Disruption of latent inhibition and perceptual learning

Rudolf Cardinal
St John's College, Cambridge.

Supervised by Dr C.H. Bennett and Professor N.J. Mackintosh, Department of Experimental Psychology, University of Cambridge, Downing Street, Cambridge CB2 3EB. Experiments performed with Claire Lloyd, Emmanuel College, Cambridge.

Abstract

The effect of a delay between conditioning and test on the magnitude of latent inhibition and perceptual learning was investigated using a taste aversion paradigm in rats. Rats were preexposed to a compound flavour BX and then conditioned to avoid another flavour AX. After a short or long delay, their consumption of BX and of the common element X was measured. Control groups were given no preexposure to BX.

In Experiment 1, all procedures were carried out in a context different from the home cages. It was found that groups given a single preexposure to BX before conditioning showed less generalization of the aversion to BX than control groups. This perceptual learning effect was not influenced by a long delay between conditioning and test. The test of the rats' consumption of X showed that preexposure to BX had resulted in latent inhibition of X relative to control groups, and this effect was also not influenced by the delay.

In Experiment 2, the same design was used except that all procedures were carried out in the familiar environment of the home cages. Again, it was found that preexposure to BX resulted in reduced generalization. There was some indication that the long delay disrupted the perceptual learning effect: the preexposed group that was given a long delay showed more generalization than that given a short delay, with no differences between the control groups. However, statistical analysis suggested that the effect of delay was independent of the effect of preexposure, so the results were not conclusive. The direct test of latent inhibition to X revealed no differences between any groups, and an explanation is offered for this apparent inconsistency.

Theories of perceptual learning and latent inhibition are discussed.

Introduction

Perceptual learning and latent inhibition

When animals are exposed to two neutral, complex stimuli, they are better able to learn a task in which they have to discriminate between them. This is a phenomenon known as perceptual learning. In the taste aversion paradigm, rats are conditioned to avoid a compound flavour stimulus, AX (a mixture of flavours A and X), by pairing it with an injection of lithium chloride to induce nausea. Normally, they will generalize this aversion to a similar flavour, BX; this is generally accepted to be because the two compound flavours share a common element, X. Mackintosh, Kaye & Bennett (1991) showed that if the rats have had prior exposure to the stimuli, generalization is reduced; in other words, the rats discriminate better between AX and BX.

The perceptual learning effect contrasts with another well-known phenomenon, that of latent inhibition. If a flavour has been presented to the rats with no unpleasant consequences, prior to conditioning to that flavour, the conditioned aversion will be reduced and the rats will consume more when tested. Latent inhibition is reliable and observed for all modalities of stimulus (Mackintosh, 1983).

Perceptual learning as latent inhibition of common elements

McLaren, Kaye & Mackintosh (1989) account for perceptual learning partly by using the idea of latent inhibition. As mentioned, generalization between two stimuli depends on the extent to which they share elements in common. When two stimuli AX and BX are preexposed, the element common to both (X) will be subject to stronger latent inhibition than those unique to one stimulus (A and B), as a result of having been sampled more often. Therefore, when AX is paired with an aversive unconditioned stimulus (US), the aversion will condition more to the unique element (A) than the common element (X), reducing generalization. While this mechanism is most relevant to the present experiments, which use a single preexposure, McLaren *et al.* (1989) proposed two additional reasons for perceptual learning. First, as elements of a complex stimulus are sampled associations will form between them, increasing the likelihood that a random sample of elements will consistently activate an accurate representation of the stimulus (‘unitization’). Second, while the associations formed between the unique and common elements of both stimuli will tend to increase generalization via the common elements, this is countered by the formation of inhibitory connections between the two sets of unique elements.

In confirmation of this theory, Mackintosh *et al.* (1991) showed that generalization between two compound flavours is dependent on the presence of elements common to both stimuli: animals did not generalize a conditioned aversion from flavour A (saline) to flavour B (sucrose), but addition of flavour X (lemon) to both the flavours caused generalization. Preexposure to both compound flavours AX and BX before conditioning to AX reduced generalization to BX on test, in part by causing differential latent inhibition of the common element (the common element is experienced more than the unique elements and is latently inhibited most). However, this mechanism is not sufficient to account for the entire perceptual learning effect, which still occurs if the common element present during preexposure is different from that present during conditioning. In line with the theory of McLaren *et al.* (1989), Mackintosh *et al.* hypothesized that inhibitory associations between the two unique elements formed during preexposure, providing an additional mechanism for perceptual learning.

Bennett, Wills, Wells & Mackintosh (1994) used a paradigm in which preexposure to just one compound flavour BX, before conditioning AX, was found to reduce generalization to BX on test. This can easily be

explained in the light of previous results: exposure to BX results in latent inhibition of the elements B and X, so an aversion conditioned to AX will condition more strongly to A than to X and there will be less generalization to BX. Their results confirmed that the reduced generalization occurs following a single preexposure, in a way that is consistent with a theory of latent inhibition of common elements. By testing consumption of X directly, they showed that the degree of generalization depends on the strength of the aversion to X, which is reduced not only by preexposure but also by a single extinction trial to X alone after conditioning. In these experiments, the effect of preexposure to B and X separately was not distinguishable from that of preexposure to the compound BX. This suggests that latent inhibition of separate elements is sufficient to reduce generalization in this paradigm, with no need to postulate latent inhibition of a "configural cue" that results from the conjoint presentation of B and X. A single preexposure to only one of the flavours does not allow inhibitory associations to form between A and B, and such associations are not required to explain the results.

