Learning about the CS during latent inhibition: Preexposure enhances temporal

control

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Abstract

In three experiments rats were given nonreinforced preexposure to an auditory stimulus, after which this stimulus and a second, novel cue were paired with food. Lower rates of conditioned responding were observed to the preexposed stimulus across the three experiments, indicative of latent inhibition. The degree to which animals used these cues to time the occurrence of food delivery was also examined. Paradoxically, the response slopes - indicating the rate of increase in responding over the course of the CS - were greater for the preexposed than for the novel cues, consistent with the suggestion that the preexposed stimulus exerted greater temporal control. Moreover, this was the case irrespective of whether the duration of the cue during preexposure differed from that during conditioning. These results suggest that although CS preexposure retards conditioning, it may enhance timing. The findings are discussed in terms of current models of conditioning and timing.

Conditioning - learning that an environmental cue (conditioned stimulus, or CS) reliably signals an outcome (an unconditioned stimulus or US, often of motivational value) - is found across the animal kingdom. It is indicated by an elevation of *conditioned responding* during CS presentation, indicating increased anticipation of the US. However, other kinds of learning also occur during conditioning tasks. For example, animals learn to use the CS to *time* US occurrence: when the CS is of a fixed duration, subjects respond at increasingly high rates as the time of US delivery approaches (e.g. Kirkpatrick & Church 2000); moreover, if the CS is presented for an extended period without reinforcement, there is a peak of responding at the point at which the US would normally occur, resulting in an inverted-U, Gaussian-type function (Roberts, 1981).

Although conditioning and timing have been shown to occur side-by-side in the same task (e.g. Balsam, Drew & Yang, 2002; Kirkpatrick & Church, 2000), the theoretical frameworks devised to explain these effects have tended to focus on one of the two phenomena (e.g. Gallistel & Gibbon 2000; Jennings et al., 2013; Kirkpatrick 2014; Kirkpatrick & Church, 1998; Savastano & Miller 1998). Theories of conditioning are often *associative*, attributing it to the formation of an association between the mental representations of the CS and the US, allowing presentation of the CS to activate the US representation and elicit a conditioned response. Such theories offer little explanation of timing effects (except for real-time models - see below). In contrast, some timing theories do offer an account of conditioning, but as a by-product of the timing process (see Kirkpatrick, 2014 for a recent review). For example, hybrid time-based models assume that the CS is represented in a form that

varies as a function of time[1], which allows the time of reinforcement to be accurately encoded. In contrast, information-processing models assume that conditioning stems from sensitivity to temporal information in the conditioning episode. For example, scalar expectancy theory (Gibbon & Balsam, 1981; cf. Rate Expectancy Theory, Gallistel & Gibbon, 2000) assumes the animal computes the difference between the expected time to reinforcement in the *presence* of the CS, and in its absence; if the former value exceeds the latter by some criterion, the decision is made to respond (cf., Balsam & Gallistel, 2009; Kirkpatrick & Church, 2003).

Because of the theoretical gulf between associative and time-based accounts of conditioning, phenomena central to the associative perspective have been largely neglected by timing theories. One example is *latent inhibition* - that nonreinforced preexposure to a stimulus retards acquisition of conditioned responding to that stimulus, relative to a nonpreexposed cue (Lubow & Moore, 1959). Many associative theories interpret latent inhibition as a retardation in learning, resulting from a drop in the *associability* of the CS (e.g. Mackintosh 1975; McLaren & Mackintosh, 2002; Pearce & Hall 1980; Wagner 1981). This may occur either as a function of the predictive ability of the CS (Mackintosh 1975; Pearce & Hall 1980), or the degree to which it is predicted, either by the context (Wagner 1981), or by elements of the stimulus itself (cf. McLaren & Mackintosh, 2002). Others view it as a *performance* effect, produced by competition between a CS-->nothing association formed during preexposure and the CS-->US association established during conditioning (Bouton,

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[1] For example, because CS onset initiates a series of memory traces (Machado, 1997), or activity in a series of oscillators with different periods (Church & Broadbent, 1990), or some other time-based process that uniquely defines different portions of the CS (see also e.g. Grossberg & Schmajuk, 1989; Staddon & Higa, 1996)

1993; Savastano, Yin, Barnet & Miller, 1998). Time-based accounts of conditioning, in contrast, say little about nonreinforced preexposure. Hybrid models typically focus on the temporal relation between specific states of the CS representation and *reinforcement*, while although information-processing theories assume information is gathered over a broad time window, which could include the preexposure phase (e.g. Balsam & Gallistel, 2009; Gallistel and Gibbon 2000), it is unclear whether they predict latent inhibition. For example, according to SET nonreinforced preexposure increases the expected time to reinforcement both in the presence of the CS *and* in its absence, leaving the ratio of these values unchanged relative to an untreated control group[2] (see also RET: Gallistel & Gibbon, 2000).

Our aim was thus to examine the effect of stimulus preexposure on timing. While most associative theories do not account for timing explicitly, they could explain it by assuming that different portions of the CS condition to different degrees according to their proximity to the US; conditioning would therefore be at a maximum at the point of US delivery. This interpretation suggests that any factor retarding acquisition of conditioning will also retard acquisition of timing. Other associative theories have been formulated to incorporate timing directly; for example, the temporal coding hypothesis (Matzel, Held & Miller, 1988) proposes that associations incorporate information about the CS-US pairing - including the temporal relationship between them (e.g. Barnet, Grahame & Miller 1993; Barnet, Cole & Miller 1997; Blaisdell, Denniston & Miller 1998). Although temporal coding does not explicitly address latent inhibition, if stimulus preexposure were to produce

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[2] whereas a control group that is simply placed in the context would, according to this account, learn faster than either of these conditions.

an association between the CS and no outcome, it would predict that the duration of the stimulus would also be encoded. When the CS is then paired with the US, potential for competition between these two associations arises (e.g. Bouton, 1993), which these authors have argued is the source of latent inhibition (cf. Savastano et al., 1998). Temporal coding thus predicts that this interference, and hence latent inhibition, will be greater when the duration of the CS during preexposure and conditioning match - and the same might also apply to timing ability.

In contrast, time-based theories of conditioning make no clear predictions. While hybrid models say little about nonreinforced exposure, information processing models like SET predict that timing occurs only *after* conditioned responding has been acquired (although see e.g. Balsam, Drew & Yang, 2002; Kirkpatrick & Church, 2000). Thus *if* they could predict latent inhibition they should also predict retardation of timing - but it is not clear they can predict latent inhibition in the first place.

In summary, the temporal coding hypothesis predicts that timing will be retarded by preexposure, and that this effect will be maximal when the duration of the stimulus during preexposure matches that during conditioning. Associative theories interpreting latent inhibition as an effect on learning may also predict it will retard timing (e.g. Mackintosh 1975; McLaren & Mackintosh, 2002; Pearce & Hall 1980; Wagner 1981), although (compared to temporal coding) the boundary conditions of this are unspecified. Finally, time-based theories of conditioning make no clear predictions about the effect of preexposure on either conditioning or timing effects.

Experiment 1

In the first experiment two groups of rats were preexposed to an auditory CS, after which this and a second, novel cue were each reinforced after 40s with two food pellets. We anticipated preexposure would result in latent inhibition - slower learning to the preexposed than to the novel CS. The groups' treatment differed in the preexposure phase. Although both groups received the same total duration of CS exposure, for Group *Same* CS presentations were always of a fixed, 40-s duration, exactly as during conditioning, whereas for Group *Different* the preexposed CS varied in duration from trial to trial according to a uniform distribution with an average of 40s), such that it was either longer or shorter than, but never equal to, 40s. Thus Group *Different* - unlike Group *Same* - did not have any experience of the conditioning duration (see Table 1).

INSERT TABLE 1 ABOUT HERE

In both groups we anticipated seeing latent inhibition - slower acquisition of conditioned responding to the preexposed CS - and we expected it to be similar in magnitude because total CS exposure was equated: Ayres et al. (1992) found this factor was the most important determinant of the degree of latent inhibition, and that matching the CS duration during preexposure and conditioning had little effect (contrary to the predictions of temporal coding). But the key issue was whether preexposure would also impair learning to *time* the duration of the preexposed CS - and if so, whether this would depend on the temporal properties of preexposure. If

timing is impaired *whatever* the duration of the CS during preexposure, then timing

the preexposed cue should be similarly affected in both groups; but if it depends on

preexposure to the *conditioning duration* (as temporal coding predicts), then timing

should be worse in Group Same, but *not* in Group Different. To assess timing we

computed the rate of conditioned responding in each 1-s bin of the CS, and

computed the *slope* of this function. Timing would be evident as an increase in rate

of conditioned responding as US delivery approaches, resulting in positive slopes. In

contrast, if there is no timing (as is found, for example, early in training or when the

stimulus is of variable duration), response rates are steady over the course of the CS,

and slopes are close to zero (e.g. Jennings et al., 2013)[3]. As we were interested in the

effect of latent inhibition on timing, it was critical that we could demonstrate a

reliable latent inhibition effect. Although preexposure retards conditioning, it

typically does not reduce asymptotic levels of responding, meaning latent inhibition

is a transient phenomenon that dissipates with extended testing. For this reason we

restricted the number of training sessions, so that asymptotic levels of responding

had not been reached. However, orderly response slopes do not develop

immediately, but require a certain amount of training to emerge. Thus we tested the

animals at around the point at which responding was just about to asymptote, which

in the experiments that follow was between 4 to 6 training sessions.

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[3] We did not use the peak procedure to evaluate timing as latent inhibition is a transient phenomenon, and this method can require a large number of trials to generate sufficient data; latent inhibition is also context-specific, and the introduction of peak trials could produce a context change that would further attenuate the effect.

