group that got a few widely spaced sessions ($F(2,15)= 3.72$, $p=.049$). The source of this difference was the significantly lower performance of the group that got many daily sessions compared to the group that got a few widely spaced sessions, as determined by Tukey's HSD post-hoc comparison ($p=.04$). This discrepancy was not due to a difference in baseline poking because the mean number of pokes during the pre-CS period of the first two extinction trials was comparable for all groups ($F(2,15)=0.31$, $p=.74$). The cause was most likely a coincidental reduction in conditioned responding in the mice of the group given many daily sessions. We have observed similar bi-directional fluctuations post acquisition before (Papachristos & Gallistel, 2006). Despite their different initial performance, all groups took an average of 29.5 to 30.3 trials to reach the extinction criterion ($F(2,15)= 0.02$, $p=.98$), and by the end of extinction conditioned responding was completely eliminated for all groups ($F(2,15)=0$, $p=1$).

Spontaneous recovery was defined as greater responding during the first trial of the recovery test than during the last two-trial block of extinction. As can be seen in the figure, none of the groups showed robust recovery 1 week after extinction; a two-way Anova with Test (last extinction block vs. first trial of the 1-week test) treated as the

within-subjects variable, and Group as the between-subjects, confirmed this by showing no main effect of Test ($F(1,15)=2.39$, $p=.14$) and no Group x Test interaction ($F(2,15)=0.32$, $p=.73$).

Three weeks post extinction there was robust spontaneous recovery for all groups except for the group that received only a few daily sessions. The same mixed models Anova revealed significant main effects of Test ($F(1,15)=48.4$, $p<.001$), Group ($F(2,15)=11.62$, $p=.001$), and their interaction ($F(2,15)=11.03$, $p=.001$). The source of this interaction was the absence of any spontaneous recovery in the group with the few daily sessions ($t(5)=0.27$, $p=.40$, one-tailed), as opposed to the two other groups.

In summary, we observed that distributing the same number of signal-reward pairings across more sessions or spacing the same acquisition sessions along a wider interval augmented the chances for spontaneous recovery 3 weeks after extinction. This is a finding that many theories of spontaneous recovery cannot accommodate. Trial-based associated models usually attribute spontaneous recovery to the dissipation with time of an inhibitory process that developed during extinction, which counteracted the excitatory properties of the CS (Pearce & Hall, 1980; Wagner, 1981). Since all groups in this experiment received the same number of acquisition trials, and very similar number of extinction trials and exposure to the context, both associations (excitatory and inhibitory) should be of equal strength in both groups. Therefore, the time-dependent fading of the inhibition should progress at equal rates and no differences should be observed in spontaneous recovery testing after any delay. We will return to the evaluation of these models during the General Discussion.

Similarly, interference theories have difficulties accounting for this effect. Although stated in different terms, interference theories emphasize the role of extinction memory as the main contributor of spontaneous recovery. As the time passes, the extinction memory becomes harder to retrieve because its activation threshold rises with time (Kraemer & Spear, 1993), or its temporal context becomes increasingly dissimilar (Bouton, 1991, 1993, 2004), and thus spontaneous recovery appears. Since all groups in this experiment received identical extinction-testing delays, no differences should be anticipated.

Devenport's Temporal Weighting Rule (Devenport, 1998) anticipates the finding that the group given many trial-poor sessions show more recovery than the group given a few trial-rich daily sessions, only if the session, as opposed to the trial, is the unit of experience that enters into the weighted averaging process. However, TWR wrongly predicts that spacing the same number of sessions should, if anything, hurt spontaneous recovery, since it moves the acquisition experiences farther into the past, thus diminishing their positive influence on the weighted average at the time of testing. Our data indicate that spacing the sessions enhances spontaneous recovery, in a similar way that spacing trials enhances conditioning.

Skinner's hypothesis (Skinner, 1950) that spontaneous recovery is an artifact of incompletely extinguished early-session cues cannot accommodate both findings either. It correctly anticipates that increasing the number of sessions augments spontaneous recovery. According to this hypothesis, the temporal proximity of the first couple of trials with the handling and transportational cues makes those early-session CSs qualitatively different than the remaining CSs in the session. Therefore, delivering more sessions

enhances the conditioning of those early-session CSs, while a single extinction session is inadequate to extinguish them. However, it is not clear how this explanation can account for the session-spacing manipulation. One would imagine that a 1-day interval or a 4-day interval between subsequent sessions should not make much of a difference in the rate at which those CSs enter into associations with the compound handling and transportational cues. Therefore, without making some rather strong assumptions, this hypothesis too fails to account for the augmenting results of the session-spacing manipulation.

Estes' statistical theory of learning (Estes, 1955b) is the only model of spontaneous recovery that correctly anticipates the enhancing effects of both manipulations. According to his theory, spacing the trials or the sessions, allows more elements to become conditioned with the US. Therefore, both experimental groups showed enhanced recovery simply because at the time of testing a larger proportion of conditioned elements were sampled, compared to the control group.

It is rather surprising that 1 week post extinction none of the groups showed any spontaneous recovery. This rest interval is usually large enough to produce robust recovery in the same preparation in the rat (Rescorla, 2007). A more careful look at the data showed that at least in the group given many daily trial-poor sessions the absence of spontaneous recovery could be attributed to its unusually elevated baseline poking at the start of the first spontaneous recovery test. Figure 4 shows the mean number of pokes during the 10-s periods preceding the two extinction blocks and each test trial. Baseline poking remained low for all groups except for the pre-CS period of the first test trial one week after extinction for the group with the many daily trial-poor sessions. A one-way Anova revealed that the three groups differed in the number of pokes during the pre-CS

period of that first trial ($F(2,15)=4.66$, $p=.03$). In specific, the group with the many daily trial-poor sessions had higher baseline poking than the group with the few daily trial-rich sessions ($p=.04$) and marginally higher than the group with the few spaced trial-rich sessions ($p=.05$), as determined by Tukey's HSD post-hoc tests. In other words, contextual conditioning was particularly high in the group with the many sessions at the time of the first test and it masked their recovery of responding to the CS 1 week post extinction.

We cannot determine what caused this elevation of contextual conditioning in the group with the many daily trial-poor sessions. Although the same group had a higher number of pokes throughout the variable part of the first ITI during the first test, a one-way Anova showed no significant difference between the groups ($F(2,15)=3.02$, $p=.08$). Moreover, this elevation was largely due to a single mouse in that group, which poked 36 times (20 pokes more than the second best performer) before the onset of the first pre-CS period. Thus, we believe that the elevated contextual poking was just a fluke, and not the result of manipulating the session number. However, to further confirm our hypothesis that the positive effect of the number of sessions on spontaneous recovery can be obtained a week after extinction without being contingent on a recovery of contextual conditioning, we decided to replicate this particular finding.

Experiment 2

The goal of this experiment was to investigate whether the positive effect of increasing the number of sessions could be obtained during the first test of spontaneous recovery. So far, this effect was only observed during the second test. Although in Experiment 1 the group with the many daily trial-poor sessions showed a trend for more vigorous responding to the CS 1 week post-extinction, their contextual conditioning also recovered, decreasing their overall elevation score. To eliminate an elevated base-rate, we gave all mice a session of context extinction on each of the two days prior to the 1-week test.

Method

Subjects and Apparatus. The subjects were 12 male C57Bl/6 mice obtained from Harlan (Indianapolis, IN). They were about 10 weeks old and weighed between 21.2 and 24 g when the experiment started. They were maintained on the same manner as in Experiment 1. The apparatus was the same as in Experiment 1.

Procedure. The procedural details during all phases of training and testing were those of Experiment 1 except where noted. Mice were randomly assigned into two groups of 6 subjects. During conditioning, the group with the few trial-rich sessions received six daily sessions (one per day) of 10 rewarded trials with the white noise, while the group with the many trial-poor sessions received 24 daily sessions of 10 such trials. A single extinction session one day after the last day of conditioning employed the same criterion as in Experiment 1. On the 5^{th} and 6^{th} day following extinction, all mice were placed in the experimental chambers for 1 hr. No stimuli were presented during those sessions,

with the exception of the house light, which remained on throughout. Finally, both groups were tested for spontaneous recovery 1 and 3 weeks post extinction, each test being identical to the tests of Experiment 1.

Results and Discussion

Extinction. The picture of extinction responding was very similar to the one from Experiment 1. Figure 5 shows the extinction step plots of all mice, as created by the change point algorithm. Extinction started after a variable delay but became complete almost immediately. The median onset latency for all subjects was again 4.5 trials, while the third quartile was only 6 trials. Impressively, all but one mouse had a dynamic interval of 0 trials. Thus, the vast majority of the subjects completed extinction as soon as they initiated it.

The session-number manipulation did not have an effect on any of the extinction parameters. None of the t-tests that compared the two groups on each parameter approached significance (all ps>.20). Finally, the only significant correlation obtained was between the dynamic interval and the extinction trial (r=.85, p<.001), indicating the obvious fact that a long dynamic interval predicts a late extinction trial. Again, the initial performance was not found to relate to any of the extinction parameters.

Spontaneous Recovery. Figure 6 shows the performance at the beginning and end of extinction, and throughout the course of the two tests. The two groups responded at similar rates during the start (t(10)=0.34, p=.74) and end of extinction (t(10)=0.56, p=.59). Moreover, there was no difference in the mean number of trials to reach the

extinction criterion (Group many trial-poor sessions: 28.5, Group few trial-rich sessions: 34.3, $t(10)=1.53$, $p=.16$).

The results of most interest come from the test session given a week later. That day found robust recovery on the first test trial for the group given many trial-poor sessions ($t(5)=3.38$, $p<.01$, one-tailed), followed by a sharp drop to practically baseline levels. Moreover, on that first trial, the same group responded to the white noise more than the group with the few trial-rich sessions ($t(10)=3$, $p=.01$). The latter group failed to show any spontaneous recovery ($t(5)=1.30$, $p=.12$, one-tailed).

Three weeks after extinction the results look very similar. The group with the many trial-poor sessions exhibited spontaneous recovery during the first ($t(5)=5.45$, $p=.001$, one-tailed) and second trial ($t(5)=2.37$, $p=.03$, one-tailed). Moreover, a two-way Anova with group treated as the between-subjects factor and test trial as the within-subjects factor found a main effect of group ($F(1,10)=10.11$, $p=.01$), indicating that during these two trials the group with the many trial-poor sessions responded more than the group with the few trial-rich sessions.

In summary, Experiment 2 replicated one of the main findings of Experiment 1 that distributing the same number of CS-US pairings across more sessions augments spontaneous recovery from extinction. The contextual extinction prior to the first test might have obscured any recovery in the group that received few sessions, while it did not in the group with the many sessions.

Experiment 3

Experiment 1 showed that spontaneous recovery is enhanced when acquisition has been prolonged by distributing trials across more sessions or by spacing trial-rich sessions more widely. The effect of the session number manipulation seemed to have a slightly bigger effect than that of the session spacing manipulation, although this was not established statistically. One might assume that it did given that there was a tendency for higher responding during the first CS presentation of the 1-wk test, which was though masked by an unusually elevated baseline. Similarly, responding throughout the second test was higher in the group that got many trial-poor daily sessions than in the group that got a few widely spaced trial-rich sessions, although again this difference was not statistically significant. The question of whether there is a difference in the size of the effect that the two manipulations have on spontaneous recovery has some theoretical importance. If the two manipulations have equal effects, then it is more likely that they both share the same mechanism of action (for example, they result in widely distributed trials, or they prolong the duration of the acquisition regime). If, on the other hand, the session number manipulation is more robust, this may indicate that whatever unique properties this manipulation possesses may be relevant parameters that a complete account of spontaneous recovery should encompass.

We tried to address this question in Experiment 3. It used a within-subjects design to compare spontaneous recovery to a stimulus that during acquisition had been presented on all training sessions with that to a stimulus presented on a random quarter of those sessions. The two stimuli had in common the total number of trials each was presented,

and the span (in days) of acquisition training, while they differed on the number of sessions in which they had appeared.

Method

Subjects and Apparatus. The subjects were 6 mice of the same sex and type as in the previous experiments. They were about 11-12 weeks old and weighed between 17.9 and 18.9 g when the experiment started. They were maintained on the same manner as in previous experiments. The apparatus was that of Experiment 1.