Disruption of latent inhibition

Many theories of latent inhibition view it as a failure of acquisition: the association formed between the conditioned stimulus (CS) and the unconditioned stimulus (US) is weaker as a result of preexposure to the CS. However, there is an alternative explanation, known as the interference account, which views latent inhibition as a failure of retrieval (Bouton, 1991; Kraemer & Roberts, 1984). It is possible that the preexposure and conditioning experiences form separate memories: the memory of preexposure interferes with retrieval of the memory of the CS–US pairing.

Reminders of different phases of the procedure can reinstate either the conditioned response (CR) or latent inhibition, which has provided support for the interference account: it is argued that these reminders promote the retrieval of one memory or the other. A reminder of the US between conditioning and test can attenuate latent inhibition (Kasprow, Catterson, Schachtman & Miller, 1984), with the proviso that the reminder context must be similar to the training context. Similarly, a reminder of the CS can "reinstate" latent inhibition (Ackil, Carman, Bakner & Riccio, 1992), though the reminder presentation is itself insufficient to cause latent inhibition. Though this has been cited as support for the interference theory (Aguardo, Symonds & Hall, 1994), the reminder was between preexposure and conditioning: such a result cannot disprove the idea of an acquisition deficit.

Aguardo, Symonds & Hall (1994) showed that latent inhibition can be disrupted if an interval of 12 days is allowed to elapse between preexposure and conditioning to a flavoured solution, or between conditioning and test. This effect was due neither to recovery of neophobia or to an effect of delay on simple conditioning. It therefore seems that the preexposure-test interval determines the magnitude of latent inhibition. This result provides strong support for the interference account, and for the suggestion that the two memories decay at different rates; it may be that memories of motivationally relevant associations are better retained than memories of unreinforced exposure to a novel stimulus. However, it is worth noting that latent inhibition was not abolished completely over long retention intervals: perhaps an acquisition defect still has a role. Unfortunately, proving the existence of an acquisition defect is much harder than proving a retrieval defect (complete forgetting of latent inhibition could prove the absence of an acquisition defect, but incomplete forgetting cannot distinguish between a residual memory of preexposure and a failure of acquisition).

Experiment 1

Given that latent inhibition can be disrupted by a delay between conditioning and test, Experiment 1 was designed to test the theory of McLaren *et al.* (1989) that perceptual learning depends, at least in part, on latent inhibition: if latent inhibition is disrupted, this theory would predict that the perceptual learning effect would also disappear. The results of Bennett *et al.* (1994) suggest that latent inhibition is the only mechanism of perceptual learning operating when a single compound flavour is pre-exposed once, or at least the most important by far.

The flavours used here are compound stimuli consisting of sucrose–lemon and saline–lemon mixtures: these flavours were selected to stimulate different classes of gustatory receptor and therefore to be as distinct as possible. Experiments of Bennett *et al.* (1994) showed that rats can discriminate the two stimuli from each other and from lemon alone. Rats show only a marginal preference for saline–lemon over sucrose–lemon, but prefer both to lemon alone.

Four groups of rats were used. Groups LONG and SHORT were both given a single preexposure to a compound flavour BX (saline–lemon), while Groups LONGC and SHORTC served as controls and were preexposed to water. Following preexposure, all groups were conditioned to avoid another flavour, AX (sucrose–lemon), by pairing it with lithium chloride injection. A delay of 12 days was then imposed for Groups LONG and LONGC, while Groups SHORT and SHORTC only experienced a 1-day delay. Consumption of BX and X was then tested. The results of Bennett *et al.* (1994) predict that Groups LONGC and SHORTC will generalize their aversion to BX, drinking little, and will avoid X on test. Group SHORT, being preexposed, should exhibit latent inhibition of X, and a perceptual learning effect in that generalization to BX will be reduced. If the findings of Aguardo *et al.* (1994) are replicated, we expect to find lower consumption of X by Group LONG than Group SHORT, reflecting a disruption of latent inhibition. If latent inhibition is disrupted and the theory of McLaren *et al.* (1989) is correct, we expect to find increased generalization to BX in Group LONG (reduced consumption of BX) compared to Group SHORT, reflecting a concomitant disruption of perceptual learning.

Table 1: Design of Experiment 1

Group:	Preexposure to:	Conditioned to:	Delay period:	Test:
LONG	BX	AX+	12 days	BX, X
LONGC	Water	AX+	12 days	BX, X
SHORT	BX	AX+	1 day	BX, X
SHORTC	Water	AX+	1 day	BX, X

Subjects and apparatus

The subjects were 32 experimentally naive male Lister hooded rats weighing between 315g and 420g before conditioning. They were housed in pairs under a natural light–dark cycle and were maintained on a 23h water deprivation schedule with free access to food. All procedures and tests were carried out in a second room fitted with eight plastic drinking cages, 30cm(L)×14cm(W)×11cm(H), with wire mesh ceilings. Fluids were administered through the front of each cage from a 50ml measuring cylinder fitted with the same rubber stopper and stainless steel drinking spout with ball bearing as used in the home cages.

Procedure

The design used is shown in Table 1. The rats were divided into four groups of 8 ("LONG", "LONGC", "SHORT" and "SHORTC"). Three flavour stimuli were chosen: 2% sucrose (stimulus A), 0.9% saline (stimulus B) and 2% lemon (by volume Sainsbury's Pure Lemon Juice) (stimulus X). In the following account, "AX" denotes a combination of stimuli A and X, and so forth.

The experimental schedules were aligned so that all groups were tested on the same days (see *Appendix 1*, in which W denotes water), i.e. Groups SHORT and SHORTC began the experiment 11 days later than the other two groups.

Acclimation. All rats began the experiment with 20-min access to water in the drinking cages; on the subsequent two days they had 10-min of access to water in the drinking cages.

Preexposure. On the next day, animals in Groups LONG and SHORT received 8ml saline–lemon (BX), while animals in Groups LONGC and SHORTC received 8ml water. All animals were allowed sufficient time to drink it all; most rats accomplished this in approximately 10 minutes. The rats were then weighed.