Method

Subjects:

Subjects were 16 male Lister hooded rats (Charles River UK) with a mean free-feeding weight of 330 g (range: 305-350 g). The rats were weighed daily and their daily food ration restricted such that they were gradually reduced to 85% of their free-feeding weights before the start of the experiment. They were maintained at this level throughout training, their target 85% level being adjusted weekly according to a growth curve so that their target weights increased gradually over the course of the experiment. Water was freely available in the home cages. They were maintained on a 12-hour light/dark cycle, with the lights turned on at 7am, and temperature was maintained at 21°C (±1); the humidity was 60% (±10%). There were 8 animals per group.

Apparatus:

All conditioning and testing procedures were conducted in 8 identical chambers (20 x 24 x 30 cm), each of which was situated in a ventilated, noise-attenuating shell (74 x 38 x 60 cm) (MED Associates). Each chamber was equipped with a speaker for delivering auditory stimuli, a houselight, a foodcup and two jewel lights, one situated on each side of the food cup. The houselight was not employed. A speaker, located on the right side of the back wall of the chamber on the opposite wall from the food cup, could deliver two 70-dB auditory stimuli (scale A, measured near the food hopper), a white noise and a 10-Hz click. A pellet dispenser (Model ENV-203)

delivered 45-mg Noyes (Improved Formula A) pellets into the food cup. Each head entry into the food cup was recorded by breaking an infra-red photobeam and recorded as a response. Med-PC for Windows (Tatham & Zurn, 1989), running on a PC, controlled experimental events.

Procedure

 Preexposure: All subjects received ten sessions of preexposure, each comprising 42 presentations of stimulus A (which for half the subjects in each group was the noise, and for the remainder the click). For subjects in Group *Same* presentations of A had a fixed duration of 40s on every trial. For subjects in Group *Different* the 42 presentations of A were of variable durations drawn without replacement from a uniform distribution, with a mean duration of 40s: thus each block of six trials comprised CS presentations of the following durations: 20, 28, 36, 44, 52 and 60s, and there were seven blocks per session. The intertrial interval in this and the conditioning phase comprised a fixed interval of 130s plus a variable interval drawn from an exponential distribution with a mean of 60s (i.e. 130+~60s).

 Conditioning: During this stage all subjects received 40 trials per session: 20 of these presentations were with CS A, to which they had been preexposed, and 20 to the novel B. For animals for which A had been the click, B was the noise, and vice versa. All CS presentations were of 40-s duration, and were preceded by a 40-s preCS period, which was a portion of the ITI during which responding was also recorded. Each CS presentation was immediately followed by the delivery of two food pellets, and the two trial types were intermixed in a semi-random order. There were five

sessions in this stage.

Data Treatment

Mean response rates during each type of trial were obtained by computing the total number of responses made during each CS type in each session, and during the corresponding preCS periods, and converting to responses per minute (rpm). Conditioned responding in each session was indexed by a *difference* score - the mean response rate during each type of CS after subtraction of the rate during the corresponding preCS periods. This provided a measure of the degree to which CS presentation elevated conditioned responding over background levels.

Timing: The total number of responses occurring in each successive 1-s time bin of each type of CS, pooled over all the conditioning sessions, was computed, and the rate of responding in each bin calculated for each rat. The resultant distributions were smoothed by taking an average over successive 5-sec bins (i.e. bins 1-5, 2-6 etc), to reduce the influence of transient variation in responding (e.g. Church et al., 1994; Tam et al., 2013; Matell et al., 2014). These rates were then normalized (divided by the total number of responses for that rat) to give the percentage of responses in each 1-s time bin, to ensure each animal contributed equally to the shape of the functions regardless of its response rate. Then a linear function was fitted to each normalized response function, and the slope determined from the best-fitting linear curve for each rat (linear fits provide a good characterization of response rates over CS duration: Jennings et al. 2007; cf. Kirkpatrick & Church 2000). A higher slope score, relative to a low or negative score, indicates that a greater

proportion of an individual's head-entry responses are distributed towards the end

of the CS, consistent with temporally accurate anticipation of US delivery; thus, slope

was used as an estimate of temporal control (Kirkpatrick & Church, 2000; Jennings &

Kirkpatrick 2006).

Data were analysed using analysis of variance; significant interactions were

explored with simple main effects analysis, using the pooled error term. Partial eta

squared (η_p^2) and its 95% confidence interval (CI) were given for significant effects

and interactions in the ANOVAs.

<p align="center">Results</p>

No data were collected during the preexposure stage.

Conditioning: The results of the conditioning phase are shown in Figure 1:

<p align="center">INSERT FIGURE 1 ABOUT HERE</p>

Rates of responding to the preexposed cue appeared lower than to the novel

stimulus in Group *Same*, indicating latent inhibition; this was less evident in Group

Different. ANOVA with preexposure (preexposed or novel), session and group (Same

or Different) as factors revealed a significant main effect of session $F(4, 56) = 54.33$,

$p < .001$, *MSe* = 13.89, η_p^2 = .79, CI = [.67, .84], which interacted with preexposure

$F(4, 56) = 3.01$, $p < .026$, *MSe* = 5.94, η_p^2 = .18, CI = [.00, .30]; nothing else was

significant, largest $F(1, 14) = 3.89$, $p = .069$, *MSe* = 24.29, η_p^2 = .22 for the

Preexposure x Group interaction. Further analysis of the Preexposure x Session

interaction revealed a significant effect of preexposure on session 2, $F(1, 70) = 14.43$,

$p < .001$, *MSe* = 9.61, but on no other session, largest $F(1, 70) = 1.12$, $p = .293$, *MSe* = 9.61 for session 3.

The Preexposure x Group interaction, although not significant ($p = .069$) is consistent with latent inhibition being weaker in Group *Different*, as is suggested in Figure 2; indeed after four sessions the Preexposure x Group interaction was significant, $p = .039$, and a significant latent inhibition effect was found in Group *Same*, $F(1, 14) = 7.72$, $p = .015$, *MSe* = 5.66, $\eta_p^2 = .47$, but not in Group *Different*, $F < 1$. Thus there is evidence that latent inhibition was weaker in Group *Different*, at least after four sessions.

The mean rate of preCS responding in each session was 4.36, 6.43, 4.50, 3.70 and 2.84 rpm for Group *Same*, and 6.10, 9.60, 7.33, 5.80, and 5.49 for Group *Different*. ANOVA with group and sessions as factors revealed only a significant effect of sessions, $F(4, 56) = 12.56$, $p < .001$, *MSe* = 5.66, $\eta_p^2 = .47$, CI = [.24, .58]; nothing else was significant, largest $F(1, 14) = 3.38$, $p = .09$, *MSe* = 73.44 for the main effect of group.

Timing: The smoothed response rates, averaged over all five sessions, are shown in the top panel of Figure 2. Response rates appeared lower to the preexposed than to the novel cue in Group *Same*, but this difference was not apparent in Group *Different*. This reflects the pattern seen in Figure 1 and again suggests a more marked latent inhibition effect in Group *Same* than in Group *Different*. There was also some suggestion that the difference in response rate between the start and the end of the CS was greater for the preexposed cue in Group *Same* than in the other conditions. This pattern was evident in the averaged slope data (lower panel of

Figure 2), where it may be seen that the mean slope for the preexposed CS in Group *Same* appeared higher than for the other conditions, which did not seem to differ substantially. ANOVA with CS and group as factors confirmed that there was a significant interaction between these two factors, $F(1, 14) = 6.62$, $p = .022$, $MSe = .0001$, $\eta_p^2 = .32$, CI = [.00, .58], and simple main effects tests revealed a difference between preexposed and novel CSs in Group *Same*, $F(1, 14) = 7.57$, $p = .016$, $MSe = .0001$, but not in Group *Different*, $F < 1$.

INSERT FIGURE 2 ABOUT HERE

Discussion

We anticipated equal latent inhibition in both groups; yet, although statistically this was the case after five sessions, the effect in Group *Different* was numerically weak, and after four sessions latent inhibition was actually present only in Group *Same*. There are reasons to expect such a difference. It is well established that latent inhibition is context-specific: if a stimulus is preexposed and conditioned in distinctly different contexts, then latent inhibition is attenuated, compared when the context of preexposure and conditioning is the same (Channell & Hall, 1983). If in Group *Different* the change between experiencing many stimulus durations during the preexposure phase to just one during conditioning constituted such a context change, this would have reduced the magnitude of latent inhibition observed.

But there are alternative explanations. First, as we saw above, the temporal coding hypothesis predicts greater latent inhibition when the durations of the preexposed and conditioned CSs match (Matzel et al., 1988). Although previous

studies have reported that this factor has little effect on latent inhibition (Ayres et al., 1992), they used only *fixed* duration stimuli, and so the result we observed could be related to the fact that the preexposed cue in Group *Different* was variable.

There are other ways using a variable CS could influence latent inhibition. For example, according to the Pearce-Hall model (1980), latent inhibition results from a drop in associability of stimuli that are reliably followed by no consequence. Although this model makes no assumptions about the temporal structure of a stimulus, one common associative approach is to conceptualise each CS as a set of shorter elements, corresponding to successive time bins that are activated in a fixed sequence after CS onset (e.g. Konorski, 1948; Wagner & Rescorla, 1972; cf. Pavlov, 1927; Vogel et al., 2003). In a CS that is always of the same duration, the first element will reliably predict the second, which will reliably predict the third and so on. Thus, each element is being reinforced by the next according to a continuous reinforcement schedule; hence, while the associability of the final element will fall because it reliably predicts no consequence, that of preceding elements could also fall because each reliably predicts the element that follows it. In contrast, if CS duration varies from trial to trial, each element is no longer reliably followed by the next - and this will result in the individual stimulus elements maintaining their associability, which could therefore reduce or eliminate the latent inhibition effect. An attempt to discriminate between these possibilities was the aim of Experiment 2.

