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Publication Date:
2007

DOI:
https://doi.org/10.26190/unsworks/17505

The role of the intertrial interval in the loss of context conditioned fear responses.

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This thesis has been submitted in fulfillment of the requirements for the degree of Doctor of Philosophy (Ph.D.)

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2007

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Abstract

Eight experiments examined the role of the intertrial interval in the extinction of conditioned fear to a context. Rats were shocked in one context (A) but not in another (B) and freezing responses to Context A were extinguished. The interval between extinction trials was spent in the home cages. Experiments 1a and 1b showed that massed extinction trials produced better response loss but worse learning than spaced trials. Experiment 2 demonstrated that the interval between the final extinction trial and test mediated the level of responding on a test exposure. Experiments 3 and 4 showed that the duration of the extinction trial affected long term response loss, whereby long durations facilitate response loss compared to shorter durations. Subsequent experiments (Experiments 5 to 8) demonstrated that the first in the series of massed extinction trials reduced the associability of subsequent trials. Associability was restored by alternating extinction trials between Context A and Context B. The results are discussed in terms of the role accorded to self-generated priming in the models developed by A. R. Wagner (1978; 1981).

Acknowledgements

First and foremost I would like to thank my supervisor, Fred Westbrook, for his support and encouragement throughout my candidature. His commitment to furthering my understanding of science was relentless and greatly appreciated. I would also like to thank the School of Psychology for providing me with the means and opportunity to complete this degree. Finally, I would like to thank my friends and family for their endless support, encouragement and friendship throughout the course of my degree.

Publications

The material from this thesis has been submitted as:

Li, S. H., & Westbrook, R. F. (in press). Massed extinction trials produce better loss of context conditioned fear responses but worse learning than spaced trials. *Journal of Experimental Psychology: Animal Behavior Processes.*

CONTENTS

CHAPTER 1

Introduction

The typical procedure used to produce extinction of Pavlovian conditioned responses involves exposing subjects to a positive contingency between a stimulus and a motivationally significant outcome [an unconditioned stimulus (US)]. Then this contingency is broken by presentation of the now conditioned stimulus (CS) in the absence of its associated US. The well-documented outcome of such CS alone presentations is a loss or extinction of conditioned responding (for review see Rescorla, 2001a; Delamater, 2004). Although well-documented, the processes underlying this extinction of responding remain poorly understood. Understanding these processes is important for several reasons. First, the extinction of conditioned responses is a fundamental observation in Pavlovian conditioning. Any theory of the processes by which a contingent relation results in the acquisition of responding must address the loss of these responses when this relation is broken. Second, the simplicity of the procedure (a stimulus is presented) and the reliability of the outcome (loss of responding) offers the prospect of identifying the underlying neurobiological substrate. Finally, extinction is a critical component of exposure-based treatments of various disorders in people, including anxiety and addictions. Therefore, understanding the processes underlying extinction will contribute towards the development of theoretical treatments of Pavlovian conditioning, identify what has to be explained at the neurobiological level, and result in more effective treatment of various learned disorders.

In order to understand the processes underlying extinction attention must be paid to two points in time: t1, the time at which the animal is given the opportunity to learn (i.e., CS alone presentations) and t2, the time at which what the animal has learnt is assessed (common test; see Rescorla & Holland, 1976; Rescorla, 1988). This allows a common assessment of what is learnt under differing extinction conditions. We arrive therefore at three questions: what are the conditions of CS alone presentations at time 1 that produce response loss at time 2? What are the conditions of test that reveal the loss in conditioned responding? And what are the processes of learning that mediate the response loss?

Conditions of training

Breaking the positive contingency by CS alone presentations is but one of several procedures that reduce conditioned responding. Others include additional CS-US training in the presence of another excitatory CS (overexpectation), training the CS with an affectively opposite US (counterconditioning), and breaking the CS-US contingency by presenting the CS and the US in a random fashion.

The Rescorla-Wagner model (Rescorla & Wagner, 1972) provides a general account of such response loss. It identifies learning with a single construct, associative strength (V), and holds that changes (Δ) in V are given by Equation 1:

$$\Delta V_{CS} = \alpha\beta(\lambda - \Sigma V),$$

where α and β are rate parameters associated with the target CS and US, respectively, ΣV is the summed Vs of all CSs present on a given trial, and λ (which has a positive value on occasions when the US occurs and a value of zero on occasions when the US does not occur) is the asymptotic associative strength

supported by the US. Thus, acquisition of a CS-US association is regulated by the positive discrepancy between λ and ΣV, and loss of that association is a consequence of the negative discrepancy between the current value of λ and ΣV.

The omission of a predicted US produces a negative discrepancy but the occurrence of a US can also produce that discrepancy. One illustration is the phenomenon of overexpectation. Rescorla (1970) separately trained light-shock and tone-shock associations. Then one group of rats received additional light-tone compound conditioning trials, while other groups either continued to receive tone-shock pairings or did not receive additional training. Subsequent tests of the tone revealed less conditioned responding (interruption of appetitive responding) by the rats trained with the tone in combination with the light than by those subjected to additional tone-shock pairings or those not receiving any additional training. Thus, training a stimulus in compound with another stimulus that has been trained separately with the same US reduced conditioned performance (see also Kehoe & White, 2004; Lattal & Nakajima, 1998, Rescorla, 2006; 2007). The Rescorla-Wagner model predicted this effect. The presentation of two excitatory stimuli in compound increases the ΣV while maintaining the original λ value (because the same US is presented). This results in a negative discrepancy between λ and ΣV that lowers the associative strength of the stimuli present on that trial.

Another way of breaking the contingency is to uncouple the CS from the US by presenting each in a random relation to the other. For example, Keller, Ayres and Mahoney (1971) presented the CS and the US in a random fashion following a conditioning session. Initially rats were trained to lever press for a sucrose reward. They were then exposed to a conditioning procedure in which the noise was paired

with shock. Subsequently, the noise and shock were presented in a random, non-contingent fashion. The extent to which CS presentations suppressed lever pressing for the sucrose reward was measured. It was observed that the application of the random procedure produced initial responding to the CS that reduced as the session proceeded (see also, Ayres, Benedict & Witcher, 1975; Rescorla, 1972). These results can be understood in terms of the associative strength of the background context during the course of non-contingent presentations of the CS and US. Specifically, exposure to random presentations of the CS and US meant that reinforcement of the CS early in the session occurred when the background context has not been fully conditioned thus preserving the association between the CS and the US. However, as the session proceeded the background became fully conditioned so that presentation of the CS in conjunction with the background resulted in a level of conditioning (ΣV) that exceeded the value supported by the reinforcer (λ). The negative discrepancy between λ and ΣV thus results in a decrease in the associative strength of the CS thereby reducing conditioned responding over the course of random CS and US presentations (but see Rescorla, 2000).

Counterconditioning is the replacement of the original outcome with a second outcome that is affectively opposite (e.g., replacing a shock US with a food US). This produces a loss in the originally trained response. The Rescorla-Wagner model can be modified to explain counterconditioning by adopting the assumption that excitatory associations in one motivational system are equivalent to inhibitory associations in the opposite motivational system. In terms of the model, they have opposite algebraic signs. Thus, when an aversive CS is reinforced with an appetitive US, the algebraic sign is reversed to produce a negative ΔV. Transformation of the

aversive CS into an appetitive CS erases its association with the aversive US, which acts to reduce the aversive conditioned response (see Krank, 1985). For example, Scavio (1974) established a tone as an aversive CS for a nictitating membrane response in rabbits by pairing it with a shock. When the tone was subsequently paired with an appetitive US (the delivery of water) the level of aversive responding was reduced. Competition between the incompatible responses was not responsible for the loss of aversive responding as both appetitive and aversive responses occurred when they are statistically independent. In addition, the response loss was more rapid than an extinction control, which is consistent with a larger discrepancy (λ-ΣV) produced by reinforcement in an opponent system.

Extinction of course is the prototypical example of loss produced by a negative discrepancy. According to the Rescorla-Wagner model λ is reduced to zero when the CS is presented in the absence of the US. This results in a negative discrepancy between λ and ΣV and thus reduces the associative strength of the CS. When the decrease in associative strength is governed by this rule it produces a negatively accelerated function as shown in Figure i. It is clear from the figure that loss in associative strength is greater on the initial trials when the discrepancy between the anticipation of the US and the absence of reinforcement is greatest.

Rescorla (2001b) recently examined the degree to which non-reinforcement reduces associative strength during the initial extinction trials compared to the latter extinction trials. Rats received pairings of four different CSs with a food US (A+, B+, C+ and D+). In two separate extinction sessions all rats received non-reinforced exposures to two of the conditioned stimuli. Specifically, in the first session, A and

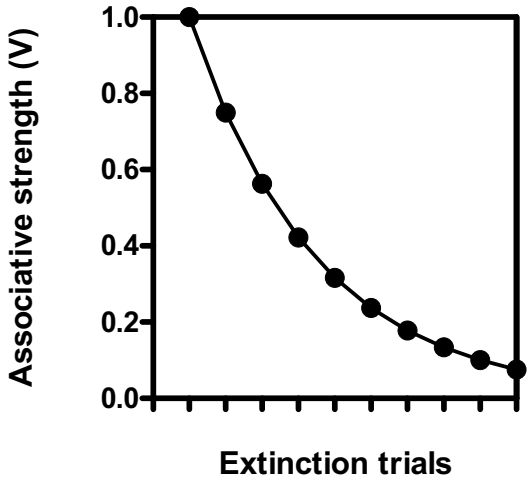

Figure i. The rate of decline in associative strength (V) across extinction trials according to the Rescorla-Wagner model (Rescorla & Wagner, 1972).

B were presented eight times in the absence of reinforcement and in the second session, A received a further four non-reinforced presentations and C received an initial four non-reinforced presentations. In order to control performance-scale differences between each CS and assess associative changes produced during the second extinction session, test consisted of presenting the CSs in compounds AD and BC (see Rescorla, 2001b). This method ensured that compounds AD and BC had each received an equal number of extinction trials. However, in the second extinction session, the A element in the AD compound had received four trials that were the final four in the series of twelve, whereas the C element in the BC compound received its initial four extinction trials. Any differences in AD and BC can therefore be attributed to differences in the loss in associative strength to C on the initial extinction trials relative to A on the later trials. Rescorla (2001b) observed less responding to BC compared to AD confirming that initial extinction trials produce a greater loss in associative strength compared to later extinction trials. Thus, these results confirm the predictions made by the Rescorla-Wagner model that reductions in associative strength follow a negatively accelerated course.

The Rescorla-Wagner model is also able to account for the augmentation of response loss produced when a target stimulus is extinguished in compound with an excitatory stimulus, or conversely, impaired by extinction in compound with an inhibitory stimulus. In one experiment, Rescorla (2000b) paired one auditory (A) and two visual (V1 and V2) with a food US. One group of rats received non-reinforced compound presentations of V1 and A, whereas another group received non-reinforced presentations of V2 alone. In a subsequent test of the visual stimuli, V1 elicited less responding than V2. This suggests that extinction in the presence of

another excitor produces a greater decrement in associative strength relative to a stimulus extinguished individually. This effect has also been observed in a conditioned eyeblink response experiment where the target CS was extinguished concurrently with either a strong or a weak excitatory stimulus (Wagner, 1969); and in conditioned fear in rats (Monastra & Sisemore, 1978). In each case, extinction of a target CS in the presence of an excitor facilitated response loss (but see Pearce & Wilson, 1991 for the opposite result using autoshaping in pigeons).

Extinction with a concurrent inhibitor impairs response loss. Rescorla (2003) trained rats to peck two key lights (A and B) for food and also trained a tone (T) to be a conditioned inhibitor by presenting it in compound with an excitor in the absence of reinforcement. During extinction the key light was presented either in the presence of the tone (e.g., AT-), or in the presence of an equally familiar but neutral noise stimulus (e.g., BN-). On a subsequent test of A and B, the stimulus nonreinforced in the presence of the neutral stimulus (B) displayed greater levels of response loss compared to the stimulus extinguished in the presence of the conditioned inhibitor (A). A CS is therefore protected from extinction when presented with a conditioned inhibitor in the absence of reinforcement (see also Kamin, 1969; but see Lovibond, Davis & O'Flaherty, 2000).

The Rescorla-Wagner model explains both of these effects in terms of the magnitude of the discrepancy between λ and ΣV. Specifically, extinction of a CS in the presence of another excitatory cue increases ΣV and thus the size of the negative discrepancy between λ and ΣV. In the example provided by Rescorla (2001), nonreinforcement of the V1A compound resulted in a larger discrepancy between λ and ΣV relative to nonreinforcement of V2. This resulted in a greater loss in

associative strength to the V1A compound, and consequently reduced responding to V1 relative to V2 on the subsequent test. In contrast, in the experiment reported by Rescorla (2003) nonreinforcing the CS (A) in the presence of a conditioned inhibitor reduced ΣV and therefore decreased the discrepancy between λ and ΣV. Consequently, there was less loss in associative strength to A compared to the control stimulus that was extinguished in the presence of a neutral stimulus.

Thus, according to the Rescorla-Wagner model, the conditions for learning during extinction is a negative discrepancy between what is expected and what actually occurs. When this discrepancy is large there is a large reduction in associative strength relative to when the discrepancy is small. This model is successful in explaining a number of training protocols that reduce conditioned performance, such as overexpectation, counterconditioning and extinction. This model is, however, unable to account for results that reveal a preservation of associative strength after extinction. There are a number of testing procedures that have revealed that the effects of extinction are often temporary suggesting that extinction does not remove all of the original learning. These results are inconsistent with the assumption that changes in associative strength are permanent and represented by a single construct (V).

Conditions of test

Extinguished responding can be restored in the absence of retraining. Several post extinction procedures restore responding to an extinguished CS. These include manipulating the passage of time between non-reinforcement and test, the presentation of a novel, but neutral CS, testing outside the extinction context, and US

alone presentations. The restoration of extinguished behaviour by these procedures suggests that some, if not all, the learning that took place during acquisition survives the extinction process.

Spontaneous recovery of responding has been known since Pavlov (1927). A typical procedure involves extinguishing a previously acquired conditioned response and testing the level of conditioned performance after a passage of time. Spontaneous recovery is observed when conditioned performance is greater following the retention interval compared to at the end of extinction training (for a review see Rescorla, 2004). For example, Rescorla (1997) demonstrated spontaneous recovery in a Pavlovian magazine approach experiment with rats. In this experiment two CSs were paired with food and then extinguished. CS1 was extinguished immediately after conditioning, while CS2 was extinguished after a delay. The level of conditioned performance to each CS was then tested immediately after extinction of CS2. This procedure compared conditioned performance between stimuli that were tested at different intervals after extinction but whose conditioning to test intervals was the same. On test there was more conditioned performance to CS1 than to CS2, indicating that a longer interval between extinction and test produces greater levels of spontaneous recovery. This effect has been demonstrated in a variety of procedures and across species, including the extinction of a conditioned taste aversion (Rosas & Bouton, 1996) and conditioned fear (Quirk, 2002) in rats and autoshaped pecking responses in pigeons (Robbins, 1990).

Disinhibition refers to the recovery of conditioned performance resulting from the presentation of a novel, but neutral stimulus. There are, however, few demonstrations of disinhibition in the literature (e.g., Bottjer, 1982; Brimer, 1970,

1972; Hearst, Franklin & Mueller, 1974) and an equal number where disinhibition was not demonstrated (e.g., Boakes, 1973; Boakes & Halliday, 1975; Hendry, 1982). Consequently, it is not wise to rely heavily on this phenomenon to provide support for the suggestion that original learning remains intact after extinction. There is however, evidence that suggests conditioned performance after extinction is modulated by the presence of contextual cues during test that have a history of reinforcement.

Bouton and King (1986) examined the effects of testing an extinguished CS in a previously shocked context or in a non-shocked context. Across a series of experiments they found that conditioned fear responding (conditioned suppression) was restored when the CS was tested in the previously shocked context relative to the non-shocked context. Performance, however, to a non-extinguished CS, a latently inhibited CS and a partially reinforced CS, which were matched in their levels of responding to the extinguished CS, was not facilitated (see Bouton, 1984). Thus, some of the original learning survived extinction and its expression during test was determined by the reinforcement history of the test context.

Similarly, conditioned performance to an appetitive CS is more likely to be expressed after extinction when it is tested in the presence of contextual cues with an associative history. Swartzentruber and Rescorla (1994) trained a flashing light stimulus (X) to be a conditioned facilitator by reinforcing pigeons pecking a keylight in its presence and not in its absence (XA+/A-). Two other key lights were then trained using either an extinction procedure (C+, C-) or preexposure procedure (D-, D+). C and D were trained so that their levels of responding were equivalent. Responding to the two target keylights (C and D) was then compared in either the

presence of the conditioned facilitator or in the presence of a control stimulus. The test revealed that there was more responding to the extinguished stimulus (C) when tested in the presence of the conditioned facilitator relative to the control stimulus, while responding to the preexposed stimulus (D) was modulated less by the facilitator. These results are consistent with those described in the case of conditioned fear (Bouton & King, 1986) as extinguished responding was restored when tested in the presence of contextual cues with a history of reinforcement.

The role of contextual cues in the unmasking of conditioned performance is illustrated by the context specificity of extinction. Renewal refers to the restoration of conditioned performance when the CS is tested outside of the context where it was extinguished (for reviews see Bouton, 1993; 2002; 2004). For example, Bouton and Bolles (1979) conditioned a CS in one context (A) and extinguished it in another (B). They found that when the extinguished CS was returned to the original conditioning context (A) responding to the CS returned. In a second experiment conditioning was conducted in Context A, extinction in Context B, while test took place in a third novel context (C). Again, conditioned performance was restored to the extinguished CS (Bouton & Bolles, 1979; Harris, Jones, Bailey & Westbrook, 2000). Conditioning and extinguishing the CS in the same context, and then testing the CS in a second context also renewed the conditioned response (Bouton & Ricker, 1994). Thus, when the CS is tested outside of the extinction context there is a renewal of the conditioned performance.

The final example of a post extinction procedure that restores conditioned performance is reinstatement. This is where extinguished behaviour is reinstated by reexposure to the original US in the absence of the CS. Reinstatement has been

demonstrated with both aversive USs (Rescorla & Heth, 1975; Westbrook, Iordanova, McNally, Richardson & Harris, 2002) and appetitive USs (Bouton & Peck, 1989; Bouton, Rosengard, Achenbach, Peck & Brooks, 1993). This effect suggests that unsignalled US presentations reveal the original learning that is masked by extinction training. Reinstatement, however, depends on the context in which US alone presentations occurred. Specifically, reinstatement is more likely to occur when the US presentations and test occur in the same context compared to when US presentation occur in a different context from where extinction and test occur (Bouton & King, 1983; Bouton et al., 1993; Westbrook et al., 2002). Furthermore, if an extinction session is inserted between noncontingent US presentations and test the reinstatement effect is reduced (Bouton & Bolles, 1979). These conditions of test that influence the reinstatement effect suggest that reinstatement is not the removal of the mask imposed by extinction, but rather the influence of context-US associations, formed during US alone exposures, on the extinguished stimuli on test (Bouton et al, 1993).

It has, however, been demonstrated that new CS-US associations produced by US alone presentations can also mediate the reinstatement effect. Specifically, Westbrook et al. (2002) paired a CS with shock in Context A and extinguished the CS in that context. Rats then received exposures to the shock in either A or a different context (B) before being tested to the extinguished CS in a third context (C). They found that more fear was reinstated when the rats were shocked in the conditioned and extinguished context (A) than when rats were shocked in the different context (B). Subsequent experiments revealed that reinstatement to the extinguished CS when tested in a novel context (C) reflected the formation of new

associations between the CS and the US as a consequence of US presentations in the extinction context.

These phenomena demonstrate how the conditions of test are likely to modulate performance to an extinguished stimulus. The conditions of test that have demonstrated the restoration of responding to an extinguished stimulus include the time and place of test, relative to extinction training and the reinforcement history of the test context. It has, however, been noted by several authors (see Rescorla, 2001; Delamater, 2004) that these procedures rarely result in a complete recovery of performance, that is, responding that is equivalent to that produced by a non-extinguished control group. In circumstances when the restoration of performance is equivalent to a non-extinguished control other differences may explain the results. For example, Quirk (2002) extinguished conditioned fear and assessed the levels of conditioned responding (freezing) either the same day, or 1, 2, 4, 6, 10 or 14 days after extinction. When tested 10 days after extinction the levels of freezing were comparable with those elicited on the first two extinction trials. However, the assessment compared responding under different test conditions. For example, there were differences in the interval between original training and test, resulting in differences in the age of animals and time spent in their home cages on a food deprivation schedule. Thus, while the results suggest a complete restoration of conditioned responding, confounding factors related to test conditions prevent one making such a conclusion.

Incomplete restoration of conditioned performance raises the possibility that some associative loss does take place during extinction, though sufficient associative strength is preserved to allow a recovery of responding. In contrast to this view,

however, is evidence that extinction leaves the excitatory association established during conditioning completely intact. For example, Delamater (1996) compared the ability of an extinguished and a non-extinguished CS to control an instrumental response for an outcome that was the same as the one previously paired with the extinguished CS. Two different CSs (A and B) were paired with different reinforcing outcomes, after which one of the CSs was extinguished (e.g., A). Rats were then given a choice between lever pressing and chain pulling instrumental responses that had previously been trained with the same outcomes as A and B, respectively. This choice test occurred in the presence of either the extinguished stimulus (A) or the non-extinguished stimulus (B). It was observed that the instrumental choice was determined by its shared outcome with the CS. Furthermore, the extinguished CS resulted in as much instrumental performance as the non-extinguished CS. Thus, it was concluded that the CS-US associations were fully preserved after extinction (see Rescorla, 1996 for the same results using a selective outcome devaluation procedure).

A further demonstration of the preservation of originally learned CS-US associations through an extinction procedure comes from an experiment that devalued the US (Rescorla, 1996). In this experiment two CSs were each paired with a distinctive outcome, pellets and sucrose. One group then received non-reinforced presentations of both CSs, while the other group were spared this treatment. All rats then received pairing of both CSs with a common third outcome. For both groups one of the original outcomes was then paired with LiCl, after which performance to both CSs was assessed. The results showed that for both groups presentation of the CS increased responding. Furthermore, the magnitude of this increase was greater

for the CS whose original outcome had not been paired with LiCl (i.e., devalued). These results suggest that the original CS-US association was not eliminated through training the CS with another outcome.

The results from these assessment procedures suggest that associations acquired during conditioning are mostly, if not completely preserved after extinction training in spite of the fact that the CS fails to elicit responding. This is inconsistent with the view that extinction results in the erasure of the association between the CS and the US. Most contemporary theories of extinction have assumed that the response loss observed during extinction is a result of new learning, rather than permanent associative loss.

Theories of extinction

There are many contemporary theories of extinction that account for the retention of the original acquisition learning by allowing for the presence of a second process that interferes with the expression of conditioned performance. This second process has been conceptualised as conditioned inhibition between the CS and the US (Wagner, 1981); an elevation in the threshold of US activation (Konorski, 1948; Rescorla, 1979, 1985); an opponent motivational state resulting from the absence of the US, such as relief in the case of aversive conditioning (Denny, 1976, 1991) or frustration in the case of appetitive conditioning (Amsel, 1958); or the formation of a second contrasting memory that is activated depending on the similarity of the test context with the extinction context (Bouton, 1993). In each of these cases, the second process produces changes that exist concurrent with the original associations formed during conditioning.

For example, Wagner's Sometimes Opponent Process (SOP) Model (Wagner, 1981; Wagner & Brandon, 1989) implements the common-error term of the Rescorla-Wagner model, but assumes that an additional process results in the formation of inhibitory associations between the CS and the US. According to the SOP model associative learning occurs when stimulus representations are in an active state (i.e. in either A1 or A2), whereby stimulus representations simultaneously in A1 form an excitatory association, while a stimulus representation in A1 and a stimulus representation in A2 will form an inhibitory association. An association that has formed between two stimuli, a CS and a US, renders the CS capable of eliciting the US representation directly into A2, thus generating conditioned responding. Over consecutive presentations of the CS in the absence of the US, however, an inhibitory association is formed between them as the CS representation is in A1 and the US representation is in A2. This renders the C so that the CS becomes unable to incapable of eliciting the CR. This results in a decrease in conditioned responding, that is, extinction.

Conditioned inhibition and extinction procedures share common properties. For example, both involve non-reinforcement in the presence of an excitatory CS, and both result in a decrease in the conditioned response. The conceptualisation of the second process during extinction as conditioned inhibition suggests that a conditioned inhibitor and an extinguished CS should share common properties, namely, they should both pass summation and retardation tests. Rescorla (1979) examined the inhibitory processes produced by extinction training by presenting a novel stimulus in compound with a fearful stimulus during extinction training. Subsequently, the novel stimulus passed both summation and retardation tests,

indicating the presence of conditioned inhibition. Despite this, there is little evidence that a conditioned inhibitor exhibits the same properties as an extinguished CS when confronted with other test conditions. For example, the properties of a conditioned inhibitor are not lost with the passage of time (Rescorla, 2005) or a change in context (Bouton & Nelson, 1994; Nelson & Bouton, 1997; cf. Mackintosh, 1974) and an inhibitory CS appears to modulate performance to other stimuli via indirect, rather than direct, associations with the US (see Rescorla, 1985).

Rescorla (1979, 1985) has proposed that changes in performance after initial conditioning are the result of changes in the threshold at which the US representation can be activated. Changes in this threshold depend on the discrepancy rule describe by the Rescorla-Wagner model (e.g. Rescorla, 1985, p. 323) and occur alongside the already established excitatory association. Thus, conditioning produces a permanent excitatory association and subsequent changes in performance are due to increases or decreases in a hypothetical threshold governing the activation of the US representation. This view assumes that both extinction and conditioned inhibition increase the threshold of US activation. The elevation occurs because repeated CS alone presentations activate the representation of the US, thus generating a discrepancy between what is expected and what actually occurs (the absence of the US). A decrease in conditioned performance is therefore a result of a deterioration of the US representation. This deterioration may also represent forgetting, or a devaluation of the US equivalent to a reduction in US strength or a loss of emotional salience. This view also assumes that extinction produces a permanent effect on the threshold, compared to conditioned inhibition that produces a transitory effect. Specifically, a conditioned inhibitor elevates the threshold when it is present,

whereas an extinguished stimulus elevates the threshold for other stimuli associated with the same US whether it is present or not.

An alternative to inhibitory learning is that during extinction new excitatory associations are formed (Konorski, 1967; Rescorla & Solomon, 1967). According to this view, the omission of an anticipated reinforcer, such as food or shock, elicits a motivational response that it opposite to that elicited when the reinforcer is present, such as frustration (Amsel, 1958) or relief (Denny, 1974, 1991). This opposing motivational response then forms an association with the stimuli present during nonreinforcment, and consequently impairs conditioned responding. For example, in the case of appetitive conditioning the absence of the food reward during extinction training produces a frustration response that is then capable of forming an association with the CS, the context in which non-reward occurs, and the preceding conditioned response. When such associations are formed concurrent with those formed during conditioning, the decremental impact of these subsequent associations are limited to that stimulus, response and context. These theories are therefore able to account for why responding to an extinguished CS is likely to be modulated by the prevailing context, or the presence of other modulators.

There is substantial evidence for the presence of an aversive state in the absence of an anticipated reward. For example, extinction of an appetitive CS enhances aggression towards conspecifics in pigeons (Azrin, Hutchinson & Hake, 1966) and in rats (Tondat & Daly, 1972). In addition, there is evidence that motivational responses are conditioned to neutral stimuli presented during the omission of reinforcement. For example, both Wagner (1963) and Daly (1974) found that stimuli present at the time of nonreinforcement acquired aversive properties,

becoming capable of promoting escape learning. There is, however, little compelling evidence that the omission of an aversive US elicits relief (but see Denny, 1991). Thus, a motivational learning account explains a variety of effects in the extinction of an appetitive CS, but the lack of direct evidence for a corresponding opponent response in the extinction of an aversive CS renders motivational accounts of the loss of fear speculative.

Bouton (1993; 2002) offers an alternative, memory-based explanation of extinction. He proposes that conditioning and extinction are represented as distinct memories, a CS-US and a CS-No US memory, respectively, and that performance to an extinguished CS is determined by which of these memories is retrieved (see Bouton, 2004 for review). These memories are formed against backgrounds that include not just the physical context where conditioning and extinction occurred but also the internal state of the subject as well as the temporal context (change in context resulting from the passage of time). In determining which memory is retrieved, Bouton (1993) suggests that the background present at test acts as an occasion setter (Holland, 1985) or a facilitator (Rescorla, 1985), favouring the retrieval of the association that was formed against a similar contextual background.

Consistent with this account is a fear conditioning experiment conduced by Morris et al (2005). Rats were trained to discriminate between a shocked (A) and a non-shocked context (B). In a third context the rats received a CS-shock pairing followed by a CS extinction trial. Finally, rats were re-exposed to either Context A or Context B after which they were tested for freezing to the CS in a fourth context. The interval (spent in the home cages) between re-exposure to the context and test was either short (2 min) or long (24 hr). Conditioned performance was restored to

the extinguished CS at the short but not the long interval and depended on re-exposure being to the dangerous context (A) but not the safe context (B). The restoration of conditioned performance also depended on rats being tested with an extinguished CS, rather than a non-conditioned CS or a conditioned but non-extinguished CS. Morris et al (2005) suggested that recent exposure to a dangerous context restored the fearful background under which the original CS-US association had formed, thus retrieval of the conditioning memory was favoured over the extinction memory (e.g., Bouton, 2002) resulting in the reinstatement of fear to an extinguished CS.

Summary

The most influential account of the mechanisms that underlie associative learning is the Rescorla-Wagner model. This model proposes that learning will occur when there is a discrepancy between what is expected and what actually occurs. The more unexpected or surprising the event, the more that is learnt about it. In this way an organism will establish associations between events that represent relations among events in their world. According to the model, the acquisition of a CS-US association is regulated by a positive discrepancy between λ and ΣV, while the extinction of that association is regulated by a negative discrepancy between λ and ΣV. Error correction models, such as the Rescorla-Wagner model, that evoke a single construct [associative strength (Rescorla & Wagner, 1972)] to explain both acquisition and extinction of an association, assume that extinction involves the unlearning of the original association. However, a range of studies have revealed that under certain testing conditions conditioned responding is restored. These results

suggest that much, if not all, the original CS-US association remains intact following extinction. It has therefore been suggested that extinction is the result of new learning that co-exists with the original acquisition learning and acts to mask the expression of conditioned performance. Various models have represented this new learning as the formation of a direct or gated inhibitory CS-US association, a change in the threshold or US activation, an excitatory association between the CS and an opponent motivational state.

CHAPTER 2

The role of the intertrial interval in extinction context conditioned fear responses

One of the variables that influence response loss is the interval between CS alone presentations [the intertrial interval (ITI)]. Studies comparing responding by subjects exposed to different intervals between extinction trials have found that short intervals produce more rapid loss than longer ones (e.g., Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980; Pavlov, 1927, pp. 52 - 53; Reynolds, 1945). This could mean that short intervals produce better learning than longer ones. However, subjects in these studies differed not only in the interval between CS alone presentations (the interval between training trials) but also in the interval used to assess its effects (the interval between training and test trials). Therefore, the differences in response loss could have been due to the effects of the interval on the expression of learning rather than on the learning itself.