Procedure. To improve stimulus discrimination, this experiment used three stimuli: white noise, flashing house-light (0.5-s on, 0.5-s off), and diffuse hopper light. Two of these stimuli served as conditioned stimuli, while the third one had always the opposite outcome, that is, it was not reinforced during conditioning, but it was during extinction. The role of the stimuli was counterbalanced across subjects. One role was that of what we will call the routinely present CS. The routine CS occurred equally often in every session. The second role was that of what we will call the intermittently frequent CS. The intermittently frequent CS only occurred in 25% of the sessions, but in those sessions, it occurred 4 times more frequently than the routine CS. The third role was that of the control CS. This CS occurred in every training session for one quarter of the number of trials of the other CS(s) (that is, it was presented once for every four trials with either of the other two CSs). In training sessions, it was never reinforced; in the extinction session, it was always reinforced. Its role was to prevent stimulus generalization between the other two CSs, that is, to prevent the subject from drawing the

conclusion that whenever a stimulus came on, it predicted food, regardless of the identity of the stimulus.

Acquisition: Initial training lasted for 28 sessions (one session per day). Sessions started with a variable 180-s ITI, followed by an unsignaled fixed 10-s pre-CS interval, at the end of which a 10-s trial started. 75% of the sessions contained 8 trials with the routine CS and 2 trials with the control CS. The other 25% of the sessions contained 8 trials with the routine CS, 32 trials with the intermittently frequent CS, and 10 trials with the control CS. With the exception of the first and last conditioning session, which belonged to this type, the choice of the other 5 such sessions was individually randomized for each mouse. The order of trial type within all sessions was determined randomly for each mouse by the computer program that controlled the experiment, without any restrictions imposed.

Extinction: The day after acquisition ended, mice received a single extinction session, which contained 20 non-reinforced presentations of each of the two CSs, and 10 rewarded presentations of the control CS, with random order. This alternation in the latter stimulus' outcome aimed to maintain some level of reinforcement, and accelerate extinction.

Spontaneous Recovery Tests: All mice were tested for spontaneous recovery 1 and 4 weeks after extinction. Each test included two non-reinforced trials with each of the two extinguished CSs. During each test, for half of the mice the order of the stimuli followed an ABBA pattern while for the other half it was reversed (BAAB). Each mouse received both orders across the two tests. There were no presentations of the control CS in any of the tests.

Results and Discussion

Figure 7 shows responding to the two conditioned stimuli during the course of extinction and the two tests. Mice responded indistinguishably to the two stimuli at the onset of extinction. The curves remained almost superimposed throughout extinction, and at the end there was no difference in responding to the two stimuli ($t(5)=0.55$, $p=.60$).

One week later, responding to the two stimuli recovered slightly during the first trial and marginally exceeded that shown at the final extinction block for both stimuli (Routine CS: $t(5)=2.02$, $p=.05$; Intermittently frequent CS: $t(5)=2.05$, $p=.05$, one-tailed). Four weeks post extinction there was a trend for recovery only in the routine CS but it did not reach significance ($t(5)=1.73$, $p=.07$, one-tailed). Moreover, although the mice tended to respond to it more than they did to the intermittently frequent CS, the difference between the two stimuli at the first trial of the test did not approach significance either ($t(5)=2$, $p=.1$).

A closer look at the data during the first trial of the 4-week test revealed that the absence of a difference in responding to the two stimuli was due to the violation of normality in the distribution of elevation scores for the intermittently frequent CS. Figure 8 shows the cumulative distributions of the elevation scores during the first trials of that test for each stimulus. As the figure shows, 5 out of 6 mice did not respond at all to the intermittently frequent CS. In this case, the t-test is not the appropriate statistic for the comparison of responding to the two stimuli. Thus, we decided to perform this stimulus comparison using a Bayesian analysis.

The rationale behind Bayesian statistics was explained during the description of the change-point algorithm. Briefly, there are two alternative hypotheses. Under the null-

hypothesis, responding to the two stimuli during their respective first trial of the 4-week test does not differ. That is, the elevation scores to the routine CS and the intermittently frequent CS, when each was presented for the first time during that test, come from the same distribution. The alternative hypothesis states that responding to one CS is different than the other. In order to compare the two hypotheses and compute how much more likely one is from the other, we computed the likelihood of the data under each hypothesis.

To do so, we first hypothesized that the underlying distribution is the Skellam distribution, which is the distribution of the difference of two Poisson-distributed variables –the variables being the counts of pokes during the CS and the pre-CS periods. It is a two-parameter distribution, the parameters being the rates of poking in the presence of the CS and during the pre-CS period. The Skellam probability density function that best fits the data provides for each elevation score a likelihood. This likelihood is the probability density at the location specified by the datum (elevation score). The overall likelihood of the data under a given model is the product of these likelihoods.

Under the null hypothesis, all 12 data points come from the same Skellam distribution. Under the alternative hypothesis, there is a different Skellam distribution for the six data with each CS. The parameters for each of the three Skellam distributions were calculated using the maximum likelihood estimate function in Matlab. Once we computed the overall likelihood for each of the two models we used the Schwarz criterion (Schwarz, 1978) to correct for the additional two parameters of the alternative hypothesis.

For the first trial of the 4-week test, the Bayes Factor was 178 in favor of the alternative hypothesis. In other words, the alternative hypothesis was 178 times more

likely than the null hypothesis. This is decisive evidence for the alternative hypothesis. The Bayes Factor for the second trial of the same test was 33, again in favor of the alternative hypothesis. Because Bayes Factors are in essence relative likelihoods, one can combine them multiplicatively to determine the overall odds in favor of a model. Doing so for the two trials of the 4-week test, the odds (5,874) overwhelmingly support the hypothesis that the mice responded more to the routine CS than the intermittently frequent CS.

To summarize, there was equally weak spontaneous recovery to both stimuli one week after extinction, followed by a trend for small recovery only to the routine CS three weeks later. Although conventional statistics showed that the latter effect was not significant, a Bayesian analysis provided decisive evidence for it. Thus, it is rather safe to conclude that the session-number manipulation is, if anything, slightly stronger than session spacing in promoting spontaneous recovery from extinction. Both manipulations result in increasing the total duration (in days) of the acquisition regime. However, the slightly stronger effect of the session-number manipulation may be taken to indicate that the session is an important unit of experience and therefore increasing the number of sessions may contain an additional mechanism, which acts independently of the resulting increase in the overall acquisition duration.

Experiment 4

The previous experiments suggested that the number of sessions is an important parameter for spontaneous recovery. With total trial number held constant, more initial conditioning sessions augment spontaneous recovery. This property may partially be attributed to the resulting increase of acquisition, because spacing trial-rich sessions has almost the same effect. However, it would not be premature to conclude that there is an additional source for this effect. One possibility is that the number of conditioning sessions is an important parameter for spontaneous recovery. We speculated that if session is an important unit of experience, the number of trials within a session might be an irrelevant parameter. Experiment 4 was designed to evaluate this possibility. Two groups of mice received the same number of conditioning sessions but differed in the number of trials per session. Group Many Trials was trained with four times more trials per session than Group Few Trials. Both groups were given a single extinction session and subsequently tested for spontaneous recovery. If the number of sessions is the only determinant of spontaneous recovery, both groups should exhibit equivalent recovery, since they don't differ on that regard. If on the other hand the number of trials within a session contributes to spontaneous recovery, then Group Many Trials should show higher recovery.

Method

Subjects and Apparatus. The subjects were 12 mice of the same sex and type as in Experiment 1. They were about 11 weeks old and weighed between 18.1 and 21 g

when the experiment started. They were maintained on the same manner as in Experiment 1. The apparatus was that of Experiment 1.

Procedure. The procedural details during all phases of training and testing were those of Experiment 1 except where noted. Mice were randomly assigned into two groups of 6 subjects. During conditioning, Group Few Trials received eight daily sessions (one per day) of 10 rewarded trials with the white noise, while Group Many Trials received 8 daily sessions of 40 trials. Note that since the same 3-min average ITI was given to both groups, session durations differed also by a factor of four in these groups. A single extinction session one day after the last day of conditioning employed the same criterion as in Experiment 1. All mice were tested for spontaneous recovery 1 and 3 weeks post extinction. Both tests were the same as the respective tests of Experiment 1.

Results and Discussion

Extinction. Figure 9 contains the algorithm-generated extinction step plots from all mice. The overall picture from the previous experiments of an abrupt extinction after a variable delay still held. The median onset latency was 3 trials, while the third quartile was 5 trials. The same values for the dynamic interval were both 0. In fact, only 2 out of 12 mice (17%) exhibited a dynamic interval. Given the small value of these two extinction measures, it was only expected that the extinction trial would be similarly small (its median and third quartile were 3.5 and 6 trials, respectively).

Figure 10 shows the means of the two groups on the three extinction measures. Despite a four-fold difference in the number of acquisition trials between the two groups, they did not differ in any of these variables (all ps>.13). Additionally, both groups

responded similarly during the start of extinction; that is, they did not differ in the slope of the first segment of the cumulative record ($t(10)=1.15$, $p=.28$).

The correlation analysis found a very high positive correlation between the extinction trial and the onset latency ($r=.98$, $p<.001$) and a more interesting correlation between the initial level of performance (that is, the slope of the first step) and the dynamic interval ($r=.68$, $p=.02$). This is the first time that we obtained evidence for a positive relationship between the initial strength of responding and the abruptness of extinction. Thus, contrary to our previous experiments, in this study a higher rate of responding at the onset of the extinction session predicted a lower extinction rate. However, this correlation relies heavily on the performance of the two mice that actually exhibited a dynamic interval, and thus should be interpreted with caution.

It should be noted that the lack of a reliable difference between the two groups in the initial level of responding is rather surprising. Group Many Trials received four times more conditioning trials in acquisition. Yet, as Figure 11 shows, there was a trend on the opposite direction; the group that received the fewer conditioning trials tended to respond more vigorously during the first two extinction trials. This difference, although not significant, is opposite to what was expected by most trial-based conditioning models. This is also true for Estes' statistical theory of learning, which predicts that the initial magnitude of responding in extinction is an exponential function of the number of reinforcements (Estes, 1955b; Homme, 1956).

Spontaneous Recovery. Figure 11 shows responding during the beginning and end of extinction, and the two spontaneous recovery tests. The groups responded at similar levels both at the start ($t(10)=1.68$, $p=.12$) and end ($t(10)=1.83$, $p=.10$) of

extinction. Moreover, they did not differ in the number of trials to reach the extinction criterion ($t(10)=0.07$, $p=.94$).

The results of interest come from the two spontaneous recovery tests. One week after extinction none of the groups showed any recovery. It is rather surprising that as it appears in the graph Group Few Trials responded overall at a higher rate than Group Many Trials. However, only on the fourth trial did this difference approach statistical significance ($t(10)=2.12$, $p=.06$). Three weeks after extinction conditioned responding in both groups recovered substantially on the first trial, but dropped sharply immediately after. On the first trial both Group Many Trials ($t(5)=3.78$, $p<.01$, one-tailed) and Group Few Trials ($t(5)=3.27$, $p=.01$, one-tailed) exceeded their performance shown at the end of extinction. Moreover, although there was a trend for Group Few Trials to respond at a higher rate than Group Many Trials, this difference did not reach statistical significance ($t(10)=1.88$, $p=.09$).

In summary, the results of this experiment argue against a role of trial number as a main determinant of spontaneous recovery. The two groups received eight conditioning sessions, but Group Many Trials received four times as many trials as Group Few Trials. Thus, a four-fold difference on this parameter did not have an effect on the magnitude of spontaneous recovery from extinction. The predictions of trial-based models are at odds with these findings. Such models emphasize the importance of trials for the formation and the strengthening or weakening of associations. Thus, they capitalize on the assumption that more conditioning trials should result in higher excitatory associative strength. The latter either should take longer to decline during extinction (Mackintosh, 1975; Rescorla & Wagner, 1972) or should require more time before it is cancelled out

by an opposite (inhibitory) association that develops during extinction (Pearce & Hall, 1980; Wagner, 1981). In the absence of any differences on the number of extinction trials, these models would predict that at the end of extinction the group that received more conditioning trials should have a higher net associative strength which should cause higher recovery. Estes' model (Estes, 1955b) similarly fails to predict the lack of a difference in spontaneous recovery between the two groups. Under similar trial spacing protocols, the model stipulates that spontaneous recovery is an increasing function of the number of reinforcements. Thus, it too expects Group Many Trials to produce more recovery.