The second notable finding was the effect of preexposure on timing. Contrary to our expectation, no impairment in temporal control was observed - rather, the mean slope of the preexposed CS was greater in Group *Same*. Thus, the group that showed the most robust latent inhibition effect also displayed greater temporal

control after preexposure in these animals. Experiment 2 also aimed to replicate this finding.

Experiment 2

Two groups of rats, Group *Same* and Group *Different*, were preexposed to *two* stimuli, A and a control stimulus C (see Table 1). For Group *Same*, A was preexposed at a fixed 20s duration, and C with a variable duration that was either longer or shorter but never equal to 20s. Group *Different* received the converse arrangement, A being variable and C fixed. Then both groups were conditioned to A, and also to a novel cue B, both with a fixed 20s duration. We reduced the mean CS duration from 40s to 20s to equate the total amount of stimulus exposure with that given in Experiment 1. Although this also halved the amount of preexposure given to the target stimulus A, previous work has shown that this is more than enough required to give a robust latent inhibition effect.

As all animals experienced stimuli of both variable *and* fixed durations during preexposure, this should equate the similarity of the preexposure and conditioning contexts more closely than in Experiment 1. Thus, if the apparent failure to observe robust latent inhibition in Group *Different* of Experiment 1 was due to a context change for Group *Different* but not Group *Same*, then latent inhibition should be equally strong in both groups in the present study. But if it was due to the change in the temporal nature of the to-be-conditioned stimulus between preexposure and test (cf. Matzel et al., 1988), or the ability of variable stimulus presentations to maintain associability during preexposure (cf. Pearce & Hall, 1980), then latent

inhibition should be greater in Group *Same* than in Group *Different*, just as in the previous experiment.

Method

Subjects and Apparatus:

Subjects were 16 male Lister hooded rats (Charles River UK) with a mean free-feeding weight of 352 g (range: 329-372 g). They were housed and food restricted exactly as in Experiment 1. The apparatus was identical to that of Experiment 1, except for the addition of a third auditory stimulus, a 75-db 4-kHz tone.

Procedure:

Preexposure: Subjects received ten sessions of preexposure, each comprising 42 presentations of A, and 42 of C. A and B were counterbalanced between the click and the noise, as in Experiment 1, and C was always the tone. The trials were arranged in 12-trial blocks, each comprising 6 presentations of A and 6 of C. For subjects in Group *Same* presentations of A had a fixed duration of 20s on every trial, while C's duration was drawn without replacement from one of the following durations: 10, 14, 18, 22, 26 and 30s. For Group *Different* these treatments of A and C were reversed. The intertrial interval in this and the conditioning stage comprised a fixed interval of 40s and a variable interval drawn from an exponential distribution with a mean of 40s (40+~40s).

Conditioning: The five conditioning sessions each comprised 20 trials with A

and 20 with B, intermixed in a semi-random order. Both stimuli were presented at a fixed 20-s duration, and followed by the delivery of two food pellets; the 20s period of the ITI immediately preceding each CS presentation constituted the preCS period.

Data Treatment This was identical to that in the previous experiment.

<div align="center">Results</div>

Conditioning: The results of the conditioning phase, summarised in Figure 3, suggest that latent inhibition was present in both groups; ANOVA with preexposure (preexposed or not), session and group (Same or Different) as factors revealed a significant main effect of preexposure $F(1, 14) = 7.54$, $p = .016$, $MSe = 16.84$, $\eta_p^2 = .35$, CI = [.01, .60], and also of session $F(4, 56) = 39.91$ $p < .001$, $MSe = 19.58$, $\eta_p^2 = .74$, CI = [.59, .80]; nothing else was significant, largest $F(4, 56) = 1.37$, $p = .255$, $MSe = 19.58$.

<div align="center">INSERT FIGURE 3 ABOUT HERE</div>

The mean rates of preCS responding in each sessions were 5.00, 8.63, 7.28, 6.10 and 4.86 rpm for Group *Same*, and 4.42, 11.44, 5.63, 7.76, and 4.98 for Group *Different*. ANOVA with group and sessions as factors revealed a significant main effect of sessions, $F(4, 56) = 12.45$, $p < .001$, $MSe = 11.78$, $\eta_p^2 = .47$, CI = [.24, .58]; nothing else was significant, largest $F(4, 56) = 2.15$, $p = .087$, $MSe = 11.78$ for the Session x Group interaction.

Timing: The smoothed response rates, averaged over all five sessions, are shown in the upper panel of Figure 4. Responding at the start of the preexposed stimuli appeared to be lower than to the novel cues, an effect that dissipated as the CS continued; this is consistent with the preexposed stimulus having steeper slope functions than those for the novel cues.

INSERT FIGURE 4 ABOUT HERE

A similar conclusion was suggested by the averaged slope data (Figure 4 lower panel): slopes were higher for preexposed than for novel cues in both groups. ANOVA with preexposure and group as factors revealed a significant main effect of preexposure, $F(1, 14) = 4.92$, $p = .044$, $MSe = .006$, partial $\eta^2 = .26$, CI = [.00, .54]; nothing else was significant, $Fs < 1$. Thus, the response slopes were greater following the preexposure treatment consistent with a higher level of temporal control.

Discussion

Just as in Experiment 1, Group *Same* was conditioned to a stimulus duration which they had experienced during preexposure to that CS, while Group *Different* was not. In this study, unlike in Experiment 1, robust latent inhibition - lower rates of conditioned responding to the preexposed cue - was evident in Group *Different*. The main difference was that in the present experiment *both* groups had experience of variable duration stimuli during preexposure, which aimed to equate preexposure

and conditioning contexts more closely for Group *Different* than was the case in

Experiment 1. The fact that minimising the likelihood of context-specificity in Group

Different allowed the emergence of strong latent inhibition suggests that this was

the factor reducing the size of this effect in Experiment 1, rather than because

preexposure to a variable stimulus maintains its associability (Pearce & Hall, 1980),

or because a match between CS durations in preexposure and conditioning

maximises latent inhibition (Matzel et al., 1988). That the degree of latent inhibition

observed is little affected by a mismatch in stimulus duration between preexposure

and conditioning is consistent with previous findings by Ayres et al. (1992).

The present experiment also replicated the finding from Experiment 1 that

the mean slope of the response distributions was greater for the preexposed than

for the novel cues - and this was true in both groups. This result is not consistent

with the predictions of the conditioning theories outlined above, none of which

predicted the enhancement in temporal control that such a difference seems to

reflect. In Experiment 1 this effect was evident in Group *Same* although not in Group

Different - despite the fact that both underwent stimulus preexposure. But in

Experiment 1 latent inhibition appeared markedly smaller in Group *Different*, an

effect that was supported by analysis of the data after four sessions. The suggestion

is that the enhancement in the slope of the response function might be a by-product

of latent inhibition itself.

We argued above that if learning to time depended on successful association

formation, then theories viewing latent inhibition as a retardation in association

formation (e.g., Pearce & Hall, 1980; Mackintosh, 1975; McLaren & Mackintosh,

2002; Wagner, 1981) might also predict a deficit in timing. But this analysis, although

perhaps plausible at face value, is possibly naïve. Associative theories often consider the CS as being composed, at least in part, of constituent elements that are activated in a consistent temporal sequence (Vogel et al., 2004). If early portions of the CS condition less effectively than later ones, this would result in increasing levels of conditioned responding as the US approaches, and produce the characteristic timing function. The steeper slope observed in the preexposed CS could, then, be interpreted in terms of differential latent inhibition accruing to the various portions of the CS. If more latent inhibition were suffered by initial portions of the CS than by later ones, this could selectively retard conditioning at the start of the CS, and produce the pattern of results we observed; discussion of potential mechanisms for this will be taken up below. Alternatively, the effects of latent inhibition might be less evident when levels of responding are lower - such as at the *start* of the CS, which will condition less well because the US is temporally distant. Both suggestions require us to assume that conditioning, and perhaps also latent inhibition, can occur relatively independently to earlier and later portions of the CS.

One aspect of our results could help us to evaluate this suggestion. In Group *Same* all temporal elements of the CS would have been preexposed on the same number of occasions, as the entire stimulus was present on every preexposure trial. In contrast, for subjects in Group *Different,* preexposure trials with the target stimulus were either longer or shorter than the to-be-conditioned stimulus, so that while earlier elements of the CS were present on every preexposure trial, later ones were not. If the degree of latent inhibition to a CS element is determined by the amount of preexposure it receives, then these earlier portions should have conditioned less well than the later ones, accentuating the slope of the response

function. This analysis predicts that the slope for the preexposed cue should have been greater in Group *Different* than in Group *Same*.

Although there was no evidence of this, it may be that the previous experiments were insufficiently sensitive to detect such a difference. The discrepancy in preexposure between the start and the end of the stimulus in Group *Different* was not that great - the first quarter of the CS was present on 100% of the preexposure trials, and the final quarter on approximately 67%, a difference of only 33%. Moreover, it is possible that latent inhibition produced by this amount of preexposure was close to ceiling, making differences difficult to detect. The aim of the third experiment was to replicate the effect of preexposure on timing, and at the same time enhance the chances of observing the contribution of such a mechanism.

Experiment 3

Experiment 3a

Experiment 3a was a replication of Experiment 2, but with some modifications (see Table 1). First, preexposure was altered to increase the difference in exposure to the initial and final portions of the CS. Two groups of rats, Group *Same* and Group *Different*, were both preexposed to A and C. For Group *Same* A was always of 20s duration, whereas C was 10s on half the occasions it was presented, and 30s on the remainder. Group *Different* experienced the converse arrangement. Then, as in Experiment 2, both groups were conditioned to A and a novel stimulus B,

both of which were followed by food after 20s.