One way to distinguish between these alternatives is to train at different intervals but test at common ones. Any differences in responding at the common test intervals must be due to the learning produced by the different training intervals (see Davis, 1970; Davis & Wagner, 1968, 1969; Rescorla, 1988, for discussion). Rescorla and Durlach (1987, Experiment 2) used just such a design to study the effects of the interval on learning. Pigeons were exposed to pairings of CS1-food and CS2-food in two distinctive contexts (A and B). The interval between CS-food trials was equally often short (10-s) and long (2-min). Then CS1 was extinguished in Context A and

CS2 in Context B. The interval between CS1 presentations in Context A was short (10 s) and that between CS2 presentations in Context B was longer (2 min). Finally, the pigeons were tested in Context C with intermixed 10-s and 2-min intervals between presentations of each CS. Rescorla and Durlach (1987) confirmed that the short interval between presentations of CS1 in Context A was more effective in producing response loss than the longer interval between CS2 presentations in Context B. They also found that the short interval was more effective than the longer one in promoting the learning that underlies long-term response loss. The pigeons responded less to CS1 than to CS2 when subsequently tested in Context C at common intervals between CS presentations (see also Cain, Blouin, & Barad, 2003).

The design used by Rescorla and Durlach (1987) confounded the duration of the interval between CS alone presentations with the time spent in the context between these presentations. This confound is theoretically significant as the time spent in the context between CS alone presentations can mediate the effect of the interval between these presentations on learning. Contemporary theories of Pavlovian conditioning identify associative formation with prediction error, and many assume that all of the cues present on a trial, including those provided by the background or context, are used to compute this error (e.g., Rescorla &Wagner, 1972; Wagner & Rescorla, 1972; Wagner, 1981; Wagner & Brandon, 1989). In extinction, the omission of the predicted US constitutes the error, and the magnitude of this error determines the amount of learning. For instance, the Sometimes Opponent Process (SOP) theory (Wagner, 1978, 1981) and the Affective Extension of SOP (AESOP;Wagner & Brandon, 1989) identify this learning with the formation of inhibitory associations between the CS and US. These are formed when CS

elements are in the focus of working memory (an A1 state of activity) concomitantly with US elements in the periphery (an A2 state of activity), and the strength of the resulting associations is determined by the proportion of US elements in A2. SOP and AESOP explain the results reported by Rescorla and Durlach (1987) in terms of differences in the predictive value of the context at the time of CS presentations. Specifically, the context (B) where the interval between CS2 presentations was long (2 min) would have undergone more extinction and, hence, a greater loss in predictive value, than the context (A) where the interval between CS1 presentations was shorter (10 s). Therefore, the proportion of US elements excited to A2 by the combined action of Context B and CS2 would have been less than that excited by Context A and CS1. The consequence of these differences in the proportion of US elements in A2 is that stronger inhibitory associations accrued to CS1 than to CS2. Hence, subjects responded less to CS1 than to CS2 when subsequently tested in Context C with common intervals between CS presentations (see Rescorla & Durlach, 1987, for discussion).

The results reported by Rescorla and Durlach (1987) show that a short interval between CS alone presentations produces better learning than a longer one. But, as noted, these results were the product of a procedure that confounded this interval with time spent in the context between the presentations. Westbrook, Smith and Charnock (1985) broke this confound by removing subjects from the training context between extinction trials. Rats were made sick after ingestion of a novel flavor in a distinctive chamber and then exposed to that flavor on two occasions but were not made sick. The interval between these flavor extinction trials was relatively short (0.5 hr), moderate (3 hr), or long (24 hr) and this interval was spent outside the

training context in the home cages. Finally, all rats were tested with the flavor in the chambers at a common retention interval (24 hr after the second extinction trial) also spent in the home cages. The test revealed that intake was positively related to the interval between the extinction trials: that is, a longer interval produced greater extinction of the learned flavor aversion than shorter intervals. Morris, Furlong and Westbrook (2005, Experiment1) reported similar results for a context conditioned fear response (freezing). Rats were shocked in a distinctive context and then subjected to two extinction trials. The interval between these trials was short (2 min), moderate (12 hr) or long (24 hr). Finally, rats in each of these conditions were subjected to common intervals between the second extinction trial and testing. The intervals between the two extinction trials and between the second trial and test were spent in the home cages. Rats exposed to the longest (24 hr) interval between the two extinction trials froze less than those exposed to the short (2 min) interval when rats from each condition were tested 24 hr after the second extinction trial. Thus, a long interval between extinction trials again produced better learning than a shorter interval.

Error correction models such as SOP and AESOP can also explain the better learning produced by a long interval. As noted previously, these models explain the better learning produced by short intervals in terms of the greater proportion of US elements excited to A2 by the combined action of the context and CS. Other things being equal, that is, the proportion of elements excited to A1 by the CS, inhibitory learning is regulated by the proportion of US elements concomitantly present in A2. However, other things are not always equal. SOP and AESOP also assume that CS elements decay from A1 to A2 and, critically, that elements in A2 are less able to be

excited to A1 than when such elements are in a state of inactivity (I). Essentially, A2 serves as a comparator that regulates whether a stimulus presentation (e.g., a CS) can excite its elements to A1. Thus, any manipulation that reduces the proportion of CS elements excited to A1 will also reduce the associability of that CS. One such manipulation is the duration of the interval between extinction trials. If this interval is such that trial N+1 overlaps with CS elements in A2 from the preceding trial (N), then associative formation is impaired relative to an interval where there is no such overlap. Thus, according to SOP and AESOP, a loss of associability underlies the impairment in learning produced by a short interval between extinction trials (Morris et al., 2005; Westbrook et al., 1985).

SOP and AESOP do not just predict that a CS undergoing a loss in associability is impaired in its ability to enter into an inhibitory association with the US: They also predict that such a CS loses its ability to elicit conditioned responding. SOP and AESOP predict this loss in conditioned responding because a CS is assumed to elicit conditioned responding by its excitation of US elements to A2. Moreover, this excitation of US elements to A2 occurs when CS elements are in A1 but not when CS elements are in A2. Therefore, a CS whose presentation on trial N+1 overlaps with CS elements in A2 from the preceding trial N will be less able to elicit conditioned responding than when there is no such overlap. And, of course, that CS on trial N+1 will be less able to enter into the inhibitory association with the US that underlies the long-term loss of conditioned responding. Thus, SOP and AESOP predict that the duration of the interval between extinction trials exerts contrasting effects on conditioned responding and learning: short intervals will be

more effective in reducing conditioned responding but less effective in promoting learning than longer ones.

The present experiments studied the effects of the intertrial interval on extinction of fear responses (freezing) to a context CS. Rats were trained to discriminate between a context (A) where shock occurred and a second context (B) where shock did not occur. Then they were repeatedly exposed to Context A in the absence of shock. Each of these extinction trials was 2 min in duration and the interval between them was either short (4 min) or long (24 hr). This interval was spent in the home cages. Morris et al. (2005) reported that a short interval between two such extinction trials produced worse learning than a longer interval when rats from each condition were tested after a long interval. This result was thus observed with the minimum number of extinction trials required to study the effects of the intertrial interval on learning. However, that number of training trials was not sufficient to assess the rate at which fear responses was lost across different intertrial intervals. Therefore, the initial experiments trained rats with short or longer intertrial intervals until fear responses were lost in order to determine any differences in the rate at which this loss occurred. Then rats were subjected to additional tests at common intertrial intervals in order to determine the effects of the duration of the intertrial interval on learning.

Experiment 1a and 1b

These experiments had two aims. The first was to assess the effects of the duration of the interval between extinction trials on the rate of response loss. Half of the rats were trained with a short (4-min) interval between extinction trials (Groups

Massed) while the remainder was trained with a longer (24-hr) interval (Groups Spaced). The second aim was to assess the effect of the duration of the interval between training trials on learning. Half of the rats trained at the short interval and half trained at the longer interval were tested with a short interval between additional extinction trials (Groups Massed-Massed and Spaced-Massed, respectively). The remaining rats trained with the short or the longer intervals were tested with the longer interval between extinction trials (Groups Massed-Spaced and Spaced-Spaced). The intervals between training trials and between test trials were spent in the home cages.

Rats were trained to discriminate between two contexts. In one of these (A) shock occurred and in the other (B) shock was not presented. This was done to ensure that the changes in behavior (the development of freezing responses) were due to the pairings of Context A and shock rather than to non-associative processes engaged by exposures to the contexts and/or shocks. Then rats received extinction training. In Experiment 1a, this consisted in a daily non-shocked exposure to Context A across ten days for rats in Groups Spaced, and a daily non-shocked exposure to Context B across these days for rats in Groups Massed. Rats in Group Spaced received Massed exposures to Context B prior to their final extinction trial in Context A on day 11, whereas rats in Groups Massed received 11 massed extinction trials in Context A on that day. Control rats received spaced extinction trials in B followed by massed extinction trials in B. All rats received additional extinction trials in Context A across subsequent testing. Half of the rats in each condition received massed extinction testing (Groups Massed-Massed, Spaced-Massed and No Extinction-Massed), while the remainder received spaced extinction testing (Groups

Massed-Spaced, MassedSpaced-Spaced and No Extinction-Spaced). Experiment 1b was identical except that rats in Groups Massed received extinction training on the day when those in Groups Spaced began their spaced training.

The design used in Experiment 1a arranged that rats spent equivalent amounts of time in each context across extinction training. They also received equivalent exposures to short and long intervals. However, the identity of the context separated by these intervals differed. Extinction trials in Context A were separated by the longer interval in the case of rats in Groups Spaced and a short interval in the case of rats in Groups Massed, whereas extinction trials in Context B were separated by a short interval in the case of rats in Groups Spaced and the longer interval in the case of rats in Groups Massed. In Experiment 1a, rats in the Massed condition received massed exposures to Context A on the day when rats in the Spaced condition received their massed exposures with Context B and the final exposure to Context A. Therefore, rats in these conditions were subjected to additional test exposures at the same time but differed in terms of the interval of time between the termination of discriminative conditioning and the initiation of extinction trials with Context A. Rats in the Spaced condition began this training on the day following the termination of conditioning whereas those in the Massed condition began extinction training of Context A 11 days after the termination of conditioning. Experiment 1b was identical to Experiment 1a except that rats in the Massed condition received massed extinction trials with Context A on the day following the termination of discriminative conditioning. Rats in this condition proceeded into massed testing on that same day and into spaced testing on the following day.

Method

Subjects. Subjects were 72 experimentally naïve male Wistar rats (*Rattus Norvegicus*) in Experiment 1a and 48 such rats in Experiment 1b. They were obtained from a local supplier (Gore Hill Research Laboratories, Sydney) and weighed between 350 and 450 g at the onset of the experiment. Rats were housed in groups of eight in plastic boxes (67-cm length x 40-cm width x 22-cm height) with food and water continuously available. The boxes were kept in an air-conditioned colony room maintained on a 12:12-hr light-dark cycle. Each rat was handled for approximately 3 min per day for four days prior to the start of the experiment. The experimental procedures followed the ethical guidelines established by the American Psychological Association and were approved by the Animal Care and Ethics Committee of the University of New South Wales.

Apparatus. Two separate rooms each containing four experimental chambers served as Contexts A and B. In both rooms, each chamber was located in a separate compartment of a wooden cabinet, with the doors of the cabinet open to allow observation. In one room, the chambers measured 30 cm (width) x 29 cm (height) x 25 cm (length) . The side walls and ceiling of these chambers were constructed of aluminium which had been painted white, while the front and back walls were constructed of clear Perspex. A sheet of white cardboard covered the outside of the back wall. The floor consisted of 16 steel rods, 6 mm in diameter, spaced 18 mm apart, centre to centre. A removable tray located 5 cm below the rods contained cat litter as bedding *(Farmland, Australia)*, onto which 0.5 mL of rose oil (*Cara-Mia, Sydney*) solution was sprinkled prior to each session, providing a distinctive odor.

Each chamber was cleaned using tap water upon removal of a rat. The room was lit by a white fluorescent tube mounted in the ceiling (36W/W41, Thorn, Australia).

In the other room, the chambers measured 30 cm (width) x 30 cm (height) x 27 cm (length). The sidewalls and ceiling of these chambers were constructed of aluminum which had been painted black while front and back walls were constructed of clear Perspex. Each of these chambers were located in separate compartment of a wooden cabinet, the inside walls of which were painted black. The floor of each chamber consisted of stainless steel rods, 2 mm in diameter, spaced 10 mm apart, centre to centre. A removable tray located 5 cm below the rods contained paper-pellet bedding (Fibrecycle, Mudgeeraba, Australia*)*, onto which four drops of eucalyptus oil (Sheldon Drug Co., Sydney) were placed to provide a distinctive odor. The chambers were cleaned with tap water upon removal of each rat. The room was illuminated by a red fluorescent tube located in the ceiling. The identity of the chambers (white or black) that served as Contexts A and B were fully counterbalanced within groups.

Unscrambled AC 50Hz shock, from a constant current generator, could be delivered to the floor of each chamber in both rooms. The current available to each floor could be adjusted by an in-line milliampere meter. The background noise level was 65 dB as measured by a sound level meter (A scale; Model 2235; Bruel-Kjaer Instruments, Marlborough, MA) with the microphone located in the center of the chamber. The behavior of each rat was recorded by a camera mounted on the wall facing the chambers. The camera was connected to a video recorder and monitor located in another room in the laboratory. This room also contained the equipment for controlling shock presentation to the chambers in both rooms.

Scoring and statistics. All exposures to the contexts were videotaped, and the levels of freezing were measured with a time sampling procedure in which the rat's behavior was scored as freezing or not freezing every 2 s. Freezing was defined as the absence of all movement, except those related to breathing (Fanselow, 1980). The percentage of all samples scored as freezing was determined for each rat. Two observers, one of whom was unaware of the rat's treatment condition, scored the videotaped record of each rat. The inter-rater reliability for this and the remaining experiments was high. The correlation coefficients were > 0.9 in each experiment. Unless indicated otherwise, the data in this and the remaining experiments were analyzed with a contrast testing procedure that controlled the Decision-Wise Error rate at $\alpha = 0.05$ with the procedure described by Hays (1963).

Procedure. The procedure for Experiments 1a and 1b is represented in Table 1 and 2, respectively. In each experiment, rats were trained to discriminate between a shocked (A) and non-shocked (B) context. For half of the rats, the shocked context was the white chambers and the non-shocked context was the black chambers, while for the remaining rats, shock occurred in the black chambers but not in the white ones. On the first day, rats were placed in Context A for 9 min, and a 0.25 mA shock was delivered at 60, 180, 300 and 420 s, before being returned to their home cage 120 s after the last shock. In the afternoon of the same day, the rats were placed in Context B for 9 min without being shocked, before being returned to their home cage. Across Days 2 – 8, rats received a daily shocked exposure to A and a daily non-shocked exposure to B. These exposures alternated irregularly so that exposure to A and B occurred equally often in the morning and in the afternoon session. Shock intensity was increased by approximately 0.05 mA per day, reaching a

maximum intensity of 0.8 mA by the eighth conditioning session. From Day 6, the duration of exposure to each context was reduced to 120 s, and a single shock was delivered 60 s after placement in A. From Day 7, rats received a single treatment each day, either a shocked exposure in Context A or a non-shocked exposure in Context B. By Day 10 there had been a total of eight shocked exposures to Context A and eight non-shocked exposures to Context B. All experimental procedures took place between 9-11a.m. and 4-6p.m. At the end of discriminative conditioning, rats were assigned to six groups in each experiment (n = 12 in Experiment 1a and n = 8 in Experiment 1b) matched for their levels of freezing in Context A.

Extinction training began on Day 11. In Experiment 1a, rats in Groups Spaced received a daily, 2 min extinction trial in Context A across Days 11- 21, whereas those in Groups Massed and No Extinction received a daily extinction trial across these days in Context B. On Day 22, rats in Groups Spaced received ten massed extinction trials in Context B followed by their final extinction trial in Context A. Those in Groups Massed received 11 massed extinction trials in Context A while rats in Groups No Extinction received 11 massed extinction trials in Context B. Each trial was 2 min in duration and the interval between the trials was 4 min. Rats spent this interval in their home cages.

Massed extinction testing began 4 min after the final training trial for rats in Groups Spaced-Massed, Massed-Massed, and No Extinction-Massed. It consisted in 11 consecutive extinction trials in Context A. Each trial was 2 min in duration, the interval between trials was 4 min and rats spent this interval in their home cages. Spaced extinction testing began 24 hr after the final training trial for rats in Groups Spaced-Spaced, Massed-Spaced, and No Extinction-Massed. It consisted in a daily 2

min extinction trial in Context A across 11 days. Thus, the interval between these trials was 24 hr and rats spent this interval in their home cages.

In Experiment 1b, rats in Groups Spaced-Massed, Spaced-Spaced, No-Extinction-Massed and No Extinction-Spaced were treated in the manner described for these groups in Experiment 1a with the exception that Groups Spaced-Massed and Spaced-Spaced did not receive massed exposures to Context B. The difference between the two experiments was that rats in Groups Massed began their extinction training on the day when those in Groups Spaced began their extinction training. That is, on the day following termination of discriminative conditioning. Rats in Groups Massed received 11 extinction trials in Context A on that day. Each trial was 2 min in duration and the 4-min interval between trials was spent in the home cages. Rats in Groups Massed-Massed proceeded to an additional 11 extinction test trials in Context A. These began 4 min after the final training trial and were conducted in the manner described previously. Rats in Group Massed-Spaced began spaced testing on the day following their massed training. This consisted of a daily 2 min extinction trial in Context A across 11 days.

Results

In each experiment, discrimination training was successful. On the final exposures, the mean (±*SEM*) percentage levels of freezing to the A and B contexts in Experiment 1a were 63.93 (2.1) and 1.82 (0.9), respectively, while in Experiment 1b they were 70.70 (2.4) and 0 (0), respectively. There were no differences between the levels of freezing in the chambers that served as Context A or Context B. The mean (±*SEM*) levels of freezing across extinction training and testing for each of the groups in Experiment 1a are shown in Figure 1. The left panel shows the levels of

Context conditioning	Extinction	Test
A+/B-	[24-hr] A- (x10) B- (x10) A- [4-min]	[4-min] A-
		[24-hr] A-
	[24-hr] B- (x10) A- (x10) A- [4-min]	[4-min] A-
		[24-hr] A-
	[24-hr] B- (x10) B- (x10) B- [4-min]	[4-min] A-
		[24-hr] A-

Table 1. This table represents the consecutive phases in Experiments 1a. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures to Context A.

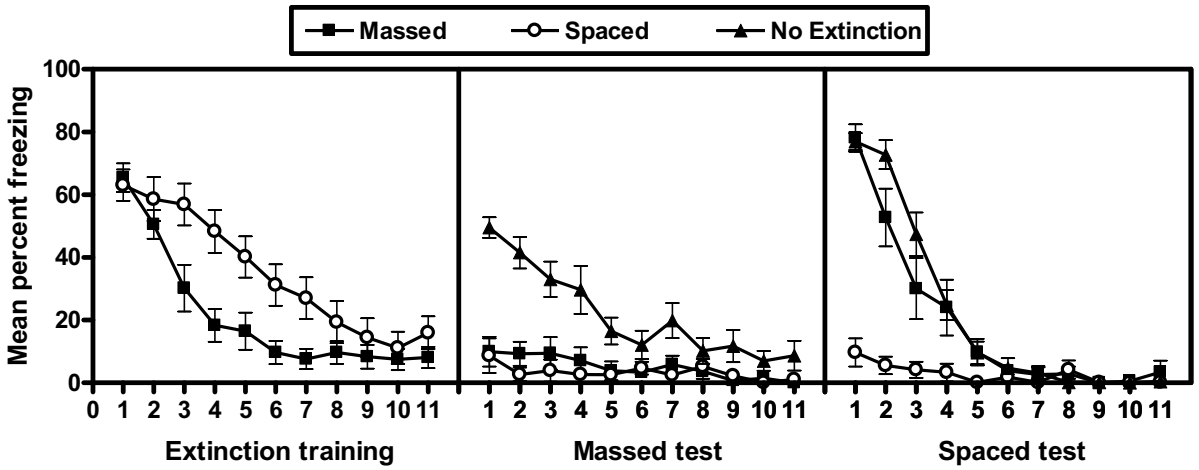

Figure 1. Mean (±SEM) percentage of time spent freezing across extinction trials and test trials. Extinction training began 24 hrs after the final day of discrimination training for Groups Spaced and 10 days after the final day of discrimination training for Groups Massed. The rats spent the interval between extinction trials and test trials in their home cages. Rats in Groups Massed had received a 4-min interval between extinction trials; those in Groups Spaced had received a 24-hr interval between extinction trials. Half the rats in each condition received a 24-hr interval between test trials, and the remainder received a 4-min interval between test trials. Rats in Groups No Extinction received either a 4-min or 24-hr interval between test trials in the absence of prior extinction training.

freezing for rats in Groups Spaced and Massed across extinction training in Context A; the center panel shows the levels of freezing across massed testing for rats in Groups Massed-Massed, Spaced-Massed, and No Extinction-Massed; the right panel shows the levels of freezing across spaced testing for rats in Groups Massed-Spaced, Spaced-Spaced, and No Extinction-Spaced. Inspection of the left panel shows that freezing declined across massed and spaced extinction training and suggests that this decline was more rapid when the interval between trials was short (massed) rather than long (spaced). The statistical analysis confirmed that the decline in freezing was significant, F (1, 50) = 361.34, F_{critical} = 4.03, and that rats in Groups Massed lost freezing more rapidly than did rats in Groups Spaced, F (1, 50) = 12.56.

Inspection of the middle panel shows that rats in Group No Extinction-Massed froze more than those that received massed testing after massed or spaced extinction training, and that rats in these latter groups exhibited similar low levels of freezing across massed testing. The statistical analysis confirmed that rats in Group No Extinction-Massed froze significantly more than rats in the remaining groups, F (1, 37) = 43.06, F_{critical} = 4.11, and that there were no statistically significant differences between the levels of freezing among rats in Groups Massed-Massed and Spaced-Massed, $F < 1$. There was a significant linear trend, F (1, 37) = 73.09, showing that freezing decreased over test trials. There was also a significant interaction between the linear trend and the contrast testing for group differences, F (1, 37) = 54.14, which, from inspection, is due to the decline in freezing among rats in the control group (Group No Extinction) and the absence of any such decline among rats in the extinction groups (Groups Massed and Spaced). There were no

significant differences in the rate of extinction across testing for rats in Groups Massed and Spaced, $F < 1$.

Inspection of the right panel shows that rats in Group Spaced-Spaced continued to exhibit low levels of freezing across spaced extinction testing but that rats in Group Massed-Spaced showed an almost complete recovery of lost freezing. Rats in this latter group showed levels of freezing across extinction testing similar to those by rats in Group No Extinction-Spaced. The statistical analysis confirmed that rats in Group Spaced-Spaced froze significantly less than rats in Groups Massed-Spaced and No Extinction-Spaced, $F(1, 37) = 53.96$, $F_{critical} = 4.11$, and that there were no significant differences between the levels of freezing from rats in Groups Massed-Spaced and No Extinction-Spaced, $F < 1$. A significant linear trend, $F(1, 37) = 255.83$, confirmed that freezing decreased across testing. There was a significant interaction between the linear trend and the contrast testing for differences between Group Spaced-Spaced and the other two groups, $F(1, 37) = 92.84$. This confirms that freezing remained low across testing for rats in Group Spaced-Spaced and declined from a substantial level across testing for those in the two other groups. There were no significant differences between the rates of decline across testing among rats in Groups Massed-Spaced and No Extinction-Spaced, $F < 1$.

The mean (±*SEM*) levels of freezing across extinction training and testing for each of the groups in Experiment 1b are shown in Figure 2. The left panel shows the levels of freezing for rats in Groups Spaced and Massed across extinction training in Context A; the center panel shows the levels of freezing across massed testing for rats in Groups Massed-Massed, Spaced-Massed, and No Extinction-Massed; the

Context conditioning	Extinction	Test
A+/B-	[24-hr] A- (x10) A-	[4-min] A-
		[24-hr] A-
	A- (x10) A- [4-min]	[4-min] A-
		[24-hr] A-
	[24-hr] B- (x10) B- (x10) B- [4-min]	[4-min] A-
		[24-hr] A-

Table 2. This table represents the consecutive phases in Experiments 1b. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures to Context A.

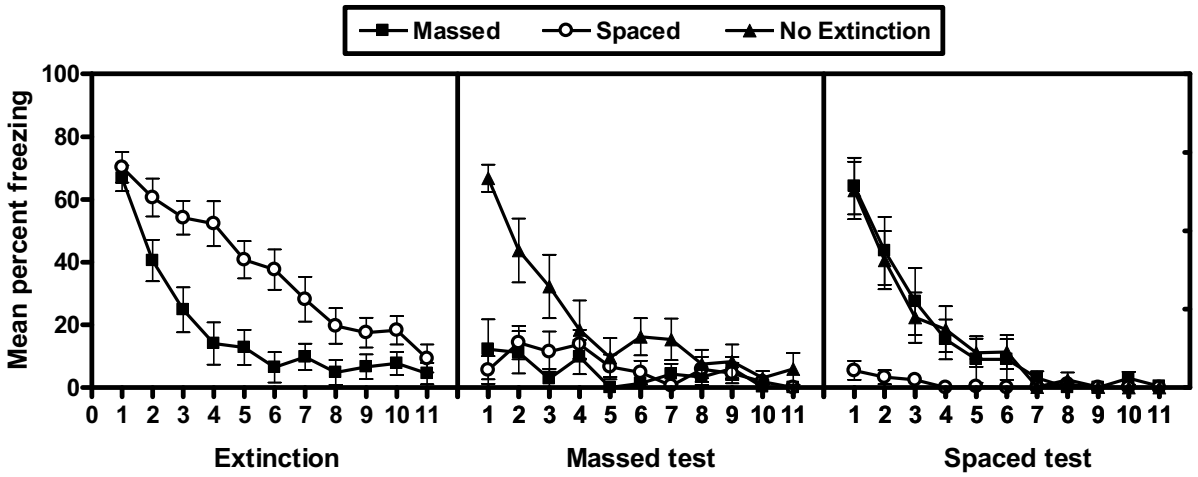

Figure 2. Mean (±SEM) percentage of time spent freezing across extinction trials and test trials. Extinction training began 24 hrs after the final day of discrimination training for Groups Spaced and Groups Massed. The rats spent the interval between extinction trials and test trials in their home cages. Rats in Groups Massed had received a 4 min interval between extinction trials; those in Groups Spaced had received a 24 hr interval between extinction trials. Half the rats in each condition received a 24-hr interval between test trials, and the remainder received a 4-min interval between test trials. Rats in Groups No Extinction received either a 4-min or 24-hr interval between test trials in the absence of prior extinction training

right panel shows the levels of freezing across spaced testing for rats in Groups Massed-Spaced, Spaced-Spaced, and No Extinction-Spaced. Inspection of the left panel shows that freezing declined across massed and spaced extinction training and suggests that this decline was more rapid when the interval between trials was short (massed) rather than long (spaced). The statistical analysis confirmed that rats in Groups Massed froze significantly less than those in Groups Spaced, F (1, 28) = 11.79, $F_{\text{critical}} = 4.20$. There was also a significant linear trend, F (1, 28) = 163.15, confirming that freezing declined across extinction testing.

The statistical analysis confirmed what is clear from inspection of the center panel: Rats in Group No Extinction-Massed froze significantly more than those in Groups Massed-Massed and Spaced-Massed, F (1, 21) = 39.81, $F_{\text{critical}} = 4.33$, and rats in these latter groups did not differ significantly in their levels of freezing, $F < 1$. The significant linear trend, F (1, 21) = 30.67, shows that freezing declined across testing, while the significant interaction of the contrast that tested Group No Extinction-Massed against the two other groups (Groups Massed-Massed and Spaced-Massed), F (1, 21) = 19.73**,** shows that the decline was specific to rats in Group No Extinction-Massed.

The statistical analysis again confirmed what is clear from inspection of the right panel. Rats in Group Spaced-Spaced froze significantly less across spaced testing than rats in Groups Massed-Spaced and Non Extinction-Spaced, F (1, 21) = 19.49, $F_{\text{critical}} = 4.33$, and rats in these latter groups did not differ significantly in their levels of freezing, $F < 1$. The significant linear trend, F (1, 21) = 81.77, shows that freezing declined across testing. There was also a significant interaction between the contrast testing for linear trend and the contrast testing for differences between rats

in Groups Spaced-Spaced and those in the two other groups (Groups Massed-Spaced and No Extinction-Spaced), F (1, 21) = 32.34. This confirms that freezing declined among rats in Groups Massed-Spaced and No Extinction-Spaced while remaining at a low level among those in Group Spaced-Spaced. There was no significant interaction between the linear trend and the difference between rats in Groups Massed-Spaced and No Extinction-Spaced, $F < 1$.

Discussion

These experiments have shown that rats subjected to a short (4-min) interval between extinction trials lost fear responses (freezing) to a context CS more rapidly than those exposed to a longer (24-hr) interval. They also showed that this difference in response loss was not due to the better learning produced by massed training. In fact, that training produced little or none of the learning that underlies long-term response loss. Rats trained with the short interval and tested with the longer interval (Group Massed-Spaced) froze just as much as control rats (Group No-Extinguished-Spaced), and rats in both of these groups froze more than rats trained and tested with the longer interval (Group Spaced-Spaced). The difference in learning produced by massed and spaced training is the opposite of that reported when training confounded the interval between extinction trials with the time spent in the context between the trials (Rescorla & Durlach, 1987). However, the present result replicates those reported when subjects spend the interval between trials in their homes cages (Morris et al., 2005; Westbrook et al., 1985). In these cases, massed training is less effective than spaced training in producing the learning that underlies long-term response loss.

Experiment 2

According to SOP and AESOP, short intervals impair the ability of the context CS representation to enter into the central focus of working memory. Consequently, context alone exposures fail to activate the US representation into an A2 state of activity resulting in: 1) a failure to elicit the conditioned response, and 2) an inability to form CS-US inhibitory associations. Accordingly, this deficit in learning is only expressed when tested a long interval after massed extinction training because short intervals impede conditioned responding .That is, unlike a short interval, a longer interval enables the context CS representation to re-enter the A1 state and activate the US representation into A2. Thus, the aim of the current experiment was to assess the time it takes for the context CS representation to decay to inactivity, thereby resulting in a test exposure eliciting the conditioned response.

Rats were trained to discriminate between two contexts, one in which they were shocked (A), and another in which they were not shocked (B). All rats then received massed extinction training, which has been shown to produce ineffective extinction learning (Experiments 1a and 1b). Testing consisted of an additional 2-min context exposure 4 min, 1 hr, 3 hr, 6 hr or 24 hr after the final massed extinction exposure. A recovery in the freezing response should indicate that the context CS representation had decayed to an inactive state prior to the test presentation. In contrast, if the freezing response remains low the context CS representation remains in an A2 state preventing activation of the US representation and thus conditioned responding.