It is possible that the 80 acquisition trials were enough to fully condition mice in Group Few Trials. Thus, the absence of any difference in spontaneous recovery might have been due to the fact that the excitatory associations for both groups were practically equal at the onset of extinction. Nonetheless, if trial number did contribute to spontaneous recovery, one should expect that Group Many Trials should show at least a trend for higher recovery especially 1 week post extinction, when performance was maintained at the low end of the scale. In the absence of such a trend, the current findings are supportive of our hypothesis that the number of sessions and/or the duration of acquisition are more important parameters for spontaneous recovery than the number of conditioning trials.

Experiment 5

Experiment 4 found no differences in spontaneous recovery between two groups that received the same number of sessions but differed by a factor of four on the total number of trials and context exposure. Although this finding argues against a role of trial number in spontaneous recovery, one cannot exclude the possibility that this parameter may have an effect after all at lower values. In other words, if the number of conditioning trials within a session drops below a critical value, it may hurt not only conditioning, but also spontaneous recovery. In this case, the hypothesized superior role of session as an important unit of experience may be contingent on it containing a minimum number of trials. A direct evaluation of this hypothesis would entail a replication of Experiment 4, with the exception that Group Few Trials would receive only one or two trials per session. However, such a design is almost certainly doomed to fail, because, in the absence of an increased ITI in that group, their session duration would be too short to allow mice to obtain the reward in a timely manner during the first couple of sessions. If on the other hand the two groups were equated on context exposure (session duration), the results would be confounded by the trial-spacing effect. To avoid these complications, Experiment 5 was designed to pit the number of sessions against the number of trials. Thus, we trained two groups on the magazine approach procedure. The session-rich-trial-poor group received four times more sessions but also four times fewer total trials than the session-poor-trial-rich group. Both groups were subsequently put through extinction and then tested for spontaneous recovery. If the number of sessions and/or the acquisition duration is a more important parameter than the number of trials, then it should stimulate more recovery in the session-rich-trial-poor group.

Method

Subjects and Apparatus. The subjects were 12 mice of the same sex and type as in Experiment 1. They were about 10-12 weeks old and weighed between 19 and 21 g when the experiment started. They were maintained on the same manner as in Experiment 1. The apparatus was that of Experiment 1.

Procedure. The procedural details during all phases of training and testing were those of Experiment 1 except where noted. Mice were randomly assigned into two groups of 6 subjects. During conditioning, the session-rich-trial-poor group received 28 daily sessions (one per day), each containing 2.5 trials on average (2 or 3 trials alternating between adjacent sessions). The session-poor-trial-rich group received 7 daily sessions of 40 trials each. A single extinction session one day after the last day of conditioning employed the same criterion as in Experiment 1. All mice were tested for spontaneous recovery 3 and 18 days after extinction. Both tests were identical to the tests of Experiment 1.

Results and Discussion

Extinction. Figure 12 contains the step-plots of all mice from the extinction session, as generated by the algorithm. The story is the same as it was in previous experiments. Sharp declines to virtually asymptotic levels were observed after delays that varied from mouse to mouse. The median mouse took 5.5 trials to start extinguishing, while 75% of the mice needed 11 trials. The dynamic interval was spectacularly absent in 75% of the subjects. The same quartile of the extinction trial was 13.5 trials.

Figure 13 shows the means of the groups on the three extinction measures. As can be seen in the figure, the two groups did not differ in any measure (all ts<1). Thus, the manipulation did not affect the rate at which extinction proceeded. Moreover, the groups did not differ on the initial level of performance, as determined by the algorithm (t<1).

Finally, the extinction trial once again correlated positively with the dynamic interval (r=.89, p<.001). Interestingly, the initial level of extinction correlated positively with the dynamic interval (r=.72, p<.01) and negatively with onset latency (r=-.87, p<.001). Thus, it appeared that stronger responding at the outset of extinction predicted an earlier decline in responding followed by a prolonged transition till extinction became complete. The correlation of initial level of responding with the dynamic interval, obtained from this and the previous experiment, argues in favor of the common assertion of trial-based models that the rate of extinction depends on the initial level of responding. However, as in the previous experiment, here too this correlation depended largely on two mice, the only ones that displayed a dynamic interval. Therefore, it should be treated with caution.

Spontaneous Recovery. Figure 14 shows responding during the beginning and end of extinction, and the two spontaneous recovery tests. The groups responded at similar levels both at the start ($t(10)$=0.51, p=.62) and end ($t(10)$=1.46, p=.18) of extinction. Moreover, they did not differ in the number of trials to reach the extinction criterion ($t(10)$=0.08, p=.94).

The results of interest come from the two spontaneous recovery tests. Three days after extinction none of the groups showed any recovery. This interval was chosen to ensure that the session-poor-trial-rich group would respond at a low rate, so that any

enhancing effect of the session number manipulation could be easily detected. However, this choice of delay proved unfortunate, since it was too short to allow any recovery in the session-rich-trial-poor group as well. As can be seen in the figure, only on the second trial did the mice of the session-rich-trial-poor group show a trend of higher responding than their controls, but this difference did not reach statistical significance ($t(10)=2.07$, $p=.07$).

Eighteen days after extinction responding recovered for both groups during the first (Session-rich-trial-poor group: $t(5)=3.46$, $p<.01$; Session-poor-trial-rich group: $t(5)=2.27$, $p=.04$, one-tailed) and second ($t(5)= 3.02$, $p=.01$; $t(5)=2.92$, $p=.02$, one-tailed) test trials. More importantly, the session-rich-trial-poor group responded at a higher rate than the session-poor-trial-rich group during the first three trials of that test. A two-way Anova with trial as the within-subjects factor and group as the between-subjects showed significant main effects of the trial ($F(2,20)=4.41$, $p=.03$) and the group ($F(1,10)=7.57$, $p=.02$) factors.

The results from this experiment suggest that session number may be a more important parameter for spontaneous recovery than trial number. Having four times more sessions and four times fewer trials was slightly more effective than the reverse in promoting spontaneous recovery. However, the magnitude of this effect was not as big as one would expect if only the session was the important unit of experience. Note that the session-rich-trial-poor group significantly outperformed the session-poor-trial-rich group only when the first three trials of the 18-d test were taken into account. However, this difference should be treated with caution because on the 3rd trial both groups had already returned to levels of responding comparable to those each had shown at the end of

extinction. It was only during the first two trials that both groups' recovery was maintained at a high level. The superior responding of the session-rich-trial-poor group during these first two trials approached but did not reach significance ($F(1,10)=4.59$, $p=.06$).

In conclusion, the results of this experiment provide some evidence for the hypothesis that session, as opposed to trial, may be a more important parameter for spontaneous recovery. However, the evidence is not very robust. Until this finding is replicated, the results should be treated with caution. Future experiments should aim at remedying two drawbacks of this design. First, the extinction-test intervals should be chosen to ensure robust recovery of the session-rich-trial-poor group. Second, the number of trials in the same group should be kept at the minimum, possibly at just one trial per session, in order to avoid asymptotic conditioning of this group's subjects. Such a design may be more appropriate for a direct and conclusive evaluation of this hypothesis.

General Discussion

This dissertation used a conditioned magazine approach in the mouse to investigate whether and how various temporal parameters in acquisition, such as the distribution of trials into sessions and the distribution of sessions over time, would affect spontaneous recovery from extinction. Experiment 1 found that distributing the same number of acquisition trials across more sessions, or spacing the same conditioning sessions more widely, augmented spontaneous recovery. The effect of the former manipulation was especially strong during the second post-extinction test, while in the first test an elevated base-rate masked the recovery to the extinguished CS. Using a contextual extinction manipulation before the first test, Experiment 2 replicated the finding that conditioning trials enhance spontaneous recovery from extinction when they are distributed across more sessions. Experiment 3 aimed to compare the effect size of the session-number and session-spacing manipulations using a within-subjects design. Experiment 4 found that when the session number in acquisition is constant, a four-fold decrease in the number of trials did not hurt spontaneous recovery. Experiment 5 made a more direct comparison of whether the number of trials or sessions is a more important parameter for spontaneous recovery and found that having four times more sessions and four times fewer trials was at least as effective, if not more, as the reverse in promoting spontaneous recovery.

A variety of theoretical models of spontaneous recovery were presented in the introduction. The purpose of our experiments was not to test the predictions of every single model and accept the one(s) that can account for the majority of the findings. In fact, as it became clear in the introduction, no model so far can provide a fully

satisfactory account of spontaneous recovery. Nevertheless, these results add some new findings to the slowly growing research on spontaneous recovery, which an adequate explanatory account should be able to address.

The results obtained were inconsistent with **generalization decrement** accounts (e.g., Capaldi, 1967). These accounts attribute spontaneous recovery to the ambiguous state of the context at the time of testing. The observation of Experiments 1 and 2 that spontaneous recovery is enhanced in mice that receive the same number of trials across more sessions poses serious difficulties to this account. Since this manipulation did not affect the total exposure to the context, mice given many trial-poor or few trial-rich sessions should be equally (un)able to classify the test context as signaling reward or no reward. The same reasoning can be applied to explain the failure of this account to predict the enhancing effects of the session-spacing manipulation in Experiment 1.

Interference accounts (e.g., Bouton, 1991; 1993; Kraemer & Spear, 1993) have similar difficulties explaining these main findings. These accounts emphasize the role of retrieval processes during testing to explain spontaneous recovery. However, both rely on a time-induced change in the extinction memory, but not the acquisition memory. Our manipulations did not target extinction. Thus, the observed differences between the groups of Experiment 1 cannot be explained by a time-induced increase in the threshold for the activation of the extinction memory (Kraemer & Spear, 1993) or a time-induced change in the internal state of the animal since extinction (Bouton, 1991; 1993).

The **early-session cue** account of spontaneous recovery (Burstein, 1967; Skinner, 1950) can predict most of the findings in this study, with the exception of the session-spacing effect. Regarding the session-number manipulation, the administration of more

conditioning sessions could have resulted in better conditioning of the first CS(s), thereby causing stronger recovery during testing. For this account, the number of trials within a session is not important for spontaneous recovery because it is only the first couple of CSs that cause recovery. This is why Experiment 4 found no differences between two groups that received the same number of sessions but differed in the trial number. Using the same rationale, this account explains why session number is may be a more important parameter for spontaneous recovery than trial number (Experiment 5).

However, Skinner's hypothesis (1950) cannot account for the enhancing effects of the session-spacing manipulation (Experiment 1). Group Few Spaced Sessions received the exact same number of trials and sessions as the control group and thus should show equal recovery. Additionally, stronger evidence against this account has been obtained by studies which showed that recovery is not constrained to appear at the beginning of sessions (Robbins, 1990; Thomas & Sherman, 1986). In unpublished work in our laboratory, using the same procedure and species as in the reported experiments, we also observed recovery in the middle of a session, after three non-reinforced trials with another formerly trained and extinguished CS. Finally, perhaps the most serious objection to Skinner's proposition comes from its inability to predict the main feature of spontaneous recovery that it increases with the passage of time after extinction (Mackintosh, 1974; Rescorla, 2004a; Thomas & Sherman, 1986). Our own experiments showed this repeatedly: spontaneous recovery was always stronger on the second test, even though the intervening test constituted a brief extinction session.

The **temporal weighting rule** (Devenport, 1998; Devenport et al., 1997) is similarly challenged by the finding that session-spacing enhances spontaneous recovery

(Experiment 1). This manipulation results in endowing all but the last acquisition sessions with smaller weights, thereby diminishing their influence on the weighted average and resulting in smaller, if anything, recovery. However, TWR can accommodate the rest of our findings. In specific, assuming that the session and not the trial is the important unit of experience that enters into the averaging process, this account predicts that the number of sessions, regardless of the number of trials within a session, affects spontaneous recovery positively. Thus it explains why Experiment 4 found no differences between two groups that received the same number of sessions, and also why in Experiment 5 the four-fold increase in the same variable tended to outperform the four-fold decrease in the number of trials.

Estes' **statistical theory of learning** (Estes, 1955a, 1955b) shares the same success with TWR in accounting for most of our results. This model is unique among associative models in specifying the time-induced changes that occur throughout learning. Any trial-spacing manipulation, according to this model, results in a higher number of conditioned elements in the total population. During the single extinction session a small subset of these elements will become unconditioned. After extinction, the exchange of elements between the sample and the population that occurs, allows more conditioned elements to be included in the test sample, thus causing more recovery to the extinguished CS.