For Group *Different* the first quarter of the 20-s CS was present on 100% of the preexposure trials, and the final quarter on 50%, giving a discrepancy of 50% - greater than the 33% differential in Experiment 2. In addition, the total amount of preexposure was halved from that in Experiment 2; this aimed to reduce the possibility that latent inhibition was close to ceiling, potentially masking any differences that might be present.

Method

Subjects and Apparatus:

Subjects were 16 male Lister hooded rats (Charles River UK) with a mean free-feeding weight of 296 g (range: 276-310 g). They were housed and food-restricted exactly as in Experiment 1. The apparatus was identical to that of Experiment 1.

Procedure:

Preexposure: All subjects received ten sessions of preexposure, each comprising 20 presentations of stimulus A, and 20 of stimulus C, intermixed in a semi-random order. For subjects in Group *Same* presentations of A had a fixed duration of 20s on every trial, while C's duration was 10s on half the occasions on which it was presented, and 30s on the remainder. For subjects in Group *Different* these arrangements were reversed (see Table 1). In all other respects this stage was

identical to that of the previous experiment.

Conditioning: Conditioning sessions were identical to those of the previous experiment. There were six sessions in this stage.

Data Treatment: In the conditioning stage one rat from Group *Same* was identified as an outlier, responding substantially more to the preexposed cue than to the novel cue[4], and so was omitted from all subsequent analyses. Moreover, possibly as a result of the deliberately reduced number of preexposure trials, latent inhibition was less robust in the present experiment, only being evident after six sessions; thus the data from six rather than five conditioning sessions are considered in the analyses below. In all other respects data analysis was identical to that of the previous experiment.

Results

Conditioning: The results of the conditioning phase are shown in Figure 5:

INSERT FIGURE 5 ABOUT HERE

Latent inhibition was again apparent in both groups: ANOVA with

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[4] A latent inhibition score was derived for each rat, by subtracting response rates to the preexposed cue from those to the novel cue, summed over all the conditioning sessions. This score was more than two standard deviations below the mean for this animal.

preexposure (preexposed or not), session and group (*Same* or *Different*) as factors revealed a significant effect of preexposure $F(1, 13) = 6.41$, $p = .025$, $MSe = 36.27$, η_p^2 = .33, CI = [.00, .59] and also of session, $F(5, 65) = 26.13$ $p < .001$, $MSe = 45.61$, $\eta_p^2 =$ .67, CI = [.50, .73]; nothing else was significant, largest $F(5, 65) = 1.83$, $p = .120$, MSe = 15.38.

The mean rates of preCS responding in each sessions were 9.60, 6.36, 12.98, 9.84, 7.35 and 8.65 rpm for Group S, and 10.94, 5.57, 12.10, 11.22, 11.59 and 8.87 for Group D. ANOVA with group and sessions as factors revealed only a significant effect of sessions, $F(5, 65) = 5.64$, $p < .001$, $MSe = 25.31$, $\eta_p^2 = .30$, CI = [.09, .42]; nothing else was significant, largest $F(5, 65) = 1.06$, $p = .388$, $MSe = 25.21$ for the Session x Group interaction.

Timing: The smoothed response rates, averaged over all six sessions, are shown in the upper panel of Figure 6. In Group *Same* responding was lower to the preexposed than to the novel cue at the start of the CS, but this difference dissipated over the course of the stimulus; in contrast, in Group *Different* responding appeared to be lower to the preexposed cue throughout the entire stimulus. Moreover, any differences between the two groups were in responding to the novel CS - the timing functions for the preexposed CSs were approximately superimposed. The averaged slope data are presented in the lower panel of Figure 6; slopes were again higher for the preexposed cues, evidenced by a significant effect of preexposure, $F(1, 13) =$ 5.25, $p = .039$, $MSe = .007$, $\eta_p^2 = .29$, CI = [.00, .56]; although this effect seemed clearer in Group *Same*, the Group x Preexposure interaction was not significant $F(1, 13) = 2.17$, $p = .164$, $MSe = .007$. Nothing else was significant, $F < 1$.

INSERT FIGURE 6 ABOUT HERE

Discussion

In this experiment latent inhibition was again similar in the two groups. We also replicated the effect on temporal control shown in the previous experiments: response function slopes were significantly greater for the preexposed cues in both groups of animals.

Nonetheless there was no evidence that this enhancement in slope was more pronounced in Group *Different*; if anything the effect was weaker in these animals. Thus Experiment 3b tried a more extreme strategy to facilitate observation of this effect. It may be that *any* preexposure to a portion of the CS produces enough latent inhibition to make differences in conditioning difficult to observe, and that the only way to observe a gradation in conditioning to different portions of the CS is to ensure that one portion is preexposed while the other is not. This logic motivated Experiment 3b. All subjects were preexposed to one cue, A. For Group *Same* presentations of A were either of 10- or 40-s duration, with an equal number of each. Group *Different* received the same number of preexposure trials, but all stimulus presentations were of 25-s duration, thus equating total duration of exposure in the two groups. In the conditioning phase all rats were conditioned to both A and the novel cue B, with presentations of both stimuli being followed by food after 40s.

Subjects in Group *Different* never experienced a stimulus longer than 25s

during the preexposure phase; thus, although the first quarter of A was preexposed

on 100% of trials in both groups, the end of the 40s CS was preexposed in Group

Same, but *never* in Group *Different* - making the difference in preexposure to the

first and last quarters of the conditioned duration 100%. We also increased the

amount of preexposure from the reduced level given in the previous experiment, to

maximise this differential effect. Finally, there was no preexposure to stimulus C in

the present experiment, to preclude any generalisation of latent inhibition obscuring

the results. For example, if subjects in Group *Different* had been preexposed to C

with durations of 10 and 40s (in parallel with Experiment 3a), the final portion of

stimulus C would have been preexposed, even if the final portion of A had not. If this

were to generalise more effectively to A, which had also been experienced during

the preexposure phase, than to B, which had not, this might minimise the

differential latent inhibition between the start and the end of stimulus A at test in

Group *Different*.[5]

Experiment 3b

Method

Subjects and Apparatus:

Subjects were 16 male Lister hooded rats (Charles River UK) with a mean free-

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[5] This strategy introduced the risk of there being a greater context change between preexposure and test for Group *Different*, which had never experienced a 40s stimulus before the conditioning phase, than in Group *Same*, which had. But conversely subjects in Group *Same* experienced two stimulus durations during preexposure, but only one at test - meaning that in this respect it underwent a greater context change between preexposure and test than subjects in Group *Different*, which experienced only one stimulus duration throughout. It was hoped that these effects would offset each other, and roughly equate the similarity between preexposure and test contexts in the two groups.

feeding weight of 405 g (range: 364-436 g). They were housed and food-restricted

exactly as in Experiment 1. The apparatus was identical to that of Experiment 1.

Procedure:

Preexposure: All subjects received ten sessions of preexposure, each

comprising 40 presentations of stimulus A. A and B were counterbalanced between

click and noise, as in the previous experiments. For Group *Same* presentations of A

had a fixed duration of 40s on half the trials and 10s on the remainder; these trials

were intermixed in a semi-random order. For subjects in Group *Different* all CS

presentations were of 25s duration. The intertrial interval comprised a fixed interval

of 115s and a variable interval drawn from an exponential distribution with a mean

of 60s. In all other respects this stage was identical to that in the previous

experiments.

Conditioning: Conditioning sessions were identical to those of Experiment 1.

There were four sessions in this stage.

Data Treatment This was identical to that of the previous experiments. The total

duration of preexposure of the target stimulus A was 166 minutes, considerably

more the 66 minutes of exposure given in Experiment 3a. This might be expected to

increase the levels of latent inhibition and thus allow it to be observed at an earlier

point in training - and this proved to be the case. Thus the data were analysed over

four sessions in the present experiment.

Results

Conditioning: The results of the conditioning phase are shown in Figure 7:

INSERT FIGURE 7 ABOUT HERE

Robust latent inhibition was apparent in both groups; ANOVA with

preexposure (preexposed or not), session and group (Same or Different) as factors

revealed a significant effect of session, $F(3, 42) = 47.10$, $p < .001$, $MSe = 17.45$, $\eta_p^2 =$

.77, CI = [.61, .83], preexposure $F(1, 14) = 13.03$, $p = .003$, $MSe = 14.16$, $\eta_p^2 = .48$, CI =

[.08, .69], and interaction between these two factors, $F(3, 42) = 6.88$ $p = .001$, $MSe =$

4.59, $\eta_p^2 = .33$, CI = [.08, .48]; nothing else was significant, largest $F(1, 14) = 1.14$, $p =$

.305, $MSe = 4.59$.

The mean rates of preCS responding in each session were 4.16, 8.46, 5.44,

and 4.13 rpm for Group *Same*, and 4.02, 8.10, 4.05, and 3.03 for Group *Different*.

ANOVA with group and sessions as factors revealed only a significant effect of

sessions, $F(3, 42) = 19.53$, $p < .001$, $MSe = 7.41$, $\eta_p^2 = .58$, CI = [.34, .69]; nothing else

was significant, $Fs < 1$.

Timing: The smoothed response rates, averaged over all four sessions, are shown in

the upper panel of Figure 8. As in Experiment 3a, there was no obvious sign that

conditioning at the end of the CS was greater in Group *Different* than in Group *Same*

- the difference in responding to preexposed and novel cues appeared greater in

Group *Same* than in Group *Different* across the entire course of the CS.