Conditioning. On the conditioning day, the rats were all given 8ml sucrose–lemon (AX) which was followed immediately by an intraperitoneal injection of isotonic lithium chloride (0.15M, 10 ml/kg).

Delay. Groups LONG and LONGC were then given 12 days in their home cages where they were allowed access to water daily; Groups SHORT and SHORTC were given 1 day before testing. This ensures that at least 48h has elapsed between conditioning and test, for LiCl injection causes rats to consume less fluid for several hours and there is sometimes a compensatory increase on the subsequent day (Aguardo *et al.*, 1994).

Reacclimation. All groups were then given one day during which they received 10-min access to water in their drinking cages. This allows recovery of fluid consumption in the experimental apparatus and extinction of any conditioning to that context. It also ensures that the test environment is equally familiar to all groups.

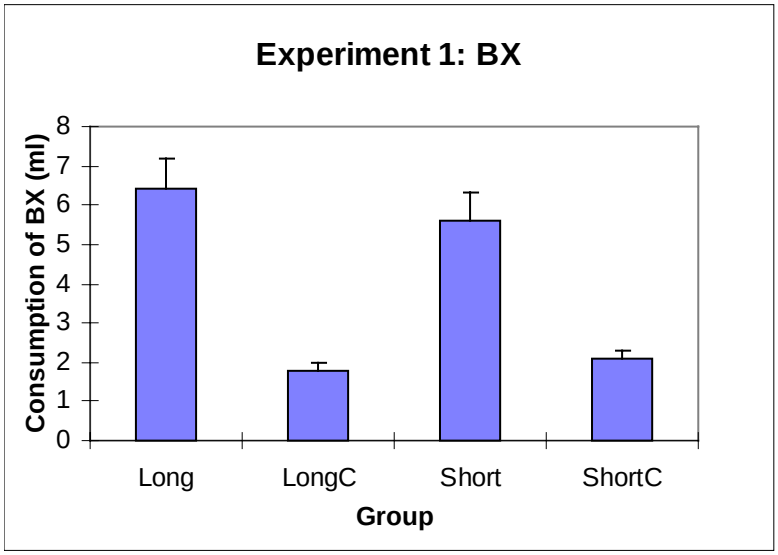
Test. On the following day all animals were tested in the drinking cages for consumption of saline–lemon (BX), and on the next two days they were tested for consumption of lemon (X) alone. The tests lasted 10 min. Note that testing of the rats' consumption of BX constitutes an extinction procedure which might be expected to reduce the rats' aversion to X on the following day.

Results

Throughout the statistical analysis, all tests were two-tailed and used a significance level of $p < 0.05$ unless otherwise stated. The data for one animal in Group SHORTC were omitted from the following analysis due to spillage of fluid during a test trial.

The water consumption records for the sessions prior to conditioning were checked to ensure that all groups drank equivalent amounts: a mixed design analysis of variance (ANOVA), using recording day as a repeated measures factor, revealed no difference in water consumption between groups ($F < 1$).

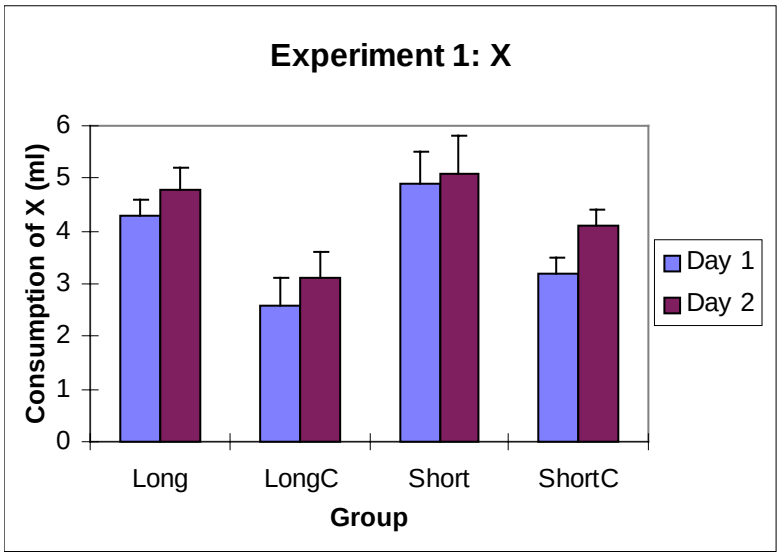
Figure 1



Mean consumption of BX on test is shown in Figure 1 (in all figures, error bars show standard error of the mean). An ANOVA on the consumption data, using preexposure and delay as factors, showed that the preexposed

animals (Groups LONG and SHORT) drank more than those in the two control groups, $F_{1,27} = 48.55$, but there was no effect of delay or any interaction (Fs < 1).

Figure 2



Mean consumption of X on test is shown in Figure 2. A mixed design ANOVA, with preexposure and delay as unrelated factors and test day as a repeated measures factor, again showed an effect of preexposure where animals in Groups LONG and SHORT drank more than animals in the two control groups, $F_{1,27} = 11.24$. There was also an effect of test trial: a general increase in consumption on the second test day, $F_{1,27} = 10.24$. This indicates extinction to X. There was no effect of delay ($F_{1,27} = 2.17$) and there were no interactions (Fs < 1.18).