Method

Subjects and apparatus. Subjects were 48 experimentally naïve male Wistar rats (*Rattus Norvegicus*), weighing between 350 and 450 g, of the same sex, strain and obtained from the same source as in previous experiments. They were kept under the conditions described previously.

Procedure. The experiment consisted of three distinct phases: discrimination training, extinction and test that are represented in Table 3. It was conducted in two replications, each of which included 4 rats from each group. In the first phase rats were trained to discriminate between a shocked (A) and non-shocked (B) context using the same procedure as described in previous experiments. All experimental procedures took place between 9-11 a.m. and 4-6 p.m. At the end of discriminative conditioning, rats were assigned to the various groups in such a way as to match their levels of freezing in Context A. Extinction training began 24 hrs after discrimination training and consisted of 11 2-min exposures to Context A separated by an interval of 4 min that was spent in their home cages for all groups. Test consisted of a non-reinforced exposure to Context A. The interval between extinction training and test was varied so that rats were tested 4 min, 1 hr, 3 hrs, 6 hrs, or 24 hrs after the final extinction exposures (Groups 4min, 1hr, 3hr, 6hr, and 24hr, respectively). Rats underwent the massed extinction phase at a time that allowed all groups to be tested at a common time (e.g., between 4-5 pm).

Results

Discrimination training proceeded such that by the eighth conditioning trial rats showed substantial freezing to Context A and very little freezing to Context B [mean (±*SEM*)]: 60.88 (3.2) and 4.50 (2.0), respectively. There was no difference in

Context conditioning	Extinction	Test				
		4min	1hr	3hr	6hr	24hr
A+/B-	A- (x10) A- [4-min]	A-				
	A- (x10) A- [4-min]		A-			
	A- (x10) A- [4-min]			A-		
	A- (x10) A- [4-min]				A-	
	A- (x10) A- [4-min]					A-

Table 3. This table represents the consecutive phases in Experiments 2. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures.

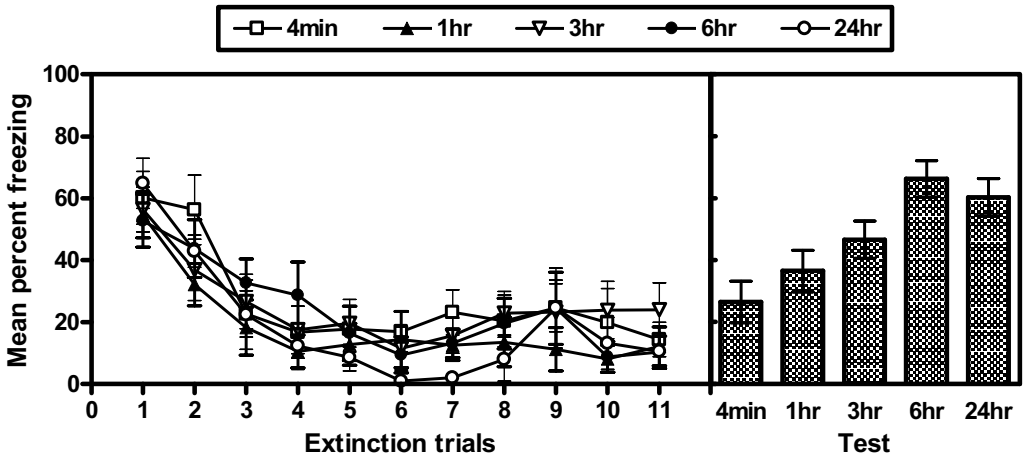

Figure 3. Mean (±SEM) percentage of time spent freezing across extinction trials and on the test trial. Rats spent the interval between extinction trials in their home cages. All rats had received a 4-min interval between extinction trials. Those in Group 4-min were tested 4 min after the final extinction trial; those in Group 1hr were tested one hr after the final extinction trial; those in Group 3hr were tested three hrs after the final extinction trial; those in Group 6hr were tested six hrs after the final extinction trial; and those in Group 24hr were tested 24 hrs after the final extinction trial.

levels of conditioning to the two counterbalanced contexts so the data was summated. The left and right panels of Figure 3 show the mean (±*SEM*) percentage levels of freezing for each 2-min exposure to Context A during massed extinction training and the level of freezing on test, respectively. The major impression from the left panel of the figure is that freezing to Context A decreased gradually over the 11 extinction trials and did not differ between groups. The right panel of the figure shows the level of freezing on test was positively related to the length of the interval between the final extinction trial and test, i.e., long intervals (3, 6, or 24 hrs) produced higher level of freezing than short intervals (4 min or 1 hr). These impressions were supported by the statistical analysis. There was a significant linear trend, F (1, 34) = 67.24, F_{critical} = 4.13, confirming that freezing decreased over extinction trials. The decline in the freezing response and the overall level of freezing did not differ between groups, $F < 2.13$. On test there was a significant difference in the level of freezing between the long interval groups (Groups 3hr, 6hr, and 24hr) and the shorter interval groups (Groups 4min and 1hr), F (1, 34) = 20.86, F_{critical} = 4.13. There was a significant difference in the level of freezing between Groups 24hr and 6hr and Group 3hr, F = 4.65. There were no reliable differences between Groups 4min and 1hr, or between Groups 24hr and 6hr, $Fs < 1.29$.

Discussion

These results show that longer intervals between the final extinction trial and test produce a recovery in the freezing response after training with a short interval. The greatest recovery in the freezing response was produced when testing occurred after an interval of 6 and 24 hrs. Furthermore, performance on the interval test did not differ between the short interval groups (Groups 4min and 1hr) or between the

long interval groups (Groups 6hr, and 24hr), indicating that there are no differential effects produced from these sets of values. A 3hr interval seemed to lie somewhere between the two sets of intervals in terms of the levels of responding produced during test. These results indicate that it takes up to 6 hrs for the context CS representation to completely decay from an active to an inactive state.Therefore, intervals between context exposures that are less than 6 hrs reduce adequate processing of the context CS representation thus reducing its associability and impairing learning.

Experiment 3

The previous experiments have demonstrated that short intervals between extinction exposures result in little long term response loss and suggest that short intervals engage processes that facilitate response loss while undermining the learning and memorial processes that mediate long-term response loss (e.g., Wagner, 1981). A question yet to be answered is: what role does the duration of the non-reinforced context exposure play in producing response loss during massed extinction? According to SOP the context (CS) representation remains in the highest level of activation (A1) while the subject is exposed to it, thus promoting the formation of inhibitory associations between the CS and the US. Therefore, according to SOP, extinction trials that are long in duration should produce more inhibitory associations and therefore more response loss than extinction trials that are short in duration.

The purpose of the current experiment was to investigate the amount of response loss produced using a short and a long interval when varying the duration

of non-reinforced context exposures. All rats were shocked in one context (A) and not in another (B). Half the rats received four 5-min context exposures that were separated by an interval of either 4 min or 24 hrs (Groups 5min/4min and 5min/24hr, respectively). The other half of the rats received two 10-min exposures to the context separated by an interval of either 4 min or 24 hrs (Groups 10min/4min and 10min/24hr, respectively). All groups received a final 2-min exposure to the context using the same interval that separated the previous exposures. A non-extinguished control group (Group No Extinction) that received exposures to a non-shocked context was included. Test involved 11 2-min exposures to the context separated by an interval of 24 hrs. SOP and AESOP predict that the level of response loss will be greatest in the two spaced groups (Groups 5min/24hr and 10min/24hr). In addition, Group 10min/4min should show greater response loss than Group 5min/4min and finally Groups 10min/4min and 5min/4min should show more response loss than Group No Extinction and these differences can be attributed to the differences in the length of the initial context exposure.

Method

Subjects and apparatus. The subjects were 40 experimentally naïve male Wistar rats (Rattus Norvegicus) obtained from the same source and maintained under the same conditions as described in previous experiments. The two contexts were the black and white chambers described in previous experiments, counterbalanced.

Procedure. The experiment consisted of three distinct phases: discrimination training, extinction and test (represented in Table 4). In the first phase rats were trained to discriminate between a shocked (A) and non-shocked (B) context using the same procedure as described in previous experiments. All experimental procedures

took place between 9-11 a.m. and 4-6 p.m. Rats were assigned to 5min/24hr, 10min/24hr, 5min/4min, 10min/4min and No Extinction groups after conditioning in order to match levels of freezing to Context A.

On the afternoon of Day 11 spaced extinction began. Rats in Groups 5min/24h and 5min/4min were placed in Context A (the previously reinforced context) and Context B (the previously non-reinforced context), respectively, for 5 min before being returned to their home cage. This continued each afternoon, spaced 24 hrs apart, for four days, until freezing to A extinguished in the 5min/24h group. Rats in Groups 10min/24h and 10min/4min received handling equivalent to Groups 5min/24h and 5min/4min across Days 11 and 12. On the afternoon of Day 13 rats in the Groups 10min/24h and 10min/4min were placed in Context A and Context B, respectively, for 10 min, before being returned to their home cage. This was repeated 24 hrs later. Rats in the control group received four exposures to Context B separated by 24 hrs, which lasted 5 min or 10 min, equating to a total of 20 min exposure to B over the four days.

On the afternoon of Day 15 massed extinction began. Rats in Group 5min/24h were placed in Context B four times. Each exposure lasted 5 min and exposures were separated by a 4-min interval. Rats in Group 10min/24h were placed in Context B twice. Each exposure lasted 10 min and exposures were spaced apart by 4 min. Rats in Group 5min/4min were placed Context A four times. Each exposure lasted 5 min and was separated by a 4 min interval. Rats in the 10min/4min group were given two Context A exposures each lasting 10 min and separated by a 4 min interval. Rats in the control group received four exposures to Context BA lasting 5 or 10 min and separated by 4 min. All rats were returned to their home cages when

not in the specified context. Four min after the final context exposure, all rats received a final 2-min exposure to Context A. Test consisted of 11 2-min exposures to Context A separated by 24 hrs. They began 24 hrs after the final massed extinction trial for all rats.

Results

Conditioning proceeded such that by the eighth conditioning trial rats showed substantial freezing to Context A and very little freezing to Context B [mean (*SEM*)]: 48.53 (4.1) and 1.58 (1.6), respectively. There was no difference in the level of freezing to the two counterbalanced A contexts so the data was summated ($F < 1$). The left hand panel of Figure 4 shows the mean (±*SEM*) percentage levels of freezing to Context A during extinction training in 2-min blocks. On inspection of the figure, the level of freezing to Context A decreased gradually over extinction exposures to the context. This decline in the freezing response does not appear to differ between groups. These observations were supported by the statistical analysis. There was a significant linear trend, $F(1, 28) = 185.83$, $F_{critical} = 4.20$, confirming that averaged across groups, freezing decreased over extinction trials. Neither the rate of decline nor the overall level of freezing differed between the groups ($Fs < 1$).

The data of major interest are from the test exposures to Context A. The right hand panel of Figure 4 shows the mean (±*SEM*) percentage levels of freezing to Context A over each 2-min test exposure. It is clear from the figure that when tested 24 hrs after the final extinction trial long intervals between context exposures (Groups 5min/24h and 10min/24h) displayed low levels of freezing short intervals (Groups 5min/4min and 10min/4min) irrespective of the duration of the extinction trials. However, short intervals between extinction trials produced lower levels of

Context conditioning	Extinction	Test
A+/B-	[24hr] A-(5-min) x4 — A-(2-min)	[24-hr] A-
	[24hr] A-(10-min) x2 — A- (2-min)	[24-hr] A-
	[4-min] A-(5-min) x4 — A-(2-min)	[24-hr] A-
	[4-min] A-(10-min) x2 — A-(2-min)	[24-hr] A-
	[4-min] B-(5m x4 or 10m x2) — B-(2-min)	[24-hr] A-

Table 4. This table represents the consecutive phases in Experiments 3. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures of Context A.

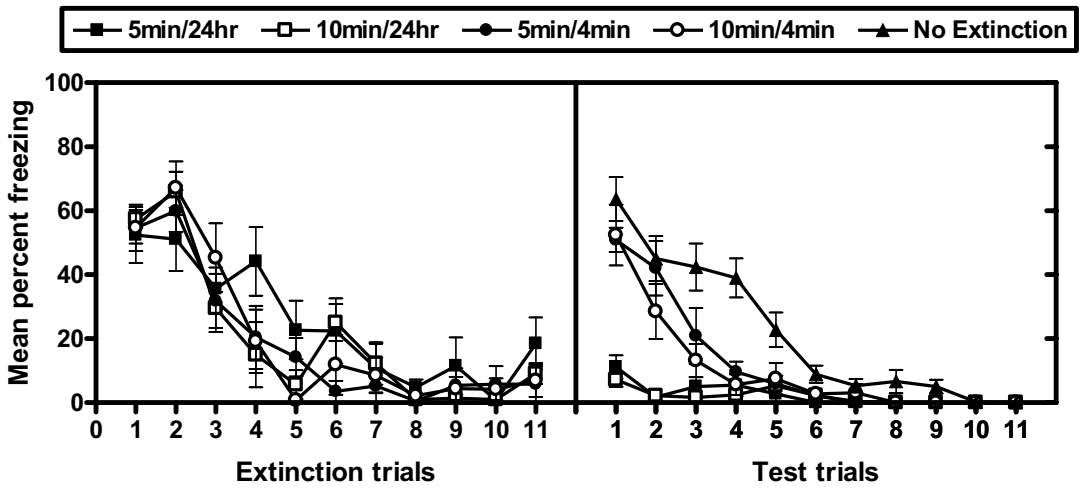

Figure 4. Mean (±SEM) percentage of time spent freezing across extinction trials (in 2-min blocks) and test trials. The rats spent the interval between extinction trials and test trials in the home cage. During extinction training rats in Groups 5min/24hr and 5min/4min received four 5-min and one 2-min exposure to the context separated by an interval of 24-hrs and 4-min, respectively; those in Groups 10min/24hr and 10min/4min had received two 10-min and one 2-min exposure to the context separated by an interval of 24-hr or 4-min, respectively. All groups received a 24-hr interval between test trials. Rats in the No Extinction group received a 24-hr interval between test trials in the absence of prior extinction training

responding than the non-extinguished group. These observations were supported by the statistical analysis, which consisted of a contrast testing procedure that controlled the Familywise error rate at $\alpha = 0.05$ with the Bonferroni procedure (Harris, 1994).

This analysis differed from previous analyses because a set of orthogonal contrasts could not be constructed to test the hypotheses. Rats that received the 4-min interval between context exposures and the non-extinguished group froze significantly more than rats that received a 24-hr interval between context exposures, F (1, 35) = 45.55, Bonferroni $F_{critical}$ (k = 4, df = 35) = 6.95. The level of responding in Groups 5min/4min and 10min/4min did not differ, F (1, 35) = 2.03, nor did the level of responding differ between Groups 5min/24h and 10min/24hr ($F < 1$). However, Groups 5min/4min and 10min/4min froze significantly less than the non-extinguished control group, F (1, 35) = 12.08. There was a significant linear trend, F (1, 35) = 126.57, indicating that responding decreased over test trials. There was also a significant interaction between the linear trend and the contrast testing for differences between Groups 5min/4min, 10min/4min and No Ext and Groups 5min/24hr and 10min/24hr, F (1, 35) = 54.01. This finding confirms that short intervals between extinction exposures and no extinction training produce a gradual reduction in conditioned responding across test trials compared to longer intervals that produce stable low levels of freezing.

Discussion

These results indicate that long intervals between extinction trials produce greater response loss compared to shorter intervals and short and long intervals produce the same level of response loss regardless of whether extinction trials were 5 min or 10 min in duration. However, short intervals between extinction trials of 5

min and 10 min in duration produced greater long term response loss compared to a non-extinguished control group. This experiment does not provide direct support for the predictions made by SOP and AESOP as longer trial durations did not produce more response loss compared to shorter trial durations. However, long intervals between extinction trials produce a complete loss in responding, thus the effect of the trial duration would only be seen under massed training conditions. Finally, equivalent levels of response loss produced by short intervals between extinction trials that were 5 and 10 min in duration may have been due to an inadequate difference between the selected trial durations

Experiment 4

The results of the previous experiment demonstrated that 5 min and 10 min long massed extinction trials did not produce different levels of response loss but facilitated response loss compared to a non-extinguished control. Previous experiments (Experiment 1a and 1b) have demonstrated that massed extinction trials that are 2 min in duration do not produce greater response loss than a non-extinguished control. Therefore, based on the results of previous experiments 2 min and 10 min long massed extinction trials should produce different levels of response loss. The aim of the current experiment was to test this prediction by comparing the level of long-term response loss produced by massed extinction trials that are 2 min and 10 min in duration.

Rats were shocked in one context (A) but not in another (B). They then received non-reinforced exposures to the conditioned context. Group Massed received 11 2-min exposures separated by an interval of 4 min, while the

10min/4min group received two 10-min exposures separated by an interval of 4 min. Both groups received a final 2-min context exposure 4 min after the previous exposures. The interval between context exposures was spent in the rat's home cage. A non-extinguished control group was included. Test consisted of 11 2-min exposures to the context separated by an interval of 24 hr. If long context exposures facilitate response loss then Group 10min/4min should freeze less than Group Massed, which should not differ from the non-extinguished control (based on the results of previous experiments).

Method

Subjects and apparatus. The subjects were 32 experimentally naïve male Wistar rats (Rattus Norvegicus) obtained from the same source and maintained under the same conditions as described in previous experiments. The black and white chambers described in previous experiment were used as Contexts A and B.

Procedure. The experiment consisted of three distinct phases: conditioning, extinction and test (shown in Table 5). In discrimination training rats were trained to discriminate between a shocked (A) and non-shocked (B) context using the same procedure described in previous experiments. Rats were assigned to Massed, 10min/4min and No Extinction groups after conditioning in order to match levels of freezing to Context A. On the afternoon of Day 11, rats in the Massed group and the No Extinction group received 11 2-min exposures to Context A and Context B, respectively. Each exposure was separated by an interval of 4 min that was spent in their home cages. Rats in the 10min/4min group received eight 2-min exposures to Context B that was followed by two 10-min exposures to Context A. Each exposure was separated by an interval of 4 min that was spent in the rat's home cage. Test

consisted of 11 2-min exposures to Context A separated by an interval of 24 hrs that began 24 hrs after the final massed extinction trial for all rats.

Results

Discrimination proceeded such that by the eighth trial rats showed substantial freezing to Context A and very little freezing to Context B [mean (*SEM*)] : 63.59 (3.9) and 6.53 (3.4), respectively. There were no differences in the levels of conditioning to the two counterbalanced contexts so the data was summated. The left hand panel of Figure 5 shows the mean (±*SEM*) percentage levels of freezing to Context A across extinction trials in 2-min blocks. Inspection of the figure suggests that the level of freezing to Context A was similar for both groups although Group Massed showed a more rapid initial decline in freezing than Group10min/4min. The analysis revealed that there were no differences between groups in the level of freezing to Context A, $F < 1$. There was a significant linear trend, F (1, 14) = 47.00, $F_{\text{critical}} = 4.60$, confirming that freezing decreased over extinction training for both groups. There was a significant interaction between the linear trend and the contrast testing for differences in freezing between Group Massed and Group 10min/4min, F (1, 14) = 4.76. This finding indicates that the decrease in freezing across extinction training was greater for Group Massed compared to Group 10min/4min. The right hand panel of Figure 5 shows the mean (±*SEM*) percentage level of freezing to Context A over each 2-min test trial. The figure reveals that Groups Massed and No Extinction displayed equivalently high initial levels of freezing compared to Group 10min/4min. All three groups show a gradual decline in the freezing response over test trials. These observations were supported by the statistical analysis. Rats in Groups Massed and No Extinction froze significantly more than rats in

Context conditioning	Extinction	Test
A+/B-	[24hr] A-(2-min) x10 — A- (2-min)	[24-hr] A-
	[4-min] A-(10-min) x2 — A-(2-min)	[24-hr] A-
	[4-min] B-(2-min) — B-(2-min)	[24-hr] A-

Table 5. This table represents the consecutive phases in Experiments 4. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures to Context A.

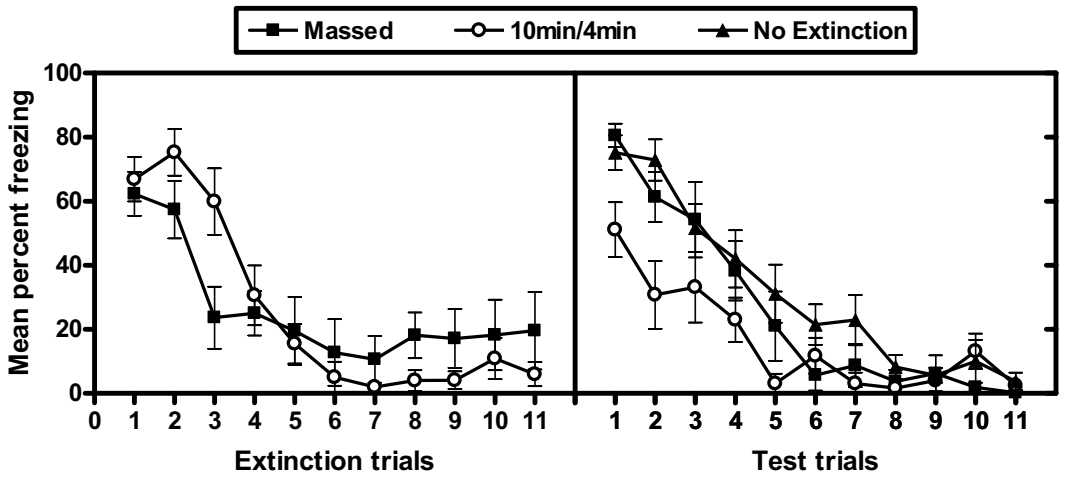

Figure 5. Mean (±SEM) percentage of time spent freezing across extinction trials and test trials. The rats spent the interval between extinction trials and test trials in the home cage. During extinction training rats in Group Massed had received 11 2-min context exposures; those in Group 10min/4min had received two 10-min and one 2-min exposure to the context. Both groups received a 4-min interval between extinction trials and a 24-hr interval between test trials. Rats in Group No Extinction received a 24-hr interval between test trials in the absence of prior extinction training.

Group 10min/4min, F (1, 21) = 7.12, $F_{critical}$ = 4.33. The level of freezing between Groups No Extinction and Massed did not differ, F (1, 21) = 1.14. There was a significant linear trend, F (1, 21) = 255.37, confirming that freezing decreased over test trials for all groups. There was also a significant interaction between the linear trend and the contrast that tested for differences between Groups No Extinction and Massed and Group 10min/4min, F (1, 21) = 17.95, indicating that the decrease in freezing across test trials was more rapid for Groups No Extinction and Massed compared to Group 10min/4min.

Discussion

These results replicate those produced in the previous experiment by demonstrating that the duration of the context exposure affects long term response loss. Specifically, longer trial durations facilitate long-term response loss compared to short trial durations. The level of responding produced by extinction trials that were short in duration did not differ from the level of responding displayed by non-extinguished control rats. These results also support the predictions made by the SOP model that context exposures that are long in duration promote the formation of inhibitory associations between the context (CS) and the US thus producing greater response loss compared to context exposures that are shorter in duration.

Experiment 5

Experiments 1a and 1b demonstrated that massed training produced better response loss but worse long-term learning than spaced training. SOP and AESOP explain this effect in terms of a loss in the associability of the context CS across the massed trials. The interval between these trials was such that trial N+1 overlapped

with the context CS in A2 from trial N. This overlap reduces the proportion of context CS nodes excited to A1 that, in turn, reduces the proportion of US nodes associatively excited to the A2 state. This leads to a loss of conditioned responding and impairment in inhibitory associations. However, massed training did not just impair inhibitory associations: it did not appear to have produced any such associations. The degree of impairment produced by massed training was surprising. The initial trials should have produced some learning even if they reduced associability across the subsequent trials.

The present experiment provided a further study of the effects of massed training on long-term learning. The aim was to identify the number of massed trials required to produce a loss of associability. The design consisted in training the discrimination between the shocked (A) and the non-shocked Context (B) and then subjecting rats to extinction trials in Context A. Those in Group Spaced received a single trial each day across 11 days, whereas those in Groups Massed-3, Massed-6, and Massed-11 received three, six, or 11 massed extinction trials each day across the 11 days. The levels of freezing on the initial trial each day provides a measure of the long-term learning produced by one extinction trial or by three, six, or 11 massed trials on the preceding day. If associability is lost after a single trial, then the levels of freezing on the initial trial across each of the days will be similar in all the groups; if the initial three trials produce learning but then a loss in associability, the levels of freezing will be higher among rats in Groups Spaced than in the remaining groups, and so on. The second aim was to assess whether the rate of response loss across the massed trials on a given day was influenced by the number of such trials on the preceding day. Thus, if six or 11 massed trials produced better response loss than

three such trials, rats in Groups Massed-6 and Massed-11 will freeze less on trials 2 and 3 than those in Group Massed-3; if 11 massed trials produced better response loss than six trials, rats in Group Massed-11 will freeze less across trials 4, 5 and 6than those in Group Massed-6.

Method

Subjects and apparatus. The subjects were 32 experimentally naïve rats (350 - 450 g), of the same sex and strain, and obtained from the same source as those in Experiments 1a and 1b. They were maintained in the conditions described previously. The two contexts (A and B) used in this experiment were the white and black chambers described previously.

Procedure. Table 6 depicts the consecutive phases of this experiment. Rats were trained to discriminate between a shocked (A) and a non-shocked (B) context according to the protocol used previously. The chambers serving as A and B were counterbalanced. Then rats were exposed to Context A in the absence of shock. Each day for 11 days, rats in Groups Massed-3, Massed-6, and Massed-11 received, respectively, 3, 6, or 11 extinction trials in Context A. Each trial was 2 min in duration and the interval between trials was 4 min. This interval was spent in the home cages. On each of these days, rats in Group Spaced received a single 2-min extinction trial in Context A. Rats in Groups Spaced, Massed-3 and Massed-6 groups were handled after their daily extinction trial(s) in order to equate for the amount of handling received by rats in Group Massed-11.

Results

Discrimination training was successful. The mean (*SEM*) percentage levels of freezing prior to shock in A and those in B on the final discrimination sessions were

73.23 (4.1) and 33.74 (5.8), respectively. There were no differences in the levels of freezing to the two counterbalanced contexts (within each group) and data were collapsed across the counterbalancing. There were no significant differences between groups in the levels of freezing to the A or B contexts (*Fs* < 1). The data of major interest are the levels of freezing across the 11 days of extinction. These are shown in Figure 6. Panel A shows the mean (±*SEM*) percentage levels of freezing by rats in each of the four groups on the initial trial each day across the 11 days; Panel B the levels of freezing by rats in the three Massed groups averaged across Trials 2 and 3 each day; Panel C the levels of freezing by rats in Groups Massed-6 and Massed-11 averaged across Trials 4 – 6 each day; while Panel D shows the levels of freezing by rats in Group Massed-11 averaged across Trials 7 – 11 each day across the 11 days.

An inspection of Panel A suggests that the levels of freezing on Trial 1 declined across the 11 days of extinction but that the rate of decline was largely unaffected by the number of extinction trials within each day. Panels B and C show that the levels of freezing on Trials 2 – 3 and Trials 4 – 7, respectively, also declined across days but that the rate of this declines was again unaffected by the number of daily extinction trials. Finally, Panel D shows that freezing averaged across Trials 7 – 11 among rats in Group Massed-11 declined across days.

Separate analyses compared the levels of freezing by rats in each of the groups on Trial 1, by rats in the three Massed groups on the average of Trials 2 and 3, and by those in Groups Massed-6 and Massed-11 on the average of Trials 4 – 6. Finally, a within-group analysis compared the levels of freezing averaged over Trials 7 – 11 by rats in Group Massed-11 across days. The analysis of Trial 1 revealed a significant linear trend, confirming that the levels of freezing declined across days, *F*

Context conditioning	Extinction session/day (Days 11-21)
A+/B-	[4-min] A- (x10) A-
	[4-min] A- (x5) A-
	[4-min] A- (x2) A-
	A-

Table 6. This table represents the consecutive phases in Experiments 5. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures

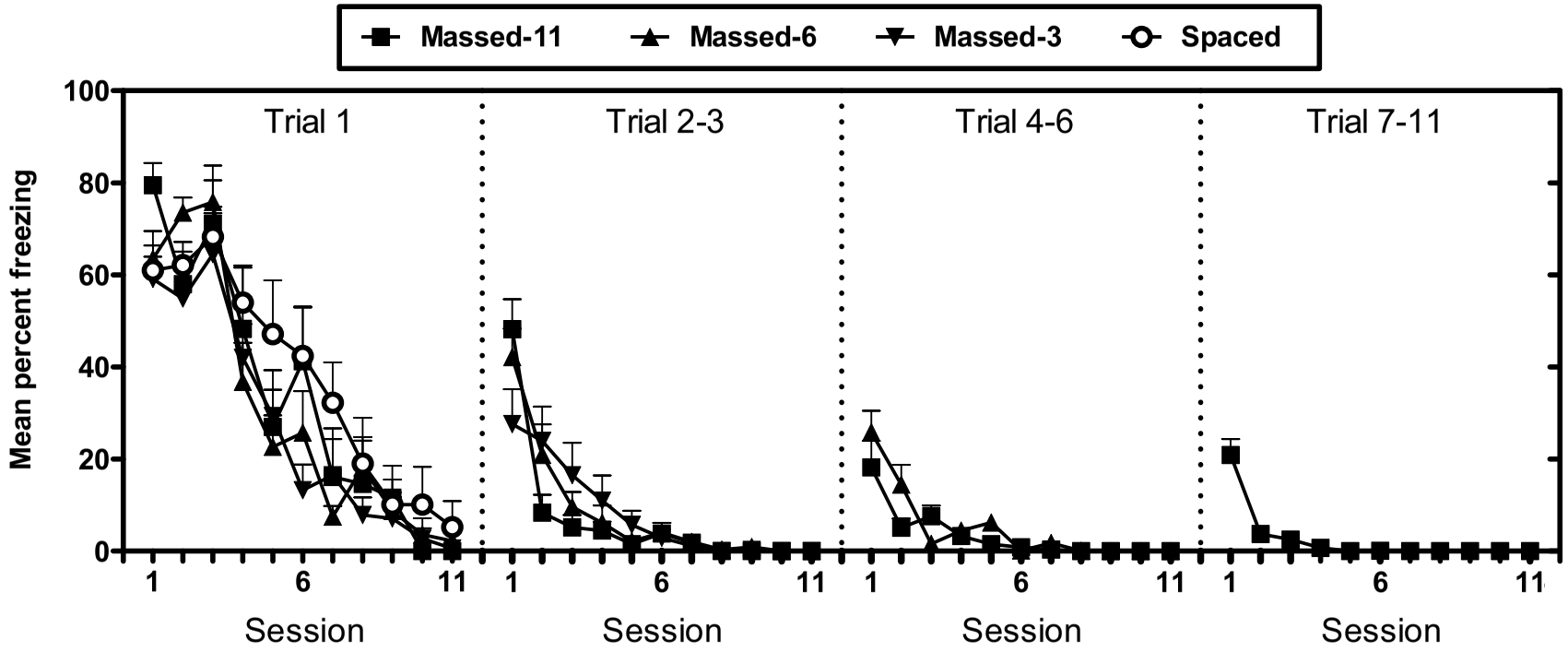

Figure 6. Mean (±SEM) percentage of time spent freezing in each of the four groups on the initial trial each day across the 11 days; for the Groups Massed averaged across Trials 2 and 3 each day; for Groups Massed-6 and Massed-11 averaged across Trials 4 – 6 each day; and for Group Massed-11 averaged across Trials 7 – 11 each day across the 11 days. All rats had received a 4-min interval between extinction trials that was spent in their home cages; those in Group Spaced had received one exposure to the context each day; those in Group Massed-3 had received three exposures to the context each day; those in Group Massed-6 had received six exposures to the context each day; and those in Group Massed-11 had received eleven exposures to the context each day. .