What Estes' model fails to accommodate is the lack of a difference between two groups that received the same number of sessions differing only in the number of trials within that session (Experiment 4). Estes's account predicts that under identical trial-spacing protocols, spontaneous recovery is a positive (exponential) function of the

number of reinforcements (Estes, 1955b; Homme, 1956). Thus, a four-fold difference in that variable should if anything hurt spontaneous recovery, contrary to what we observed.

The picture is less clear when evaluating models that attribute extinction to the development of **non-associative inhibition** (Hull, 1943; Pavlov, 1927; Rescorla, 1979; Rescorla & Cunningham, 1978; Robbins, 1990) or **associative inhibition** between the CS and the conditioned response (Rescorla, 1993b, 1997). These models focus only on extinction, the part of the training protocol that was not manipulated in our experiments. Thus, without resorting to further assumptions, they would fail to capture the current findings.

The majority of the **trial-based associative models** are also seriously challenged by these data (e.g., Mackintosh, 1975; Rescorla & Wagner, 1972). In order to account for the effects of trial spacing on conditioning, they assume that the CS and the context compete for associative strength (i.e., Barela, 1999). Longer ITIs produce contextual extinction, thereby allowing the CS to acquire more associative strength than the one it would have accrued with massed ITIs. However, all the groups in Experiments 1 and 2 were equated for context exposure. Therefore, such models cannot explain the main findings about the enhancing effects of spreading trials out over more sessions or spreading sessions out over more time.

Trial-based associative models that have incorporated a **CS-processing** mechanism, such as the SOP model by Wagner (1981) seem to have a better fate. According to SOP a stimulus is represented by a number of elements that can be in any of three states: inactive, active, and refractory. When the CS occurs, some of its elements in the inactive state enter the active state. From there, there is a constant decay of elements

to the refractory state, and from the refractory state back to the inactive, where they remain until the CS appears again. Excitatory conditioning takes place when elements of both CS and US representations happen to be in the active state. If the CS elements are in the active state but the US elements in the refractory state, conditioned inhibition occurs instead. A very dense trial protocol hurts learning because the US elements from the previous trials do not have enough time to decay to the inactive state but instead remain in the refractory state, where they can support inhibitory conditioning to the CS. By remaining in the refractory state, they also limit the number of US elements that can become active upon presentation of the US, thereby reducing the excitatory strength accumulated to the CS.

The mechanism proposed by SOP could be responsible for the session-number manipulation. The 24-hr intervals between successive sessions are long enough to allow the majority of the CS and US elements to be in the inactive state before conditioning starts. More such intervals, as the result of increasing the session number, are beneficial for learning and also augment the chances for spontaneous recovery. The same rationale could explain the results of Experiment 4, in which more trials per session almost hurt conditioning and spontaneous recovery. However, nothing could be said for the session-spacing manipulation. It is conceivable that even if the parameter for the probability of elements in the refractory state decaying in the inactive state is very small, it should still support conditioning at least during the first trial of each session. Thus, inserting a 4-d intersession interval should not have a better effect than a 1-d interval.

SOP is a fairly complex model with many free parameters, which endow it with high explanatory power. However, its value as an adequate account of our data is limited

because it heavily depends on a particular subset of parameters. If the current findings hold across a number of procedures, which use different experimental parameters, it is unlikely that the same set of free parameters would suffice to explain all data. On the same grounds, the use of a high number of free parameters in any model of learning is discouraged because it allows the model to make unspecific and sometimes contradictory predictions. If, for example, Experiment 4 had found that the number of trials in a session positively correlated with spontaneous recovery, SOP would predict that by adjusting its parameters to allow the CS to recover its conditionability too quickly.

Finally, it is worth noting that the session-number manipulation's effect on spontaneous recovery resembles the trials-per-session effect (TPSE). The TPSE refers to the observation that distributing the same number of trials across more sessions increases the rate of conditioning. It has been thoroughly documented in rabbit eyeblink conditioning (Kehoe & Macrae, 1994), but has also been observed in autoshaping experiments with rats (Papini & Dudley, 1993) and pigeons (Papini & Overmier, 1985). If the session number manipulation in our experiment resulted in higher associative strength between the CS and the US by means of the TPSE, that could explain why it also produced more recovery in Experiments 1 and 2. However, it would not explain why the session-spacing manipulation in Experiment 1 had also the same effect. Moreover, it would similarly fail to account for the results of Experiments 4 and 5, in which there was a four-fold difference in the total number of trials between the two groups.

The possibility that the session-number manipulation resulted in the TPSE is rather slim, but it cannot be excluded. If the TPSE had been obtained in our experiments, it would have expedited conditioning in the respective group(s), an effect that we did not

observe. Moreover, in Experiments 1 and 2, performance during the first two trials of extinction was equal for the group with the many trial-poor sessions and the group with the few daily trial-rich sessions. Particularly in Experiment 1, conditioned responding for the former group at the start of extinction was very low, arguing against the possibility that this group was better conditioned than its control. Nevertheless, our experiments were not designed to address the possibility of differential conditioning, and thus did not include proper common tests for group comparisons during acquisition. Therefore, it is still possible that the effect was present but we failed to detect it.

Number of Sessions and the Temporal Weighting Rule

Experiments 4 and 5 provided evidence that the number of trials within a session is not the primary determinant of spontaneous recovery. Instead, the number of sessions appeared to be a more important parameter. There is reason to believe that the session may be an important unit of experience. The observation of the TPSE (Kehoe & Macrae, 1994; Levinthal, 1973; Papini & Dudley, 1993; Papini & Overmier, 1985) supports this argument. Moreover, changes in conditioned responding usually occur at the beginning of a new session (Gallistel et al., 2007b; Papachristos & Gallistel, 2006). Finally, using between- and within-subjects experiments with rats, mice, and pigeons, Gottlieb (in press) found that an eight-fold variation in the number of trials during acquisition did not have an effect on conditioning. The majority of his results were better accounted for by time-centered models of conditioning (e.g., Gallistel & Gibbon, 2000), for which the number of trials within a session is not a meaningful parameter, as opposed to trial-centered models.

Of all the models of spontaneous recovery that have been presented, TWR is the only one that can account for all the findings obtained from manipulating the number of sessions (but not their spacing). Therefore we believe that a fully successful account of spontaneous recovery should incorporate some of the features of TWR.

Perhaps its most attractive feature is that it is time-scale invariant. The term refers to the fact that if one multiplies all the temporal intervals in a conditioning protocol by the same factor, the result does not change (Gallistel, 2000). In other words, the time unit in which one measures the duration of trials and intertrial intervals (ITIs) is not important. What is relevant is the proportion between the two intervals (and proportions are unitless). Time-scale invariance has been obtained in many aspects of conditioning, ranging from the timing of the conditioned response (Gibbon, Baldock, Locurto, Gold, & Terrace, 1977) to even the rate of acquisition (the inverse of the number of reinforcements to acquisition) (Gallistel & Gibbon, 2002). Its observation has been proven awkward for traditional associative models, because most of them divide time into discrete trials (Gallistel, 2000). The notion of a trial assumes that there is a critical time-window in which the CS and US have to occur in order to become associated. Accordingly, if one prolongs only the duration of the trial (lengthens the time window while keeping the ITI fixed), the rate of conditioning becomes slower. The flaw in such an account is the idea of a trial (critical time window) because it imposes a time scale on the conditioning protocol. Thus, it becomes unable to explain why lengthening the trial and the ITI by the same factor (e.g., quadrupling both intervals) does not affect the rate of conditioning.

TWR is time-scale invariant because according to the model what matters is not the absolute but the relative time distance of the two experiences (acquisition and extinction) from testing. Since each experience is weighted by its recency (the inverse of the time passed since that experience), changing the time units will not change the result of the weighted average. In other words, if a single acquisition session ended 4 hrs while an extinction session ended 2 hrs before testing, the quality of the CS as a predictor of reward would be the same as if the time since acquisition was 4 days, while since extinction 2 days.

Note that any account of spontaneous recovery that invokes a time-changing process (i.e., fading of inhibition, change in temporal context, rise of activation threshold, etc.) would fail to offer time-scale invariance, because the time-changing process imposes a time scale on the experimental protocol. In other words, such a process subjects the appearance of spontaneous recovery to a fixed delay. For example, if time changes the temporal context, it must do so within a specific delay. Of course, because none of these models are given quantitative expression, the only way one can infer how much a delay is needed between extinction and testing for the appearance of spontaneous recovery is by inspecting the behavioral data. Suppose that one finds that a delay *t* is sufficient to produce spontaneous recovery. A subsequent change (e.g., lengthening) in the interval between acquisition and extinction, if adequately big, could hurt the appearance of spontaneous recovery, as Rescorla (2004b) showed. Thus, the same delay *t* from extinction that once was sufficient to cause a change in the temporal context now is unable to produce the same effect. Again, the flaw relies on the use of a single time changing process of fixed temporal dynamics.

A New Hypothesis

Recently, Papachristos and Gallistel (2006) offered an alternative interpretation of spontaneous recovery that shares some features with TWR. According to their proposition, spontaneous recovery is a behavioral indication of the animal's uncertainty about the current status of the signal-reward relation. The animal is assumed to track the degree of temporal stability (stationarity) in its world. For a substantial period (acquisition) it experiences a positive predictive relation between a signal and a reward every time it is placed in the experimental chamber. Subsequently, there is a period (extinction, usually shorter in length) in which the signal is not followed by the reward. In the course of extinction the animal detects the change in the contingency and at some point it stops responding to the signal. Following a resting interval, the animal is placed back on the same experimental chamber and presented with the same signal. There is no *a priori* way of knowing whether the stimulus-reward relation will hold. It is quite possible that a single failure (extinction) of an otherwise stable process (CS predicts US) has outlived its lifespan. Based on this expectation, the animal decides to respond and recovery of the extinguished behavior is observed.

Inherent in the above explanation is the assumption that animals act as change detectors. This assumption has been supported by recent findings on matching in the mouse (Gallistel et al., 2007b). According to these findings mice track the rates of reward obtained from two independent locations and allocate their investment (time visiting each location) accordingly, so that the returns (income divided by invested time) from the two locations are equated. Transient changes in the local rates of reward, due to the random-rate process that determines the interval after which a visit in a location will pay off, are

detected as fast as they in principle could, and mice immediately adjust their policy according to the updated rates.

The temporal stability hypothesis also depends on the assumption that during acquisition the animals obtain an estimate of the rate of US occurrence in the presence of the CS (Gallistel, 1990). Deviations from this estimate (such as an extinction contingency), depending on how long they last, can be treated as either noise (short-lived changes) or as evidence of a longer-lasting change in the signal-food contingency (if they persist). When an animal has had a history of conflicting experiences, its decision on whether the post-change contingency still holds depends primarily on two factors: its *relative recency* compared to the recency of the pre-change experience and its *relative duration* (how long it lasts relative to the duration of the pre-change experience). A relatively recent change is more likely to be still in effect, thus shortly after extinction responding is still depressed. However, if the post-change experience lasted considerably shorter than the pre-change experience, it is very likely that its effect was only temporary and that things may have regressed to the longer-lasting pre-change experience. Under these conditions, spontaneous recovery is observed.

Recency, similarly to TWR, is simply the inverse of the elapsed time since each observation. It requires that animals keep track of the time since each foraging experience, an assumption with strong empirical support (Clayton & Dickinson, 1998, 1999; Clayton et al., 2001, 2003). Relative *duration* is based on the assumption that animals add intervals between similar foraging experiences; that is, they calculate the duration of a period under which there is no change in contingencies. The intervals that enter into this computation are not the intervals between trials but between sessions.

Unlike intersession intervals, ITIs are too short to allow any confident estimates about temporal stationarity. After all, an event that is repeated after 5 min does not allow as strong inferences about its generality as an event that is repeated after 5 days. The latter is more likely to be a stable event because it was true for a much longer period.

In summary, our hypothesis about spontaneous recovery is based on TWR's assumption that experiences are weighted according to their recency. However, our hypothesis also emphasizes the importance of duration of each of the conflicting experiences. The duration of an experience depends mainly on the temporal distribution of the occasions (sessions) of that experience. Thus, unlike TWR, our hypothesis predicts that, other things being equal, a more widely spaced acquisition protocol should increase the magnitude of spontaneous recovery after extinction because it increases the weight of the long-lasting acquisition regime.