Discussion

Bennett *et al.* (1994) showed that habituation of neophobia is not solely responsible for increased consumption of BX in preexposed animals. They demonstrated that when AX and lithium chloride injection were unpaired, rats preexposed to BX drank only very slightly more than controls, and this difference extinguished rapidly. Also, preexposure to BX did not affect the strength of the aversion to AX. Therefore, our results provide evidence of the expected perceptual learning effect, in that preexposure to BX reduces generalization. Furthermore, the degree of generalization is consistent with the strength of the aversion to X, which reflects latent inhibition in the preexposed groups. This supports McLaren *et al.*'s (1989) theory of perceptual learning. However, there were no differences between Groups LONG and SHORT, so we failed to find any evidence of disruption of latent inhibition or perceptual learning with a 12-day interval between conditioning and test.

Experiment 2

Although we found support in Experiment 1 for McLaren *et al.*'s (1989) theory, we did not see the effect reported by Aguardo *et al.* (1994). As we were attempting to disrupt latent inhibition in order to see if perceptual learning is disrupted too, we repeated Experiment 1 using conditions more similar to those used by Aguardo *et al.*; namely, with all procedures carried out in the rats' home cages.

Subjects and apparatus

The subjects were 32 experimentally naive male Lister hooded rats weighing between 255g and 355g before conditioning. This time, the rats were housed singly. Otherwise, all details were as for Experiment 1.

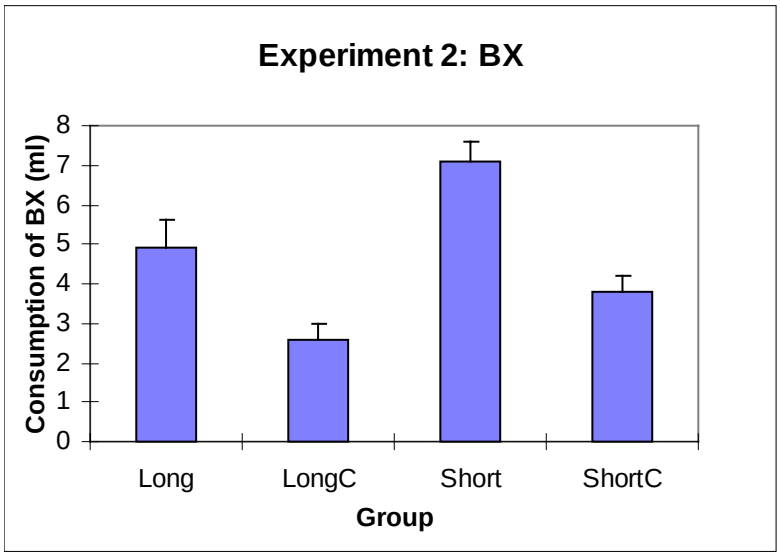
Procedure

As in Experiment 1, but all procedures took place in the animals' home cages.

Results

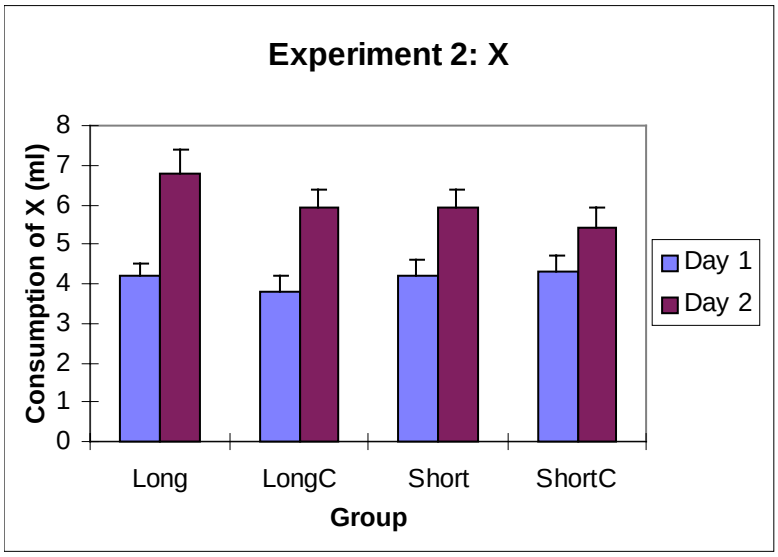
The data for one animal from Group LONG were omitted from the analysis, as it consumed a great deal more than all others in its group. Its datum of 2.0ml had a standard residual of 3.66 and was thus considered an outlier; all three results from this rat were discarded from further analysis. (This did in fact alter the effect of delay on BX from insignificance at the $p = 0.05$ level to significance at the $p = 0.01$ level, indicating the undue influence of this rat on the results as a whole.) As before, the water consumption records prior to conditioning were checked for constancy, and there was no difference in water consumption between groups ($F_{3,27} = 1.85$).

Figure 3



Mean consumption of BX is shown in Figure 3. An ANOVA on the consumption data, using preexposure and delay as factors, revealed a significant effect of both preexposure, $F_{1,27} = 35.95$, and delay, $F_{1,27} = 12.49$, on consumption of BX, with no interaction between preexposure and delay ($F_{1,27} = 1.11$). While it is not strictly appropriate to examine simple effects in this situation, they can be masked by a non-significant interaction. Analysis of simple effects showed that Group LONG drank less than Group SHORT, while there was no significant difference between Groups LONGC and SHORTC. This is weak evidence for a disruption of perceptual learning. (In addition, of course, the main effect of preexposure meant that Group LONG drank more then Group LONGC, and Group SHORT more than Group SHORTC.)

Figure 4



Mean consumption of X is shown in Figure 4. A mixed design ANOVA, using preexposure and delay as unrelated factors and test day as a repeated measures factor, revealed a significant effect of test day, $F_{1,27} = 171.92$, indicating extinction, and an interaction between delay and test day, $F_{1,27} = 10.73$. There was no effect of either preexposure or delay (Fs < 1), no interaction between test day and preexposure ($F_{1,27} = 3.38$) and no three-way interaction ($F < 1$). The interaction between delay and test day suggests that extinction – the difference between the two test days – was more pronounced in the delay condition; this might result if conditioning itself was weakened over the delay period. However, that conclusion is not supported by the BX data.