The slope data are shown in the lower panel of Figure 8; once again slopes were higher for the preexposed cues: ANOVA with preexposure and group as factors revealed a significant effect of preexposure, $F(1, 14) = 4.62$, $p = .049$, $MSe = .001$, $\eta_p^2 = .25$, CI = [.00, .53]; nothing else was significant, $Fs < 1$.

INSERT FIGURE 8 ABOUT HERE

Discussion

The results of Experiment 3b mirrored those of Experiment 3a: regardless of whether subjects had experienced the conditioning duration during the preexposure phase, they showed equivalent latent inhibition, and equivalent enhancement in the slope of the preexposed cue's response function. Despite the greater differential exposure to the start and end portions of the CS in Group *Different* relative to that in Group *Same*, there was no sign that this influenced the pattern of results.

We argued above that an associative analysis could explain the enhancement in temporal control after CS preexposure in a number of ways, provided it assumes that the CS can be conceptualised as comprising independent elements that occur in a fixed temporal order, and can suffer latent inhibition and undergo conditioning relatively independently. But if this were the case, one would expect that artificially manipulating the amount of exposure to different portions of the CS could produce differential amounts of latent inhibition, and hence differential levels of conditioning. We could not find any evidence for such an effect, which casts doubt

on this assumption.

General Discussion

Taken together the results from all four experiments supported the assertion that when latent inhibition was obtained, the slope of the preexposed stimulus was higher than that of the novel cue. In Experiments 2, 3 and 4 we found lower levels of responding to the preexposed than to the novel stimulus, and this was accompanied by greater values of slope for the preexposed cue. The results from Experiment 1 were slightly more complex, in that for Group *Different* there was a latent inhibition effect[6] but no difference in slope. However, numerically the latent inhibition effect seemed weak in these animals, and analysis of the data after four rather than five training sessions suggested that neither latent inhibition nor an effect of preexposure on slope was present in this group. Taken together these results are consistent with the proposal that the latent inhibition effect produced by stimulus preexposure is accompanied by an increase in temporal control.

However, one potential reservation with this conclusion relates to the inevitable confound between the slope differences we observed and response rate - in all the experiments we examined slope at a point in training at which latent inhibition was present. This raises the possibility that, because response rates were higher to the novel than to the preexposed CSs, a ceiling effect in responding to the

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[6] more precisely, there was a preexposure x sessions interaction that did not interact with group; we did not examine latent inhibition in Group Different separately.

novel stimulus could have been responsible for the shallower slopes observed to this cue. To examine this possibility we conducted six further training sessions in Experiment 3b, and examined how slopes changed in the later parts of training. The conditioning scores and timing slopes were computed as described above, but in five 2-session blocks; the resultant data are presented in Figure 9. First, it is evident from the data in the top panel that latent inhibition is most apparent in the first two training blocks; thereafter the main difference seems to be higher rates of responding in Group Same. The slopes, shown in the lower panel, do not show the same pattern, in that they do not appear to asymptote but continue increasing as training progresses. There is also an indication of higher slopes in the preexposed cues, but only in the first two training blocks. The data from the first two blocks correspond to the data that were analysed above; analysis of the data from the final three blocks is presented here. ANOVA performed on the conditioning scores, with preexposure (preexposed or not), block and group (Same or Different) as factors revealed no significant effects or interactions, largest $F(1, 14) = 3.75$, $p = .07$, $MSe = 112.28$ for the effect of group; neither the effect of block nor any interaction with this factor was significant. This confirms the suggestion that response rates were at asymptote during these final training blocks. A corresponding analysis for the slopes revealed a significant main effect of block, $F(2, 28) = 5.21$, $p = .012$, $MSe = .001$, $\eta_p^2 = .27$, CI = [.02, .47], confirming that slopes were still increasing, and this interacted with preexposure and group, $F(2, 28) = 6.52$, $p = .005$, $MSe = .001$, $\eta_p^2 = .32$, CI = [.04, .51]; nothing else was significant, $Fs < 1$. The interaction was explored further with two group x block analysis conducted on the slopes for the preexposed and nonpreexposed cues. For the preexposed cues this revealed a significant main effect

of block, $F(2, 28) = 8.44$, $p = .001$, *MSe* $< .001$, $\eta_p^2 = .38$, CI $= [.08, .56]$; nothing else

was significant, largest $F(1, 14) = 1.18$, $p = .30$, *MSe* $< .003$. A corresponding analysis

for the nonpreexposed cues revealed nothing significant, largest $F(2, 28) = 2.31$, $p =$

.12, *MSe* $< .001$. In other words, despite the fact that response rates had reached

ceiling in both preexposed and nonpreexposed cues, slopes continued to increase,

but only for the preexposed stimuli. This is not the pattern that would be predicted if

the higher levels of slope in the preexposed cues had been due to a ceiling effect in

responding to the novel CS.

INSERT FIGURE 9 ABOUT HERE

Thus the present results suggest that latent inhibition training does not

impair temporal control - but that in fact preexposure to a stimulus appeared to

enhance the ability of subjects to track the time to US delivery. Furthermore, in most

cases this was unrelated to the temporal distribution form or duration of the cue

that was preexposed - the effect was equally evident regardless of whether or not

the subjects are preexposed to the conditioning duration. Finally, we found no

evidence that the timing response function was an emergent property of differential

latent inhibition to the end of the cue - casting doubt on the suggestion that a CS

may be conceptualised as a fixed sequence of independently conditionable

elements. Even when the start of the conditioned CS had been preexposed but the

end was completely novel - which should foster greater conditioning to the end than

to the start of the CS - there was no evidence that this resulted in greater values of

slope. It should be noted that we were only able to manipulate exposure to the end

of the cue, not to its onset, so it is still logically possible that our effects stemmed

from a differential loss of associability at the start of the preexposed stimuli. This is

particularly suggested in Experiment 2, for which the smoothed response rates in

each 1-s bin of the stimuli appeared lower in the preexposed than the novel cues at

the start of training, differences which disappeared in later portions of the stimulus

(top panel of Figure 4); however the other experiments did not show such a clear

pattern in this regard (top panel of Figures 2, 6 and 8). Moreover, if differential

exposure to the start of the CS could bias the response slope function, then one

might expect manipulations in exposure to the end of the CS to have a similar effect,

and yet we found no evidence of this in Experiment 3. Nonetheless, this possibility

must remain as a possible alternative interpretation of our results.

Of course our conclusion of better temporal control relies solely on the slope

measure employed in these studies. Although a greater difference in differential

responding between the start and end of the CS seems indicative of more accurate

timing, it would be far more convincing if we could demonstrate the effect with

different measures of timing ability (for example, the peak procedure). However,

obtaining good data from the peak procedure requires a considerable amount of

training, and in the current studies this would mean training beyond the point at

which latent inhibition had dissipated. Moreover, the peak procedure entails

introducing a significant number of longer, nonreinforced trials, which could

potentially create a context change, which would also attenuate the latent inhibition

effect.

These results do not fit easily within any theoretical framework. The only

associative models of conditioning that offer an explanation of timing do so by

conceptualising stimuli in the componential manner we have described above, as (at least in part) a fixed sequence of constituent elements (e.g. Sutton & Barto, 1990; Vogel, Brandon & Wagner, 2003). In principle these theories could explain the enhancement in temporal control after preexposure in terms of differential latent inhibition in different portions of the CS. For example, Vogel et al. (2003) proposed a specific componential way of describing the CS, but the learning rules they employed are based on those of Wagner's SOP model. According to this account (e.g. Wagner, 1981; cf. McLaren & Mackintosh, 2002), latent inhibition occurs because the CS comes to be predicted by other cues, either those of the experimental context or of the stimulus itself; the stronger these associations the stronger the latent inhibition effect. Moreover, the model can also predict that such associations will form more effectively at the start of the stimulus. Wagner's SOP theory assumes that initial presentation of the CS leaves the stimulus in an active state that is highly associable, but with time it lapses into a secondary activation state in which it cannot act as a CS or a US in excitatory associations. This would predict that both the experimental context and elements of the stimulus itself will become more strongly associated with the start of the CS than the end, resulting in the start of the CS suffering more latent inhibition. But if such a mechanism were responsible for the enhancement of temporal control produced by preexposure, one should be able to produce such differential latent inhibition artificially, as we attempted in Experiments 3a and 3b. Yet these manipulations appeared to have no effect on the slope of the response function.

The alternative accounts of timing fare no better; for example, although the temporal coding hypothesis (e.g. Barnet, Arnold & Miller, 1991) can, with added

assumptions, explain latent inhibition, it regards timing as a by-product of conditioning, rather than independent of it. Thus it is hard to see how preexposure, which results in conditioning being less effective in producing the conditioned response, should have the opposite effect on timing. Finally there is also, as we noted above, a large class of time-based models that have been adapted to explain conditioning effects - but it is not clear such accounts can predict latent inhibition, let alone the effect of preexposure on timing. It seems our results present a potential theoretical challenge to theories of both conditioning and timing effects.

In summary, many of the studies that seek to investigate how conditioning and timing interact have focused on learning processes engaged during training that involves reinforcement (e.g. Kirkpatrick & Church 1998; although see e.g. Savastano & Miller, 1998). Although this focus has led to a rapid expansion in time-based theories (see Kirkpatrick, 2014 for a recent review), learning phenomena such as latent inhibition, where training occurs in the absence of reinforcement, have tended to be neglected. In this sense the results presented above are completely novel, and also challenging: theoretical models, irrespective of the tradition from which they derive, are unable to adequately account for our findings. Nevertheless, as noted by Kirkpatrick (2014), the speed at which models in the field are evolving suggests that these findings may fuel further theoretical development.