(1, 28) = 662.01, $F_{critical}$ = 4.20. However, there were no statistically significant between group differences in the levels of freezing, largest F (1, 28) = 1.14, nor were there any statistically significant group x trend interactions, largest F (1, 28) = 2.00. The analyses of the levels of freezing by rats averaged over Trials 2 – 3 and averaged over Trials 4 – 6 revealed significant linear trends, F (1, 21) = 43.49, $F_{critical}$ = 4.33 and F (1, 14) = 22.80, $F_{critical}$ = 4.60, respectively. This confirms that these levels of freezing declined across days. But the rate of decline in these levels did not differ among groups: There were no statistically significant trend x group interactions (*Fs* < 1) nor any statistically significant between-group differences (Fs < 1, and F (1, 14) = 2.66, respectively). The within-group analysis of the levels of freezing averaged over Trials 7 – 11 by rats in Group Massed-11 revealed a significant linear trend, F (1, 7) = 20.56, $F_{critical}$ = 5.60, confirming that the levels of freezing across these trials declined across days.

Discussion

These results show that rats subjected to a single, daily extinction trial lost fear responses across days just as rapidly as did rats that received three, six or 11 daily massed extinction trials. This suggests that a single extinction trial each day was just as effective as multiple massed trials in promoting long-term learning. This, in turn, implies that the first of the massed trials impairs the associability of the remaining trials and, thereby undermines the inhibitory learning assumed to underlie long-term response loss. The present results have also shown that rats subjected to three massed extinction trials lost fear responses within each day just as rapidly as those receiving six or 11 massed trials. And rats subjected to six massed trials lost fear responses within each day just as rapidly as did rats receiving 11 such trials.

Thus, the rate of response within a day was unaffected by the number of such trials on the preceding day. This could mean that rats did not learn to use the initial trials as a signal for extinction of later ones or, perhaps, that rats subjected to three massed trials acquired just as much occasion-setting as did those that received six or 11 such trials.

Experiment 6

The failure to detect any differences in the rate at which freezing responses declined on the initial trial each day suggests that extinction learning occurs on the first of the massed trials and that little or no additional learning occurs across the remaining trials. According to SOP and AESOP, the conditions for learning are present on the first but not on subsequent trials. On the first trial, inhibitory learning occurs because representations of the context CS and the US are each concomitantly active in A1 and A2 states, respectively, but this learning is reduced on subsequent trials as the representation of the context has decayed into an A2 state. SOP and AESOP (like the Rescorla-Wagner model) propose that associative formation (excitatory or inhibitory) is regulated by prediction error and that all the cues present contribute to this error. Thus, the presence of an excitatory CS on a context extinction trial will increase that error and thereby the amount of extinction learning accruing to the context (and that CS). But, critically, this increase in the amount of extinction to the context (or the CS) will only occur if the CS is presented on the first of the massed context extinction trials. Extinction learning will be increased on the first trial because both the context and the CS excite US representations to an A2 state of activity according to SOP and AESOP. Or, it will be increased because

associative change is regulated by λ - ∑V where λ equals zero and ∑V equals the sum of the excitatory strengths (V) of the context and CS according to Rescorla-Wagner. However, extinction learning will not be increased when the excitatory CS is located on subsequent context extinction trials because the context representation is in A2 and, therefore, cannot excite its associated US representation to the A2 state of activity. Or, extinction learning will not be increased because the salience (α) of the context has declined to zero and is therefore unable to contribute its V value to the error term.

The present experiment studies these predictions. The design consisted in training rats to discriminate between a shocked context (A+) and a non-shocked context (B-). Then they received pairings of a noise and shock (N+) in a third context (C). Massed extinction training consisted of eleven non-shocked exposure to Context A. Each trial was 2 min in duration and trial were separated by a short interval (4 min) spent in the home cages. Rats in Groups First, Middle and Last received a 30-s presentation of the noise CS during the first, middle (6th) or final (11th) extinction trial in Context A, respectively. Rats in the Massed group underwent massed extinction in the absence of any CS presentations Testing consisted of six additional context extinction trials spaced 24 hr apart. These began 24 hrs after the final training trial. Finally, rats were tested with the noise CS in a fourth neutral context (D).

Method

Subjects. The subjects were 32 experimentally naïve rats (350 - 450 g), of the same sex and strain, and obtained from the same source as those in Experiments 1a and 1b. They were maintained in the conditions described previously.

Apparatus. Four contexts were used in this experiment. The two contexts (A and B) used for discrimination training were the white and black chambers described in the previous experiment. A set of four chambers in a third room were used for CS conditioning. Each chamber was 21 x 23 x 23-cm (width, height, length). The front and back walls and lids were made of clear Perspex, and the side walls were made of aluminum. The floor consisted of stainless steel rods, 2mm in diameter, spaced 13 cm apart (center to center), with a removable tray containing bedding below. The floor and side walls of each chamber were clean upon the removal of each rat with a 1% acetic acid solution. An incandescent (60W) bulb located in the ceiling illuminated the room. A speaker (160 mm diameter wideband width) was attached to the ceiling. The speaker was wired to a generator that could provide a white noise CS that consisted of a 68 dB, 10-Hz spike (rise time < 1.0 μs and a decay time of 250 μs). Unscrambled AC 50Hz shock from a custom-built constant current generator could be delivered to the floor of each chamber. The background noise level in each of the chambers was 65 dB

The context used for testing the white-noise CS consisted of two plastic chambers (44-cm length x 26-cm width x 16-cm height). The front wall of each chamber was constructed of Perspex. The floor and the side and rear walls were made of black plastic, and the roof was made of stainless steel rods. These chambers were located on the roof of the wooden cabinet that contained the chambers used to condition the white-noise CS. The background noise in each of these chambers was 69 dB. The white noise and background noise were measured in the manner described previously. Each chamber was located in a separate compartment of a

wooden cabinet, with the doors of the cabinet open to allow observation in the manner described previously.

Procedure. The experiment consisted of four phases shown in Table 7: discrimination training, CS conditioning, massed extinction and testing. Phase 1 consisted of the discrimination training described in previous experiments, with the exception that CS conditioning was conducted Day 7, after which discrimination training resumed as usual. Two CS conditioning trials took place on Day 7, one in the morning, the other 6-hrs later in the afternoon. Each trial consisted in subjects being exposed to a third context. Ten minutes after placement in this context, they received a 30-s, 68dB white noise that co-terminated in foot shock (0.5-s x 0.6 mA). Rats remained in the context for a further nine minutes and 30-s before being removed and returned to their home cages. Rats were assigned to Massed, First, Middle and Final groups after conditioning in order to match levels of freezing to Context A and the CS.

Massed extinction consisted of 11 exposures to Context A. Each exposure was 2 min in duration and exposures were separated by an interval of 4 min. This interval was pent in the home cages. Rats in the First, Middle and Final groups received a 30-s presentation of the white noise CS during either the first, middle (6^{th}) or final (11^{th}) exposure to Context A. On these exposures, rats received a 30-s CS presentation that began 1 min after placement in the context. Rat remained in the context for a further 30 s and were returned to their home cage. Rats in the Massed group underwent massed extinction in the absence of any CS presentations. Test consisted of six exposures to Context A. Each was 2 min in duration and the interval between exposures was 24 hr. This interval was spent in the home cages. These

additional, spaced extinction tests began 24 hrs after the final massed extinction trial. On the day following the final spaced extinction test, rats were tested with the CS. This test consisted of exposing each rat to the plastic chambers for 30 s before presenting the noise CS for 120-s, after which rats were returned to their home cages.

Results

Discrimination training was successful: On the final sessions, rats showed substantial freezing in Context A but not in Context B [Mean (*SEM*)]: 62.02 (3.9) and 12.93 (4.4), respectively. There were no significant differences between the counterbalanced contexts ($F < 1$). CS conditioning was also successful: There was substantial freezing to the 30-s CS on its second presentation [Mean (*SEM*): 47.42 (4.7)] and very little freezing to the context in the preceding 30 s interval [Mean (*SEM*): 3.71 (2.7)].

Figure 7 shows the data of major interest. The left panel shows the mean (±*SEM*) percentage levels of freezing across massed extinction training; the center panel shows the levels of freezing across spaced extinction tests; the right panel shows the levels of freezing in the period before and during the CS test. Inspection of the left panel shows that freezing to Context A decreased gradually over the 11 extinction trials. It is clear that the noise CS elevated freezing when presented on the 6^{th} (Group Middle) and 11^{th} (Group Last) context extinction trial. The statistical analysis revealed a significant linear trend, $F\ (1, 26) = 46.59$, $F_{critical} = 4.23$, confirming that freezing decreased across massed extinction trials. There were no significant differences in the rate of decrease between groups ($Fs < 1$). An analysis of the levels of freezing before and during the CS presentations revealed that the differences between these levels were greater on the 6^{th} and 11^{th} presentations than

Context cond'ning	Extinction	Context test	CS test
A+/B-	[4-min] [A-CS-] A- A- A- A- A- A- A- A- A- A-	[24-hr] A-	[24-hr] CS-
	[4-min] A- A- A- A- A- [A-CS-]A- A- A- A- A-	[24-hr] A-	[24-hr] CS-
	[4-min] A- A- A- A- A- A- A- A- A- A [A-CS-]	[24-hr] A-	[24-hr] CS-
	[4-min] A- A- A- A- A- A- A- A- A- A- A-	[24-hr] A-	[24-hr] CS-

Table 7. This table represents the consecutive phases in Experiments 6. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Context test consisted of six exposures to Context A.

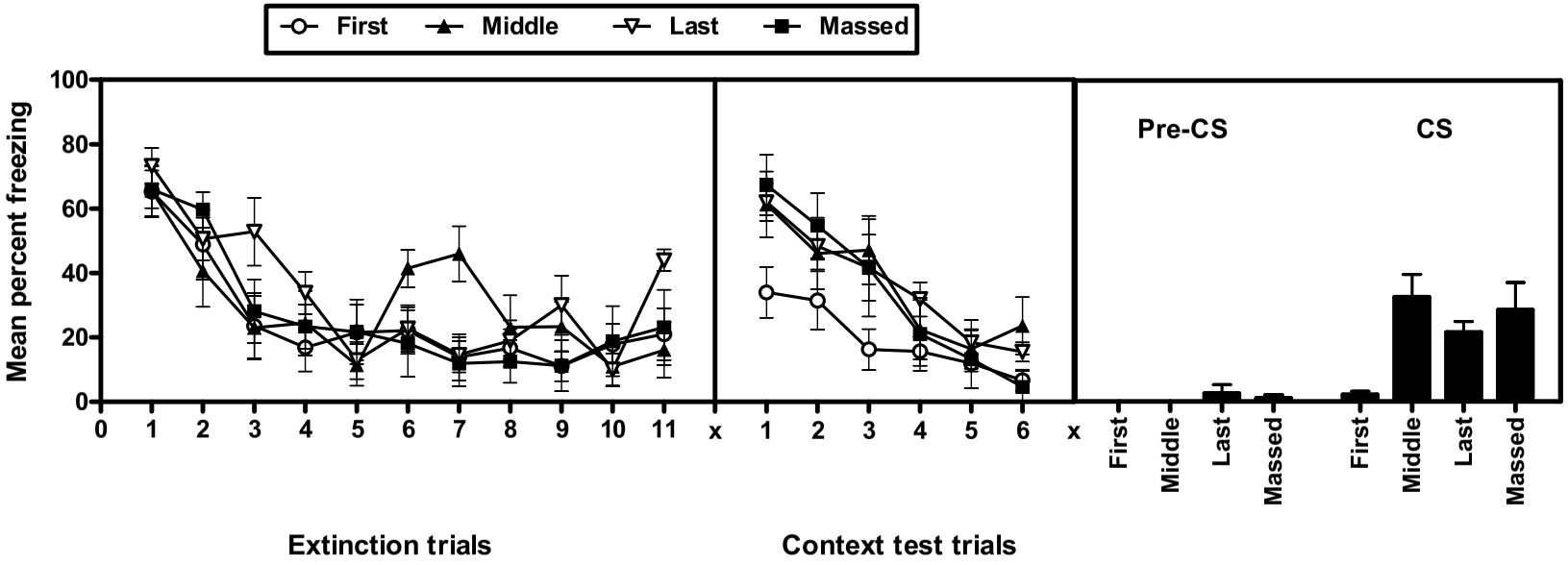

Figure 7. Mean (±SEM) percentage of time spent freezing across massed extinction training (left panel), space extinction test trials (centre panel), and the period before and during the CS test (right panel). The rats spent the interval between extinction trials and test trials in their home cages. All groups had received a 4-min interval between extinction trials and a 24-hr interval between test trials. Rats in Groups First, Middle and Last received a 30-s presentation of a white noise CS during the first, middle (6th) or last (11th) context presentation of extinction training, respectively. Rats in the Massed group underwent massed extinction in the absence of any CS presentations.

on the 1st presentation, F (1,19) = 34.88, $F_{critical}$ = 4.38, and that there were no significant differences between the pre CS or between the CS levels of freezing on the 6th and 11th presentations, $F < 1$.

Inspection of the center panel suggests that presentation of the CS on the 1st context extinction trial produced better learning than presentation on later trials or without a presentation on any trial. The statistical analysis confirmed that rats in Group First displayed significantly less freezing across the spaced extinction tests than rats in the remaining groups, F (1, 26) = 4.45, $F_{critical}$ = 4.23. There were no significant differences between the levels of freezing among the remaining groups, $Fs < 1$. The significant linear trend, F (1, 26) = 156.40, confirmed that the levels of freezing declined across test trials. The linear trend interacted significantly with the differences between Group First and the remaining groups, F (1, 26) = 8.00, which, from inspection, reflects the lower levels of freezing with which rats in Group First entered spaced extinction testing. No other interactions were significant, $Fs < 3.14$.

Inspection of the right panel shows that there was little or no freezing when rats were exposed to the plastic chambers used as the context for CS testing. In contrast, there was substantial freezing to the noise CS except among those rats that had been exposed to the noise on the initial context extinction trial. The statistical analysis confirmed these observations. There were no significant differences between groups in the levels of freezing to the context prior to the onset of the CS, $Fs < 1$. It also showed that rats in Group First froze significantly less to the noise CS than rats in the remaining groups, F (1, 26) = 14.04. There were no other differences between groups, $F < 1.82$.

Discussion

These results show that the location of the noise CS on the initial context extinction trial produced greater learning about both the noise and the context than when it was located on later trials. The location of the noise CS on the initial context extinction trials also produced more learning about the context than when the noise was not presented on any trial. Learning about the context thus benefited from the presence of the noise but only when the context itself supported such learning. The initial context trial enjoyed that benefit but not the other trials. These results thus confirm that long-term extinction learning occurs on the first in a series of massed extinction trials but does not occur on the subsequent trials.

Experiment 7

Self-generated priming constitutes a mechanism by which the first in a series of massed trials undermines the ability of subsequent trials to produce extinction learning. However, the previous experiment did not provide direct evidence for such a mechanism. It simply showed that extinction learning occurred on the first of the massed trials and that there was no detectable evidence for such learning on the subsequent trials. Evidence for such a mechanism entails showing that the amount of learning produced by exposure to the context on trial N+1 is less when it follows trial N than where there had been no preceding trial. Experiments 3 and 4 demonstrated that a long trial duration facilitates long term response loss compared to a short trial duration. If self-generated priming prevents subsequent trials from producing extinction learning it can be assumed that it was the initial extinction trial alone that was responsible for this facilitated response loss and subsequent extinction trials were redundant. In addition, experiments in this laboratory have shown that the

duration of an extinction trial influences long-term extinction learning. For instance, rats exposed for 20-min to a dangerous context in the absence of shock froze less when tested 24 hr later than rats exposed to that context for 2-min (Laurent, Marchand, & Westbrook, in progress).

The present experiment uses this finding to provide evidence for self-generated priming as a mechanism for the effects of massed training on long-term extinction learning. Rats were trained to discriminate between two contexts, one of which (A) was shocked and the other (B) was not shocked. Then rats received extinction training in Context A. Two groups received a single non-shocked exposure to Context A. The duration of this extinction trial was 2 min for rats in Group 2-min and 20 min for those in Group 20-min. The intention was to confirm that a 2-min exposure was less effective in producing long-term extinction learning than a 20-min exposure. Two other groups received two non-shocked exposures. The total amount of time spent in the context was the same in each of these groups but the manner in which this time was distributed was different. The initial exposure (trial N) was 2 min and the second (trial N+1) was 20 min in duration for rats in Group 2<4>20, whereas the duration of trial N was 20 min and that of trial N+1 was 2 min for those in Group 20<4>2. The interval between these exposures was short (4 min) and was spent in the home cages. A fifth group (No Extinction) did not receive any extinction but were handled. Finally, rats were tested. This consisted of a daily 2-min non-shocked exposure to Context A across 11 days. This interval between the daily trials was spent in the home cages. Rats subjected to a 20-min extinction session (Groups 20-min and 20<4>2) should acquire better extinction learning than those that received a 2-min extinction session (Group 2-min). However, the question of interest is the amount of learning by rats in Group 2<4>20. If the amount learned

is determined by the total time spent in the to-be-extinguished context, then rats in Group 2-min should have learned less than those in Groups 20min, 20<4>2 and 2<4>20. If the amount learned across the 20-min exposure is impaired by the preceding 2-min exposure, then rats in Group 2<4>20 should have learned similar amounts as those in Group 2-min and this learning should be less than that by rats in Groups 20-min and 20<4>2.

Method

Subjects and apparatus. The subjects were 40 experimentally naïve rats (300 – 450 g) of the same sex, strain, from the same source, and maintained under the same conditions as described previously. The two contexts used were the white and black chambers described in the previous experiments.

Procedure. The experiment consisted of discrimination training between a shocked (A) and non-shocked (B) context, extinction in Context A, and spaced testing of Context A. These phases of training are represented in Table 8. Rats were trained to discriminate between Contexts A and B using the same procedure described in previous experiments. On the last three days of discrimination training all rats received additional daily exposures to plastic buckets for approximately 5 min. All experimental procedures took place between 9-11a.m. and 4-6p.m. Rats were assigned to the 2-min, 20-min, 2<4>20, 20<4>2 and No Extinction groups after conditioning in order to match levels of freezing to Context A.

For the 20<4>2 and 2<4>20 groups extinction training consisted of two exposures to the conditioned context (A). The interval between the exposures was 4 min and was spent in the home cages. Rats in the 20<4>2 group received a 20-min exposure followed by a 2-min exposure, while rats in the 2<4>20 group received a 2-min exposure followed by a 20-min exposure. Rats in the 20-min and 2-min groups

received a 20-min exposure to A followed by a 2-min exposure to a plastic bucket and a 2-min exposure to A followed by a 20-min exposure to a plastic bucket, respectively. The No Extinction group received two exposures to the plastic buckets. The duration of one exposure was 2 min and that of the other was 20 min. For half the rats in Group No Extinction the 2-min exposure preceded the 20-min exposure, and for the other half the 20-min exposure preceded the 2-min exposure. On the following day, testing began. This consisted in a daily 2-min extinction session in Context A across 11 days.

Results

Discrimination training was successful: On the final sessions, freezing was substantial in Context A and negligible in B [Mean (*SEM*)]: 62.87 (4.5) and 9.92 (4.9), respectively. The data of major interest are the levels of freezing across extinction training and testing in Context A. These are shown in Figure 8. The left panel shows the mean percentage (±*SEM*) levels of freezing across blocks of 2-min during extinction training: the freezing by rats in Groups 2-min and 2<4>20 on the initial 2-min exposure; by rats in Groups 2<4>20, 20-min, and 20<4>2 across the 20-min exposure; and by rats in Group 20<4>2 on their final 2-min exposure. Inspection of freezing across the 20-min exposure reveals a gradual decline in the freezing response but little or no differences in the rate of this decline. The statistical analysis confirmed these observations. There was a statistically significant linear trend, F (1, 21) = 75.76, $F_{\text{critical}} = 4.33$, but no statistically significant differences in the levels of freezing between groups ($Fs < 1$) or in the interaction between linear trend and groups, F (1, 21) < 1.93.

Context conditioning	Extinction	Test
A+, B-, P-	[4-min] A-(2-min) P-(20-min)	[24-hr] A-
	[4-min] A-(20-min) P-(2-min)	[24-hr] A-
	[4-min] A-(2-min) A-(20-min)	[24-hr] A-
	[4-min] A-(20-min) A-(2-min)	[24-hr] A-
	[4-min] P-(2 or 20-min) P-(2 or 20-min)	[24-hr] A-

Table 8. This table represents the consecutive phases in Experiments 7. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures to Context A.

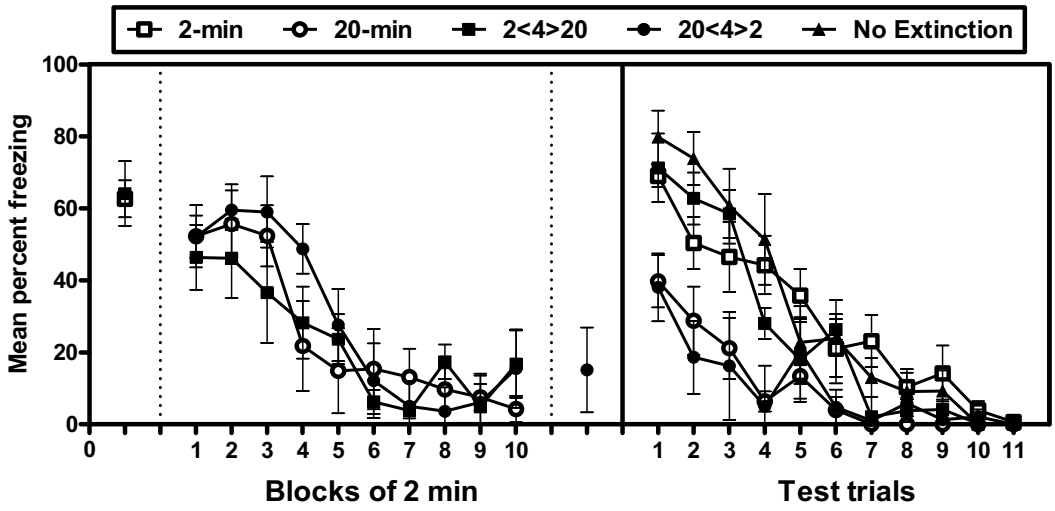

Figure 8. Mean (±SEM) percentage of time spent freezing across 2-min blocks during extinction training: the freezing by rats in Groups 2-min and 2<4>20 on the initial 2-min exposure; by rats in Groups 2<4>20, 20-min, and 20<4>2 across the 20-min exposure; and by rats in Group 20<4>2 on their 2-min exposure. The rats spent the interval between extinction trials and test trials in their home cages. During extinction training rats in the 2-min and 20-min groups received a single 2-min and 20 minute exposure to the context, respectively; those in the 2<4>20 and 20<4>2 groups received a 2-min exposure followed by a 20-min exposure, and a 20-min followed by a 2-min exposure to the context, respectively. For both groups the exposures were separated by an interval of 4 min. All groups received a 24-hr interval between test trials. Rats in the No Extinction group received a 24-hr interval between test trials in the absence of prior extinction training.

The right hand panel shows the mean percent (±*SEM*) levels of freezing in Context A across the 11 days of spaced testing. It is clear that rats in Groups 20-min and 20<4>2 froze less across the initial tests than did rats in the remaining groups. It is also clear that freezing declined across test days in all groups. These data were analyzed with a contrast testing procedure that used the Bonferroni inequality to control the Family Wise error rate at $\alpha = 0.05$ (Harris, 1994). The analysis confirmed that rats in Groups 2-min, 2<4>20 and No Extinction froze significantly more than those in Groups 20-min and 20<4>2, F (1, 35) = 32.82, $F_{critical} = 6.95$. There were no statistically significant differences between the levels of freezing by rats in Groups 2-min, 2<4>20 and No Extinction, ($Fs < 1$), nor between the levels of freezing by rats in Groups 20-min and 20<4>2 groups, ($F < 1$). There was a significant linear trend, F (1, 35) = 312.45, showing that freezing decreased across test trials. There was also a statistically significant interaction between the contrasts testing for the linear trend and the differences between Groups 2-min, 2<4>20 and No Extinction versus Groups 20-min and 20<4>2 groups, F (1, 35) = 39.72. From inspection of the figure, this interaction reflects the higher initial levels of freezing and, hence, the greater decline among rats in Groups 2-min, 2<4>20 and No Ext. None of the other interactions reached conventional levels of statistical significance, F (1, 35) < 3.02.

Discussion

These results confirm that the duration of an extinction trial influences long-term response loss: rats subjected to a 20-min trial froze less across subsequent spaced testing than rats exposed to a 2-min trial. Thus, the longer extinction trial produced more extinction learning than did the short trial. More importantly, they have also provided evidence that the order of exposure to the short and the longer

trials also influences learning. Rats subjected to a 20-min exposure followed a few minutes later by a 2-min exposure subsequently exhibited levels of freezing similar to those by rats subjected to the 20-min trial alone. Essentially, the additional 2-min trial contributed nothing to the learning already produced by the immediately preceding 20-min trial. However, rats subjected to a 2-min exposure followed shortly by a 20-min exposure subsequently froze at the same levels as rats that received a 2-min trial or rats not subjected to any extinction training. Thus, the initial 2-min extinction trial impaired the long-term learning otherwise produced by the 20-min extinction trial. Effectively, the 20-min trial contributed nothing to whatever learning had already occurred on the immediately preceding 2-min trial. The associability of the context had been lost.

Experiment 8

The previous experiment demonstrated that learning occurs on the first of a series of massed trials and that the duration of this trial determines the amount of that learning. More importantly, it also showed that this trial reduces the amount learned on the subsequent trial: the short duration trial impaired the ability of the subsequent long duration trial to produce learning. Thus, the first context trial causes a loss in the associability across subsequent trials. What restores associability? According to SOP and AESOP, associability is regulated by the proportion of context nodes that are in an A2 state of activity: the more such nodes are still in that state from the context exposure on trial N , the less the context is able to excite its nodes to A1 on trial N+1 and, hence, the smaller will be the resulting inhibitory learning; the less such nodes

are in A2, the more the context excites its nodes to A1 and, hence, the greater will be the resulting inhibitory learning.

The present experiment studied the effects of alternating extinction trials in Contexts A and B on long-term learning. The rationale for this manipulation is that there was a negative correlation between these contexts during the initial discrimination training. Each of these contexts consists of unique and common elements (AX and BX) such that exposure to Context B would excite a representation of A to an A2 state of activity. This establishes an inhibitory association from B to A which means that subsequent exposures to B will act to suppress the A2 activity of A. Thus, alternations of Contexts A and B should reduce the proportion of A nodes in an A2 state of activity because exposure to B will exert an inhibitory influence on the nodes that have decayed into A2 from exposure to Context A. Rats were trained in the discrimination between a shocked (A) and a non-shocked (B) context. On the day following the end of this training, all rats received six extinction trials in Context A. These trials were preceded by six extinction trials in either the similar, B context (Group Blocked Similar) or the different context, the plastic buckets (Group Blocked Different). The six extinction trials in Context A alternated with six extinction trials in either the similar (B) context (Group Alternation Similar) or the different (plastic buckets) context (Group Alternation Different). Each trial was 2 min in duration and the interval between trials was 4 min. This interval was spent in the home cages. Finally, rats were tested. This consisted in a daily 2-min extinction trial in Context A across eight days.

Method

Subjects and apparatus. The subjects were 32 experimentally naïve rats of the same sex, weight, from the same source and maintained under the same conditions as in previous experiments. During the course of the experiment one rat was excluded due to equipment failure. Three contexts were used. The contexts used for discrimination training were the same as in previous experiments. The third context was a set of four white plastic buckets (26-cm diameter x 26-cm height). The floor of the buckets contained cardboard bedding that was replaced after the removal of each rat. The behavior of the rats in this context was not recorded.

Procedure. The consecutive phases of Experiment 8 are shown in Table 9. Discrimination training between the shocked (A) and the non-shocked (B) contexts was that described previously. Rats were then assigned to groups matched on the basis of the levels of freezing in Context A. On the day following the final sessions of discrimination training, all rats received six extinction trials in Context A. These extinction trials in A alternated with exposures to Context B for rats in Group Alternation Similar while the extinction trials in A were alternated with exposures to plastic buckets for rats in Group Alternation Different. Alternations were organized so that the first and last extinction trials were in A. Two additional groups received six exposures to Context A. These exposures were preceded by five exposures to Context B for rats in Group Blocked Similar or five exposures to plastic buckets for rats in Group Blocked Different. Each exposure to any of the three contexts (A, B or the plastic buckets) was 2 min in duration. The interval between exposures was 4 min and the rats spent this interval in the home cages. Testing began on the following day. It consisted of a daily 2-min extinction trial in Context A across eight days.

Results

Discrimination training was successful: on the final day, rats showed substantial freezing in Context A [64.79 (3.8)] and little freezing in Context B [8.96 (3.4)]. The data of major interest are the levels of freezing in Context A across massed extinction training and spaced testing. These are shown in the left and right panels, respectively, of Figure 9. The statistical analysis of the levels of freezing across massed extinction training revealed a statistically significant linear trend, F (1, 27) = 81.972, F_{critical} = 4.21. This indicates that the levels of freezing declined across massed training. However, there were no statistically significant interactions between linear trend and group differences, $Fs < 1$, nor any statistically significant differences among the groups, largest F (1, 27) = 2.15. Rats in Groups Blocked Similar and Alternation Similar froze very little (< 10%) in Context B during extinction training. The levels of freezing did not differ between the groups, F (1, 13) = 2.24, F_{critical} = 4.67, nor was there a linear trend or interaction, $Fs < 1$ (data not shown).