Discussion

The results again exhibit a perceptual learning effect, in that preexposure to BX reduces generalization. (Once more, this result depends on the control findings of Bennett *et al.* [1994] described above.) While there is some evidence for a disruption of perceptual learning, in that there is a difference between Groups LONG and SHORT in consumption of BX on test with no difference between Groups SHORTC and LONGC, the ANOVA suggests that there is a general effect of delay that decreases consumption, regardless of whether the group was preexposed (it might be that the conditioned aversion itself strengthened during the delay, but the lack of a significant difference between the two control groups prevents us from concluding that). To summarize, we find no conclusive evidence of forgetting of perceptual learning with a 12-day interval. However, we speculate that we are seeing the early stages of forgetting, where Group LONG's consumption is decreasing but has not reached statistical significance.

In this experiment, the differences in consumption of BX were *not* reflected by similar differences in X. As extinction to X was observed without a difference between groups emerging, we cannot attribute the failure to observe a difference on day 1 of the X test to a floor effect (the rats all drinking some 'minimum' value). The data would support the suggestion that consumption of BX was independent of aversion to X, which would imply that a mechanism other than latent inhibition of common elements underlies reduced generalization when all procedures take place in the rats' home cages. However, there is another difference from Experiment 1: the magnitude of extinction to X is noticeably greater (might the conditioned aversion itself be slightly weaker in the familiar context?). This raises the possibility that the rats' response to X was greatly affected by the previous day's extinction trial to BX. In this situation it would be unwise to use the X data to support any conclusion.

General discussion and conclusions

To summarize, in Experiment 1 we demonstrated that preexposure to a compound stimulus BX reduces generalization to it of an aversion conditioned to AX. The direct test of latent inhibition to X supported the theory of McLaren *et al.* (1989) that perceptual learning depends in part on latent inhibition of common elements. No disruption of latent inhibition or perceptual learning was seen. In Experiment 2, we again observed perceptual learning, and speculated that we might be seeing some disruption as a result of the delay between conditioning and test, though the results were not conclusive. The direct test of latent inhibition was not informative for reasons discussed above.

As we did not see a clear disruption of latent inhibition in either experiment, we could not test the prediction that perceptual learning is forgotten at the same time. However, our results do provide support for a theory of perceptual learning based on latent inhibition of common elements, and they pose the question: why was latent inhibition not forgotten?

The experiments of Aguardo *et al.* (1994) differed from our Experiment 1 in three main ways: (1) they used the same stimulus, saccharin, as the preexposure stimulus and the CS; (2) they preexposed their animals on three days, where we used a single preexposure; (3) in their experiment, all procedures took place in the home cage.

Effects of stimulus on latent inhibition

Might the use of a preexposure stimulus that is different from the CS improve retention of latent inhibition? It is known that preexposure to a compound stimulus, only one element of which is to be conditioned, attenuates latent inhibition. Reed (1995), who used visual stimuli and an appetitive conditioning paradigm, showed that preexposure to BX followed by conditioning to X produced less latent inhibition than preexposure to X did. It might be that our use of BX produced less latent inhibition of X than occurred in the experiments of Aguardo *et al.* (1994). However, these studies have not addressed the question of the resilience of the memory of preexposure, and it would appear counter-intuitive that a weaker latent inhibition effect would last longer.

Similarly, Aguardo *et al.* (1994) gave their subjects more experience of the stimulus before conditioning. But this should cause stronger latent inhibition, and again it appears unlikely that stronger latent inhibition would be more easily disrupted by a delay.

It is also possible that lemon is simply more salient than saccharin, and so all experiences of it are remembered better. A stronger memory of the preexposure phase for lemon, compared to saccharin, might explain why a delay disrupts the memory of saccharin preexposure more easily than that of lemon.

Effects of context on latent inhibition

It is known that a change in context between preexposure and conditioning can attenuate latent inhibition. However, Hall & Channell (1986) found that latent inhibition transfers across contexts when the context change consists of a move from one home cage to another between preexposure and conditioning. Other contextual changes (experience of an irrelevant flavour during the exposure phase, variation in light and background noise levels) also failed to produce an effect. Context specificity of latent inhibition was found only when subjects were given daily sessions in two experimental contexts (one for preexposure, the other for conditioning), these being very different from each other and from the home cage. This pattern of results suggests that latent inhibition is context-specific unless the context itself has been preexposed, in which case it is context-independent.

However, a simple account of context specificity cannot explain a difference between our results and those of Aguardo *et al*. (1994). Firstly, all groups here had equal experience with the experimental context: we cannot appeal to differential associability of the context. Secondly, preexposure, conditioning and test all took place in the same context. If there is an effect of context, it must be that the use of an experimental context here in some way 'strengthens' the memory of preexposure. Two explanations suggest themselves. (1) It is possible that there is an arousal effect. When rats are moved to an experimental context, they are handled, carried in a cage with many other rats and placed in a relatively strange context; they are likely to be in a state of high arousal compared with rats that remain in their home cages. There is a good deal of evidence that arousal can improve memory formation (e.g. reviewed in McGaugh, 1991). (2) In the experimental context, contextual cues are less familiar; one would assume that they are more associable as a result. The extra association of CS–context might make the memory of preexposure more durable, or the context could act as a extra retrieval cue for the memory of preexposure.

Is this plausible? Certainly contextual control of a conditioned aversion is less effective when the contextual stimuli are familiar (Archer, Sjödén & Nilsson, 1985). Wagner (1976) suggested that the formation of a CS–context association leads to a reduction in the associability of the CS when conditioning occurs in that context, but this returns us to the theory of latent inhibition as an acquisition defect.