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Table 1. In all experiments Group Same were preexposed to the conditioning

duration, whereas Group Different were not. In Experiments 1, 2 & 3a Group

Different experienced durations (variable (var) in Experiments 1 and 2, fixed (fix) in

Experiment 3a) that were either longer or shorter than, but never the same as, the

fixed durations experienced by Group Same. In Experiment 3b Group Different only

experienced durations that were shorter than those experienced by Group Same.

Total exposure duration was always identical in the two groups.

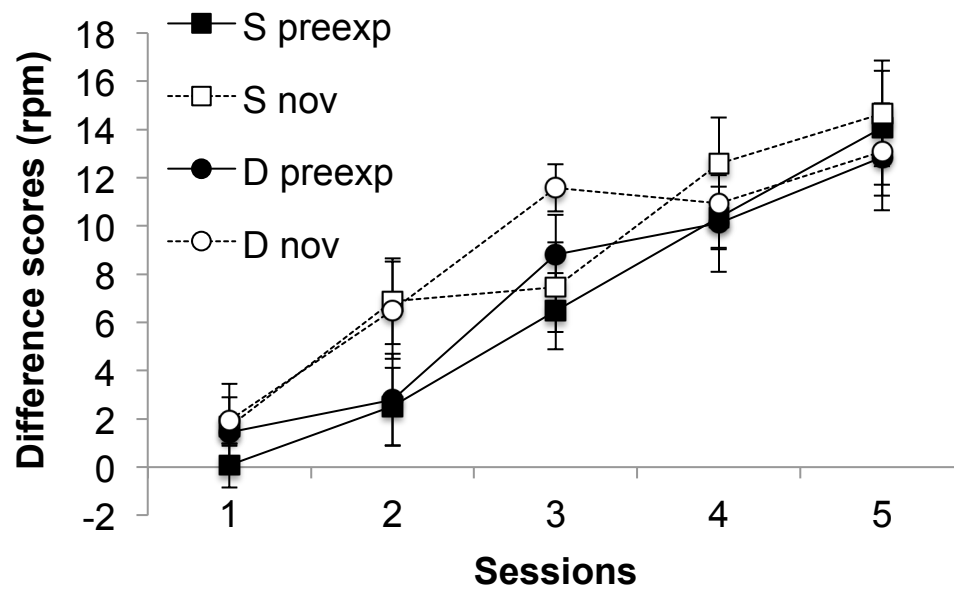| Experiment	Group	Preexposure	Conditioning
1	Same	A (fix 40)	A (fix 40) ->food B (fix 40)->food
	Different	A (var 40)	
2	Same	A (fix 20) C (var 20)	A (fix 20) ->food B (fix 20)->food
	Different	A (var 20) C (fix 20)	
3a	Same	A (20) C (10, 30)	A (20) ->food B (20)->food
	Different	A (10, 30) C (20)	
3b	Same	A (10, 40)	A (40) ->food B (40)-->food
	Different	A (25)	

Figure 1.
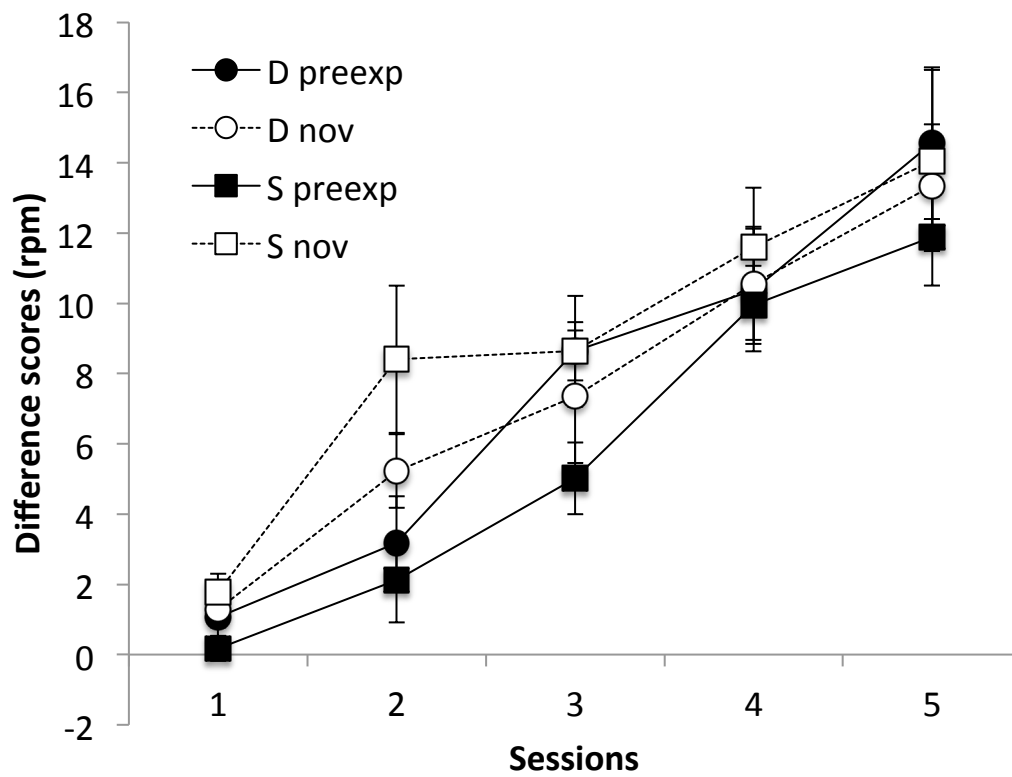
Figure 1: Group mean difference scores (response rate during CS - response rate during preCS period) for the preexposed (preexp) and novel (nov) CSs in each of the five conditioning sessions (11-15) of Experiment 1. Error bars show standard error of the mean.
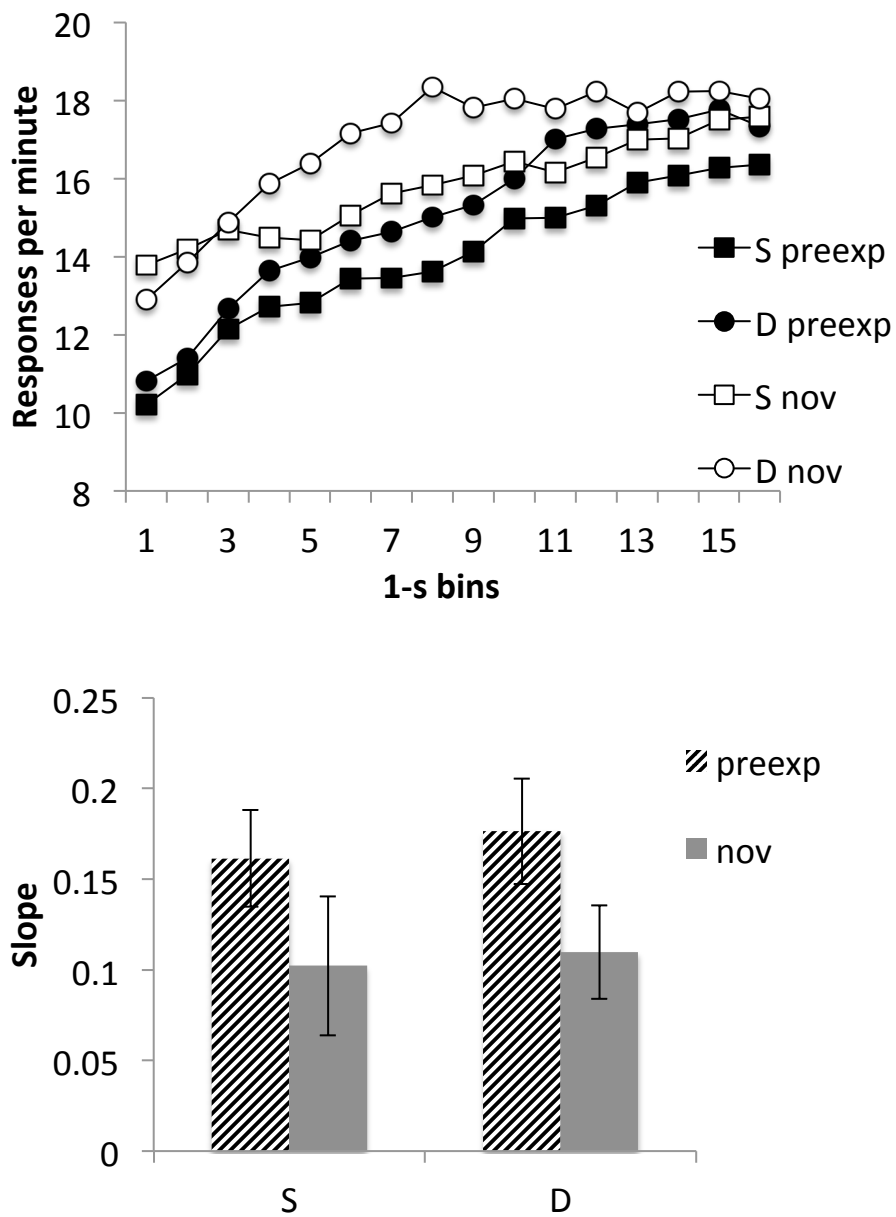
Figure 2
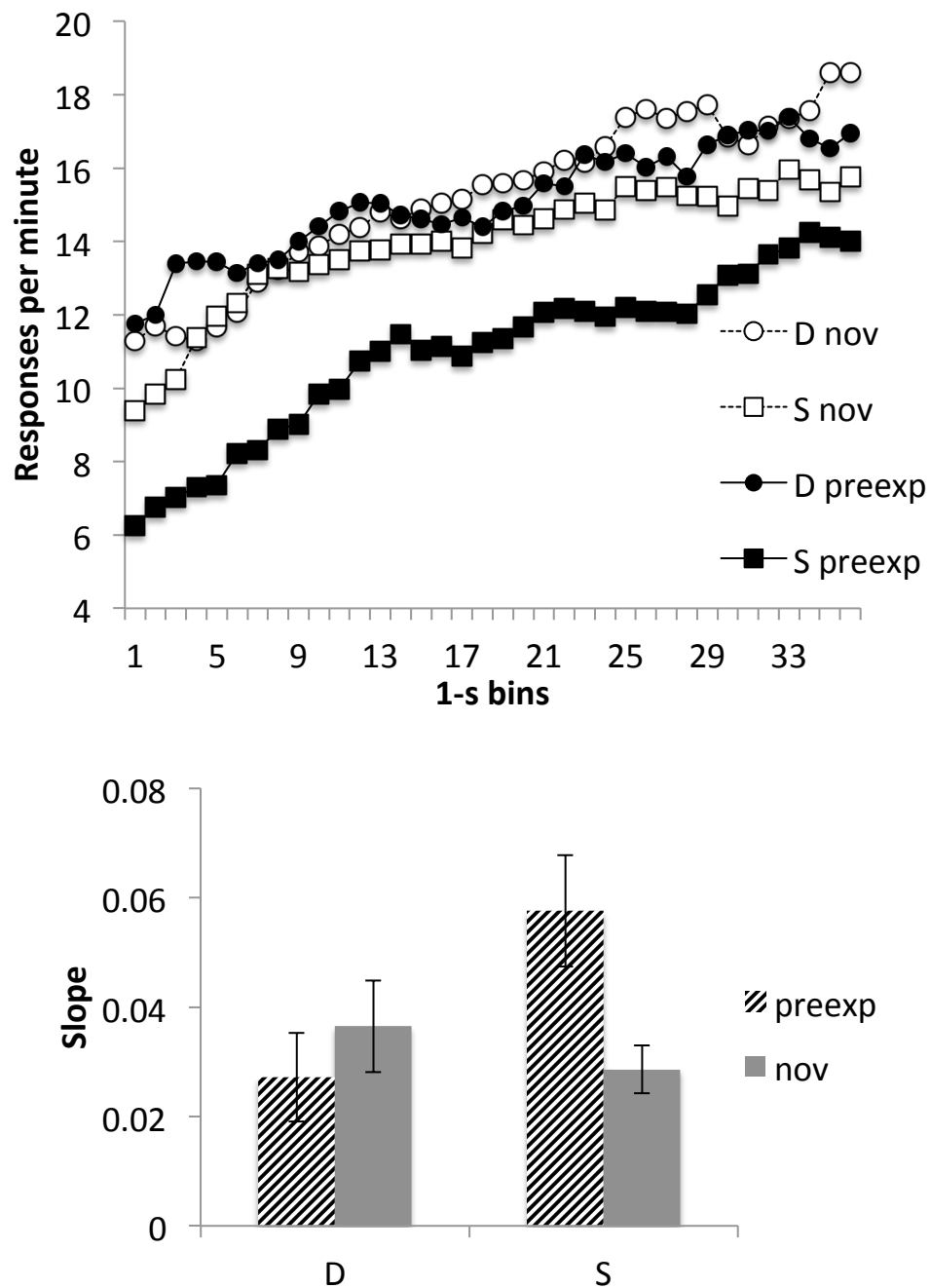
Figure 2: Group mean scores for Group Same (S) and Different (D) for the preexposed (preexp) and novel (nov) CSs from the conditioning sessions (11-15) of Experiment 1. Upper panel: Smoothed response rates in each 1-sec bin. Lower panel: Slopes. Error bars represent standard error of the mean.
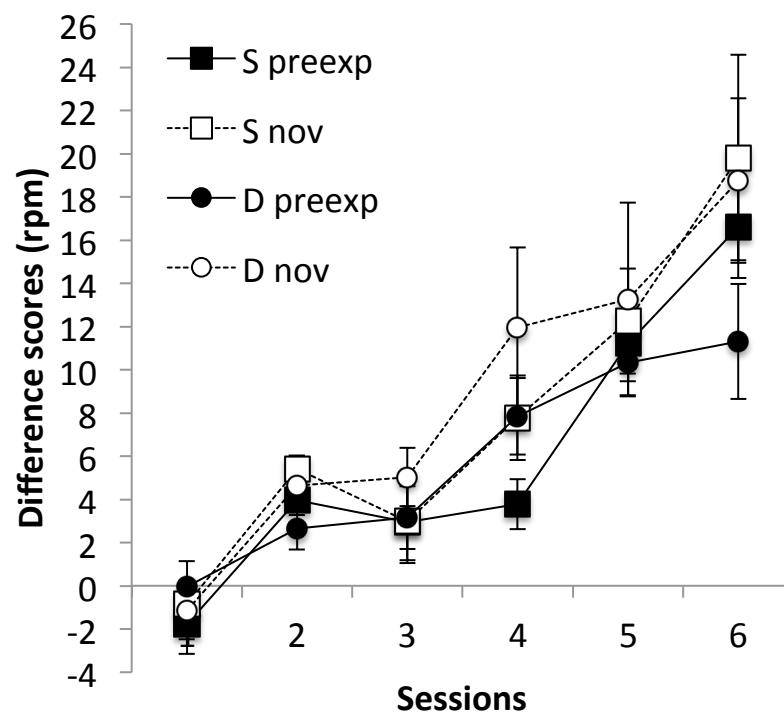
Figure 3