Inspection of the levels of freezing across spaced testing (right panel) suggests that rats subjected to alternating extinction trials in Contexts A and B (Group Alternation Similar) maintained lower levels of freezing than did rats in the remaining groups. In these latter groups, the freezing that had been lost across massed training recovered. Freezing declined across spaced tests in all groups and this decline appeared to be less rapid among rats in Group Alternation Similar. The statistical analysis confirmed that rats in Group Alternation Similar froze significantly less across spaced tests than rats in the remaining groups, F (1, 27) = 8.66, F_{critical} = 4.21. There were no statistically significant differences between the levels of freezing by rats in Group Alternation Different and those in Groups

Context conditioning	Extinction	Test
A+/B-, P-	[4-min] B- B- B- B- B- A- A- A- A- A- A-	[24-hr] A-
	[4-min] P- P- P- P- P- A- A- A- A- A- A-	[24-hr] A-
	[4-min] A- B- A- B- A- B- A- B- A- B- A-	[24-hr] A-
	[4-min] A- P- A- P- A- P- A- P- A- P- A-	[24-hr] A-

Table 9. This table represents the consecutive phases in Experiments 8. A, B and P represent Context A, Context B and Plastic Buckets, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of eight exposures to Context A.

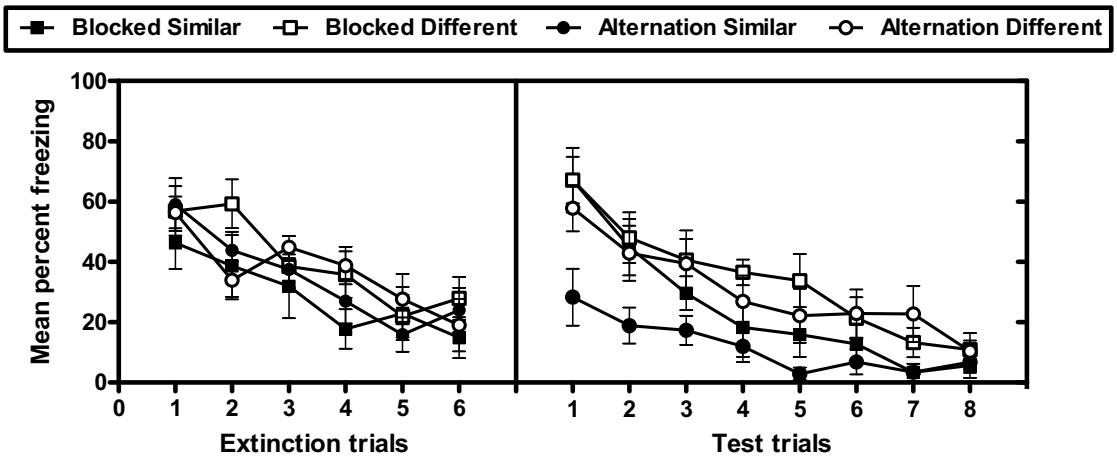

Figure 9. Mean (± SEM) percentage of time spent freezing across massed extinction trials in Context A and test trials. The rats spent the interval between extinction trials and test trials in their home cages. All groups had received a 4-min interval between extinction trials and a 24-hr interval between test trials. Rats in Groups Blocked Similar and Blocked Different received five 2-min exposures to Context B and plastic buckets, respectively, followed by six 2-min exposures to Context A. Rats in Groups Alternation Similar and Alternation Different received six exposures to Context A alternated with five exposures to Context B and plastic buckets, respectively.

Blocked, $F < 1$, nor between those in Groups Blocked Similar and Blocked Different, F (1, 27) = 1.74. The statistically significant linear trend, F (1, 27) = 158.82, shows that freezing declined across test trials. The interaction between linear trend and the contrast testing for the differences between Group Alternation Similar and the remaining groups was statistically significant, F (1,37) = 10.18. From inspection of the figure, this confirms that freezing declined more among rats in Groups Alternation Different and Blocked than among those in Group Alternation Similar. There were no statistically significant interactions between linear trend and the contrasts testing for other between-group differences, Fs < 1.

Discussion

These results show that alternations of Contexts A and B across a series of massed extinction trials produce more learning than blocked trials in Context B followed by blocked trials in A. Thus, the increased learning was not due to recent exposures to Context B. Nor was it due to alternations per se or to the longer interval between extinction trials in Context A. Rats subjected to alternations of Context A and plastic buckets learned as little as those that received a block of trials in A preceded by a block of trials in either Context B or the plastic buckets. Thus, alternation of Context A with Context B was critical for the restoration of associability otherwise lost across a series of massed extinction trials.

CHAPTER 3

General discussion

The series of experiments in this thesis studied the effects of the duration of the interval between extinction trials on the loss of context conditioned freezing responses in rats. This interval was spent in the home cages. In each experiment, rats were shocked in one context (A) but not in another (B) until freezing responses were confined to Context A. Then they were repeatedly exposed to Context A in the absence of shock until freezing responses were lost. Experiments 1a and 1b trained rats with short (4-min) or longer (24-hr) intervals between these extinction trials and then tested rats from each condition at short or longer intervals. Those trained with short intervals (Groups Massed) lost freezing responses more rapidly than those trained with longer ones (Groups Spaced). Rats trained with either the short or the longer intervals maintained response loss when tested with short intervals (Groups Massed-Massed and Spaced-Massed, respectively), as did those trained and tested with longer intervals (Group Spaced-Spaced). In contrast, rats trained with short intervals and tested with longer ones (Group Massed-Spaced) exhibited a complete recovery of lost responses. They then lost freezing responses across spaced testing at a similar rate as control rats not subjected to prior extinction training. Thus, these experiments demonstrate that massed extinction trials produce better response loss but worse learning than spaced trials.

These experiments differ in several respects from those used previously to study the effect of the interval between extinction trials on response loss. These

include the intervals selected for comparison (4 min versus 24 hr as compared to seconds versus minutes), the nature of the CS extinguished (context as compared to a discrete CS), and the location of the subjects across the interval (home cages as compared to the context where conditioning occurred). Nevertheless, the conditions used here produced effects that have some generality as the better response loss at short intervals replicate that reported in both appetitive (e.g., Rescorla & Durlach, 1987) and aversive (Cain et al., 2003) procedures. The worse learning produced by the short intervals in the present experiments also replicates effects reported previously when subjects spent the interval outside the training context (Morris et al., 2005; Westbrook et al., 1985). However, this effect on learning is the opposite of that reported when subjects spent the interval in the conditioning context (e.g., Rescorla & Durlach, 1987). In this case, short intervals produced better learning than longer ones. Thus, the source of the effect on response loss differs depending on the location of the subjects across the interval. As noted previously, the time spent in the context between extinction trials produces a greater prediction error when the CS is presented after a short than a longer interval. Thus, in this case, better response loss is due to the effect of the short interval on learning. However, when there is no such confound, as in the present case, better response loss is due to the effect of the short interval on performance.

SOP and AESOP predict the present results. According to these models, a loss in associability underlies the contrasting effects of massed extinction trials on learning and performance. This loss occurred because the duration of the interval was such that context elements on trial N+1 were still in A2 from trial N. This overlap reduces the number of elements excited to A1 by the context CS on trial

N+1. This reduction in the number of elements in A1 means that fewer are available to excite their associated US elements to the A2 state of activity that is critical for both conditioned responding and inhibitory learning. Thus, massed extinction training produced rats that were not frightened in the dangerous context. They were not frightened because they did not process the context in such a way as to enable it to retrieve the feared outcome (the shock US). However, these rats had not learned to inhibit their fear of this context and, hence, had to learn that inhibition across subsequent testing with longer intervals. Spaced extinction training also produced rats that were not frightened in the dangerous context. But these rats were not frightened precisely because they had processed that context in such a way as to have learned to inhibit their fear.

SOP and AESOP explain low levels of freezing during massed extinction training in terms an inability of the context CS representation to enter an A1 state of activity. During massed extinction and test the US representation is not elicited to an active state, thus conditioned responding is not produced. Conditioned responding is determined by the time it takes for the context CS representation to decay to an inactive state, and this is determined by the length of the interval between context exposures. Therefore, a longer interval between the final massed extinction trial and test should produce more responding than a short interval. Experiment 2 examined this prediction by varying the interval between the final extinction trial and test. The results demonstrated that shorter intervals (4 min, 1 hr and 3 hr) between the final extinction trial and test produced low levels of freezing, whereas longer intervals (6 hr and 24 hr) produced high levels of responding. These results suggest that the

context CS representation decays to inactivity between 3 and 6 hr after a context exposure.

Experiments 3 and 4 examined the effect of the duration of extinction trials during massed training on response loss. According to SOP and AESOP a longer extinction trial permits greater processing of the array of cues that constitute the context thus affording them greater opportunity to enter into inhibitory associations than if the duration of the extinction trial is short. SOP and AESOP therefore predict that longer extinction trials will result in greater learning than short extinction trials. The experiments tested this prediction by varying the length of the massed extinction trials so they were either long (10 min) or short (2 or 4 min). The results confirmed the predictions made by SOP and AESOP by demonstrating that long extinction trials facilitate long-term response loss compared to shorter extinction trials. Thus, these results show that long extinction trials produce better learning than shorter ones.

Subsequent experiments provided evidence that the first in the series of massed extinction trials produced the loss in associability. In Experiment 5, the levels of freezing on the initial trial each day declined at the same rate in rats just receiving that trial as in those for whom that trial was followed by two, five, or ten additional trials each day. This similarity in the rate at which freezing responses were lost on the initial trial each day suggests that learning occurred on the first in the series of daily massed trials but not on any of the subsequent trials. Experiment 6 provided evidence for this suggestion. Rats received the standard discrimination training between A-shock and B-no shock and were additionally trained with an excitatory CS in a third context (C). Then that CS was presented at different

locations across the series of massed extinction trials in the target context (A), specifically, on the 1st, 6th, or 11th trial. If learning occurs on the first of the massed trials but not on the subsequent trials, then the first extinction trial will contribute a V value to the common error term (0 - ∑V CS + V Context A 1st trial) but subsequent trials will not make such a contribution (effectively, α Context A = 0 on the 6th and 11th trial). Or, in the language of SOP and AESOP, the proportion of US elements excited to A2 concomitantly with CS and context elements in A1 will be greater on the 1st trial than on later ones. Therefore, more learning will accrue to both the CS and Context A when the compound is extinguished on the 1st trial than when extinguished on later trials. Exactly these results were obtained. Rats exposed to the excitatory CS on the 1st of the massed extinction trials rapidly lost freezing responses across subsequent spaced testing in Context A and exhibited little or no freezing when tested with the CS in a fourth context (D). In contrast, rats exposed to the CS on the 6th or 11th massed extinction trial did not differ from control rats in rate of response loss across subsequent spaced testing or in their (substantial) levels of freezing when tested with the CS.

These results show that learning occurs on the first of the massed trials and that little or no such learning occurs on the later trials. However, they do not show that the absence of learning on the later trials was due to the loss in associability caused by the initial trial. Experiment 7 demonstrated such an effect. Rats exposed to a short (2 min) duration extinction trial followed a few minutes later by a longer (20 min) duration trial lost fear responses across subsequent spaced testing at a similar rate as rats that only received the short trial. Moreover, these rats lost fear responses more slowly across subsequent testing than those that had just received the longer

duration trial. These results show that the duration of the initial extinction trial determined the amount of learning and suggest that the duration of the subsequent trial provides little or no additional contribution to that learning. Multiple mechanisms are likely to underlie the effect of the duration of the initial trial on learning. For instance, a longer duration affords more opportunities for processing the array of cues in such a way as to enable them to enter into inhibitory associations than does a short duration (see Experiments 3 and 4). However, preceding the longer trial with a short one appears to alter this processing, presumably by shifting it from the A1 state critical for inhibitory learning to the A2 state that impairs such learning. It is as if the associability of the context CS declines at a fixed rate independently of the location of the subjects. But why this should be the case is unclear.

According to SOP and AESOP, the stimulus processing system is organised such that elements excited to A1 decay into a refractory state (A2) that precludes their excitation to the A1 state. The dynamics of this system mediate the loss in associability produced by the initial context extinction trial: elements excited to A1 on that trial decay into A2 and cannot be re-excited on the subsequent trial. However, if these elements are themselves precluded from being in this refractory (A2) state, then the normal decay function from A1 to A2 will be altered and elements will persist in the A1 state. Experiment 8 provided evidence that alternations of Context A and Context B served just such a function. It demonstrated that rats subjected to these alternations lost freezing responses across subsequent spaced testing more rapidly than rats exposed to Context B prior to Context A exposures or rats exposed to alternations between Context A and a neutral context (plastic buckets).

The rationale underlying this manipulation was the initial discrimination training between A and B would have resulted in the formation of inhibitory associations between their distinctive features via their common associates. Evidence for such associations comes from studies in which rats received alternating exposures to solutions composed of saline-lemon and sucrose-lemon. Then they were exposed to a saline-peppermint solution and finally tested for intake of a sucrose-peppermint solution under a sodium appetite. The logic of this test is that the peppermint excites its valuable associate, saline, from a state of inactivity (I) to A2 (thereby provoking drinking), but the inhibitory association between sucrose and saline formed across pre-exposure prevents this activation (thereby reducing drinking). Exactly this result was obtained (e.g., Dwyer & Mackintosh, 2002; Dwyer, Bennett, & Mackintosh, 2001). However, the suggestion here is that the inhibitory association between B and A across discrimination training resulted in the former acting to suppress the latter's elements in A2. Effectively, exposure to Context B displaced elements of Context A from the periphery of working memory (or it inhibited the A2 activity of these elements). This means that exposure to Context A was more "surprising" following exposure Context B. This is analogous to the increase in the surprise value of a US presented after a compound composed of a novel cue and an inhibitor (Rescorla, 2004). According to the present suggestion, the increased conditioning to the novel cue is a consequence of the effect of the inhibitor on the decay of US elements from A1 to A2. It prevents their A2 activity and thus maintains the US in the A1 state critical for learning.

Implications for theories of extinction

Contemporary theories of Pavlovian conditioning assume that associative formation is regulated by prediction error. Many of these theories (e.g., Rescorla &Wagner, 1972; Wagner & Rescorla, 1972; Wagner, 1981; Wagner & Brandon, 1989) also assume that all of the cues present on a trial, including those provided by the background or context, are used to compute this error. The design used by Rescorla and Durlach (1987) confounded the duration of the interval between CS alone presentations with the time spent in the context between these presentations. This confound is therefore theoretically significant as the time spent in the context between CS alone presentations explains the effect of the interval between these presentations on learning. The experiments in this thesis broke this confound by returning subjects to their home cages between CS presentations and produced results opposite to those reported by Rescorla and Durlach (1987). Elimination of the confound means the results cannot be explained in terms of changes in prediction error resulting from differential amounts of time spent in the training context. Instead, the results are explained in terms of the learning and memorial processes described by SOP and AESOP that suggest that recovered responding during spaced testing is a result of very little learning during massed extinction training.

The memory-based explanation of extinction developed by Bouton (1993; 2002) explains a number of results produced in this thesis. He proposes that conditioning and extinction are represented as distinct memories, a CS-US and a CS-No US memory, respectively, and that performance to an extinguished CS is determined by which of these memories is retrieved (see Bouton, 2004, for a review). These memories are formed against backgrounds that include not just the physical context where conditioning and extinction occurred, but also the internal

state of the subject, as well as the temporal context (change in context resulting from the passage of time). In determining which memory is retrieved, Bouton (1993) suggests that the background present at test acts as an occasion setter (Holland, 1985) or a facilitator (Rescorla, 1985), favouring the retrieval of the association that was formed against a similar contextual background.

Morris et al (2005, Experiment 7) reported results consistent with this explanation. Rats were trained to discriminate between two contexts, in one of which a shock was presented (A) but not in the other (B). In a third context the rats received a CS-shock pairing followed by a CS extinction trial. Finally, rats were re-exposed to either Context A or Context B after which they were tested for freezing to the extinguished CS in a fourth context. The interval (spent in the home cages) between re-exposure to the context and test was either short (2 min) or long (24 hr). Conditioned performance was restored to the extinguished CS at the short but not the long interval and depended on re-exposure being to the dangerous context (A) but not the safe context (B). The restoration of conditioned performance also depended on rats being tested with an extinguished CS, rather than a non-conditioned CS or a conditioned but non-extinguished CS. Morris et al (2005) suggested that recent exposure to a dangerous context restored the fearful background under which the original CS-US association had formed, thus retrieval of the conditioning memory was favoured over the extinction memory resulting in the reinstatement of fear to an extinguished CS.

As previously mentioned, the contextual background may include the "temporal context", where the passage of time may cause a gradually changing context (e.g. Bouton, 1993). The concept of a temporal context can be extended to

include the effects of the intertrial interval. Bouton (for review, see Bouton, Westbrook, Corcoran, & Maren, 2006) has suggested that the interval between successive presentations of the CS may be encoded as part of the extinction context. According to this suggestion performance should be specific to the interval with which the subject is trained. Bouton and colleges have demonstrated this role of the intertrial interval in extinction. Specifically, Bouton and Garcia-Gutierrez (2006) found that rats that had been trained with extinction trials separated by an interval of 4 min displayed a recovery in responding when tested after a retention interval of 16 min. In contrast, rats that had received extinction trials that were separated by an interval of 16 min displayed effective long term response loss when tested after a retention interval of 16 min (see also Moody, Sunsay & Bouton, 2006). Interestingly, further research found that extinction trials separated by an interval of 16 min and tested after a retention interval of 4 min did not result in a similar recovery in conditioned responding despite the disparity between intervals. Bouton and Garcia-Gutierrez (2006) suggest that these results are evidence that the intervals between extinction trials are encoded as part of the extinction context but there are constraints regarding the conditions under which they are used.

Some of the results produced in this thesis are at least in part consistent with this explanation. For example, Experiments 1a and 1b showed that when there was a disparity between the interval between extinction trials and test trials there was a recovery in the freezing response that was not present when the interval between extinction and test trials did not differ. However, the recovery in conditioned responding occurred when the intervals between extinction trials were shorter than the intervals between test trials and not when the intervals between extinction trials

were longer than the interval between test trials. This asymmetry reflects the results produced by Bouton and Garcia- Gutierrez (2006) where a recovery in conditioned responding was dependent on the test trial intervals being longer than the extinction trial intervals. Thus, the interval between trials may be encoded as part of the extinction context and act to control performance to an extinguished stimulus, but only when the intervals between extinction trials are shorter compared to the test retention interval.

Similarly, Experiment 2 showed the recovery in conditioned responding increased as the mismatch between the interval between extinction trials (4 min) and the retention interval increased. This suggests that as the interval deviated further from the interval encoded as part of the extinction context the conditioning memory was favoured over the extinction memory. Despite Bouton's model accounting for a number of results produced by experiments in this thesis, it does not explain why the effect of massed extinction training is reduced by introducing a white noise-CS (Experiment 6) or alternating Context A with Context B during extinction training (Experiment 8). One possible explanation is that the introduction of a second stimulus during extinction training (i.e., a white noise-CS and a second context in Experiments 5 and 8, respectively) disrupts the role of trial spacing in determining which memory (conditioning or extinction) is retrieved. Furthermore, if the interval is encoded as part of the extinction context it could be argued that the results of Experiment 5 should have demonstrated that a larger number of extinction trials, thus greater experience with the interval producing context exposures in the absence of shock, should produce a more rapid loss in conditioned responding when re-exposed to short intervals on the subsequent day. However, in Experiment 5, it is

possible that response loss is facilitated with repeated exposures to the interval, but to the same extent irrespective of the number of massed extinction trials administered.

The effects of alternating presentations of Context A and Context B were explained by way of a modification of Wagner's models. But another explanation of these effects comes from Hall's negative habituation process. Hall and colleagues have shown that alternating pre-exposures to a pair of similar stimuli enhances their discriminability (see Hall, 2003 for a review). For example, intermixed exposure to AX and BX (where A and B represent the unique features of the two stimuli and X represents the shared features of the stimuli, i.e., the features they have in common) limits generalisation between AX and BX compared to a procedure in which AX and BX are pre-exposed equally often but in separate blocks of trials (e.g., Mondragòn & Hall, 2003; Symonds & Hall, 1995; 1997). In these experiments learning occurs as a result of exposure to a single event and is therefore not readily accounted for by traditional theories of associative learning. Recall that associative theories work under the assumption that the co-occurrence of activation of representations allows the formation of an association between them, thus producing new learning.

Instead, Hall (2003) suggests that exposure to compounds with unique and common features (AX and BX) results in the formation of excitatory associations, between A and X and between B and X (see McLaren, Kaye & Mackintosh, 1989). Direct activation of a compound representation through repeated exposure results in habituation, or a decrease in associability, to the compound (e.g., latent inhibition). In contrast, the consequence of presenting AX and BX in alternation is that the representation of A will be activated on the BX trials via its association with X, and

the representation of B will be activated on AX trials. Therefore, alternating stimuli ensures that the unique features of each stimulus will be associatively activated. Hall and colleges have demonstrated this associative activation of the stimulus representation restores the effectiveness, or associability, of the unique features of the stimulus (see Blair & Hall, 2003; Blair, Wilkinson, & Hall, 2004; Hall, Blair, & Artigas, 2006; Hall, Prados, & Sansa, 2005; but see Dwyer & Honey, 2007).

It is therefore conceivable that the same mechanisms are responsible for the results produced in Experiment 8. Specifically, exposure to alternations between Context A and a similar context (Context B) during extinction training attenuated habituation to the unique elements acting to maintain their associability thus allowing extinction learning. In contrast, Context A trials that were presented in succession (massed extinction training) resulted in habituation that prevented further associative learning (i.e., the formation of inhibitory associations). Therefore, it is possible that this process mediated the facilitated responding produced by alternating A and B during extinction training in Experiment 8. Equally, however, the mechanism proposed here could mediate the effects reported by Hall and colleagues. That is, it is possible that alternations of similar cues in the studies by Hall and colleagues produced the inhibitory associations among the distinctive features (A and B) that maintain their representations in the A1 state critical for associability.

An alternative account of the rapid response loss but impaired learning produced by massed extinction trials is that extinction learning occurred, yet the massing of extinction trials resulted in reconsolidation of the original fear memory, rather than consolidation of the extinction memory. Extensive research conducted by McGaugh and colleagues has shown the importance of the adrenergic system in the

modulation of memories for emotional events (e.g. Cahill & McGaugh, 1998; for review see McGaugh & Cahill, 1997). Specifically, the consolidation of a contextual fear memory is sensitive to treatments that facilitate or impair noradrenergic transmission in the amygdala, whereby blockade and facilitation of noradrenergic transmission impair and facilitate performance respectively (e.g. LaLumiere, Buen & McGaugh, 2003; Roozendaal, de Quervain, Schelling & McGaugh, 2004).

A recent study investigating the role of the adrenergic system in the reinstatement of extinguished fear was conducted by Morris, Westbrook and Killcross (2006). Rats received a noise-shock pairing which was then partially extinguished. Rats were then shocked in a distinctive context (A). Prior to a nonreinforced re-exposure to the noise, subjects were given a 30-s exposure to Context A. When tested in a novel context the following day it was demonstrated that exposure to Context A reinstated extinguished fear to the noise CS. The reinstatement of an extinguished fear was dependent on exposure to the context in which shock occurred and restricted to an extinguished CS. This reinstatement effect was blocked by the β-adrenergic antagonist propranolol, and mimicked by the administration of a β-adrenergic agonist, epinephrine. Thus, the authors concluded that exposure to Context A aroused the adrenergic system that activated and therefore reconsolidated of the conditioning rather than the extinction memory.

It is possible that a similar mechanism resulted in poor extinction learning produced by massed extinction. According to this account, the initial extinction trial would have retrieved the memory of fear conditioning and simultaneously activated the adrenergic system. As a result arousal of the adrenergic system would have facilitated the reconsolidation of the fear memory rather than the extinction memory.

Furthermore, the short interval between extinction trials would have resulted in the summation of adrenergic transmission across extinction trials, thus further facilitating the reconsolidation of the conditioned memory. Therefore, when tested 4 min after massed training performance reflected the extinction memory that resided in working memory. However, when tested 24 hrs after massed training the extinction memory would have decayed from working memory, thus performance reflected the fear memory that had been strengthened during extinction.

As the adrenergic system has been implicated in mediating changes in cardiovascular responding (Carrive, 2002), if the above-mentioned explanation is correct, cardiovascular responding should be higher in massed training compared to spaced training. Further, cardiovascular responding should increase across massed extinction training as adrenergic transmission produced from each massed exposure summates. Experiment 9 (see Appendix A) examined the effect of the intertrial interval on cardiovascular responding through the implantation of radio-telemetry probes. The results showed that, in general, spaced training produced elevated cardiovascular responding compared to massed training. In addition, cardiovascular responding gradually declined across massed training. Thus, these results do not confirm the suggestion that increased adrenergic transmission during massed extinction training retrieves the fear conditioning memory and facilitates its reconsolidation. In contrast, they suggest that the impaired processing of the context as described by SOP and AESOP result in reductions in cardiovascular responding via the same mechanism the freezing response is reduced.

The experiments in this thesis provide evidence that a loss in fear responding is not necessarily indicative of learned inhibition. Early studies investigating the

effect of the interval between extinction trials found that short intervals produce more rapid loss than longer ones (e.g., Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980; Pavlov, 1927, pp. 52 - 53; Reynolds, 1945). However, subjects in these studies were not tested after a common interval and therefore differed in terms of the interval between extinction trials as well as the interval used to assess its effects. Therefore, the differences in response loss produced in these experiments may not have reflected differences in learning but may have been due to the effects of the interval on the expression of learning.

Summary and suggestions for future research

The experiments within this thesis were conducted in response to the observation that investigations of the intertrial interval in extinction produced opposing results depending on whether the interval between extinction trials was confounded with the amount of time spent in the extinction context. Experiments that confounded these factors demonstrated that massed extinction trials produced more rapid response loss and greater learning than did spaced extinction trials (Cain et al., 2003; Rescorla & Durlach, 1987). In contrast, experiments that broke this confound by removing subjects from the extinction context during the interval demonstrated that long intervals between extinction trials produce better learning than short intervals (Morris et al., 2005; Westbrook et al., 1985). However, these experiments administered two extinction trials resulting in substantial amounts of responding on test. The current experiments therefore investigated the role of the interval between extinction trials when rats spent the interval outside of the context

during the interval when extinction training produced negligible amounts of conditioned responding.

The present experiments have demonstrated that short intervals between extinction trials produce rapid loss of conditioned responses but impairment in learning compared to long intervals. Furthermore, a number of experiments have documented a role for self-generated priming in mediating the effects of massed extinction trials. Presumably, the conditions for such self-generated priming are present when subjects spend the interval between extinction trials in the context where conditioning occurred. To be sure, such subjects also rapidly lose conditioned responses but, in contrast to the present case, such subjects also acquired the new information about the CS that underlies long-term response loss. Thus, any effects exerted by self-generated priming appear to be more than outweighed by the effects produced by prediction error in these cases. It would be of interest to know the precise conditions under which prediction error exerts its effect over the self-generated priming mechanism that impairs learning, as this information would indicate conditions that facilitate extinction learning when the interval between trials is shorter.

Another possibility is that the differing results produced by investigations of the intertrial interval are not because of different mechanisms in terms of self-generated priming and prediction error, but are related to the stimuli being extinguished. For example, it is possible that the rate of decay from one state to another, as described by SOP and AESOP, differ depending on aspects of the stimulus. A discrete CS, such as a noise, may decay more rapidly than a complex CS such as a context. Or alternatively, the discrete CS may decay more slowly than a

context CS. Either way, if a difference such as this exists it may account for the different results produced by various studies on the effect of the intertrial interval on extinction. This possibility is difficult to assess as the intervals selected for comparison differ greatly between studies. For example, studies using a context CS (Morris et al, 2005) selected intervals of 2 min and 24 hr, whereas studies using a noise or light CS (Rescorla & Durlach, 1987) selected intervals of 10 s and 2 min. This raises another alternative explanation: the decay rate of a discrete CS and a context CS may not differ. Instead, contrasting results between experiments may be produced by the different intervals selected for comparison. Therefore, to investigate this possible explanation the differential effects of the intertrial interval on the extinction of a discrete CS, such as a noise or light, could be compared to the extinction of a context when the same intervals selected for comparison and when subjects are removed from the extinction context during the interval.

Since Pavlov (1927), a variety of test procedures have demonstrated that conditioned responding can be restored after extinction training, generating the view that extinction training does not completely remove the learning produced during conditioning. On the surface, it appears that the recovery of lost responding after massed extinction training contributes to this view. However, the results of this thesis have demonstrated that the initial massed extinction trial prevents additional extinction learning. Thus, the results of this thesis, rather than prescribe to the and therefore are unable to contribute to the view that the original learning is preserved after extinction training, indicate that extinction learning does not occur.

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APPENDIX A

This appendix presents Experiment 9.

Experiment 9

It has been well documented that repeated pairings of a CS with an aversive US results in the CS becoming capable of eliciting behavioural fear responses, such as freezing (Blanchard & Blanchard, 1969; Fanselow, 1980). The ability of the CS to elicit autonomic (i.e., cardiovascular) responses, such as alterations in respiration, heart rate and arterial blood pressure has also been documented (Iwata & Ledoux, 1988). A recent study conducted by Carrive (2000) indexed behavioural (freezing, rearing, grooming and activity) and autonomic responses [heart rate (HR) and mean arterial blood pressure (MAP)] during pre-exposure and re-exposure to a conditioned context. The main findings were that re-exposure to the conditioned context produced a prolonged freezing response, a rise in MAP, and a delayed rise in HR compared to pre-exposure to the conditioned context. Interestingly, changes in HR were not in the same direction as changes in MAP during re-exposure to the conditioned context. Specifically, when freezing and MAP were at their peak HR remained low and only began to increase when freezing and MAP began to decline. Thus, increased levels of activity are associated with an accelerative effect on HR.

Previous experiments in this thesis used freezing to index reductions in fear to a conditioned context during extinction training that varied the interval between extinction trials. The use of freezing as a measure of long-term reduction of fear to the context proved somewhat unreliable as reductions in freezing across extinction trials that were separated by a short interval were not indicative of learning. The aim of the current study was to provide a more comprehensive analysis of fear reactions during massed and spaced extinction training. Specifically, the study aimed to describe the simultaneous behavioural and autonomic changes that occur in response

to extinction training with long and short intervals. To allow for the simultaneous recording of autonomic and behavioural changes and to avoid interference between the recording of autonomic parameters and the freezing response, blood pressure was recorded in freely moving rats with radio-telemetric probes.

The experiment was a 2 (massed versus spaced extinction) x 2 (massed versus spaced test) factorial design. Rats were trained to discriminate between two contexts. In one of these (A) shock occurred and in the other (B) shock was not presented. Then they received extinction training. This consisted in a daily non-shocked exposure to Context A across ten days for rats in Groups Spaced, and a daily non-shocked exposure to Context B across these days for rats in Groups Massed. Rats in Groups Spaced received massed exposures to Context B prior to their final extinction trial with Context A on day 11, whereas rats in Groups Massed received 11 massed extinction trials with Context A on that day. All rats received additional, extinction trials with Context A across subsequent testing. Half of the rats in each condition received massed extinction testing, while the remainder received spaced extinction testing. Freezing behaviour was measured and heart rate (HR) and mean arterial blood pressure (MAP) were simultaneously recorded via radio-telemetry.