Grahame, Barnet, Gunther & Miller (1994) view latent inhibition as a performance deficit resulting from CS–context associations. They restate the comparator hypothesis of Miller & Matzel (1988), as follows. Three associative links form during conditioning: (A) CS–US; (B) CS–context; (C) context–US. During testing, the CS evokes representations of the US by two routes, firstly via link A and secondly via links B and C. If the A response is relatively large, excitatory responding is expected; if the B/C links predominate, inhibitory responding is expected. This hypothesis can explain several features of classical conditioning, including latent inhibition. If preexposure to the CS strengthens link B (CS–context), responding to the CS following conditioning would be reduced but acquisition of the CS–US association would not. This captures the essence of the interference account of latent inhibition (Bouton, 1991; Kraemer & Roberts, 1984). In support of this model, Grahame *et al.* found that context extinction following CS–US pairings attenuated latent inhibition; this effect was specific to the preexposure context. Most strikingly, following preexposure to the CS in the training context, latent inhibition was reduced by further exposure to the CS outside the training context.

This theory is easily related to our situation. In our Experiment 1, the experimental context might have high associability; thus, during preexposure, strong CS–context associations were formed. In Grahame *et al.*'s (1994) scheme, this would tend to increase latent inhibition and so, perhaps, to reduce forgetting of latent inhibition.

This hypothesis makes two obvious predictions. Firstly, the magnitude of latent inhibition should be greater if preexposure and conditioning occur in novel rather than familiar contexts. Secondly, latent inhibition is more likely to be forgotten if preexposure and training take place in familiar contexts. Our Experiment 2 tested this second prediction indirectly, but it was not clear whether latent inhibition was disrupted.

Failure to observe disruption of latent inhibition does not discredit the theories of latent inhibition discussed here. It may be that latent inhibition would have been forgotten with a longer delay, though Aguardo *et al.* (1994) found forgetting with a 12-day delay, as here. Why did our rats not forget? If the arousal theory is correct, it may be that the procedure of weighing caused arousal, strengthening the memory of preexposure (Aguardo *et al.* weighed before any procedures, while we weighed at the time of preexposure). If the strength of latent inhibition

depends on contextual novelty, it may be that the home cages were not sufficiently familiar for our rats in Experiment 2 (they had been transferred from other home cages a few days before the experiment began).

In order to confirm the importance of context we would wish to replicate the experiments described here, running both context conditions simultaneously, weighing at the start of all procedures and ensuring all rats were very familiar with their home cages. It might also be advisable to increase the delay period slightly in order to ensure that any difference between the context conditions is clearly visible. If Grahame *et al.*'s (1994) theory of latent inhibition is correct, the magnitude of latent inhibition might also be expected to be smaller when all procedures take place in home cages. Should conditions be found where latent inhibition is reliably forgotten, the rats should be tested for consumption of X and BX in counterbalanced fashion to eliminate the problem caused by the BX trial resulting in extinction to X. This would enable verification of the prediction McLaren *et al.*'s (1989) theory makes that perceptual learning is forgotten at the same time as latent inhibition.

Rudolf Cardinal,
24 April 1996.
[4800 words]

References

Ackil, J.K., Carman, H.M., Bakner, L. & Riccio, D.C. (1992). Reinstatement of latent inhibition following a reminder treatment in a conditioned taste aversion paradigm. *Behavioural & Neural Biology* **58**: 232–235.

Aguado, L., Symonds, M. & Hall, G. (1994). Interval between preexposure and test determines the magnitude of latent inhibition: Implications for an interference account. *Animal Learning & Behaviour* **22**: 188–194.

Archer, T., Sjödén, P-O. & Nilsson, L-G. (1985). Contextual control of taste-aversion conditioning and extinction. In Balsam, P.D. & Tomie, A. (Eds.), *Context and Learning*.

Bouton, M.E. (1991). Context and retrieval in extinction and in other examples of interference in simple associative learning. In Dachowski & Flaherty (Eds.), *Current topics in animal learning: Brain, emotion and cognition*. Hillsdale, NJ: Erlbaum.

Bennett, C.H., Wills, S.J., Wells, J.O. & Mackintosh, N.J. (1994). Reduced generalization following preexposure: latent inhibition of common elements or a difference in familiarity? *J. Exp. Psych.: Animal Behav. Proc.* **20**: 232–239.

Grahame, N.J., Barnet, R.C., Gunther, L.M. & Miller, R.R. (1994). Latent inhibition as a performance deficit resulting from CS–context associations. *Animal Learning and Behavior* **22**: 395–408.

Hall, G. & Channell, S. (1986). Context specificity of latent inhibition in taste aversion learning. *Quarterly J. Exp. Psych.* **38B**: 121–139.

Kasprow, W.J., Catterson, D., Schachtman, T.R. & Miller, R.R. (1984). Attenuation of latent inhibition by postacquisition reminder. *Quarterly J. Exp. Psych.* **36B**: 53–63.

Kraemer, P.J. & Roberts, W.A. (1984). The influence of flavor preexposure and test interval on conditioned taste aversions in the rat. *Learning & Motivation* **19**:259–278.

Mackintosh, N.J. (1983). *Conditioning and Associative Learning*, Oxford University Press.

Mackintosh, N.J., Kaye, H. & Bennett, C.H. (1991). Perceptual learning in flavour aversion conditioning. *Quarterly J. Exp. Psych.* **43B**: 297–322.

McGaugh, J.L. (1991). Neuromodulation and the storage of information: involvement of the amygdaloid complex. In Lister & Weingartner (Eds.), *Perspectives on Cognitive Neuroscience*.

McLaren, I.P.L., Kaye, H. & Mackintosh, N.J. (1989). An associative theory of the representation of stimuli: applications to perceptual learning and latent inhibition. In Morris, R.G.M. (ed.) *Parallel distributed processing: Implications for psychology and neurobiology* (pp. 102–130).