The Abruptness of Extinction

In all of the experiments that included a between-subjects design, extinction was found to be abrupt. The median mouse took less than 6 trials to initiate extinction, but once it did, it completed it almost immediately. The step-like pattern of the individual extinction curve is strikingly similar to the pattern of acquisition, as has been reported in previous research in our lab (Gallistel et al., 2004; Papachristos & Gallistel, 2006). This resemblance invites discussions on the underlying process that mediates extinction learning. However, before doing so, it is important to address one methodological issue.

To quantify extinction, we used a modified version of a change-point detection algorithm, recently developed by Gallistel (Gallistel et al., 2001). The algorithm’s output

depicts responding as a step-plot, with each step indicating the mean elevation score during the trials that comprised the step. Therefore, the algorithm is biased in characterizing responding as a step-like process. However, nothing inherent in the algorithm constrains the number and the height of those steps. If the data showed a gradual decrease in the vigor of responding, the algorithm would portray that as a series of downward steps. Unfortunately, we were unable to employ the change-point algorithm on the group-average data, because the Skellam distribution is only defined for integer-valued data. However, in the past we showed that the original version of the algorithm was able to capture the smooth negatively accelerated transition of the group-average acquisition curve as a series of upward steps (Papachristos & Gallistel, 2006).

The observed abruptness of extinction does not necessarily imply that it is an all or none process. The function underlying extinction learning (e.g., the growth of associative inhibition) could be of any form but the abruptness of the behavioral data does not allow us to estimate either its shape or its parameters. If the underlying function is gradual, the performance function that maps it onto observable behavior must be strongly nonlinear. For the point of the performance function (threshold) that the underlying variable must reach before extinction can be observed on behavior lies very close to the point at which its behavioral effect saturates. The threshold and the saturation point form a window that allows one to estimate the progress of the underlying function by inspecting the behavioral data. However, this window appears to be very narrow. In other words, it only allows us to observe such a limited part of the underlying growth function that we cannot extract confident estimates of its shape or its parameters.

Alternatively, the observed abruptness in extinction responding may be taken as evidence for a similarly abrupt underlying extinction process. Theories like the Rate Estimation Theory (RET) (Gallistel & Gibbon, 2000) utilize a decision threshold to map learning onto behavior. According to RET, when the strength of the evidence that the CS does not predict the US anymore exceeds some decision threshold, the animal stops responding altogether. Thus, RET anticipates that extinction, like acquisition, is abrupt. Nonetheless, in the absence of data from experiments with different procedures and/or different species, the abruptness of extinction cannot yet be the default assumption.

Summary

The experiments described in this dissertation measured the effects of trial-spacing manipulations on spontaneous recovery from extinction. Using a magazine approach procedure with mice we observed that distributing the same number of trials across more sessions or spacing the same number of sessions more widely increased spontaneous recovery. The effect of the session number manipulation appeared to be, if anything, stronger than the session-spacing manipulation. Subsequent experiments showed that the number of sessions might be a more important parameter for spontaneous recovery. When number of sessions was equated, a four-fold decrease in the number of trials did not affect spontaneous recovery. Having four times more sessions and four times fewer trials was more effective than the reverse in promoting spontaneous recovery, although the effect was not as strong as when the total number of trials was equated. Finally, using a change-point detection algorithm, we quantified extinction at the level of the individual subject. Asymptotic extinction appeared abruptly after a few trials,

indicating that the gradual form of the group-average extinction curve, at least in this procedure, is probably an averaging artifact.

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Figure Legends

Figure 1. Extinction step-plots from 12 mice of Experiment 1, as generated by the change-point algorithm. Each step shows the mean elevation score during the trials between successive change points in the cumulative record (not shown).

Figure 2. Mean number of trials of the three groups of Experiment 1 on the extinction measures of onset latency, dynamic interval, and extinction trial. The error bars denote one standard error of the mean.

Figure 3. Mean elevation scores during the first and last two-trial extinction blocks, and each trial of the two spontaneous recovery tests, for Experiment 1. The error bars indicate one standard error of the mean.

Figure 4. Mean number of pokes during the pre-CS periods of the first and last two-trial extinction blocks, as well as of each trial of the two spontaneous recovery tests, for the groups of Experiment 1. The error bars indicate one standard error of the mean.

Figure 5. Extinction step-plots from all mice of Experiment 2, as generated by the change-point algorithm. Each step shows the mean elevation score during the trials between successive change points in the cumulative record (not shown).

Figure 6. Mean elevation scores during the first and last two-trial extinction blocks, and each trial of the two spontaneous recovery tests, for Experiment 2. The error bars indicate one standard error of the mean.

Figure 7. Mean elevation scores during extinction, and each trial of the two spontaneous recovery tests, for the two stimuli of Experiment 3.

Figure 8. Cumulative distributions of elevation scores during the first trial of the 4-wk test with the CS that had been presented 8 times on every conditioning session (Routine CS) and the CS that had been presented 32 times on only a quarter of the conditioning sessions (Intermittently Frequent CS). At any datum on the x-axis, the distributions show the number of mice that had an elevation score less than or equal to that particular datum.

Figure 9. Extinction step-plots from all subjects of Experiment 4, as generated by the change-point algorithm. Each step shows the mean elevation score during the trials between successive change points in the cumulative record (not shown).

Figure 10. Mean number of trials of the two groups of Experiment 4 on the extinction measures of onset latency, dynamic interval, and extinction trial. The error bars denote one standard error of the mean.

Figure 11. Mean elevation scores during the first and last two-trial extinction blocks, and each trial of the two spontaneous recovery tests, for Experiment 4. The error bars indicate one standard error of the mean.

Figure 12. Extinction step-plots from all subjects of Experiment 5, as generated by the change-point algorithm. Each step shows the mean elevation score during the trials between successive change points in the cumulative record (not shown).

Figure 13. Mean number of trials of the two groups of Experiment 5 on the three extinction measures of onset latency, dynamic interval, and extinction trial. The error bars show one standard error of the mean.

Figure 14. Mean elevation scores during the first and last two-trial extinction blocks, and each trial of the two spontaneous recovery tests, for Experiment 5. The error bars indicate one standard error of the mean.

Figure 1

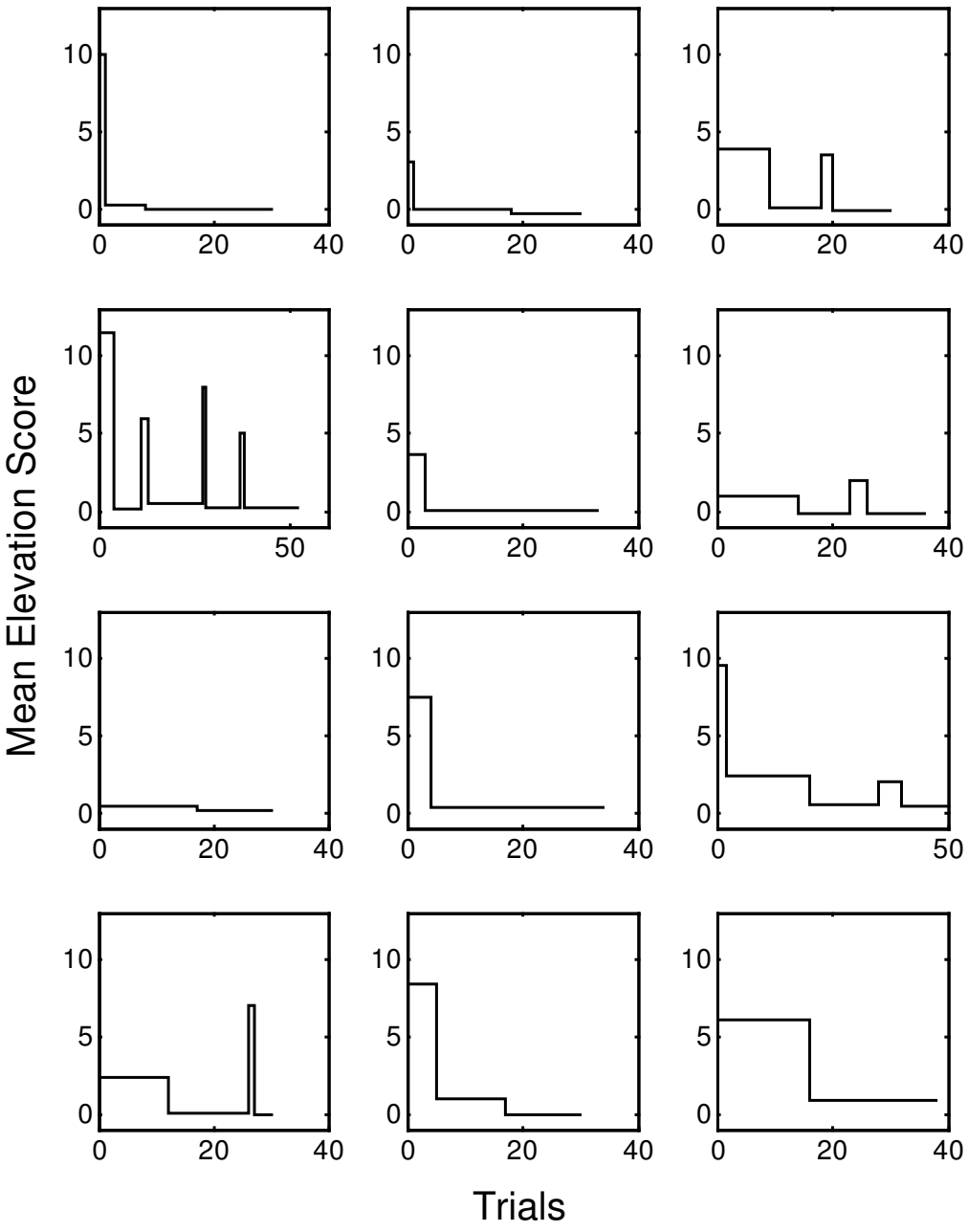


Figure 2

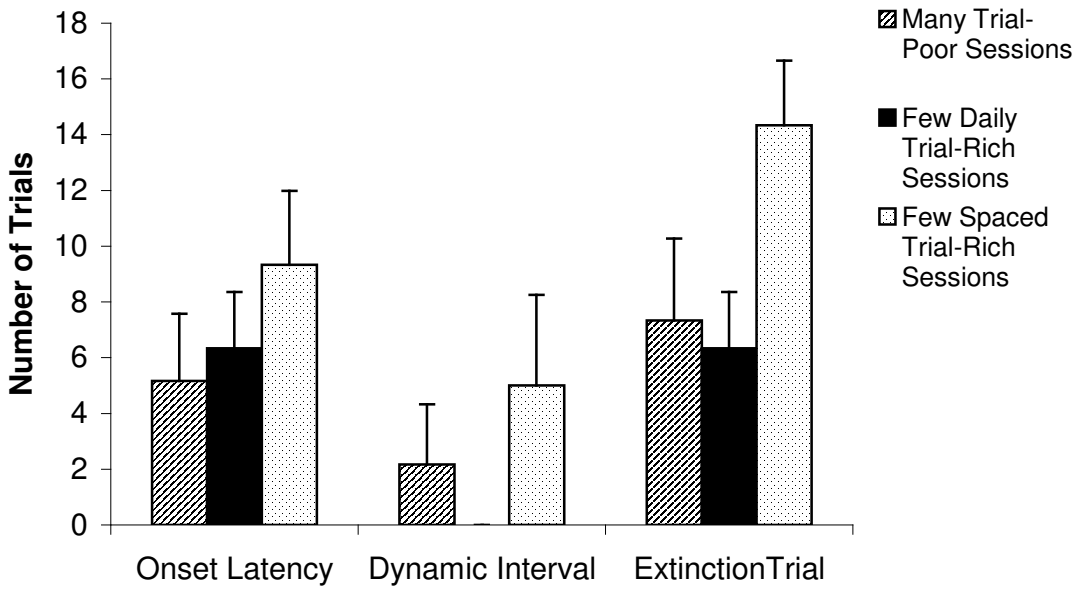

Number of Trials
18
16
14
12
10
8
6
4
2
0
Onset Latency
Dynamic Interval
ExtinctionTrial
Many Trial-Poor Sessions
Few Daily Trial-Rich Sessions
Few Spaced Trial-Rich Sessions