Method

Subjects. Subjects were 26 experimentally naïve male Wistar rats (Rattus Norvegicus) obtained from Gore Hill Research Laboratories, Sydney. Rats weighed between 350 – 450 grams at the onset of the experiment. Prior to the start of the experiment rats were housed in groups of eight in plastic boxes (67cm length x 40cm width x 22cm height). After the surgical procedure rats were housed individually in

boxes (40-cm length x 26-cm width x 16-cm height). Food and water were continuously available. The boxes were kept in an air-conditioned colony room maintained on a 12:12-hour light-dark cycle. Each rat was handled for approximately 3-min per day for fours days prior to the start of the experiment. The experimental procedures followed the ethical guidelines established by the American Psychological Association and were approved by the Animal Care and Ethics Committee of the University of New South Wales.

Apparatus.

Chambers. Two separate rooms with four experimental chambers were used as Contexts A and B. Each chamber was located in a separate compartment of a wooden cabinet, with the doors of the cabinet open to allow observation. A camera, mounted on the wall facing the experimental chambers, was used to monitor the animals' behaviour. The camera was connected to a video recorder and monitor located in another room in the laboratory, along with the equipment for controlling shock presentation. In one room the chambers measured 30x29x25cm (width x height x length) with aluminium side walls and ceiling, painted white, clear Perspex front and back walls, and white cardboard covering the back wall. The floor consisted of 16 steel rods, 6mm in diameter spaced 18mm apart, centre to centre. A removable tray located 5cm below the rods contained cat litter as bedding *(Farmland, Australia)*, onto which 1mL of rose oil (*Cara-Mia, Sydney*) solution was sprinkled prior to each session, providing a distinctive odour. Each chamber was cleaned using tap water upon removal of a rat. The room was lit by a fluorescent tube mounted in the ceiling (cool white fluorescent tube, 36W/W41, Thorn, Australia). Unscrambled AC 50Hz shock from a custom-built constant current

generator could be delivered to the floor of each chamber. The current was adjusted using an in-line milliampere meter. The background noise level in each of the chambers was 65 dB (A scale; Type 2235, Brüel-Kjaer Instruments, Marlborough, MA).

In the other room, the chambers measured 30x30x27cm (width x height x length), with aluminium sidewalls and ceiling, with clear Perspex front and back walls. The sidewalls and ceiling of each chamber were painted black, as were the walls of the cabinet housing the chambers. The floor consisted of stainless steel rods, 2mm in diameter, spaced 10mm apart, centre to centre. A removable tray located 5cm below the rods contained bedding, *(Fibrecycle, Mudgeeraba, Australia)*, onto which four drops of eucalyptus oil (Sheldon Drug Co., Sydney) were dropped, providing a distinctive odour. The chambers were cleaned with tap water upon removal of each rat. The room was illuminated by a red fluorescent tube located in the ceiling. Unscrambled AC 50Hz shock from a constant current generator could be delivered to the floor of each chamber in both rooms. The current was adjusted using an in-line milliampere meter.

Telemetry. Chambers or rat boxes were placed on top of receivers for concurrent measurement of heart rate (HR) and mean arterial blood pressure (MAP). HR and MAP were extracted automatically from the pulsatile blood pressure signal by use of the Dataquest A.R.T. Gold software (Data Sciences International, St. Paul, MN). HR and MAP were sampled every 30-s from 3-s time windows.

Procedure

Surgery. Rats were surgically implanted with radio-telemetry devices (Model TA11PA-C40, Data Sciences International, St Paul, MN) as described by Carrive

(2000). Briefly, a midline incision was made in the abdomen, and the descending aorta was exposed at the level of iliac bifurcation. The artery was punctured at this level, and the fluid-filled sensor catheter was inserted and fixed in place with tissue adhesive (3M, Animal Care Products, St Paul, MN). The body of the probe was immobilized by suturing to the ventral abdominal wall. The abdomen was closed with suture clips. The rats were injected subcutaneously with 5mg/kg of Carprofen to provide pain relief and 0.33 mL procaine penicillin to prevent infection and moved to individual plastic home cages in which they were housed for the duration of the experiment. The rats were allowed 10 days recovery prior to the start of the experiment.

Procedure. The experiment consisted of five distinct phases: baseline, conditioning, spaced extinction, massed extinction and test (see Table 10). Baseline recordings consisted of rats remaining undisturbed in their home cages from 8am until 5pm the day before the experiment started. During this time MAP and HR were sampled every 2-min. In Phase 2 rats were trained to discriminate between a shocked (A) and non-shocked (B) context. For half of the rats, the shocked context was the white chambers and the non-shocked context was the black chambers, while for the remaining rats, the black chambers were shocked and the white chambers not shocked. Discrimination training continued until levels of freezing differed substantially between the shocked (A) and non-shocked (B) contexts. On the first day, in the morning session, the animals were placed in Context A (counterbalanced between rooms) for 9 min, and a 0.25mA shock was delivered at 60, 180, 300 and 420 s, before being returned to their home cage 120 s after the last shock. In the afternoon session of the same day, the rats were placed in the other context (B) for 9

min, without being shocked, before being returned to their home cage. Each day from Days 2 to 8, rats were given a shocked exposure to A and a non-shocked exposure to B, irregularly alternated so that exposure to A and B occurred equally often in the morning and in the afternoon session. Shock intensity was increased by approximately 0.05 mA per day, reaching a maximum intensity of 0.8 mA by the eighth conditioning session. From Day 6, the duration of exposure to each context was reduced to 120 s, and a single shock was delivered 60 s after placement in A. From Day 7, rats received only conditioning treatment per day, either a shocked exposure to A or a non-shocked exposure to B, in order to reduce generalisation decrement during extinction training. By Day 10 there had been a total of eight shocked exposures to A and eight non-shocked exposures to B. All experimental procedures took place between 9-11 a.m. and 4-6 p.m. Rats were assigned to Spaced, Massed and No Extinction groups after conditioning in order to match levels of freezing to Context A.

Phase 2 began on Day 11. Rats in Groups Spaced-Massed and Spaced-Spaced were placed in Context A (the previously reinforced context) for 2 min before being returned to their home cages. Rats in Groups Massed-Spaced and Massed-Massed were placed in Context B (the previously non-reinforced context) for 2 min before being returned to their home cages. This continued each morning, spaced 24 hrs apart, for ten days, until freezing to A extinguished in the spaced extinction groups.

Massed extinction began on the morning of Day 21. Rats in Groups Spaced-Massed and Spaced-Spaced were placed in Context B ten times. Each exposure lasted 2 min and was separated by an interval of 4 min, with animals being returned

to their home cages during the interval. 4 min after the final Context B exposure, rats in Groups Spaced-Massed and Spaced-Spaced received their final 2 min exposure to Context A. Rats in Groups Massed-Spaced and Massed-Massed received 11 2 min exposures to Context A, each separated by an interval of 4 min that was spent in their home cages. Test consisted of 11 2 min exposures to Context A. For the Massed-Massed and the Spaced-Massed groups the interval between test exposures were 4 min and began 4 min after the final extinction exposure to A. For the Spaced-Spaced and the Massed-Spaced groups test exposures were separated by an interval of 24 hrs and began 24 hrs after the final extinction exposure to A.

Scoring and statistics. The conditioning, extinction and test sessions were videotaped, and the levels of freezing were measured with a time sampling procedure in which the rat's behaviour was scored as freezing or not freezing every 2 s. Freezing was defined as the absence of all movement, except those related to breathing (Fanselow, 1980). The percentage of all samples scored as freezing was determined for each rat. Two observers, one of whom was unaware of the rat's treatment condition, scored the videotaped record of each rat. The inter-rater reliability for this experiment, and all remaining experiments, was high, producing correlation coefficients in excess of $r = .9$ for all experiments.

Telemetry samples were averaged across each 2-min exposure. The average HR and MAP across the 9 hours when rats were at rest in their home cages served as baseline. Subsequent HR (beats per minute [bpm]) and MAP (mmHg) readings across the 2 min extinction and test exposures to Context A were expressed as differences from baseline. The data in this experiment were analyzed with a contrast

testing procedure that controlled the Decision-Wise Error rate at $\alpha = 0.05$ with the procedure described by Hays (1963).

Results

Figure 10 shows the mean percentage (±*SEM*) HR (top panel), MAP (middle panel), expressed as a change from baseline, and freezing (bottom panel) across baseline recordings, extinction training to the Context A, extinction training to Context B and test (left to right). Each data point represents the average of a 2 min recording period. From the figure it is clear that baseline HR and MAP were stable and did not differ between the groups. This was confirmed by the statistical analysis, $Fs < 2.90$. Discrimination training proceeded such that on the final sessions rats showed higher HR levels in Context B compared to Context A [Mean (*SEM*): 150.00 (7.8) and 127.43 (9.6), respectively], similar MAP levels in Context A and Context B [Mean (*SEM*): 32.56 (2.5) and 24.31 (2.1), respectively], and substantial freezing to Context A but not in Context B [Mean (*SEM*): 59.10 (4.4) and 18.33 (6.2), respectively].

Inspection of the A- extinction and B- extinction panels of the figure show that for HR and MAP Group Spaced displayed greater elevations from baseline compared Group Massed when extinction training was to both Context A and to Context B. In contrast, Group Spaced displayed a greater freezing response compared to Group Massed only during extinction to Context A. Freezing to Context B was equally low for both Massed and Spaced groups. These observations were confirmed by the statistical analysis. The analysis of HR when extinguished to A revealed that HR change from baseline was greater for Group Spaced than Group Massed, $F(1, 24) = 9.69$, $F_{critical} = 4.26$. The analysis also revealed a significant

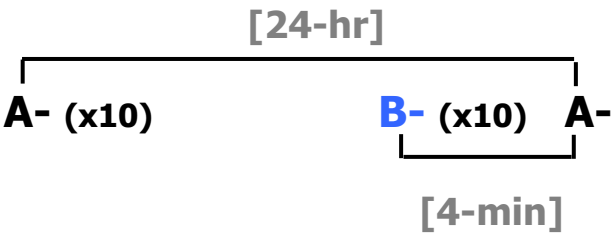
<table>
<tr><th>Context conditioning</th><th>Extinction</th><th>Test</th></tr>
<tr><td rowspan="4">A+/B-</td><td rowspan="2"> 
</td><td>[4-min] A-</td></tr>
<tr><td>[24-hr] A-</td></tr>
<tr><td rowspan="2"> 
</td><td>[4-min] A-</td></tr>
<tr><td>[24-hr] A-</td></tr>
</table>

Table 10. This table represents the consecutive phases in Experiments 9. A and B represent Context A and Context B, respectively. + and – represents footshock and the absence of footshock, respectively. / indicate alternations between exposures. Test consisted of 11 exposures to Context A.

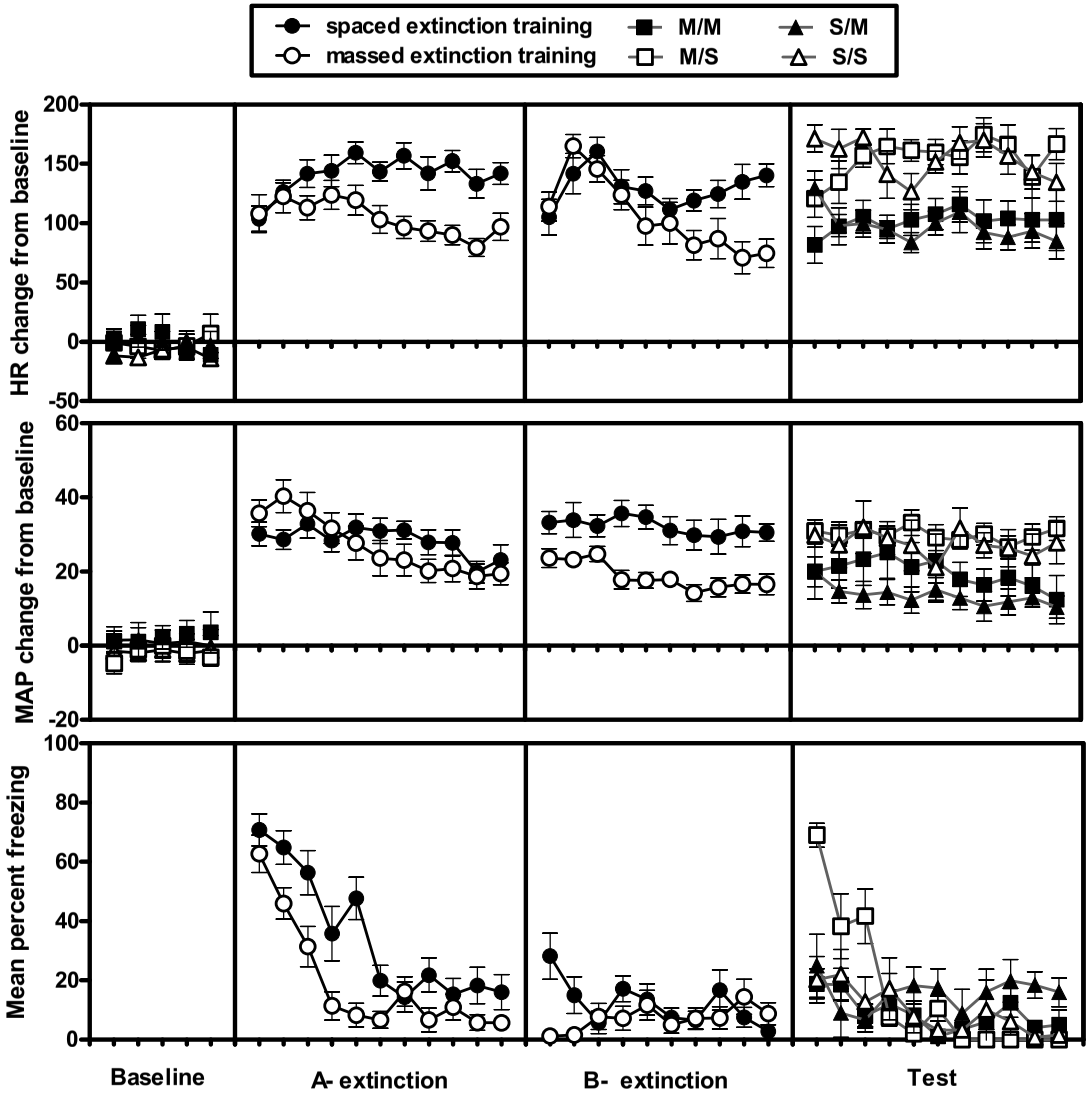


Figure 10. The mean percentage (±*SEM*) HR (top panel), MAP (middle panel), expressed as a change from baseline, and freezing (bottom panel) across baseline recordings, extinction training to the Context A, extinction training to Context B and test (left to right). Each data point represents the average of a 2-min recording period.

interaction between the contrast testing the difference between massed and spaced extinction training and the linear trend, F (1, 24) = 15.03, indicating that massed extinction produced a gradual decline in HR whereas spaced extinction produced stable levels of HR. In contrast, the analysis of HR in B- extinction found that massed and spaced extinction training produced equivalent levels of HR, $F < 1$ (this did not confirm the observation). In addition, there was an overall linear trend, F (1, 24) = 8.71, $F_{critical} = 4.26$, indicating a gradual decline in HR for both groups. There was also an interaction between the linear trend and the difference between massed and spaced extinction training, F (1, 24) = 12.40, indicating that the decline in HR was greater for the group receiving massed extinction compared to the group receiving spaced extinction. Further, massed extinction to Context A and massed extinction to Context B did not differ, F (1, 24) = 1.97, $F_{critical} = 4.26$, nor did spaced extinction to Context A differ from spaced extinction to Context B, $F < 1$.

The analyses of MAP in A- extinction revealed that massed and spaced extinction produced equivalent MAP levels, $F < 1$ (inconsistent with above mentioned observation). There was also a significant linear trend, F (1, 24) = 54.54, $F_{critical} = 4.26$, indicating that the massed and the spaced group displayed a gradual decline in MAP levels across extinction trials to Context A. An significant interaction between the contrast testing the difference in MAP levels between massed and spaced extinction and the linear trend, F (1, 24) = 13.05, indicates that massed extinction training produced a more rapid decline in MAP levels compared to spaced extinction training. The analysis of MAP in B- extinction revealed that MAP levels were significantly higher in rats that received spaced extinction training compared to rats that received massed extinction training, F (1, 24) = 14.22, $F_{critical}$ =

4.26. There was also a significant linear trend, F (1, 24) = 15.23, indicating both groups displayed a gradual decline in MAP levels over extinction trials to Context B. Finally, a separate analysis revealed that massed extinction to Context A produced higher MAP levels compared to massed extinction to Context B, F (1, 24) = 5.99, $F_{critical}$ = 4.26, whereas, spaced extinction to Context A did not differ from spaced extinction to Context B, F (1, 24) = 1.32.

The analysis of freezing in Context A revealed spaced extinction training produced higher levels of freezing compared to massed extinction training, F (1, 24) = 11.07, $F_{critical}$ = 4.26. There was also a significant linear trend, F (1, 24) = 154.92, indicating that both groups displayed a gradual decline in the freezing response across extinction trials. In contrast, the analysis of freezing to Context B revealed that massed and spaced extinction training produced equally low levels of freezing, F (1, 24) = 2.38, $F_{critical}$ = 4.26. There was not a significant linear trend, F (1, 24) = 1.07, however, there was a significant interaction between the contrast testing the difference between massed and spaced extinction and the linear trend, F (1, 24) = 10.39, indicating that spaced extinction training resulted in a more rapid decline in the freezing response compared to massed extinction training. Finally, separate analyses revealed that there was a significant difference in freezing between massed extinction training to Context A and to Context B, F (1, 24) = 24.90, $F_{critical}$ = 4.26, and between spaced extinction training to Context A and to Context B, F (1, 24) = 22.02. $F_{critical}$ = 4.26.

The data of major interest are the levels of HR, MAP and freezing during test. Inspection of the test panel shows that Groups Massed-Spaced and Spaced-Spaced displayed higher HR and MAP levels than Groups Massed-Massed and

Spaced-Massed. In contrast, Group Massed-Spaced displayed high levels of freezing compared to Groups Massed-Massed, Spaced-Massed and Spaced-Spaced, whom displayed equivalent low levels of freezing. These observations were confirmed by the statistical analysis. The analysis of HR revealed a significant main effect of test, F (1, 22) = 27.73, $F_{critical}$ = 4.30, indicating that testing under spaced conditions produced higher HR levels compared to testing under massed conditions regardless of the type of extinction training. The analysis failed to find a significant main effect of extinction, or a significant test x extinction interaction, $Fs < 1$. While the analysis failed to reveal a significant linear trend $F < 1$, a significant interaction between the extinction main effect and the linear trend was revealed, F (1, 22) = 11.21, indicating that groups that had received spaced extinction training showed a gradual decline in HR over test trials.

Similarly, the analysis of MAP revealed a significant main effect of test, F (1, 22) = 8.96, $F_{critical}$ = 4.30, indicating that spaced testing produced higher levels of MAP compared to massed testing regardless of the conditions during extinction training. The analysis failed to find a significant main effect of extinction, or a significant test x extinction interaction, $Fs < 1.18$. The analysis also revealed a significant linear trend, F (1, 22) = 13.87, indicating that all groups displayed a gradual decline in MAP levels across test trials. There was no difference between groups in the rate of this decline, $Fs < 3.45$.

The analysis of freezing failed to reveal an extinction main effect, or a test main effect, $Fs < 1$. There was, however, a significant interaction between the main effect of extinction and of test, F (1, 22) = 6.03, $F_{critical}$ = 4.30, indicating that high levels of freezing during spaced testing were dependent on rats receiving massed

extinction. There was a significant linear trend, F (1, 22) = 39.40, indicating that all groups showed a gradual decline in the freezing response. There was also a significant interaction between the extinction main effect and the linear trend, F (1, 22) = 14.14, and between the test main effect and the linear trend F (1, 22) = 23.73. This indicates that rats that had received massed extinction training displayed a more rapid decline in freezing over test trials compared to rats that had received spaced extinction, and rats that were tested under spaced conditions displayed a more rapid decline in freezing across testing compared to rats that were tested under massed conditions, respectively.

Discussion

The most important finding of this study is that test exposures that are separated by a short and a long interval produce differential effects on HR and MAP that do not directly correlated with freezing responses. Specifically, test exposures that were separated by a long interval produced high HR and MAP compared to short intervals, regardless of whether they were extinguished under short or long interval conditions. In contrast, long intervals between test exposures produced high levels of freezing compared to short intervals but only when rats were extinguished under short interval conditions. Additionally, similar changes in HR and MAP were found during massed and spaced extinction training when extinction was to Context A and to Context B despite the fact there was substantial freezing in Context A and very little freezing in Context B.

In several respects these results do not replicate Carrive's (2000) observations. For example, HR and MAP were not consistently correlated with changes in activity/freezing levels, and changes in HR and MAP were not dependent

on exposure to a conditioned context. These results can be understood if one assumes that the processing of the context during extinction training mediates the level of autonomic responding. Specifically, impaired processing of the context produces lower levels of autonomic responding compared to adequate context processing. For example, massed extinction training impairs processing of the context (see Chapter 2), thus producing rats that are not frightened in dangerous contexts and that do not elicit the freezing response. Thus, impaired processing of the context results in a reduction in autonomic and behavioural responses but also results little learning. In contrast, a long interval between context exposures maintains adequate processing of the context, thereby producing elevated HR and MAP levels. However, unlike massed extinction training, spaced extinction promotes extinction learning. So while autonomic responding remains high the conditioned freezing response gradually reduces. Interestingly, this explanation and the correlation between extinction learning and elevated autonomic responses raises the possibility that adequate processing of stimuli, and thus learning, require elevated levels of autonomic responding.

This explanation also accounts for massed extinction producing similar levels of autonomic responding irrespective of whether the presentations were to A or B (i.e., the conditioned and the non-conditioned contexts, respectively), and the equivalent levels of responding produced by spaced extinction training to A and B. Although Context B had not gained associative strength through conditioning it can still be attended to and processed in the same fashion as a conditioned context. Thus, during spaced extinction to Context B there is little freezing but autonomic responding remains elevated. In contrast, massed extinction to B produces low levels

of the conditioned freezing response and low levels of autonomic responding as the context is not being processed.

It is possible that autonomic responses are mediated by something other than the processing of the context. An explanation that may account for these similarities is that differences in handling between massed and spaced procedures may have produced the differences in autonomic responses produced by massed and spaced extinction trials. The current experiment differed from Carrive's (2000) experiment as rather than having one prolonged extinction exposure to the context there were several, short extinction trials. The handling involved in this procedure may be responsible for the differences between these experiments.

In summary, this experiment demonstrated that massed extinction trials produce lower levels of autonomic responding compared to spaced extinction trials. This result was produced regardless of whether extinction was to a conditioned context or to a non-conditioned context. These results suggest that either: 1) reduced autonomic responding during massed extinction trials is a product or impaired stimulus processing; or 2) differences in handling produced by massed and spaced extinction trials produced the observed differential effects on autonomic responding.

APPENDIX B

This appendix presents the extinction data and the test data for the test stimulus (context and CS), for each rat in each experiment. All the ANOVAs carried out in the thesis were performed using the contrast statistical package PSY2000 (K. Bird, D. Hadzi-Pavlovic, A. Isaac, © 2000). These ANOVAs are equivalent to the MANOVA approach to repeated measures data (Harris, 1994; O'Brian & Kaiser, 1985) rather than the univariate ANOVA mixed-model approach. At the level of contrast testing, the difference between these approaches is reflected in the choice of the error term for within-subjects tests: The MANOVA approach results in a separate error term for each within-subjects contrast, while the ANOVA approach provides a common error term for all within-subjects tests. Since non-sphericity implies that different within-subjects contrasts have different variances, the ANOVA approach to testing such contrasts is difficult to defend. Unless otherwise stated, contrasts were planned to be orthogonal (Hays, 1963). *Note:* When a contrast analysis is fully planned, overall tests are irrelevant.

Experiment 1a

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Massed	27	42	68	0	0	27	0	0	0	0	7
Massed	72	47	32	0	0	0	7	0	0	2	0
Massed	59	40	27	35	0	25	12	22	20	9	20
Massed	82	74	2	0	2	0	4	0	0	0	0
Massed	70	75	18	0	0	5	5	0	44	30	0
Massed	57	49	9	48	53	25	2	0	45	0	42
Massed	69	64	82	59	64	28	35	47	14	52	25
Massed	62	40	13	0	2	0	0	5	12	8	0
Massed	78	43	2	0	2	2	4	2	0	2	4
Massed	69	50	35	32	10	12	0	10	5	13	0
Massed	58	50	2	0	0	0	0	0	0	0	0
Massed	75	42	2	0	0	0	0	0	0	0	0
Massed	92	65	4	0	0	0	0	17	5	3	10
Massed	42	42	18	8	53	2	0	0	10	28	27
Massed	85	52	8	2	0	0	0	3	2	32	30
Massed	63	53	42	2	2	0	17	3	3	0	0
Massed	67	85	92	48	47	50	38	27	47	3	0
Massed	37	2	2	10	0	0	2	18	2	0	5
Massed	37	57	18	30	37	15	42	17	5	0	0
Massed	57	25	85	20	45	28	0	48	0	12	37
Massed	83	78	75	47	58	32	22	22	2	0	3
Massed	95	35	27	42	0	0	5	10	0	0	0
Massed	90	37	3	18	0	0	3	0	0	0	0
Massed	52	47	48	45	28	0	0	0	0	0	0
Massed	72	67	48	30	25	0	0	0	0	0	0
Massed	50	52	23	0	0	0	0	0	0	0	0
Spaced	57	70	45	59	19	35	39	37	0	12	3
Spaced	60	86	55	45	0	2	47	3	34	0	0
Spaced	34	75	68	23	35	49	35	12	5	0	5
Spaced	42	30	84	54	32	53	69	10	37	0	10
Spaced	50	57	57	75	27	20	0	2	0	0	0
Spaced	62	80	42	42	25	17	5	34	0	15	13
Spaced	65	50	92	68	89	55	0	0	0	0	4
Spaced	65	67	72	82	92	80	69	85	67	84	25
Spaced	95	7	0	2	54	0	2	0	0	0	42
Spaced	40	7	19	10	14	0	0	2	0	0	2
Spaced	44	20	57	27	28	22	4	19	0	0	30
Spaced	72	22	52	22	17	25	19	2	0	0	18
Spaced	47	45	50	22	27	17	19	0	0	0	0
Spaced	83	70	70	87	57	15	12	7	0	0	30
Spaced	52	28	2	3	37	2	44	17	2	0	7
Spaced	90	67	50	53	33	48	28	8	0	7	18
Spaced	93	98	100	97	95	95	95	95	85	48	0
Spaced	78	92	88	55	70	55	58	40	45	25	78
Spaced	85	75	65	38	18	13	43	7	3	2	58
Spaced	77	87	80	70	20	12	0	3	0	0	0
Spaced	37	50	42	42	38	17	12	33	2	22	5
Spaced	62	33	18	32	18	5	0	0	2	0	0
Spaced	78	77	72	82	73	62	37	55	50	32	38
Spaced	87	93	83	70	45	43	23	0	22	32	0
Spaced	52	70	63	33	37	47	35	5	0	8	3
Spaced	32	67	53	62	44	22	7	27	22	3	27

Between contrast coefficients

	Massed	Spaced
B1	1	-1

Within contrast coefficients

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	28196.252	1	28196.252	12.560
Error	112246.364	50	2244.927	
Within				
W1	162791.123	1	162791.123	361.335
B1W1	623.832	1	623.832	1.385
Error	22526.326	50	450.527	

Experiment 1a

Massed test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/M	10	14	0	0	0	0	0	0	0	0	0
M/M	0	5	17	4	0	7	24	4	7	0	0
M/M	4	10	4	12	5	0	3	25	0	0	0
M/M	8	0	0	0	2	0	0	0	0	0	0
M/M	4	45	59	52	0	5	0	0	0	0	0
M/M	57	7	34	4	0	5	7	0	0	0	0
M/M	24	27	2	2	5	0	25	0	0	0	0
M/M	0	0	5	0	0	0	0	0	0	0	0
M/M	0	0	0	0	0	0	0	0	0	0	0
M/M	0	7	2	0	0	4	0	0	0	0	0
M/M	0	0	0	0	0	0	0	0	0	0	0
M/M	0	0	0	0	0	0	0	0	0	0	0
M/M	22	5	0	18	38	22	18	19	2	19	0
S/M	0	0	7	2	0	4	0	9	0	0	0
S/M	0	0	0	0	4	9	0	10	0	0	0
S/M	4	2	0	0	7	0	5	24	10	0	0
S/M	2	4	0	0	2	10	12	20	7	2	12
S/M	2	0	0	2	4	2	4	0	12	0	0
S/M	5	0	0	4	17	23	12	0	0	0	2
S/M	62	2	0	20	0	13	0	0	0	0	0
S/M	38	27	44	5	0	0	0	0	0	0	0
S/M	0	0	0	2	0	0	0	5	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
NE/M	37	37	10	0	0	0	0	0	2	4	0
NE/M	65	45	45	12	0	0	0	0	4	37	4
NE/M	43	24	29	32	5	4	0	10	19	0	0
NE/M	48	35	9	0	0	0	0	0	0	0	0
NE/M	38	30	40	37	35	7	23	42	23	0	0
NE/M	47	15	32	30	14	4	35	8	0	0	0
NE/M	32	45	39	73	47	25	20	2	0	0	0
NE/M	47	42	43	34	25	19	10	0	0	0	0
NE/M	52	17	9	7	0	20	7	9	0	7	2
NE/M	59	42	5	3	9	0	0	0	4	0	0
NE/M	48	55	22	2	18	0	55	8	0	3	0
NE/M	63	70	73	40	37	22	40	22	40	27	22
NE/M	40	44	52	68	24	9	43	0	62	12	44
NE/M	74	80	54	77	17	59	45	42	10	8	49

Between contrast coefficients

	M/M	S/M	NE/M
B1	1	1	-2
B2	1	-1	0

Within contrast coefficients

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA massed test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	31156.404	1	31156.404	43.062
B2	193.094	1	193.094	0.267
Error	26770.320	37	723.522	
Within				
W1	14607.382	1	14607.382	73.094
B1W1	10819.532	1	10819.532	54.140
B2W1	237.404	1	237.404	1.188
Error	7394.189	37	199.843	