Miller, R.R. & Matzel, L.D. (1988). The comparator hypothesis: A response rule for the expression of associations. In Bower, G.H. (Ed.), *The psychology of learning and motivation* (Vol. 22, pp. 51–92). Orlando, FL: Academic Press.

Reed, P. (1995). Enhanced latent inhibition following compound preexposure. *Quarterly J. Exp. Psych.* **48B**: 32–45.

Wagner, A.R. (1976). Priming in STM: An information processing mechanism for self-generated or retrieval-generated depression in performance. In Tighe, T.J. & Leaton, R.N. (Eds.), *Habituation: Perspectives from child development, animal behavior, and neurophysiology* (pp. 95–128). Hillsdale, NJ: Erlbaum.

Consumption data: mean ± SEM

		Long	(ml)	LongC	(ml)	Short	(ml)	ShortC*	(ml)
16 Jan	Day 1	W (*20min*)		W *20min*		home		home	
17	Day 2	W (*10min*)	8.5 ± 0.4	W (*10min*)	7.8 ± 0.6	home		home	
18	Day 3	W (*10min*)	8.3 ± 0.5	W (*10min*)	7.6 ± 0.4	home		home	
19	Day 4	**BX**	*8ml*	W	*8ml*	home		home	
20 (Sa)	Day 5	AX+	*8ml*	AX+	*8ml*	home		home	
21 (Su)	Day 6	delay 1		delay 1		home		home	
22	Day 7	delay 2		delay 2		home		home	
23	Day 8	delay 3		delay 3		home		home	
24	Day 9	delay 4		delay 4		home		home	
25	Day 10	delay 5		delay 5		home		home	
26	Day 11	delay 6		delay 6		home		home	
27 (Sa)	Day 12	delay 7		delay 7		W (*20min*)		W (*20min*)	
28 (Su)	Day 13	delay 8		delay 8		W (*10min*)	8.5 ± 0.4	W (*10min*)	7.9 ± 0.6
29	Day 14	delay 9		delay 9		W (*10min*)	8.3 ± 0.7	W (*10min*)	8.6 ± 0.6
30	Day 15	delay 10		delay 10		**BX**	*8ml*	W	*8ml*
31	Day 16	delay 11		delay 11		AX+	*8ml*	AX+	*8ml*
1 Feb	Day 17	delay 12		delay 12		delay 1		delay 1	
2	Day 18	W (*10min*)	9.2 ± 0.3	W (*10min*)	7.9 ± 0.6	W (*10min*)	9.2 ± 0.5	W (*10min*)	10.1 ± 0.4
3 (Sa)	Test 1	BX	6.4 ± 0.8	BX	1.8 ± 0.2	BX	5.6 ± 0.7	BX	2.1 ± 0.2
4 (Su)	Test 2	X	4.3 ± 0.3	X	2.6 ± 0.5	X	4.9 ± 0.6	X	3.2 ± 0.3
5	Test 3	X	4.8 ± 0.4	X	3.1 ± 0.5	X	5.2 ± 0.7	X	4.1 ± 0.3

*After rejection of one datum, see *Results* section.

Rat masses on the day before conditioning

Rat	Day	Mass (g)	Rat	Day	Mass (g)
1	4 (19th Jan)	335	17	15 (30th Jan)	405
2	4	355	18	15	395
3	4	370	19	15	380
4	4	370	20	15	380
5	4	395	21	15	420
6	4	315	22	15	375
7	4	355	23	15	400
8	4	345	24	15	390
9	4	370	25	15	375
10	4	375	26	15	365
11	4	370	27	15	365
12	4	390	28	15	360
13	4	385	29	15	385
14	4	355	30	15	330
15	4	365	31	15	405
16	4	365	32	15	355

(volumes in ml)