Figure 3

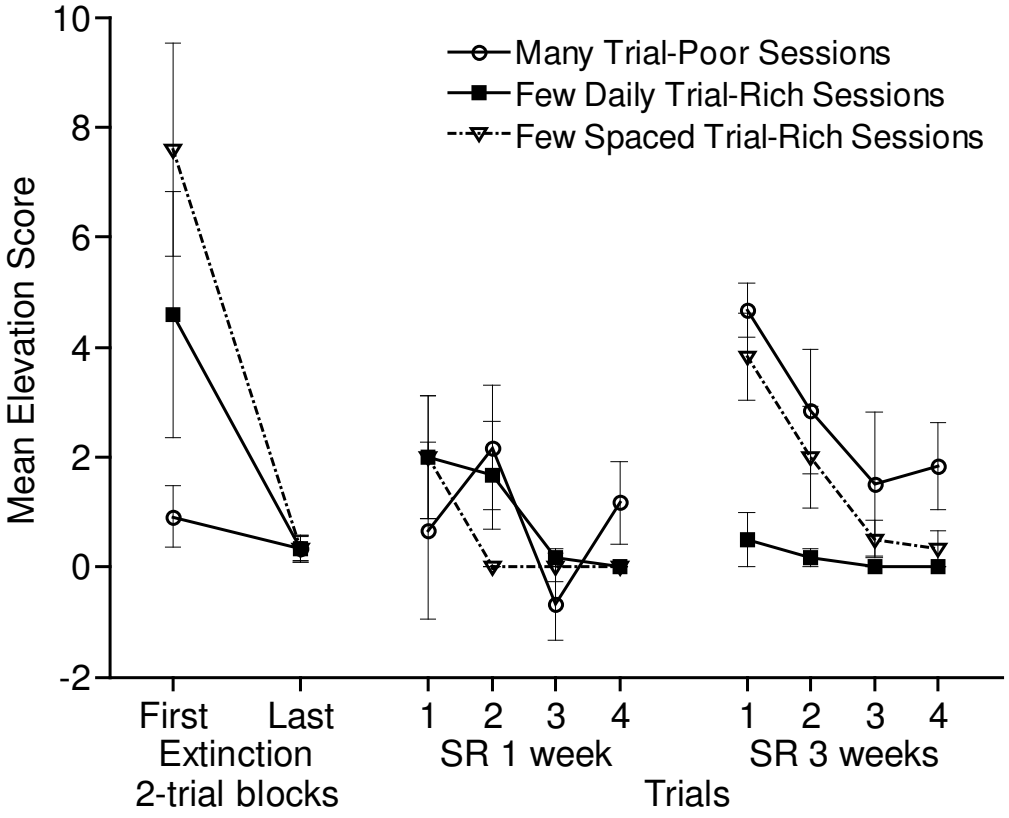

Many Trial-Poor Sessions
Few Daily Trial-Rich Sessions
Few Spaced Trial-Rich Sessions
Mean Elevation Score
10
8
6
4
2
0
-2
First
Last
Extinction
2-trial blocks
1
2
3
4
SR 1 week
1
2
3
4
SR 3 weeks
Trials

Figure 4

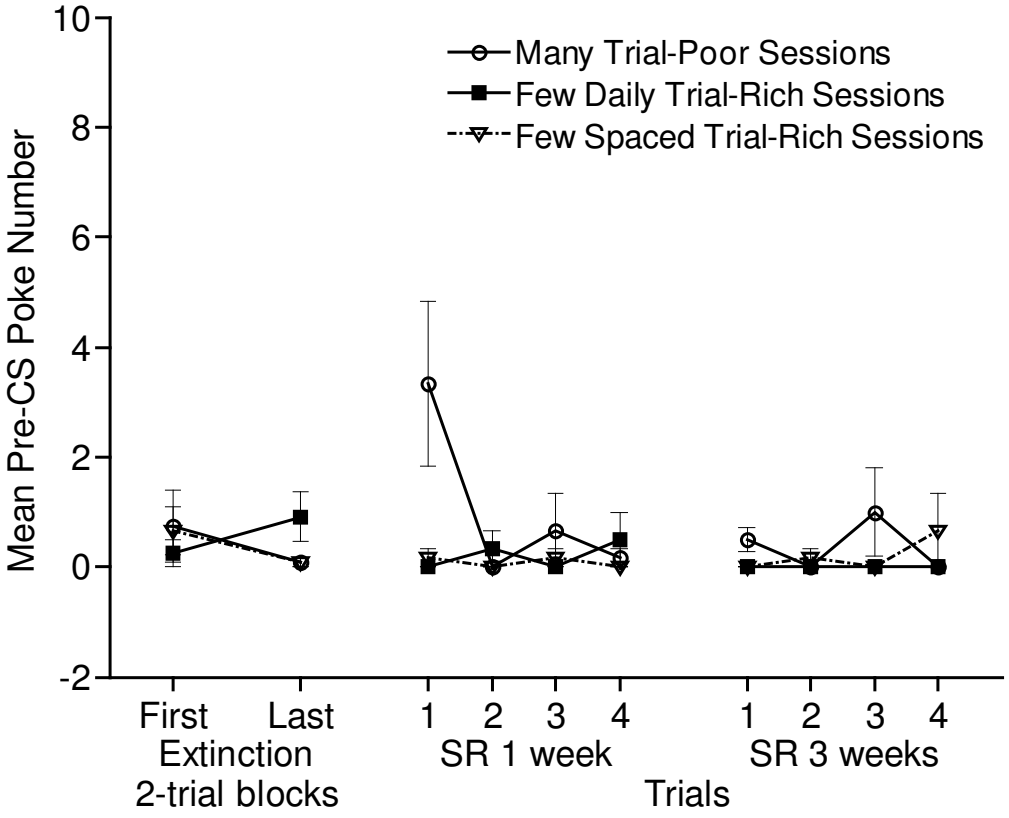

Many Trial-Poor Sessions
Few Daily Trial-Rich Sessions
Few Spaced Trial-Rich Sessions
Mean Pre-CS Poke Number
10
8
6
4
2
0
-2
First
Last
Extinction
2-trial blocks
1
2
3
4
SR 1 week
1
2
3
4
SR 3 weeks
Trials

Figure 5

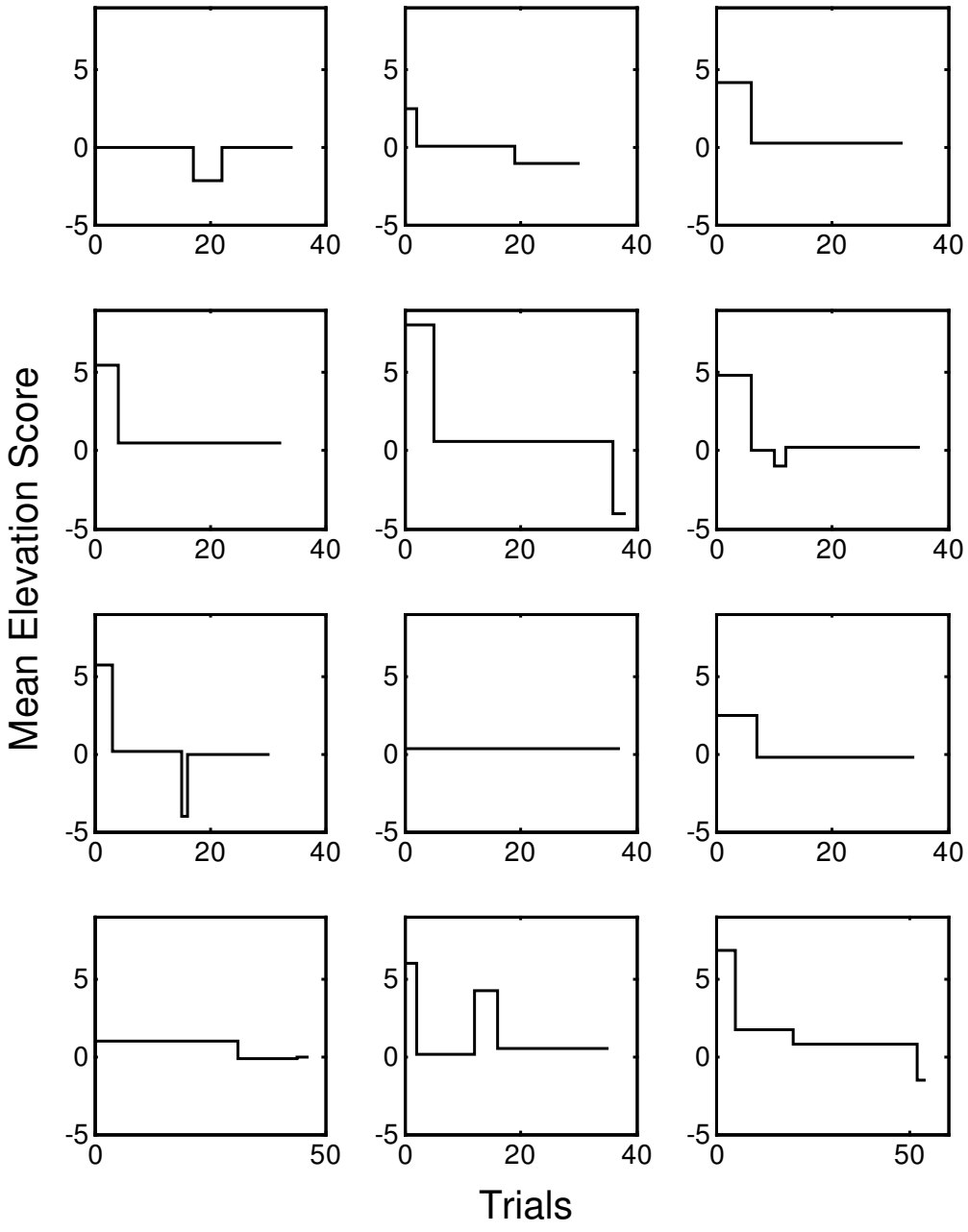


Figure 6

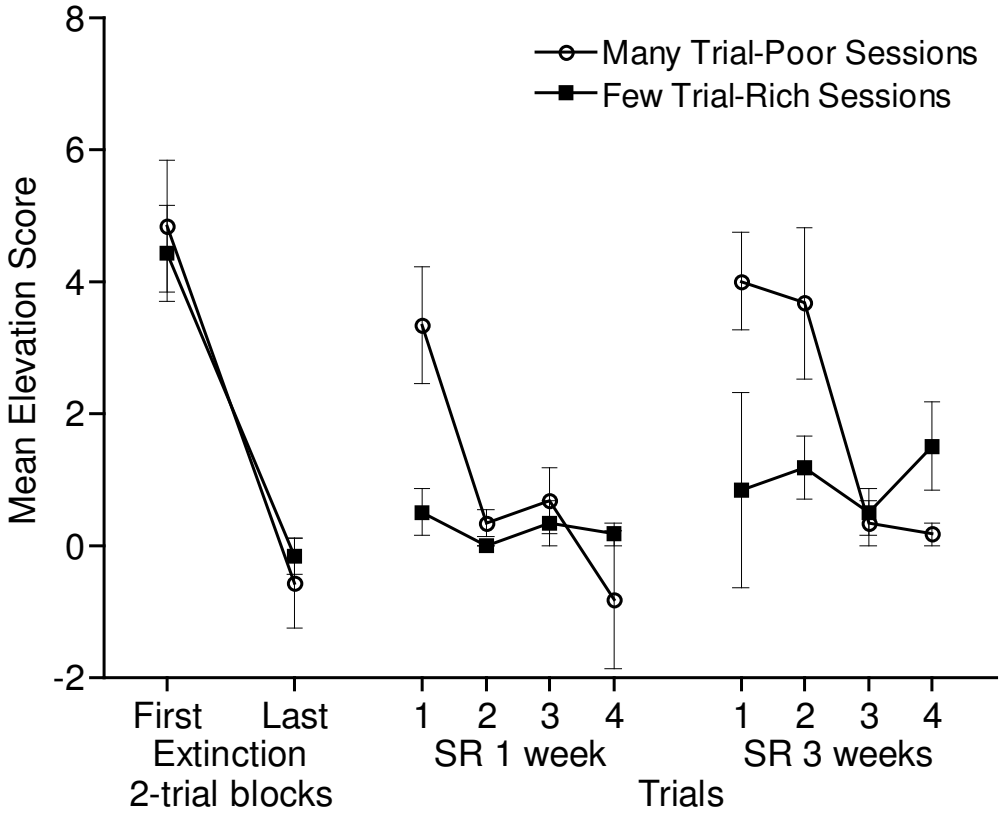

Many Trial-Poor Sessions
Few Trial-Rich Sessions
Mean Elevation Score
8
6
4
2
0
-2
First
Last
Extinction
2-trial blocks
1
2
3
4
SR 1 week
1
2
3
4
SR 3 weeks
Trials