Figure 3: Group mean difference scores (response rate during CS - response rate during preCS period) for the preexposed (preexp) and novel (nov) CSs in each of the five conditioning sessions (11-15) of Experiment 2. Error bars represent standard error of the mean.

Figure 4

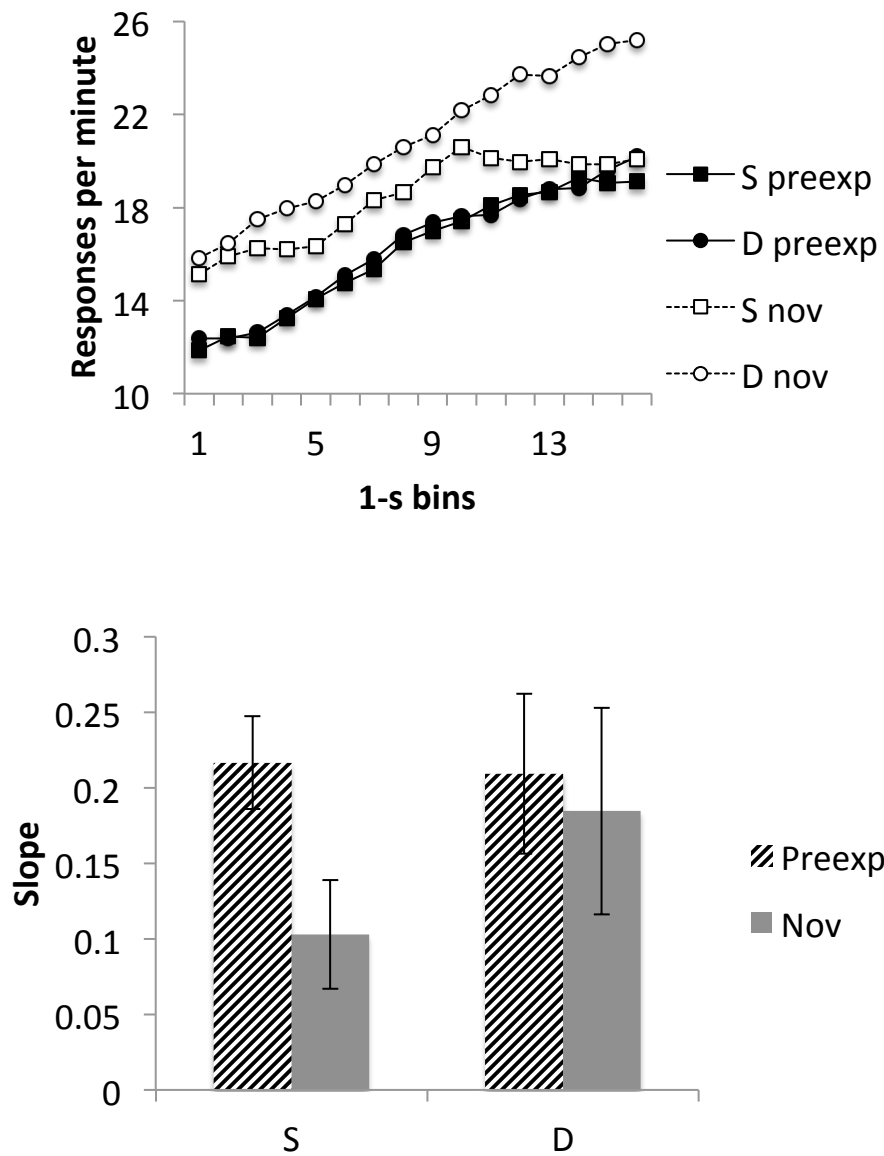Figure 4: Group mean scores for Group Same (S) and Different (D) for the preexposed (preexp) and novel (nov) CSs from the conditioning sessions (11-15) of Experiment 2. Upper panel: Smoothed response rates in each 1-sec bin of the preexposed (preexp) and novel (nov) CSs. Lower panel: Slopes. Error bars represent standard error of the mean.

Figure 5

Figure 5: Group mean difference scores (response rate during CS - response rate during preCS period) for the preexposed (preexp) and novel (nov) CSs in each of the six conditioning sessions (11-16) of Experiment 3a. Error bars represent standard error of the mean.