Spaced test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	85	5	10	0	0	0	0	0	0	0	0
M/S	77	18	0	0	0	0	0	0	0	0	0
M/S	60	15	0	7	0	0	0	2	0	0	0
M/S	88	92	80	80	0	0	0	2	0	0	0
M/S	58	80	0	3	0	0	0	3	0	0	0
M/S	92	68	2	0	0	0	0	0	0	0	45
M/S	90	80	75	67	7	0	0	0	0	7	0
M/S	70	62	55	53	25	0	0	30	0	0	0
M/S	82	47	5	0	18	0	0	0	0	0	0
M/S	95	63	5	0	8	0	0	0	0	0	0
M/S	95	92	67	57	17	0	7	0	0	0	0
M/S	75	58	73	43	50	50	28	0	0	0	0
M/S	48	5	18	2	2	0	0	0	0	0	0
S/S	3	0	0	0	0	0	0	2	2	0	0
S/S	0	0	0	0	0	0	0	0	0	0	0
S/S	0	0	0	0	0	0	2	0	0	0	0
S/S	0	0	0	0	0	0	0	0	0	0	0
S/S	28	12	8	0	0	0	0	0	0	0	0
S/S	0	2	7	0	2	0	0	0	0	0	0
S/S	0	0	0	0	0	0	0	33	0	0	0
S/S	18	17	5	8	0	0	0	20	0	0	0
S/S	0	0	0	0	0	0	0	0	0	0	0
S/S	52	32	32	35	0	20	0	0	0	0	3
S/S	0	0	0	0	0	2	0	0	0	0	0
S/S	13	2	0	0	0	2	0	0	0	0	0
S/S	12	7	2	0	0	0	0	0	0	0	0
NE/S	80	80	58	7	2	2	0	0	0	0	0
NE/S	88	80	63	23	8	8	0	0	2	0	0
NE/S	87	68	67	45	0	0	0	2	0	0	0
NE/S	70	83	57	37	0	0	0	0	0	0	0
NE/S	78	48	7	0	2	0	0	0	0	0	0
NE/S	62	80	70	53	32	13	27	0	0	0	5
NE/S	83	97	85	32	30	7	0	0	0	0	0
NE/S	82	83	28	43	0	3	0	0	0	0	0
NE/S	85	92	50	22	2	0	0	0	0	0	0
NE/S	62	75	35	27	15	15	16	0	0	0	0
NE/S	90	70	33	23	0	13	0	0	0	0	0
NE/S	75	72	77	33	0	5	0	0	0	0	0
NE/S	72	52	5	0	8	0	0	0	0	0	0
NE/S	63	38	28	2	35	0	0	0	0	0	0

Between contrast coefficients

	M/S	S/S	NE/S
B1	1	-2	1
B2	1	0	-1

Within contrast coefficients

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA spaced test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	30131.532	1	30131.532	53.955
B2	622.126	1	622.126	1.114
Error	20662.956	37	558.458	
Within				
W1	112792.899	1	112792.899	255.828
B1W1	40934.202	1	40934.202	92.844
B2W1	1380.000	1	1380.000	3.130
Error	16313.052	37	440.893	

Experiment 1b

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Massed	38	53	8	0	0	0	3	0	0	18	0
Massed	67	55	0	0	15	0	20	0	0	2	0
Massed	78	72	15	3	0	0	0	0	0	0	0
Massed	80	0	0	5	0	3	0	0	0	0	0
Massed	85	17	30	5	45	0	0	2	0	8	0
Massed	48	22	0	0	0	2	3	0	0	0	0
Massed	83	78	78	78	18	8	28	12	20	0	22
Massed	70	72	55	77	75	75	60	62	57	48	50
Massed	75	18	15	0	0	0	25	2	0	0	0
Massed	57	20	10	2	20	13	3	0	0	0	0
Massed	72	52	85	7	0	0	0	0	0	0	0
Massed	68	52	35	0	0	0	5	0	0	0	0
Massed	32	27	12	8	32	3	7	0	7	30	0
Massed	80	72	45	35	0	0	3	0	23	18	0
Massed	60	10	2	5	0	0	0	0	0	0	0
Massed	75	28	7	0	0	0	0	0	0	0	0
Spaced	70	83	63	78	67	63	70	43	27	18	7
Spaced	63	78	55	48	25	65	17	0	0	2	0
Spaced	37	30	30	32	23	7	28	7	7	42	0
Spaced	92	77	45	62	55	53	67	40	33	38	10
Spaced	90	87	87	42	43	35	0	5	2	0	12
Spaced	87	75	42	75	18	27	8	58	45	30	15
Spaced	75	70	78	75	55	68	62	60	40	45	67
Spaced	53	17	27	10	7	5	7	0	0	25	0
Spaced	100	73	72	65	47	25	2	0	0	0	0
Spaced	75	40	27	10	15	38	0	0	10	0	0
Spaced	45	57	25	55	23	0	0	0	0	0	0
Spaced	87	85	78	90	62	68	55	20	13	17	0
Spaced	62	58	58	12	32	3	5	0	0	0	0
Spaced	73	67	65	78	83	55	37	37	37	23	8
Spaced	42	15	40	22	25	28	23	12	17	8	2
Spaced	73	58	75	82	72	62	70	33	50	45	28

Between contrast coefficients

	Massed	Spaced
B1	1	-1

Within contrast coefficients

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	31977.344	1	31977.344	12.054
Error	79587.085	30	2652.903	
Within				
W1	101179.655	1	101179.655	169.805
B1W1	1497.618	1	1497.618	2.513
Error	17875.727	30	595.858	

Experiment 1b

Massed test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/M	7	25	23	23	0	8	0	10	0	0	0
M/M	0	0	0	0	0	3	0	0	0	0	0
M/M	0	0	0	0	0	0	0	17	7	0	0
M/M	0	0	0	0	0	0	0	0	13	0	0
M/M	0	0	0	0	0	0	22	0	2	2	0
M/M	0	0	0	0	0	0	0	0	27	0	0
M/M	18	17	0	18	0	0	0	0	0	0	0
M/M	73	48	0	37	0	0	13	0	0	0	0
S/M	0	7	30	0	27	27	0	0	0	13	0
S/M	0	0	10	22	8	0	0	0	17	0	0
S/M	10	8	0	2	0	0	0	30	0	0	0
S/M	2	8	0	12	0	0	0	5	20	0	0
S/M	0	40	0	28	18	0	0	0	0	2	0
S/M	0	22	45	32	0	0	5	12	0	0	0
S/M	33	28	0	5	0	12	0	0	0	0	0
S/M	0	2	7	10	0	0	0	0	0	0	0
NE/M	67	65	7	0	0	47	23	35	12	0	38
NE/M	50	5	0	23	0	0	40	12	3	13	10
NE/M	83	7	47	2	0	0	0	0	2	0	0
NE/M	67	50	40	2	0	5	42	8	42	12	0
NE/M	50	27	0	0	0	17	0	0	0	0	0
NE/M	73	65	48	30	5	27	8	5	8	0	0
NE/M	73	68	73	17	37	17	0	0	0	0	0
NE/M	71	63	43	73	35	17	10	0	0	0	0

Between contrast coefficients

	M/M	S/M	NE/M
B1	1	1	-2
B2	1	-1	0

Within contrast coefficients

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA massed test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	13461.280	1	13461.280	39.810
B2	121.114	1	121.114	0.358
Error	7100.966	21	338.141	
Within				
W1	14692.418	1	14692.418	30.661
B1W1	9453.452	1	9453.452	19.728
B2W1	10.355	1	10.355	0.022
Error	10063.084	21	479.194	

Experiment 1b

Spaced test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/M	77	83	67	7	5	0	0	0	0	7	0
M/M	40	33	42	12	2	2	0	0	0	0	2
M/M	70	80	68	45	57	48	15	0	0	15	0
M/M	90	15	0	32	0	2	2	0	0	2	0
M/M	50	45	20	0	0	0	0	0	0	0	0
M/M	70	45	3	2	0	0	0	0	0	0	2
M/M	25	0	2	0	0	0	0	0	0	0	0
M/M	92	48	18	25	8	20	8	0	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	2	0	0	0	0	0	0	5	0	0	0
S/M	5	2	0	0	0	0	0	2	0	0	0
S/M	13	15	0	0	0	0	0	2	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	0	0	0	0	0	0	0	0	0	0	0
S/M	2	0	10	0	0	0	0	0	0	0	0
S/M	22	10	10	0	3	0	0	0	0	0	0
NE/M	35	10	7	0	2	10	2	18	2	0	0
NE/M	83	28	10	0	3	3	0	2	0	0	0
NE/M	85	10	0	0	2	2	0	0	0	0	0
NE/M	20	33	0	15	0	3	0	0	0	0	0
NE/M	72	67	58	45	27	38	0	0	0	0	0
NE/M	77	75	33	42	30	30	0	0	0	0	0
NE/M	78	55	38	12	15	3	0	0	0	0	0
NE/M	53	47	33	35	10	2	0	0	0	0	0

Between contrast coefficients

	M/S	S/S	NE/S
B1	1	-2	1
B2	1	0	-1

Within contrast coefficients

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA spaced test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	12383.047	1	12383.047	19.489
B2	10.506	1	10.506	0.017
Error	13343.341	21	635.397	
Within				
W1	36877.913	1	36877.913	81.769
B1W1	14493.845	1	14493.845	32.137
B2W1	7.127	1	7.127	0.016
Error	9471.015	21	451.001	

Experiment 2

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
4m	68	22	43	2	27	5	48	58	40	75	30
4m	32	7	2	0	0	0	2	7	3	19	2
4m	83	92	2	2	0	0	0	0	0	0	22
4m	78	82	0	0	0	35	40	23	42	0	18
4m	45	72	32	15	0	32	27	40	10	5	2
4m	58	67	47	12	37	40	40	0	53	55	38
4m	87	65	38	50	43	0	3	0	37	3	3
4m	30	43	17	53	35	23	25	35	12	2	0
1h	17	22	0	0	0	0	0	0	2	2	12
1h	62	43	0	0	0	0	7	5	23	12	28
1h	58	5	0	0	3	5	15	2	0	0	0
1h	61	38	43	33	30	7	28	15	0	3	0
1h	78	42	55	25	47	68	35	62	52	33	45
1h	38	30	3	3	2	3	0	2	13	0	12
1h	52	15	5	0	0	0	2	0	0	0	0
1h	70	62	40	23	20	32	13	22	0	15	0
3h	70	62	45	3	43	0	2	25	42	22	10
3h	77	33	3	2	5	17	8	23	23	3	12
3h	68	0	0	0	0	0	2	0	0	0	15
3h	27	0	5	0	2	0	23	7	0	10	3
3h	57	55	50	30	23	17	25	60	72	53	40
3h	47	30	38	28	47	5	42	10	2	45	57
3h	32	47	15	20	0	0	0	25	2	0	0
3h	73	68	57	57	37	53	23	33	45	58	55
6h	42	43	40	38	27	8	3	3	0	5	0
6h	70	77	53	55	3	2	8	50	62	38	42
6h	43	28	30	0	0	0	2	0	0	3	0
6h	15	10	0	7	0	0	37	13	3	17	10
6h	38	20	22	0	0	0	2	8	5	0	7
6h	72	37	13	7	0	0	13	10	3	0	5
6h	57	68	63	72	58	42	23	15	63	0	3
6h	85	67	40	50	43	23	17	57	60	7	15
24h	65	35	8	53	23	3	8	0	0	10	3
24h	63	43	10	0	3	2	3	2	5	2	0
24h	28	32	5	3	12	0	0	47	17	18	32
24h	55	27	0	0	0	0	0	0	0	5	7
24h	75	53	50	12	0	0	0	0	10	0	2
24h	18	2	15	12	3	0	0	0	27	23	0
24h	78	47	72	8	0	2	0	7	67	0	22

Between contrast coefficients

	4m	1h	3h	6h	24h
B1	4	-1	-1	-1	-1
B2	0	3	-1	-1	-1
B3	0	0	2	-1	-1
B4	0	0	0	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	2384.085	1	2384.085	1.181
B2	778.858	1	778.858	0.386
B3	1714.517	1	1714.517	0.849
B4	2357.239	1	2357.239	1.167
Error	68649.062	34	2019.090	
Within				
W1	38570.866	1	38570.866	67.235
B1W1	57.875	1	57.875	0.101
B2W1	0.069	1	0.069	0.000
B3W1	1220.071	1	1220.071	2.127
B4W1	57.700	1	57.700	0.101
Error	19504.773	34	573.670	

Test data

	Interval test
4m	48
4m	15
4m	35
4m	40
4m	12
4m	50
4m	12
4m	0
1h	28
1h	60
1h	40
1h	2
1h	40
1h	20
1h	50
1h	52
3h	63
3h	50
3h	42
3h	38
3h	53
3h	10
3h	60
3h	57
6h	61
6h	85
6h	67
6h	52
6h	38
6h	77
6h	63
6h	87
24h	72
24h	25
24h	65
24h	68
24h	63
24h	62
24h	67

Between contrast coefficients

	4m	1h	3h	6h	24h
B1	3	3	-2	-2	-2
B2	0	0	2	-1	-1
B3	0	0	0	1	-1
B4	1	-1	0	0	0

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	6476.644	1	6476.644	20.855
B2	1442.897	1	1442.897	4.646
B3	132.805	1	132.805	0.428
B4	400.000	1	400.000	1.288
Error	10558.804	34	310.553	

Experiment 3

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
5m/24h	69	99	60	90	75	90	64	20	72	2	67
5m/24h	8	0	5	0	2	2	0	2	0	0	0
5m/24h	89	50	15	30	0	8	0	0	0	0	0
5m/24h	65	57	45	57	27	32	2	0	0	0	22
5m/24h	34	47	52	45	34	18	5	2	20	2	0
5m/24h	44	35	34	15	0	14	0	9	0	0	9
5m/24h	49	55	45	39	37	9	7	7	0	2	35
5m/24h	64	67	30	78	9	9	10	0	0	2	17
10m/24h	53	72	48	2	0	35	15	0	0	0	25
10m/24h	68	88	60	0	0	39	57	0	0	0	7
10m/24h	49	55	45	39	37	9	7	7	0	2	7
10m/24h	64	67	30	78	9	9	10	0	0	2	0
10m/24h	53	72	9	0	0	45	9	0	7	3	2
10m/24h	68	88	0	2	0	2	0	0	0	2	0
10m/24h	52	45	32	0	0	33	2	2	5	0	18
10m/24h	54	42	13	0	0	30	0	0	0	0	15
5m/4m	48	55	30	0	17	27	40	0	43	45	45
5m/4m	34	32	2	4	0	2	2	0	0	2	0
5m/4m	49	62	32	34	22	0	0	4	0	0	0
5m/4m	47	75	47	0	0	0	0	0	0	0	2
5m/4m	70	65	35	65	32	0	0	0	0	0	0
5m/4m	72	77	25	3	43	0	0	0	0	0	0
5m/4m	50	35	7	0	0	0	0	0	0	0	0
5m/4m	68	80	79	59	0	0	0	0	0	0	0
10m/4m	17	24	35	25	0	0	0	0	0	0	0
10m/4m	54	74	30	14	0	0	0	0	0	0	0
10m/4m	48	57	12	4	0	0	0	0	0	0	0
10m/4m	70	78	85	85	0	0	0	0	30	27	5
10m/4m	67	93	50	7	0	0	0	0	0	0	0
10m/4m	70	80	60	2	0	20	17	0	0	0	0
10m/4m	35	45	5	0	0	0	7	5	2	0	9
10m/4m	77	87	85	19	7	75	45	12	4	7	42

Between contrast coefficients

	5m/24h	10m/24h	5m/4m	10m/4m
B1	1	1	1	-3
B2	1	1	-2	0
B3	1	-1	0	0

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	63.524	1	63.524	0.041
B2	880.917	1	880.917	0.563
B3	983.273	1	983.273	0.628
Error	43832.170	28	1565.435	
Within				
W1	102351.807	1	102351.807	185.826
B1W1	212.784	1	212.784	0.386
B2W1	19.394	1	19.394	0.035
B3W1	195.111	1	195.111	0.354
Error	15422.222	28	550.794	

Experiment 3

Test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
5m/24h	27	3	0	22	0	0	0	0	0	0	0
5m/24h	3	2	0	0	0	0	2	0	0	0	0
5m/24h	3	2	0	0	0	0	0	0	0	0	0
5m/24h	20	7	0	0	0	0	0	0	0	0	0
5m/24h	7	0	12	22	22	0	0	0	0	0	0
5m/24h	23	0	23	0	0	0	0	0	0	0	0
5m/24h	3	0	0	0	0	0	0	0	0	0	0
5m/24h	3	0	5	0	0	0	0	0	0	0	0
10m/24h	10	0	2	0	0	0	0	0	0	0	0
10m/24h	8	3	0	0	0	0	0	0	0	0	0
10m/24h	3	0	0	0	0	0	0	0	0	0	0
10m/24h	10	0	0	0	3	0	0	0	0	0	0
10m/24h	0	2	2	5	25	7	0	0	0	0	0
10m/24h	0	0	0	0	0	0	3	0	0	0	0
10m/24h	8	12	2	2	2	8	0	0	0	0	0
10m/24h	18	0	7	12	12	3	0	0	0	0	0
5m/4m	58	65	33	28	8	0	0	0	0	0	0
5m/4m	37	15	5	7	2	0	0	0	0	0	0
5m/4m	63	42	18	13	13	0	0	0	0	0	0
5m/4m	58	62	70	8	8	0	0	0	0	0	0
5m/4m	35	70	20	8	10	13	0	0	0	0	0
5m/4m	52	25	0	3	0	0	0	0	0	0	0
5m/4m	53	15	0	0	0	2	0	0	0	0	0
5m/4m	92	87	82	78	23	8	0	0	0	0	0
10m/4m	78	28	28	0	0	0	0	0	0	0	0
10m/4m	27	20	0	0	0	0	0	0	0	0	0
10m/4m	30	15	2	0	0	0	0	0	0	0	0
10m/4m	48	5	12	2	40	0	0	0	0	0	0
10m/4m	52	33	0	0	2	0	0	0	0	0	0
10m/4m	82	62	33	18	2	12	22	0	0	0	0
10m/4m	17	0	0	3	3	2	0	0	0	0	0
10m/4m	85	65	30	22	13	8	3	0	0	0	0
No Ext	81	60	50	55	17	13	0	0	0	0	0
No Ext	53	20	65	62	18	13	0	0	0	0	0
No Ext	73	22	5	43	30	0	2	0	10	0	0
No Ext	45	35	20	10	0	0	0	0	2	0	0
No Ext	33	33	42	25	18	3	12	10	3	2	0
No Ext	92	63	50	43	48	7	8	0	0	0	0
No Ext	62	55	42	47	38	20	15	15	8	0	0
No Ext	70	72	65	27	13	15	5	28	17	0	0

Between contrast coefficients (non-orthogonal)

	5m/24h	10m/24h	5m/4m	10m/4m	No Ext
B1	3	3	-2	-2	-2
B2	1	-1	0	0	0
B3	0	0	1	1	-2
B4	0	0	-1	1	0

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	18912.256	1	18912.256	45.550
B2	10.023	1	10.023	0.024
B3	5013.502	1	5013.502	12.075
B4	842.188	1	842.188	2.028
Error	14531.920	35	415.198	
Within				
W1	50993.282	1	50993.282	126.565
B1W1	21760.039	1	21760.039	54.008
B2W1	38.409	1	38.409	0.095
B3W1	1500.800	1	1500.800	3.725
B4W1	1043.196	1	1043.196	2.589
Error	14101.538	35	402.901	

Experiment 4

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Massed	65	62	0	53	0	2	5	45	58	72	75
Massed	98	85	73	40	82	78	28	3	0	12	0
Massed	55	68	8	10	3	0	0	0	0	2	0
Massed	57	67	33	37	28	22	52	35	0	57	67
Massed	33	30	13	30	28	0	0	22	28	0	13
Massed	58	27	15	7	0	0	0	0	0	3	2
Massed	70	85	47	23	15	0	0	37	48	0	0
Massed	62	35	0	0	0	0	0	3	3	0	0
10min/4min	57	92	63	0	0	0	0	0	0	0	0
10min/4min	88	92	95	38	5	0	0	5	0	0	0
10min/4min	77	78	80	63	35	37	12	25	10	27	28
10min/4min	47	72	45	30	35	0	0	0	20	15	8
10min/4min	92	98	70	27	0	3	2	2	3	0	12
10min/4min	47	60	13	0	0	0	2	0	0	0	0
10min/4min	75	70	80	65	17	0	0	0	0	0	0
10min/4min	52	40	33	22	32	0	0	0	0	45	0

Between contrast coefficients

	Massed	10min/4min
B1	1	-1

Within contrast coefficients

	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	6.187	1	6.187	0.004
Error	23871.307	14	1705.093	
Within				
W1	53031.205	1	53031.205	46.997
B1W1	5365.528	1	5365.528	4.755
Error	15797.640	14	1128.403	

Experiment 4

Test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Massed	81	88	80	35	12	0	18	0	5	0	0
Massed	98	57	95	40	27	0	2	27	2	0	2
Massed	88	67	43	0	0	0	0	0	0	15	0
Massed	75	93	85	83	67	37	50	3	43	0	0
Massed	75	55	17	30	0	7	0	0	0	0	0
Massed	65	32	10	33	0	0	0	0	0	0	0
Massed	82	50	47	25	0	0	0	0	0	0	0
Massed	80	48	57	60	62	2	0	0	0	0	0
10min/4min	23	2	43	38	0	12	0	0	0	30	0
10min/4min	48	7	53	13	0	27	0	0	0	0	0
10min/4min	77	73	75	37	0	0	0	0	0	17	0
10min/4min	20	52	0	0	0	0	0	0	0	0	0
10min/4min	83	13	62	10	0	0	0	0	0	0	0
10min/4min	53	30	12	8	0	13	0	0	0	25	13
10min/4min	60	62	20	53	23	40	25	10	30	33	7
10min/4min	45	7	0	25	2	2	0	3	2	0	0
NE	68	58	25	13	0	0	0	0	2	0	0
NE	62	75	63	52	50	57	57	30	20	35	2
NE	80	78	45	28	67	23	20	13	7	42	20
NE	53	57	62	17	18	17	20	3	2	0	0
NE	78	92	38	37	13	15	3	0	2	0	0
NE	73	47	33	43	13	15	10	3	5	0	0
NE	90	83	62	63	33	30	23	7	2	0	8
NE	97	93	85	83	55	15	50	10	5	3	0

Between contrast coefficients

	Massed	10min/4min	NE
B1	-1	2	-1
B2	1	0	-1

Within contrast coefficients

	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	8976.002	1	8976.002	7.106
B2	1437.551	1	1437.551	1.138
Error	26526.170	21	1263.151	
Within				
W1	109972.546	1	109972.546	255.373
B1W1	7730.932	1	7730.932	17.952
B2W1	63.764	1	63.764	0.148
Error	9043.331	21	430.635	

Experiment 5

Trial 1 extinction data

	Session number										
	1	2	3	4	5	6	7	8	9	10	11
Spaced	62	50	57	48	17	15	2	0	0	0	0
Spaced	37	63	55	12	0	3	5	0	0	0	0
Spaced	63	60	80	88	58	73	52	28	0	0	0
Spaced	85	75	85	58	62	78	53	48	65	63	0
Spaced	63	62	65	63	82	33	27	0	3	0	0
Spaced	70	62	55	45	71	62	55	68	0	10	42
Spaced	60	70	87	60	70	55	51	0	10	0	0
Spaced	48	55	62	58	17	20	13	8	3	8	0
Massed/3	40	35	90	40	35	10	2	23	0	0	0
Massed/3	38	15	7	47	2	3	13	0	2	0	0
Massed/3	63	38	75	35	7	2	2	13	0	0	0
Massed/3	63	65	62	42	8	10	0	5	0	0	0
Massed/3	70	73	62	32	38	10	13	0	35	0	0
Massed/3	67	73	50	43	18	0	0	22	15	2	0
Massed/3	62	70	82	37	47	43	22	0	2	17	3
Massed/3	70	70	88	60	80	28	80	0	3	3	2
Massed/6	53	70	77	33	0	22	17	10	25	0	0
Massed/6	85	70	73	85	38	72	12	45	0	0	0
Massed/6	63	90	93	55	42	30	0	35	0	27	18
Massed/6	35	67	67	82	38	23	3	28	15	0	0
Massed/6	63	62	80	17	38	45	13	25	22	2	0
Massed/6	63	72	92	7	15	12	8	3	0	0	0
Massed/6	63	77	67	2	2	0	2	0	0	0	0
Massed/6	83	80	58	13	8	2	5	0	8	0	0
Massed/11	63	55	60	57	13	42	3	28	0	0	0
Massed/11	63	83	58	90	72	75	27	7	13	0	0
Massed/11	85	85	98	75	27	18	7	5	17	0	0
Massed/11	98	45	100	12	12	62	15	0	30	0	0
Massed/11	87	63	0	48	2	0	0	0	0	0	0
Massed/11	72	63	80	17	37	48	65	72	12	0	0
Massed/11	78	8	73	0	23	80	12	2	18	0	0
Massed/11	90	62	100	87	30	5	0	3	3	0	0

Between contrast coefficients

	Spaced	Massed/3	Massed/6	Massed/11
B1	3	-1	-1	-1
B2	0	2	-1	-1
B3	0	0	1	-1

Within contrast coefficients

	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA Trial 1 extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	3234.000	1	3234.000	2.521
B2	1463.335	1	1463.335	1.141
B3	308.460	1	308.460	0.240
Error	35912.341	28	1282.584	
Within				
W1	189175.575	1	189175.575	662.012
B1W1	353.834	1	353.834	1.238
B2W1	570.118	1	570.118	1.995
B3W1	79.901	1	79.901	0.280
Error	8001.235	28	285.758	

Trials 2 and 3 extinction data

	Session										
	1	2	3	4	5	6	7	8	9	10	11
Massed/3	5	20	7	5	1	0	1	0	0	0	0
Massed/3	8	2	0	4	1	0	1	0	0	0	0
Massed/3	1	1	0	6	0	6	3	0	0	0	0
Massed/3	0	0	3	2	0	5	5	0	0	0	0
Massed/3	45	19	5	3	13	2	0	0	0	0	0
Massed/3	44	27	9	10	1	2	0	0	0	0	0
Massed/3	48	43	33	1	4	1	0	0	0	0	0
Massed/3	72	81	77	59	27	9	0	0	0	0	0
Massed/6	33	33	9	0	1	0	0	0	0	0	0
Massed/6	75	75	12	6	0	0	0	0	0	0	0
Massed/6	10	10	17	20	2	14	10	0	0	0	0
Massed/6	21	21	23	23	3	0	7	0	0	0	0
Massed/6	65	14	10	0	0	0	0	0	0	0	0
Massed/6	59	14	1	0	0	10	0	0	0	0	0
Massed/6	35	2	0	0	5	5	0	0	0	0	0
Massed/6	41	0	7	1	7	4	0	0	0	0	0
Massed/11	53	12	1	3	1	0	3	0	0	0	0
Massed/11	19	21	19	16	9	15	2	0	0	0	0
Massed/11	54	8	2	3	0	0	0	0	0	0	0
Massed/11	48	2	1	12	2	0	1	0	0	0	0
Massed/11	58	2	7	0	0	3	0	0	0	0	0
Massed/11	50	24	12	0	1	14	0	0	0	0	0
Massed/11	24	0	0	0	0	0	1	0	0	0	0
Massed/11	82	0	1	4	0	0	9	0	0	0	0

Between contrast coefficients

	Massed/3	Massed/6	Massed/11
B1	2	-1	-1
B2	0	1	-1

Within contrast coefficients

	Extinction										
	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA trials 2 and 3 data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	101.500	1	101.500	0.263
B2	1.195	1	1.195	0.003
Error	8093.710	21	385.415	
Within				
W1	21609.019	1	21609.019	43.487
B1W1	28.000	1	28.000	0.056
B2W1	23.762	1	23.762	0.048
Error	10435.074	21	496.908	

Trials 4-6 extinction data

	Session										
	1	2	3	4	5	6	7	8	9	10	11
Massed/6	0	0	0	7	2	0	0	0	0	1	0
Massed/6	48	48	0	1	1	0	0	0	1	2	0
Massed/6	7	7	7	4	17	0	1	1	12	0	0
Massed/6	1	1	0	3	0	0	13	0	0	0	0
Massed/6	36	20	0	10	4	0	1	0	12	0	0
Massed/6	31	10	4	7	17	1	0	0	4	0	0
Massed/6	33	19	1	4	7	1	0	0	12	0	0
Massed/6	51	10	0	0	2	0	0	0	10	0	0
Massed/11	19	1	5	0	0	0	0	0	0	0	0
Massed/11	2	14	24	2	7	0	2	0	0	0	0
Massed/11	0	2	10	0	1	0	0	0	0	0	0
Massed/11	6	14	11	7	5	0	1	0	0	0	0
Massed/11	9	2	1	1	0	0	0	0	0	0	0
Massed/11	24	1	1	12	0	6	0	0	0	0	0
Massed/11	4	7	0	1	0	1	0	0	0	0	0
Massed/11	80	1	10	4	0	1	0	0	0	0	0

Between contrast coefficients

	Massed/6	Massed/11
B1	1	-1

Within contrast coefficients

	Extinction										
	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA trials 4-6 data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	219.678	1	219.678	2.664
Error	1154.273	14	82.448	
Within				
W1	3862.656	1	3862.656	22.795
B1W1	63.380	1	63.380	0.374
Error	2372.302	14	169.450	

Trials 7-11 extinction data

	Session										
	1	2	3	4	5	6	7	8	9	10	11
Massed/11	10	2	0	0	0	0	0	0	0	0	0
Massed/11	29	16	0	3	0	1	0	0	0	0	0
Massed/11	9	3	0	0	0	0	0	0	0	0	0
Massed/11	6	7	0	3	0	0	0	0	0	0	0
Massed/11	14	0	4	0	0	0	0	0	0	0	0
Massed/11	43	0	11	0	0	0	0	0	0	0	0
Massed/11	19	0	1	0	0	0	0	0	0	0	0
Massed/11	37	2	4	0	0	0	0	0	0	0	0

Within contrast coefficients

	Extinction										
	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA trials 7-11 data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between	195.976	7	27.997	
Within				
W1	1201.826	1	1201.826	20.562
Error	409.134	7	58.448	

Experiment 6

Extinction data

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
Massed	65	62	0	53	0	2	5	45	58	72	75
Massed	67	43	53	15	13	0	8	10	27	8	20
Massed	78	60	15	0	5	0	0	0	5	0	0
Massed	58	40	3	10	15	35	0	7	0	0	0
Massed	98	85	73	40	82	78	28	3	0	12	0
Massed	55	68	8	10	3	0	0	0	0	2	0
Massed	57	67	33	37	28	22	52	35	0	57	67
Massed	50	52	40	22	28	8	2	0	0	0	23
First	**30**	13	3	17	2	8	0	10	0	27	0
First	**97**	80	25	8	40	47	10	5	18	22	10
First	**60**	10	0	0	0	0	7	2	0	2	0
First	**67**	55	62	8	2	28	25	22	22	3	13
First	**58**	40	12	5	13	7	0	5	0	0	27
First	**63**	72	3	22	57	18	52	30	30	45	57
First	**82**	72	60	58	37	47	3	43	8	26	40
Middle	38	12	15	0	0	**30**	0	0	0	3	3
Middle	82	55	2	0	12	**48**	40	12	13	0	0
Middle	53	18	20	7	48	**12**	48	38	60	0	25
Middle	75	45	70	57	3	**50**	72	70	40	38	63
Middle	93	97	47	65	15	**48**	60	37	25	2	0
Middle	75	20	2	25	2	**45**	52	2	12	2	5
Middle	43	37	5	17	0	**57**	50	3	13	32	17
Last	67	50	37	37	10	28	38	10	32	23	**43**
Last	58	42	18	23	12	25	0	22	13	3	**35**
Last	52	30	10	42	48	17	20	20	48	35	**38**
Last	75	33	62	57	0	15	0	53	37	2	**63**
Last	77	70	70	37	15	10	2	0	15	15	**37**
Last	100	75	90	22	13	0	7	5	18	0	**45**
Last	77	65	68	50	0	65	25	30	77	0	**43**
Last	80	40	68	3	5	22	25	12	0	2	**48**