Figure 7

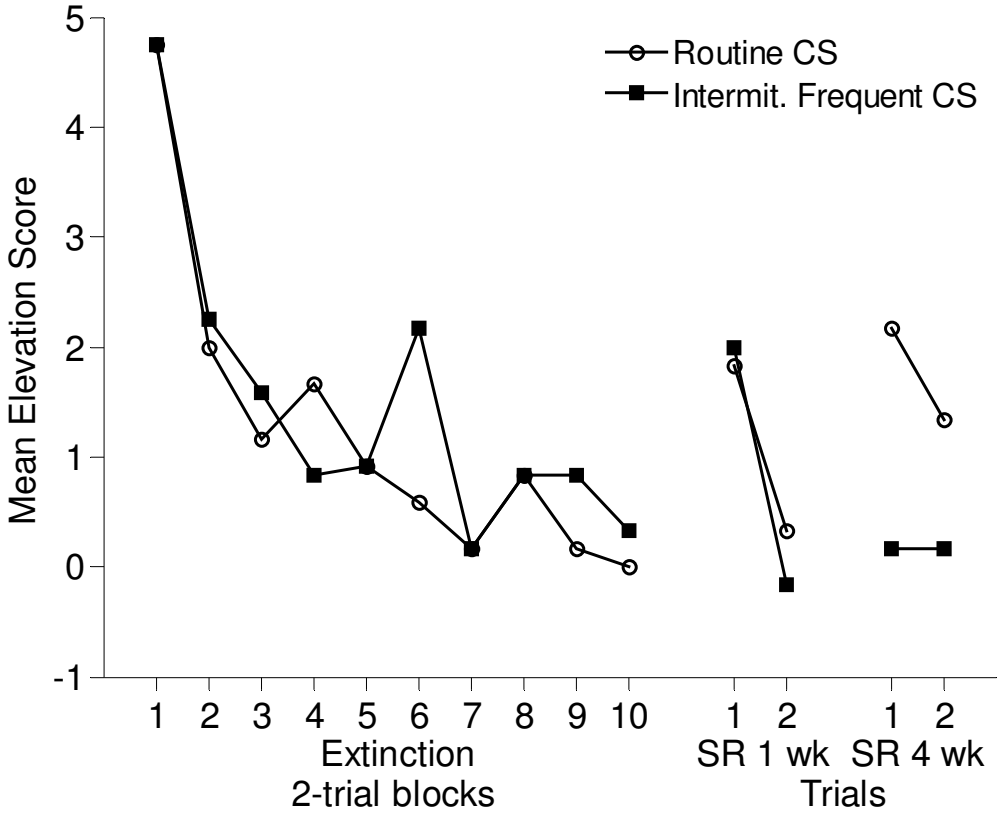

Routine CS
Intermit. Frequent CS
Mean Elevation Score
5
4
3
2
1
0
-1
1 2 3 4 5 6 7 8 9 10
Extinction
2-trial blocks
1 2
SR 1 wk
1 2
SR 4 wk
Trials

Figure 8

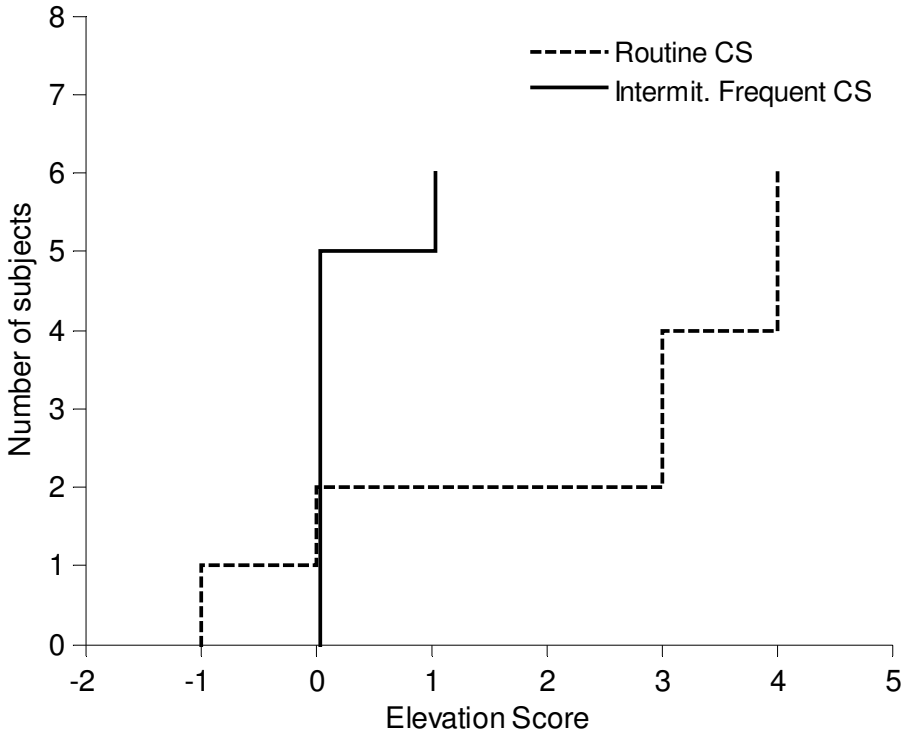

Routine CS
Intermit. Frequent CS
Number of subjects
Elevation Score
0
1
2
3
4
5
6
7
8
-2
-1
0
1
2
3
4
5

Figure 9

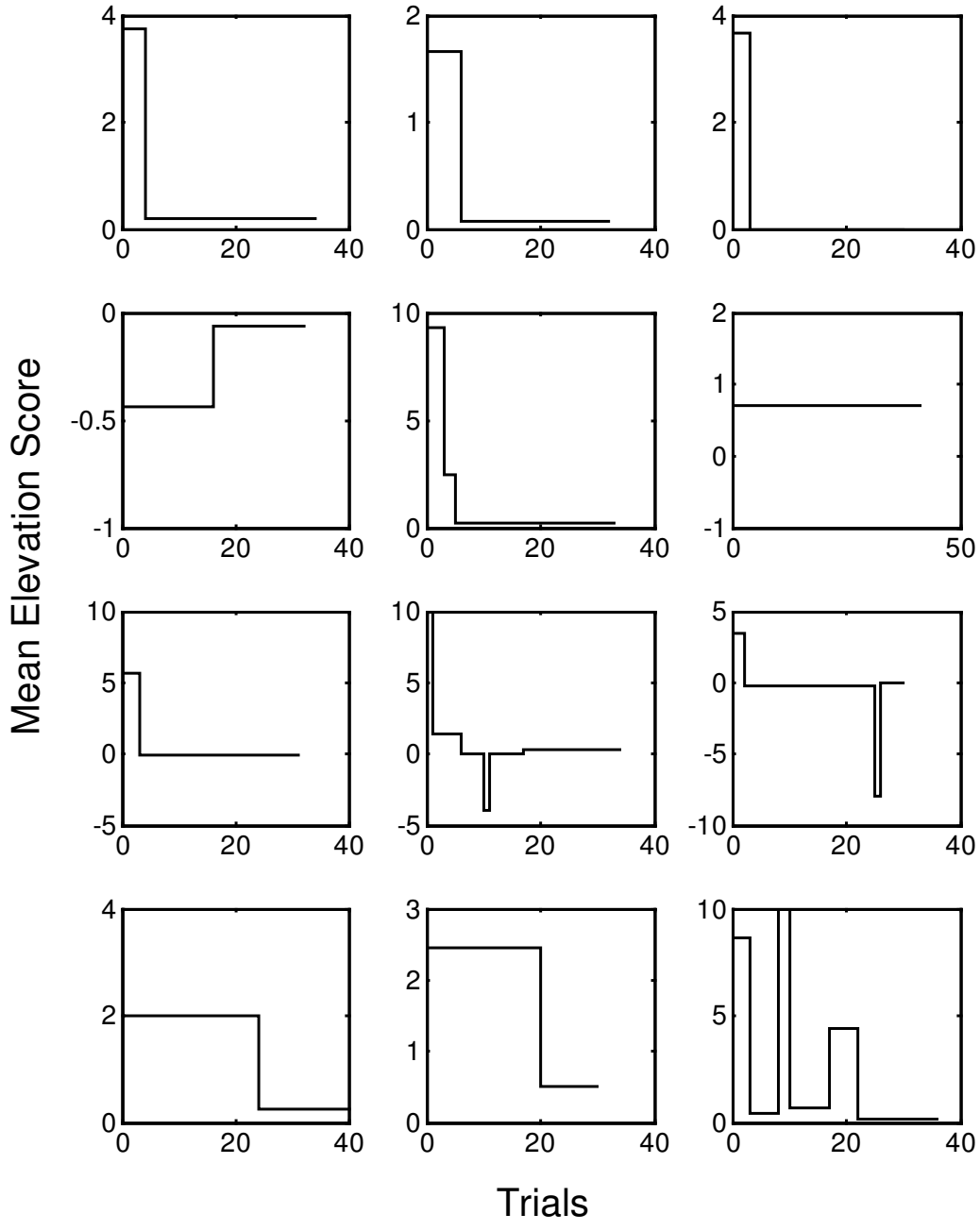


Figure 10

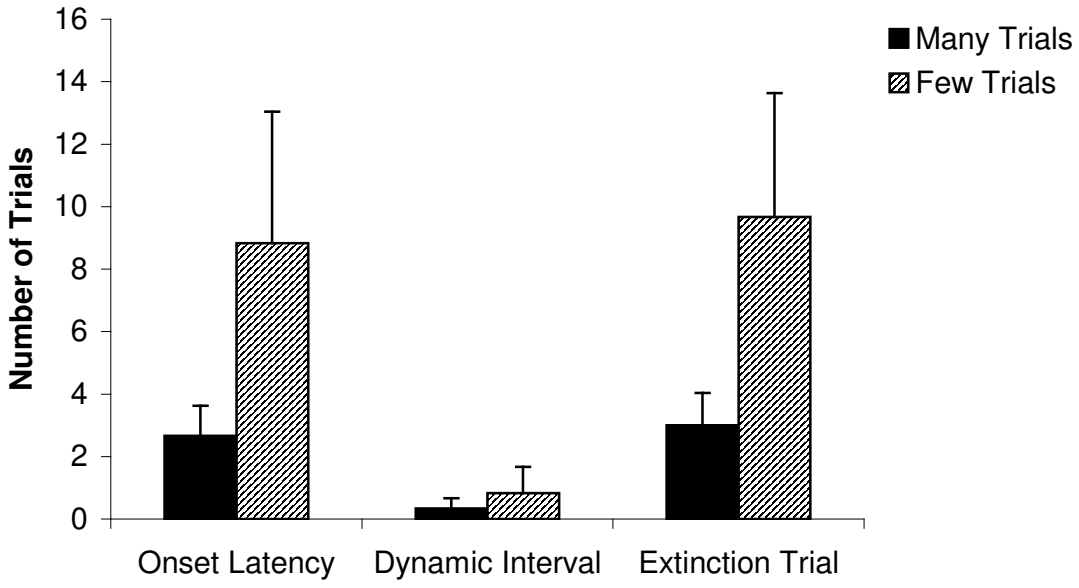

16
14
12
10
8
6
4
2
0
Number of Trials
Many Trials
Few Trials
Onset Latency
Dynamic Interval
Extinction Trial