Figure 6

Figure 6: Group mean scores for Group Same (S) and Different (D) for the

preexposed (preexp) and novel (nov) CSs from the conditioning sessions (11-16) of

Experiment 3a. Upper panel: Smoothed response rates in each 1-sec bin. Lower

panel: Slopes. Error bars represent standard error of the mean.
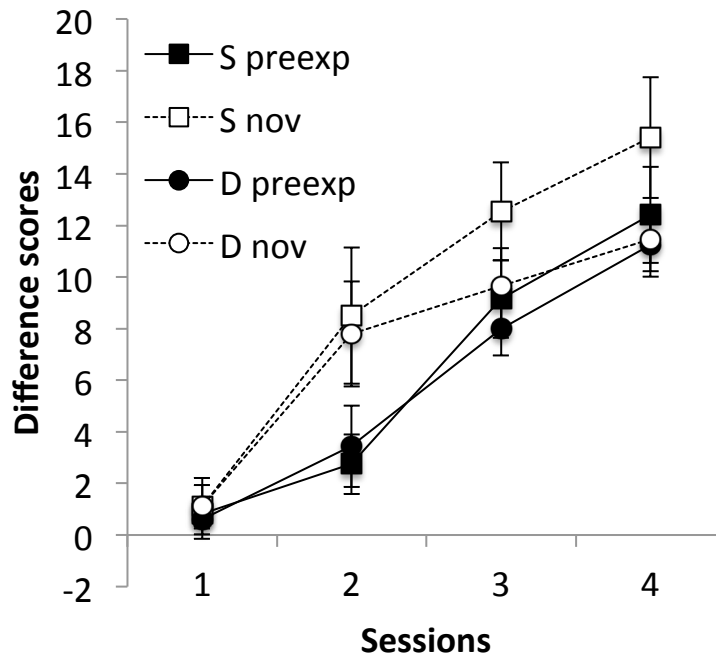
Figure 7

Figure 7: Group mean difference scores (response rate during CS - response rate during preCS period) for the preexposed (preexp) and novel (nov) CSs in each of the four conditioning sessions (11-14) of Experiment 3b. Error bars represent standard error of the mean.
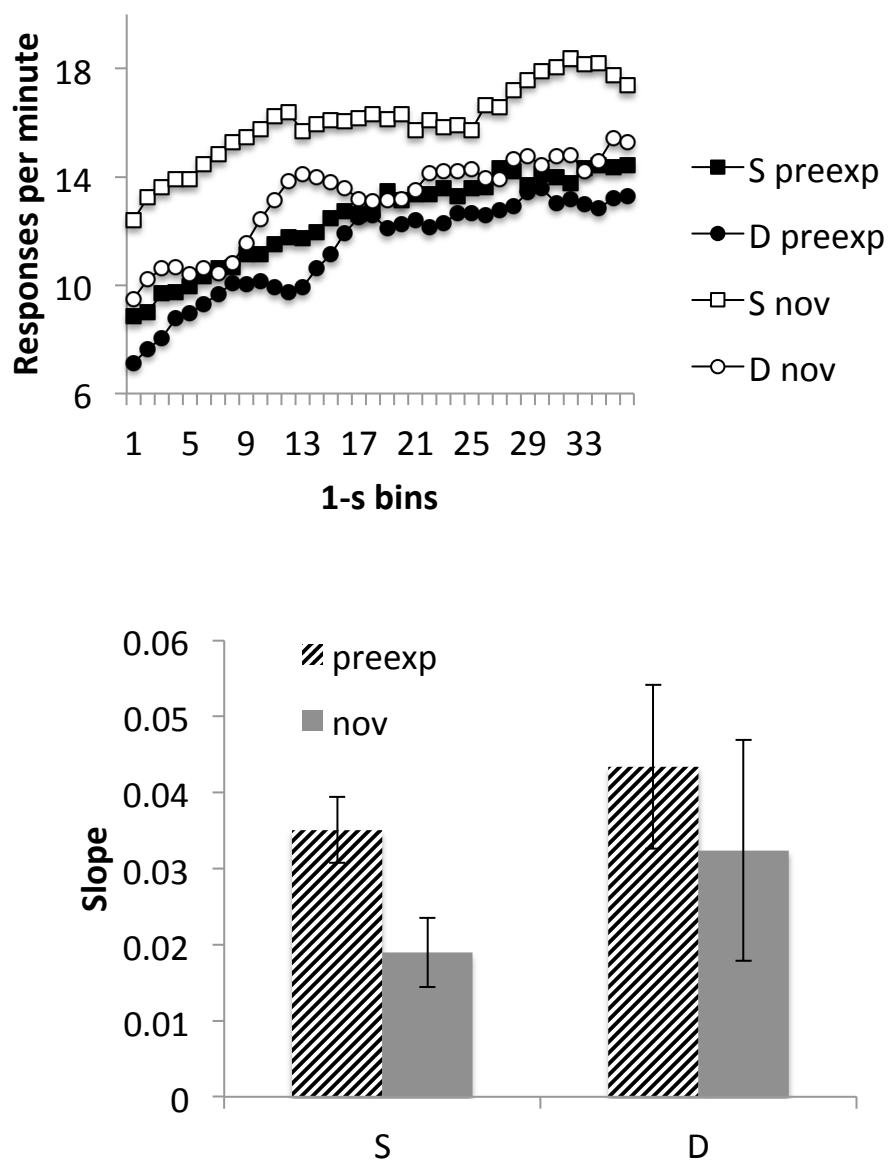
Figure 8

Figure 8: Group mean scores for Group Same (S) and Different (D) for the

preexposed (pre) and novel (nov) CSs from the conditioning sessions (11-14) of

Experiment 3b. Upper panel: Smoothed response rates in each 1-sec bin. Lower

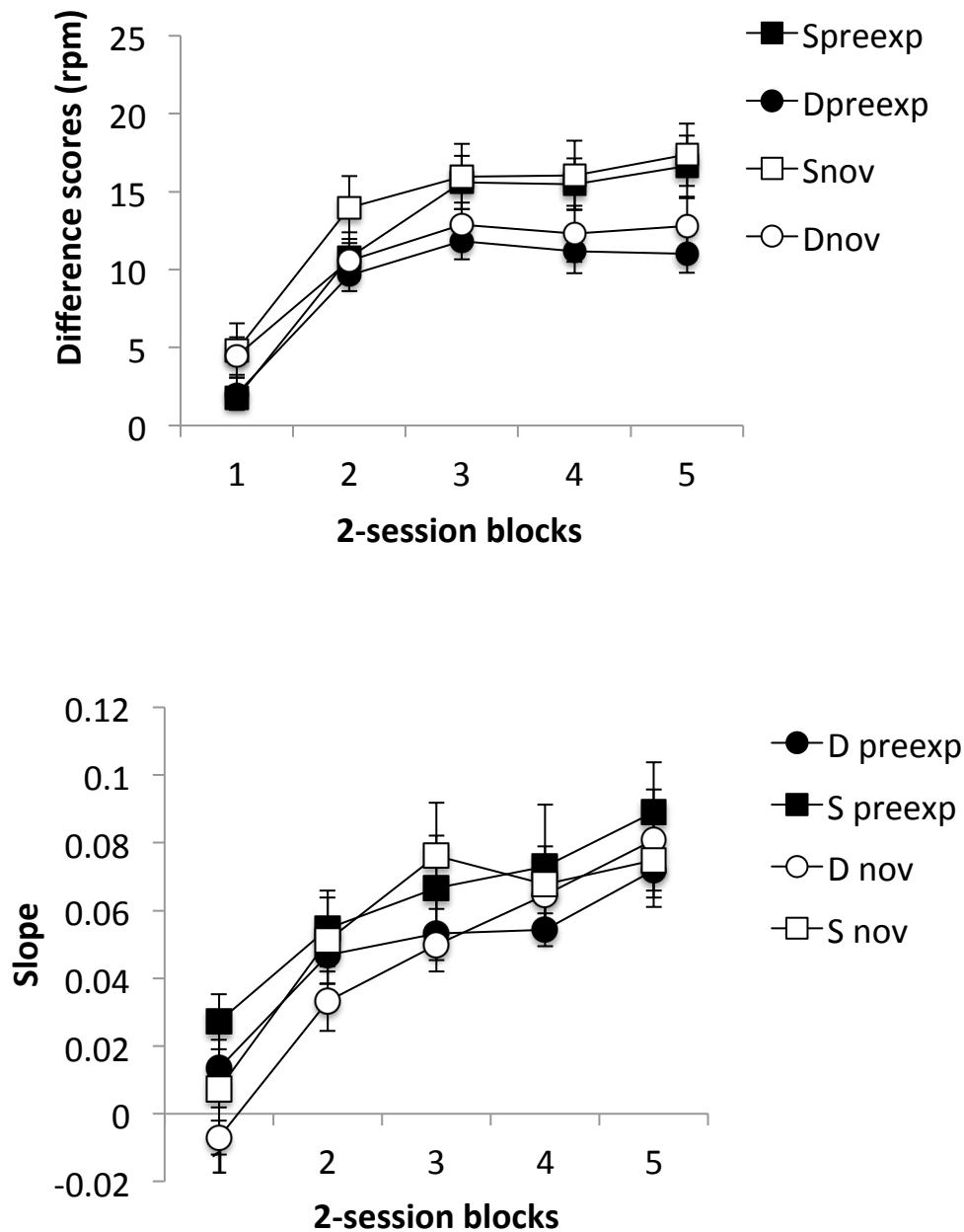panel: Slopes. Bars show standard error of the mean.

Figure 9

Figure 9: Group mean scores for Group Same (S) and Different (D) for the

preexposed (preexp) and novel (nov) CSs from the total, ten conditioning sessions

(11-20) of Experiment 3b. The data are presented in 2-session blocks. Group mean

difference scores (response rate during CS - response rate during preCS period) for

the preexposed (preexp) and novel (nov) CSs. Lower panel: Group mean slopes for

preexposed and novel cues.