Between contrast coefficients

	Massed	First	Middle	Last
B1	3	-1	-1	-1
B2	0	2	-1	-1
B3	0	0	1	-1

Within contrast coefficients

	Extinction										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	426.259	1	426.259	0.245
B2	1891.918	1	1891.918	1.087
B3	486.866	1	486.866	0.280
Error	45262.692	26	1740.873	
Within				
W1	42808.632	1	42808.632	46.586
B1W1	370.613	1	370.613	0.403
B2W1	20.480	1	20.480	0.022
B3W1	179.891	1	179.891	0.196
Error	23891.808	26	918.916	

Test data

	Test					
	1st	2nd	3rd	4th	5th	6th
Massed	81	88	80	35	12	0
Massed	27	20	18	0	0	0
Massed	72	33	0	0	0	0
Massed	35	30	12	2	0	0
Massed	98	57	95	40	27	0
Massed	88	67	43	0	0	0
Massed	75	93	85	83	67	37
Massed	63	50	0	8	0	0
First	23	18	17	13	8	15
First	30	57	47	27	18	2
First	0	32	0	2	2	0
First	48	2	28	27	5	2
First	38	5	0	0	18	2
First	32	45	10	13	18	3
First	67	61	12	28	15	23
Middle	17	7	32	0	0	0
Middle	95	92	82	71	25	23
Middle	50	48	8	22	12	18
Middle	85	62	78	32	18	72
Middle	62	58	62	22	47	27
Middle	77	43	23	2	0	20
Middle	43	13	45	8	13	5
Last	40	28	7	32	13	2
Last	55	18	7	62	12	18
Last	60	55	28	28	5	23
Last	52	33	45	20	5	17
Last	63	62	82	18	43	20
Last	93	58	57	23	10	7
Last	68	63	67	35	52	12
Last	65	70	40	37	8	25

Between contrast coefficients

	Massed	First	Middle	Last
B1	-1	3	-1	-1
B2	2	0	-1	-1
B3	0	0	1	-1

Within contrast coefficients

	Test					
	1st	2nd	3rd	4th	5th	6th
W1	-5	-3	-1	1	3	5

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	8296.965	1	8296.965	4.842
B2	190.496	1	190.496	0.111
B3	0.476	1	0.476	0.000
Error	44548.601	26	1713.408	
Within				
W1	44270.406	1	44270.406	138.682
B1W1	2190.426	1	2190.426	6.862
B2W1	1494.733	1	1494.733	4.682
B3W1	46.245	1	46.245	0.145
Error	8299.756	26	319.221	

CS test data

	Test	
	Pre-CS	CS
Massed	0	10
Massed	8	10
Massed	0	50
Massed	0	53
Massed	0	53
Massed	0	12
Massed	0	0
Massed	0	40
First	0	3
First	0	0
First	0	7
First	0	0
First	0	5
First	0	0
First	0	0
Middle	0	10
Middle	0	30
Middle	0	38
Middle	0	62
Middle	0	27
Middle	0	40
Middle	0	8
Last	0	22
Last	0	20
Last	0	15
Last	20	25
Last	0	25
Last	0	40
Last	0	12
Last	0	13

Between contrast coefficients

	Massed	First	Middle	Last
B1	-1	3	-1	-1
B2	2	0	-1	-1
B3	0	0	1	-1

ANOVA pre-CS test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	7.298	1	7.298	0.467
B2	0.326	1	0.326	0.021
B3	23.333	1	23.333	1.494
Error	406.000	26	15.615	

ANOVA CS test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	3441.021	1	3441.021	14.039
B2	12.286	1	12.286	0.050
B3	445.886	1	445.886	1.819
Error	6372.571	26	245.099	

Experiment 7

Extinction data

		Extinction (2min blocks)								
	x	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	x
2min	50										
2min	52										
2min	70										
2min	75										
2min	77										
2min	40										
2min	65										
2min	72										
20min	38	8	47	2	0	2	0	43	18	3	
20min	65	58	53	20	2	2	0	12	5	0	
20min	33	65	72	42	22	5	0	0	0	0	
20min	33	62	67	0	5	20	45	20	12	0	
20min	28	18	5	0	0	0	0	0	0	3	
20min	93	87	53	0	0	7	0	0	25	28	
20min	58	57	45	15	0	0	13	3	0	0	
20min	70	90	77	95	90	87	47	0	0	0	
2<4>20	65	48	78	50	60	43	3	17	22	10	0
2<4>20	65	52	65	53	13	17	0	0	13	2	0
2<4>20	73	32	72	57	52	3	0	0	0	0	0
2<4>20	60	37	28	5	27	38	0	0	0	0	10
2<4>20	83	52	60	38	12	5	0	8	22	17	69
2<4>20	27	50	18	0	2	23	37	3	50	8	8
2<4>20	67	70	0	32	25	57	10	3	32	0	47
2<4>20	73	30	48	58	35	3	0	0	0	2	0
20<2>4	27	62	75	57	45	55	25	2	5	38	2
20<2>4	25	22	62	62	3	2	0	0	0	0	3
20<2>4	65	70	68	0	3	7	0	0	0	0	0
20<2>4	43	32	0	0	0	0	0	0	0	0	0
20<2>4	40	47	40	27	37	5	10	18	37	58	65
20<2>4	95	90	75	57	53	18	3	3	7	28	5
20<2>4	52	80	97	100	68	0	0	3	0	0	43
20<2>4	70	73	55	87	12	10	2	3	0	0	3

Between contrast coefficients

	20min	2<4>20	20<4>2
B1	1	-2	1
B2	1	0	-1

Within contrast coefficients

	Extinction									
	1	2	3	4	5	6	7	8	9	10
W1	-9	-7	-5	-3	-1	1	3	5	7	9

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	770.133	1	770.133	0.575
B2	722.500	1	722.500	0.539
Error	28137.363	21	1339.874	
Within				
W1	66856.833	1	66856.833	75.755
B1W1	1707.071	1	1707.071	1.934
B2W1	173.094	1	173.094	0.196
Error	18533.290	21	882.538	

Test data

	Test										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
2min	63	37	10	45	18	0	3	0	0	0	0
2min	62	42	45	52	47	13	38	38	62	0	0
2min	78	30	87	77	60	57	43	13	0	3	5
2min	70	67	55	48	42	62	52	20	13	10	0
2min	81	42	10	2	8	0	2	0	5	0	0
2min	73	87	47	57	58	3	7	2	8	0	0
2min	68	38	60	33	18	5	20	3	3	18	0
2min	57	60	58	40	35	28	20	7	22	0	0
20min	60	7	48	12	30	0	0	0	0	0	0
20min	65	12	8	12	12	0	0	0	0	0	0
20min	33	8	13	0	0	0	0	0	0	0	0
20min	45	25	0	20	23	0	0	0	0	0	0
20min	7	28	3	5	0	0	0	0	0	0	0
20min	48	65	43	2	42	28	0	0	0	0	0
20min	38	15	2	0	0	2	0	0	0	0	0
20min	22	70	52	0	0	2	0	0	0	0	0
2<4>20	88	92	80	63	17	2	2	0	13	0	0
2<4>20	58	58	12	0	0	8	2	0	0	0	0
2<4>20	43	27	0	0	12	0	0	5	20	0	0
2<4>20	82	78	82	45	25	10	7	13	0	0	0
2<4>20	82	77	87	55	32	33	0	2	0	2	0
2<4>20	73	65	77	33	35	62	3	0	0	0	0
2<4>20	82	55	82	28	20	43	0	0	0	0	0
2<4>20	62	50	48	0	3	53	3	10	0	0	0
20<2>4	47	28	5	3	7	2	0	3	0	0	0
20<2>4	77	17	0	0	0	3	0	0	2	0	0
20<2>4	18	7	23	0	0	0	3	0	0	7	0
20<2>4	8	0	0	0	0	0	0	0	0	0	0
20<2>4	38	27	33	33	20	23	5	8	0	8	0
20<2>4	35	58	18	2	3	3	0	0	0	0	0
20<2>4	13	12	3	2	45	2	0	23	8	0	0
20<2>4	68	0	48	0	87	5	0	13	0	2	0
No Ext	43	73	35	30	10	12	5	8	2	0	0
No Ext	68	68	58	46	40	40	27	8	8	0	0
No Ext	88	98	85	97	25	78	30	42	42	0	0
No Ext	82	63	42	27	12	8	10	7	0	0	0
No Ext	100	92	100	88	15	0	0	0	0	0	0
No Ext	97	95	92	83	80	47	32	8	22	0	0
No Ext	95	45	35	32	0	3	0	0	0	0	0
No Ext	65	57	38	8	0	3	0	0	0	0	0

Between contrast coefficients (planned non-orthogonal)

	2min	20min	2<4>20	20<4>2	No Ext
B1	-2	3	-2	3	-2
B2	1	0	1	0	-2
B3	1	0	-1	0	0
B4	0	1	0	-1	0

Within contrast coefficients

	Test										
	1	2	3	4	5	6	7	8	9	10	11
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	34674.360	1	34674.360	32.824
B2	1053.047	1	1053.047	0.997
B3	700.006	1	700.006	0.663
B4	0.016	1	0.016	0.000
Error	36973.536	35	1056.387	
Within				
W1	149309.895	1	149309.895	312.454
B1W1	18979.512	1	18979.512	39.718
B2W1	1455.615	1	1455.615	3.046
B3W1	599.278	1	599.278	1.254
B4W1	259.914	1	259.914	0.544
Error	16725.179	35	477.862	

Extinction data

	Extinction					
	1st	2nd	3rd	4th	5th	6th
Blocked Similar	77	70	5	0	0	0
Blocked Similar	30	10	38	0	0	0
Blocked Similar	57	62	72	38	7	42
Blocked Similar	23	3	0	0	0	2
Blocked Similar	53	27	10	25	38	0
Blocked Similar	15	23	15	42	35	10
Blocked Similar	73	72	58	12	55	28
Blocked Similar	42	42	57	25	48	37
Blocked Different	47	43	30	35	12	33
Blocked Different	47	50	25	13	23	8
Blocked Different	53	52	48	35	8	15
Blocked Different	23	28	55	43	10	17
Blocked Different	92	87	2	3	2	40
Blocked Different	48	53	40	37	23	45
Blocked Different	65	73	58	67	58	7
Blocked Different	80	88	50	53	40	58
Alternation Similar	82	63	63	33	0	55
Alternation Similar	75	57	40	22	13	22
Alternation Similar	67	48	60	52	35	45
Alternation Similar	77	32	40	52	38	22
Alternation Similar	52	57	28	7	5	7
Alternation Similar	42	22	23	20	15	15
Alternation Similar	18	28	8	3	5	2
Alternation Different	32	40	36	27	15	0
Alternation Different	53	0	47	28	0	0
Alternation Different	68	22	43	42	48	12
Alternation Different	80	33	47	62	18	47
Alternation Different	50	38	33	18	0	0
Alternation Different	58	55	63	63	55	38
Alternation Different	58	45	52	40	47	2
Alternation Different	52	38	38	30	38	53

Between contrast coefficients

	Blk Sim	Blk Diff	Alt Sim	Alt Diff
B1	-3	1	1	1
B2	0	-2	1	1
B3	0	0	-1	1

Within contrast coefficients

	Extinction					
	1	2	3	4	5	6
W1	-5	-3	-1	1	3	5

ANOVA extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	2508.379	1	2508.379	2.148
B2	606.423	1	606.423	0.519
B3	111.013	1	111.013	0.095
Error	31534.643	27	1167.950	
Within				
W1	25366.731	1	25366.731	81.972
B1W1	69.494	1	69.494	0.225
B2W1	26.613	1	26.613	0.086
B3W1	177.430	1	177.430	0.573
Error	8355.353	27	309.458	

Test data

	Test							
	1st	2nd	3rd	4th	5th	6th	7th	8th
Blocked Similar	98	85	15	0	0	0	0	0
Blocked Similar	58	57	30	2	0	0	0	0
Blocked Similar	68	38	43	32	5	0	0	0
Blocked Similar	8	3	2	0	0	0	0	0
Blocked Similar	72	22	38	67	50	8	0	0
Blocked Similar	63	53	47	3	40	42	22	28
Blocked Similar	100	58	30	0	20	20	3	0
Blocked Similar	70	43	32	42	12	32	2	17
Blocked Different	58	25	18	33	25	8	8	15
Blocked Different	53	25	18	43	20	23	12	13
Blocked Different	75	45	28	27	17	5	0	2
Blocked Different	37	33	25	25	12	10	7	2
Blocked Different	98	93	83	30	85	53	35	12
Blocked Different	52	58	23	58	33	10	7	23
Blocked Different	87	50	68	45	33	45	32	18
Blocked Different	77	55	62	31	45	17	5	2
Alternation Similar	73	25	25	40	3	0	12	22
Alternation Similar	33	17	33	20	15	30	5	17
Alternation Similar	48	47	30	7	2	10	0	5
Alternation Similar	12	27	20	8	0	8	0	3
Alternation Similar	20	0	3	0	0	0	2	0
Alternation Similar	5	8	5	7	0	0	5	0
Alternation Similar	7	8	5	2	0	0	0	0
Alternation Different	52	30	33	15	2	0	0	0
Alternation Different	35	7	2	2	2	0	0	0
Alternation Different	85	67	55	33	55	38	55	20
Alternation Different	77	65	55	42	58	48	37	43
Alternation Different	28	10	15	8	0	0	0	0
Alternation Different	75	58	65	20	22	18	5	0
Alternation Different	53	58	37	73	30	47	55	20
Alternation Different	57	48	53	22	8	32	30	0

Between contrast coefficients

	Blk Sim	Blk Diff	Alt Sim	Alt Diff
B1	1	1	-3	1
B2	1	1	0	-2
B3	1	-1	0	0

Within contrast coefficients

	Test							
	1	2	3	4	5	6	7	8
W1	-7	-5	-3	-1	1	3	5	7

ANOVA test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	13604.571	1	13604.571	8.657
B2	73.500	1	73.500	0.047
B3	2738.000	1	2738.000	1.742
Error	42433.179	27	1571.599	
Within				
W1	49445.659	1	49445.659	124.065
B1W1	3582.693	1	3582.693	8.989
B2W1	950.127	1	950.127	2.384
B3W1	94.500	1	94.500	0.237
Error	10760.772	27	398.547	

Baseline data: Heart rate

	Heart rate				
	1st	2nd	3rd	4th	5th
Massed/Spaced	280	276	312	304	296
Massed/Spaced	293	296	307	290	289
Massed/Spaced	270	292	264	314	260
Massed/Spaced	258	275	258	329	250
Massed/Spaced	291	288	322	287	291
Massed/Spaced	259	284	310	266	264
Massed/Massed	307	310	308	315	314
Massed/Massed	279	279	281	285	283
Massed/Massed	423	422	433	459	409
Massed/Massed	344	317	318	309	308
Massed/Massed	321	316	328	342	394
Massed/Massed	178	231	294	141	132
Spaced/Massed	244	189	399	384	238
Spaced/Massed	295	252	307	287	294
Spaced/Massed	281	280	282	282	278
Spaced/Massed	311	329	308	300	300
Spaced/Massed	292	293	290	303	306
Spaced/Massed	269	274	278	282	278
Spaced/Massed	307	296	302	292	300
Spaced/Spaced	282	284	280	309	281
Spaced/Spaced	310	311	308	307	311
Spaced/Spaced	299	282	293	317	293
Spaced/Spaced	308	307	307	344	310
Spaced/Spaced	312	321	319	323	338
Spaced/Spaced	335	339	327	331	331

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	-1	1
B3	1	-1	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th
W1	-2	-1	0	1	2

ANOVA heart rate baseline data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	101.869	1	101.869	0.013
B2	459.435	1	459.435	0.061
B3	16444.151	1	16444.151	2.169
Error	159218.143	21	7581.816	
Within				
W1	468.927	1	468.927	0.890
B1W1	318.342	1	318.342	0.604
B2W1	21.520	1	21.520	0.041
B3W1	139.113	1	139.113	0.264
Error	11059.405	21	526.638	

Baseline data: Blood pressure

	Blood pressure				
	1st	2nd	3rd	4th	5th
Massed/Spaced	86	87	86	85	87
Massed/Spaced	84	90	108	88	87
Massed/Spaced	86	89	88	99	93
Massed/Spaced	94	98	94	95	89
Massed/Spaced	90	92	87	91	90
Massed/Spaced	75	82	81	79	81
Massed/Massed	67	68	73	77	73
Massed/Massed	82	83	82	83	83
Massed/Massed	88	96	91	90	96
Massed/Massed	79	78	84	81	81
Massed/Massed	92	97	92	96	96
Massed/Massed	79	83	82	83	82
Spaced/Massed	102	83	105	98	100
Spaced/Massed	94	94	92	93	94
Spaced/Massed	95	92	92	89	92
Spaced/Massed	88	90	89	88	88
Spaced/Massed	85	84	86	84	87
Spaced/Massed	71	75	71	72	73
Spaced/Massed	79	79	77	77	78
Spaced/Spaced	79	78	79	80	81
Spaced/Spaced	84	85	87	93	87
Spaced/Spaced	88	89	86	97	96
Spaced/Spaced	88	84	87	86	89
Spaced/Spaced	90	88	88	87	88
Spaced/Spaced	89	93	88	91	91

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	-1	1
B3	1	-1	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th
W1	-2	-1	0	1	2

ANOVA blood pressure baseline data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	14.025	1	14.025	0.056
B2	214.959	1	214.959	0.861
B3	146.692	1	146.692	0.587
Error	5245.686	21	249.795	
Within				
W1	63.264	1	63.264	7.719
B1W1	4.152	1	4.152	0.507
B2W1	0.972	1	0.972	0.119
B3W1	24.306	1	24.306	2.966
Error	172.117	21	8.196	

Extinction data: Heart rate

	Heart rate										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	484	469	457	480	429	395	381	407	387	389	409
M/S	450	404	403	409	384	376	385	398	400	388	407
M/S	356	368	395	436	426	353	357	350	359	355	323
M/S	351	425	419	403	362	378	374	365	337	345	379
M/S	405	383	381	400	446	398	402	380	377	391	385
M/S	436	418	406	394	397	391	410	381	396	378	403
M/M	517	514	478	496	466	485	439	441	416	417	409
M/M	405	477	374	414	416	401	413	408	418	396	367
M/M	409	414	420	480	426	425	427	397	435	384	432
M/M	429	473	466	501	517	480	500	475	465	461	510
M/M	341	347	379	369	383	373	375	371	360	379	383
M/M	363	410	424	410	394	394	375	392	398	387	409
M/M	446	494	466	414	509	507	418	455	427	364	448
S/M	368	438	380	432	453	409	414	339	419	344	507
S/M	440	466	455	422	442	435	444	451	390	372	418
S/M	424	444	388	450	437	480	477	446	448	445	453
S/M	400	437	468	424	410	451	425	432	449	450	409
S/M	491	427	456	495	494	490	513	495	497	470	446
S/M	405	357	397	347	450	448	479	420	469	431	402
S/M	414	423	466	445	461	445	379	388	490	431	419
S/S	408	444	449	478	484	471	475	439	445	459	475
S/S	455	436	466	510	493	465	514	508	482	496	477
S/S	466	484	510	482	493	463	500	518	503	515	489
S/S	377	455	519	506	535	456	507	510	489	470	448
S/S	402	455	509	528	528	473	493	501	501	464	507
S/S	392	455	445	416	458	465	488	458	473	450	465

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	0	0
B3	0	0	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA heart rate extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	93491.338	1	93491.338	11.979
B2	148.428	1	148.428	0.019
B3	21719.106	1	21719.106	2.783
Error	171696.902	22	7804.405	
Within				
W1	1127.708	1	1127.708	0.814
B1W1	22886.629	1	22886.629	16.518
B2W1	1650.694	1	1650.694	1.191
B3W1	2318.521	1	2318.521	1.673
Error	30481.754	22	1385.534	

Extinction data: Blood pressure

	Blood pressure										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	124	123	119	111	111	109	111	109	109	115	111
M/S	123	123	117	115	110	109	107	108	107	105	110
M/S	124	126	124	118	110	112	114	112	112	108	106
M/S	123	122	116	117	114	113	117	114	110	111	117
M/S	129	132	134	133	120	119	116	117	115	117	113
M/S	112	118	121	120	113	106	104	102	102	100	99
M/M	106	107	101	97	89	90	86	90	86	82	83
M/M	127	133	128	119	117	106	108	103	107	101	108
M/M	151	161	159	135	142	136	126	124	128	125	113
M/M	132	145	139	138	135	135	135	117	124	118	123
M/M	137	144	146	144	137	137	133	123	124	117	114
M/M	114	120	107	102	98	92	92	90	94	89	95
M/M	111	124	112	111	115	91	97	100	100	101	106
S/M	135	120	129	122	129	128	130	133	135	126	116
S/M	120	99	114	116	121	116	120	118	125	117	124
S/M	134	133	135	115	131	130	129	124	122	116	118
S/M	118	112	119	115	116	113	114	115	113	100	107
S/M	120	105	100	106	110	104	109	107	110	106	109
S/M	106	107	97	100	99	103	104	107	105	94	97
S/M	116	112	102	107	104	108	114	105	110	98	93
S/S	126	117	127	122	125	120	123	122	114	113	115
S/S	120	119	121	107	118	124	121	107	111	102	110
S/S	93	117	127	129	131	129	120	126	128	111	116
S/S	116	118	125	111	102	103	114	105	88	91	94
S/S	101	118	125	107	114	111	109	92	102	96	89
S/S	115	118	125	131	135	134	121	126	125	111	137

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	0	0
B3	0	0	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA blood pressure extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	1182.265	1	1182.265	0.825
B2	4933.410	1	4933.410	3.442
B3	1069.630	1	1069.630	0.746
Error	31534.285	22	1433.377	
Within				
W1	6391.312	1	6391.312	60.301
B1W1	1421.897	1	1421.897	13.415
B2W1	439.748	1	439.748	4.149
B3W1	100.163	1	100.163	0.945
Error	2331.789	22	105.990	

Extinction data: Freezing

	Freezing										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	12	38	33	0	0	0	13	7	0	8	17
M/S	63	40	20	5	0	0	23	7	0	7	8
M/S	70	33	42	0	17	0	0	7	0	27	2
M/S	88	87	80	25	0	0	0	0	18	17	0
M/S	70	68	45	42	12	2	2	0	18	0	0
M/S	53	30	2	0	0	0	5	0	0	0	2
M/M	82	27	22	32	0	3	5	0	0	0	15
M/M	63	18	0	0	7	8	37	0	20	0	0
M/M	52	53	38	7	21	28	43	2	3	2	0
M/M	88	48	17	0	0	22	36	8	53	0	17
M/M	77	56	8	0	47	0	0	0	12	0	2
M/M	66	45	62	3	0	0	0	0	0	0	2
M/M	31	55	43	38	0	18	38	52	13	17	10
S/M	55	62	43	7	38	23	7	38	35	25	25
S/M	78	68	60	23	67	38	12	27	15	43	13
S/M	32	38	21	3	8	0	0	0	2	0	2
S/M	80	82	68	57	80	52	30	30	12	32	50
S/M	65	67	63	22	30	3	0	0	0	27	53
S/M	70	67	63	32	43	23	0	15	3	3	48
S/M	78	52	90	73	87	53	40	67	60	70	5
S/S	42	42	17	27	32	17	0	20	0	0	0
S/S	65	33	22	0	28	15	0	0	0	0	0
S/S	95	67	55	5	22	5	2	0	2	0	0
S/S	80	90	77	35	45	18	28	30	37	22	5
S/S	80	75	57	82	68	20	17	22	23	25	0
S/S	95	98	100	95	75	0	50	38	13	2	20

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	0	0
B3	0	0	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA freezing extinction data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	12361.637	1	12361.637	8.269
B2	271.517	1	271.517	0.182
B3	2575.881	1	2575.881	1.723
Error	31392.736	21	1494.892	
Within				
W1	70029.280	1	70029.280	190.418
B1W1	499.958	1	499.958	1.359
B2W1	73.917	1	73.917	0.201
B3W1	1432.552	1	1432.552	3.895
Error	7723.073	21	367.765	

Test data: Heart rate

	Heart rate										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	472	497	478	498	482	490	479	512	505	464	494
M/S	417	448	446	474	451	468	465	488	471	438	469
M/S	361	387	437	459	468	441	413	450	466	358	428
M/S	405	402	471	448	447	451	440	443	450	426	464
M/S	411	410	437	458	448	436	476	478	462	470	478
M/S	409	414	422	401	422	424	410	426	393	428	419
M/M	423	418	408	421	422	453	426	418	469	418	387
M/M	372	441	449	438	451	431	455	467	471	478	444
M/M	379	381	472	395	419	416	461	467	416	416	518
M/M	438	441	410	456	445	466	475	444	429	488	492
M/M	359	360	405	368	373	384	367	357	372	357	355
M/M	389	418	382	413	396	395	419	371	399	391	362
M/M	462	472	461	424	463	457	458	437	422	421	411
S/M	468	434	429	437	433	455	475	470	414	441	441
S/M	453	389	405	379	395	402	359	406	365	360	360
S/M	463	430	397	389	385	419	454	403	444	400	400
S/M	419	390	420	397	367	387	429	384	382	396	396
S/M	418	378	382	397	378	396	392	383	389	368	345
S/M	371	394	366	407	393	410	375	364	381	455	369
S/M	464	421	447	407	386	382	432	387	393	385	434
S/S	476	428	481	402	433	485	449	491	442	424	433
S/S	493	485	488	499	468	496	503	516	501	477	479
S/S	539	536	525	526	476	474	528	529	530	519	515
S/S	471	463	497	433	416	439	494	451	459	450	449
S/S	505	505	485	455	491	485	507	506	491	466	450
S/S	476	492	491	464	409	463	459	460	450	455	413

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	-1	1
B3	1	-1	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA heart rate test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	315.378	1	315.378	0.041
B2	213987.091	1	213987.091	27.728
B3	241.057	1	241.057	0.031
Error	169783.649	22	7717.439	
Within				
W1	10.089	1	10.089	0.009
B1W1	11989.679	1	11989.679	11.213
B2W1	497.373	1	497.373	0.465
B3W1	142.652	1	142.652	0.133
Error	23523.124	22	1069.233	

Test data: Blood pressure

	Blood pressure										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	120	121	123	118	126	122	118	123	120	123	123
M/S	115	121	120	125	127	111	114	112	115	113	116
M/S	132	124	124	119	127	120	118	121	116	130	131
M/S	119	111	117	112	111	117	121	120	111	114	119
M/S	128	129	130	130	133	134	128	132	130	128	131
M/S	116	116	117	119	120	115	117	117	113	113	114
M/M	86	89	88	93	89	94	91	87	96	91	81
M/M	131	126	128	135	128	120	104	107	113	112	105
M/M	112	115	112	118	115	126	117	114	115	114	117
M/M	117	116	116	119	122	119	119	112	113	120	115
M/M	119	116	131	123	113	115	106	108	113	102	104
M/M	90	96	91	96	92	93	92	92	92	89	84
M/M	86	92	97	92	90	92	98	94	86	84	80
S/M	121	110	110	110	110	116	114	113	107	109	109
S/M	112	109	112	107	108	108	105	109	105	103	103
S/M	119	111	108	109	109	111	106	106	112	105	105
S/M	107	98	101	96	93	97	99	93	96	95	95
S/M	108	105	102	109	100	101	100	97	101	101	95
S/M	95	92	89	88	88	95	89	88	87	101	87
S/M	97	96	93	102	98	98	98	88	94	98	99
S/S	112	115	117	113	109	70	113	117	115	112	113
S/S	114	111	116	118	113	124	114	118	119	108	119
S/S	113	121	121	116	122	106	120	117	121	115	116
S/S	106	87	91	102	91	86	97	99	92	89	96
S/S	112	107	111	102	100	104	117	108	101	100	99
S/S	128	126	140	128	134	126	135	109	115	128	129

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	-1	1
B3	1	-1	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA blood pressure test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	201.583	1	201.583	0.174
B2	6241.674	1	6241.674	5.392
B3	1344.197	1	1344.197	1.161
Error	25465.296	22	1157.513	
Within				
W1	691.347	1	691.347	13.869
B1W1	0.447	1	0.447	0.009
B2W1	172.136	1	172.136	3.453
B3W1	37.719	1	37.719	0.757
Error	1096.693	22	49.850	

Test data: Freezing

	Freezing										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
M/S	62	8	50	5	0	25	0	0	0	0	0
M/S	77	67	65	13	10	5	0	0	0	0	0
M/S	58	13	42	2	2	10	0	0	0	0	0
M/S	82	32	35	5	0	5	0	0	0	0	0
M/S	67	58	53	18	0	0	0	0	0	0	0
M/S	68	52	5	2	0	18	0	0	0	0	0
M/M	10	33	8	17	0	2	0	2	0	0	0
M/M	13	17	27	3	8	3	13	0	0	0	0
M/M	38	28	0	27	0	0	0	2	22	2	0
M/M	23	13	18	0	18	3	0	2	15	2	0
M/M	33	37	3	30	32	0	10	13	33	5	0
M/M	0	0	0	0	0	0	0	5	0	12	0
M/M	15	2	0	7	0	0	2	18	17	7	35
S/M	5	2	10	25	13	0	2	5	33	25	3
S/M	22	3	2	8	10	0	0	3	13	20	33
S/M	0	0	30	22	28	42	2	20	2	32	13
S/M	65	58	0	48	25	30	0	5	28	5	5
S/M	20	0	2	0	2	2	0	2	8	20	35
S/M	63	0	3	8	2	20	58	60	53	0	15
S/M	0	0	0	0	48	28	0	17	2	27	8
S/S	18	45	2	60	0	0	7	0	8	3	2
S/S	0	0	0	5	0	0	0	0	0	0	0
S/S	0	0	0	0	0	0	0	0	0	0	0
S/S	30	35	48	28	23	5	8	57	25	0	5
S/S	43	18	8	2	0	10	0	2	0	0	0
S/S	30	33	18	8	23	5	5	2	3	2	3

Between contrast coefficients

	Massed/Spaced	Massed/Massed	Spaced/Massed	Spaced/Spaced
B1	1	1	-1	-1
B2	1	-1	-1	1
B3	1	-1	1	-1

Within contrast coefficients

	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th
W1	-5	-4	-3	-2	-1	0	1	2	3	4	5

ANOVA freezing test data

Analysis of Variance Summary Table

Source	SS	df	MS	F
Between				
B1	14.238	1	14.238	0.031
B2	3.656	1	3.656	0.008
B3	2768.227	1	2768.227	6.029
Error	10102.167	22	459.189	
Within				
W1	13667.098	1	13667.098	39.398
B1W1	4905.402	1	4905.402	14.141
B2W1	8232.086	1	8232.086	23.730
B3W1	1070.220	1	1070.220	3.085
Error	7631.826	22	346.901	