Figure 11

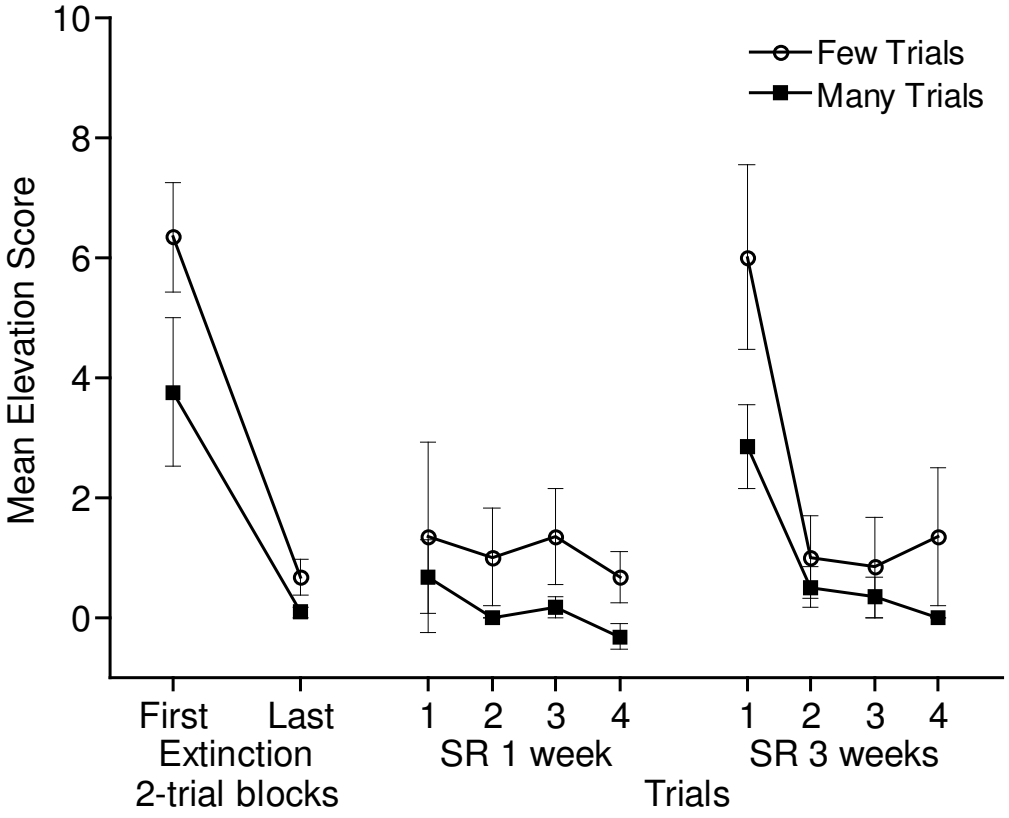

10
8
6
4
2
0
Mean Elevation Score
Few Trials
Many Trials
First
Last
Extinction
2-trial blocks
1
2
3
4
SR 1 week
1
2
3
4
SR 3 weeks
Trials

Figure 12

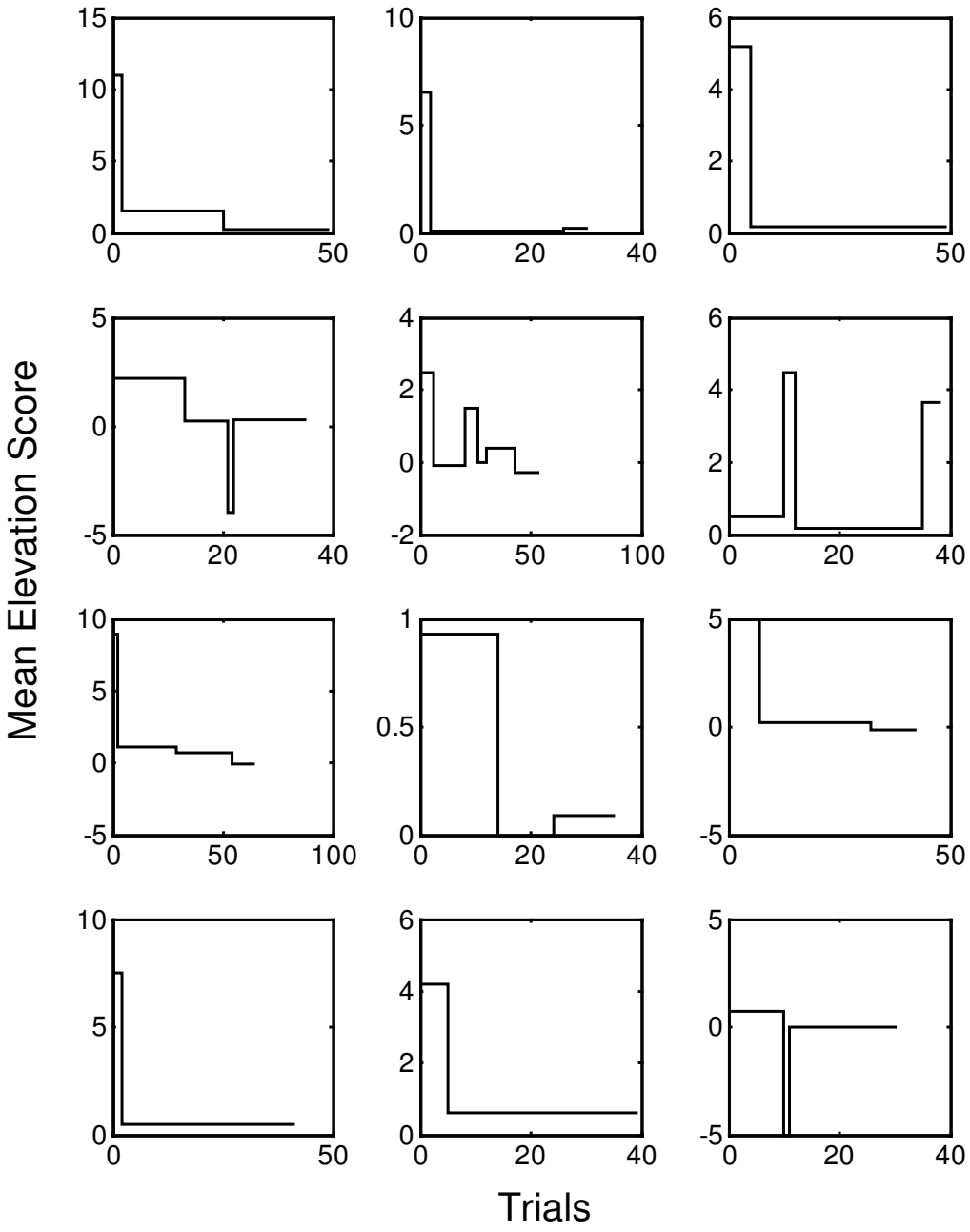


Figure 13

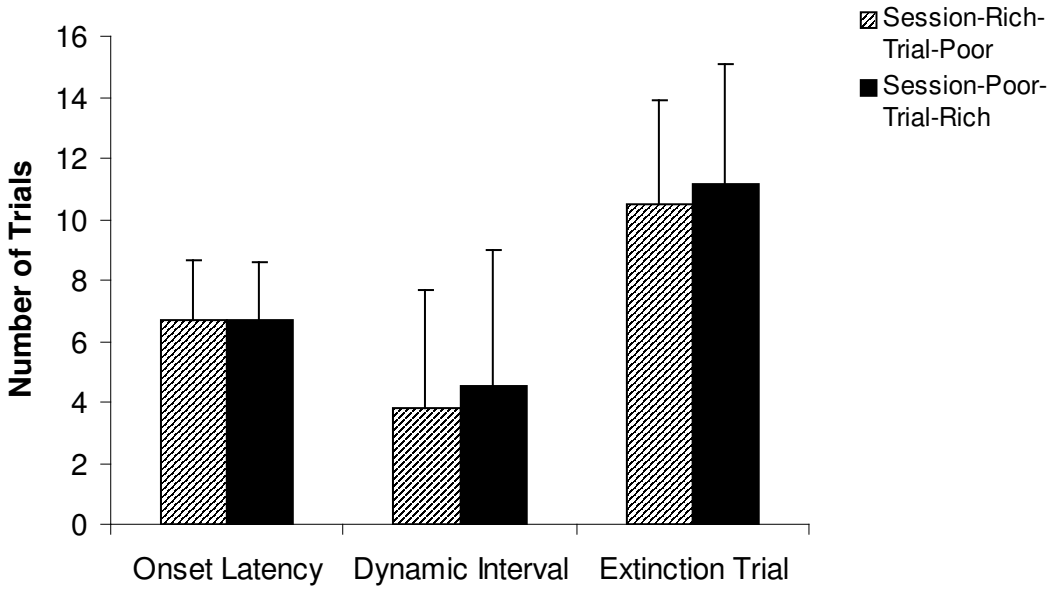
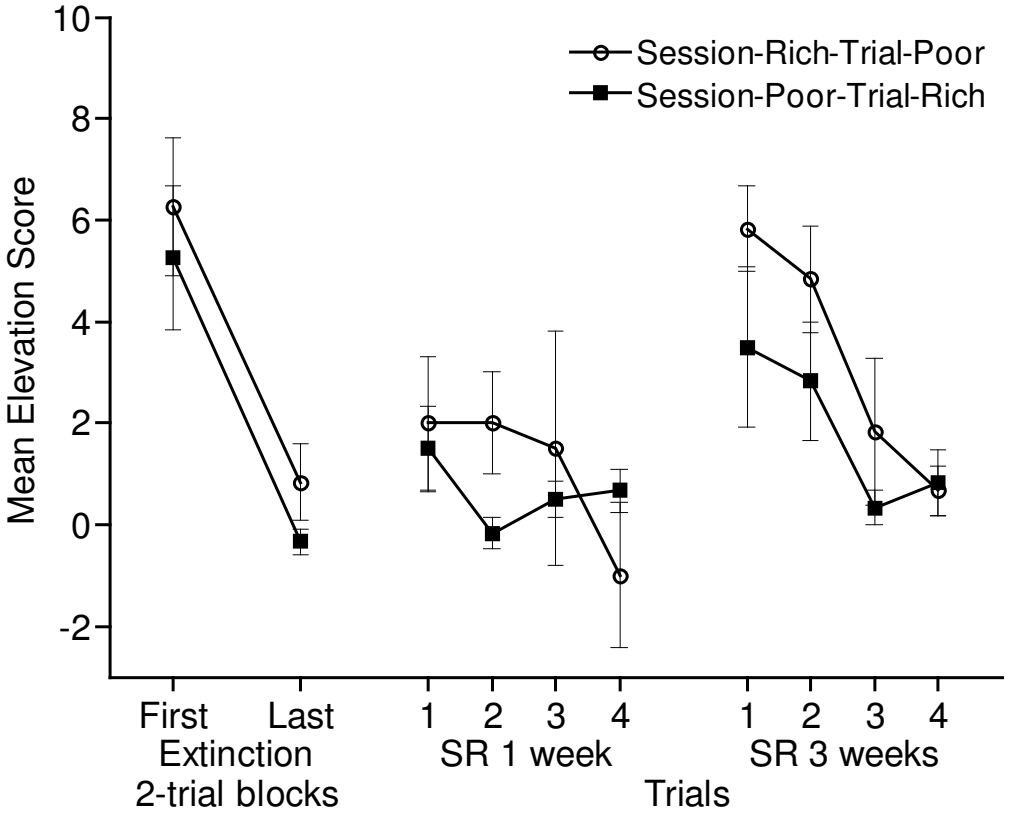

Number of Trials
16
14
12
10
8
6
4
2
0
Onset Latency
Dynamic Interval
Extinction Trial
Session-Rich-Trial-Poor
Session-Poor-Trial-Rich

Figure 14


Session-Rich-Trial-Poor
Session-Poor-Trial-Rich
Mean Elevation Score
10
8
6
4
2
0
-2
First
Last
Extinction
2-trial blocks
1
2
3
4
SR 1 week
1
2
3
4
SR 3 weeks
Trials

CURRICULUM VITAE

Efstathios B. Papachristos
Phone: 908-616-0495
E-mail: papachst@eden.rutgers.edu

Education:

2008	Ph.D., Rutgers University, Behavioral Neuroscience
2005	M.S., Rutgers University, Biopsychology and Behavioral Neuroscience
2001	B.A., University of Athens, Greece, Psychology

Honors and Awards:

2002-2007	Fulbright Scholarship
2002-2005	Alexandros S. Onassis Public Benefit Foundation Scholarship
2002-2005	Rutgers University Graduate Fellowship
2005	James Leathem Scholarship
2006-2007	Rutgers University Graduate Fellowship
2006	Gerondelis Foundation Graduate Grant

Teaching Experience:

2005-2006	Laboratory in Conditioning and Learning
2006	Conditioning and Learning

Publications:

Papachristos, E.B., & Gallistel, C.R. (2006). Autoshaped head poking in the mouse: A quantitative analysis of the learning curve, *Journal of the Experimental Analysis of Behavior, 85, 293*-308.

Gallistel, C.R., King, A.P., Gottlieb, D., Balci, F., Papachristos, E.B., Szalecki, M., & Carbone, K.S. (2007). Is matching innate? *Journal of the Experimental Analysis of Behavior, 87,* 161-199.

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