Rat	Group	Day 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Test BX	Test X	Test X
1	Long	*20min*	8.75	9.75	*8mlBX*	*8mlAX*													8.50	3.50	3.00	3.00
2	Long	*20min*	9.00	7.00	*8mlBX*	*8mlAX*													11.00	4.50	4.00	4.00
3	Long	*20min*	9.00	9.50	*8mlBX*	*8mlAX*													9.00	7.50	4.00	5.50
4	Long	*20min*	8.50	6.25	*8mlBX*	*8mlAX*													9.50	10.50	5.50	7.00
5	Long	*20min*	10.00	7.50	*8mlBX*	*8mlAX*													8.50	5.50	5.00	4.00
6	Long	*20min*	6.00	7.75	*8mlBX*	*8mlAX*													8.50	7.50	4.00	5.00
7	Long	*20min*	8.00	8.50	*8mlBX*	*8mlAX*													9.00	8.00	4.50	4.00
8	Long	*20min*	9.00	10.50	*8mlBX*	*8mlAX*													9.50	4.50	4.50	5.50
9	LongC	*20min*	7.25	8.25	*8mlW*	*8mlAX*													7.50	1.00	1.00	3.50
10	LongC	*20min*	7.00	7.00	*8mlW*	*8mlAX*													6.00	1.00	2.00	1.00
11	LongC	*20min*	10.50	8.50	*8mlW*	*8mlAX*													9.00	2.50	3.50	4.00
12	LongC	*20min*	5.00	6.00	*8mlW*	*8mlAX*													5.00	1.75	2.00	1.00
13	LongC	*20min*	7.75	6.50	*8mlW*	*8mlAX*													7.50	2.00	1.50	3.25
14	LongC	*20min*	7.50	8.00	*8mlW*	*8mlAX*													10.00	2.00	2.75	4.00
15	LongC	*20min*	7.25	8.00	*8mlW*	*8mlAX*													8.50	1.25	2.25	3.00
16	LongC	*20min*	9.75	9.00	*8mlW*	*8mlAX*													10.00	2.50	5.50	5.00
17	Short												*20min*	10.00	11.50	*8mlBX*	*8mlAX*		10.25	7.00	5.75	5.75
18	Short												*20min*	8.00	7.50	*8mlBX*	*8mlAX*		9.00	7.00	6.00	5.50
19	Short												*20min*	8.50	7.00	*8mlBX*	*8mlAX*		8.50	6.50	4.50	4.50
20	Short												*20min*	8.50	6.50	*8mlBX*	*8mlAX*		7.00	2.50	3.00	2.50
21	Short												*20min*	6.75	6.50	*8mlBX*	*8mlAX*		8.00	6.00	5.25	6.00
22	Short												*20min*	8.50	7.50	*8mlBX*	*8mlAX*		10.00	2.50	3.00	3.50
23	Short												*20min*	7.50	9.50	*8mlBX*	*8mlAX*		9.50	5.50	3.50	4.50
24	Short												*20min*	10.50	10.00	*8mlBX*	*8mlAX*		11.00	8.00	8.00	9.00
25	ShortC												*20min*	10.00	8.00	*8mlW*	*8mlAX*		12.00	2.50	3.00	3.00
26	ShortC												*20min*	8.50	10.50	*8mlW*	*8mlAX*		10.00	3.00	3.00	5.00
27	ShortC												*20min*	8.00	8.50	*8mlW*	*8mlAX*		9.00	2.00	†4.50	4.00
28	ShortC												*20min*	9.00	10.50	*8mlW*	*8mlAX*		10.50	1.50	2.75	4.00
29	ShortC												*20min*	9.00	7.50	*8mlW*	*8mlAX*		9.00	1.50	3.00	3.50
30	ShortC												*20min*	6.50	6.50	*8mlW*	*8mlAX*		9.00	2.00	3.00	4.50
31	ShortC												*20min*	6.50	8.00	*8mlW*	*8mlAX*		10.00	1.50	2.75	4.00
32	ShortC												*20min*	6.00	9.00	*8mlW*	*8mlAX*		10.00	2.50	5.00	5.00

†A noticeable amount of liquid was spilt by this rat, so its results were omitted from analysis.

Consumption data: mean ± SEM

		Long‡	(ml)	LongC	(ml)	Short	(ml)	ShortC	(ml)
8 Feb	Day 1	W (*20min*)		W *20min*		home		home	
9	Day 2	W (*10min*)	12.3 ± 0.5	W (*10min*)	9.8 ± 0.5	home		home	
10 (Sa)	Day 3	W (*10min*)	11.0 ± 0.4	W (*10min*)	9.9 ± 0.4	home		home	
11 (Su)	Day 4	**BX**	*8ml*	W	*8ml*	home		home	
12	Day 5	AX+	*8ml*	AX+	*8ml*	home		home	
13	Day 6	delay 1		delay 1		home		home	
14	Day 7	delay 2		delay 2		home		home	
15	Day 8	delay 3		delay 3		home		home	
16	Day 9	delay 4		delay 4		home		home	
17 (Sa)	Day 10	delay 5		delay 5		home		home	
18 (Su)	Day 11	delay 6		delay 6		home		home	
19	Day 12	delay 7		delay 7		W (*20min*)		W (*20min*)	
20	Day 13	delay 8		delay 8		W (*10min*)	10.3 ± 0.5	W (*10min*)	10.4 ± 0.3
21	Day 14	delay 9		delay 9		W (*10min*)	11.8 ± 0.3	W (*10min*)	10.4 ± 0.4
22	Day 15	delay 10		delay 10		**BX**	*8ml*	W	*8ml*
23	Day 16	delay 11		delay 11		AX+	*8ml*	AX+	*8ml*
24 (Sa)	Day 17	delay 12		delay 12		delay 1		delay 1	
25 (Su)	Day 18	W (*10min*)	12.0 ± 0.3	W (*10min*)	11.0 ± 0.4	W (*10min*)	13.1 ± 0.3	W (*10min*)	11.9 ± 0.4
26	Test 1	BX	4.9 ± 0.7	BX	2.6 ± 0.4	BX	7.1 ± 0.5	BX	3.8 ± 0.4
27	Test 2	X	4.2 ± 0.3	X	3.8 ± 0.4	X	4.2 ± 0.4	X	4.3 ± 0.4
28	Test 3	X	6.8 ± 0.6	X	5.9 ± 0.5	X	5.9 ± 0.5	X	5.4 ± 0.5

‡After rejection of one datum, see *Results* section.

Rat masses on the day before conditioning

Rat	Day	Mass (g)	Rat	Day	Mass (g)
1	4 (11th Feb)	280	17	15 (22nd Feb)	345
2	4	300	18	15	350
3	4	290	19	15	330
4	4	300	20	15	355
5	4	275	21	15	330
6	4	280	22	15	335
7	4	290	23	15	330
8	4	255	24	15	335
9	4	290	25	15	320
10	4	285	26	15	340
11	4	280	27	15	335
12	4	290	28	15	305
13	4	285	29	15	320
14	4	260	30	15	310
15	4	275	31	15	290
16	4	290	32	15	330

(volumes in ml)

Rat	Group	Day 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Test BX	Test X	Test X
1	Long	*20min*	9.50	9.50	*8mlBX*	*8mlAX*													10.00	†12.00	5.00	7.00
2	Long	*20min*	12.50	12.50	*8mlBX*	*8mlAX*													12.00	4.00	4.00	7.00
3	Long	*20min*	13.00	11.50	*8mlBX*	*8mlAX*													11.50	4.00	5.00	7.50
4	Long	*20min*	13.00	11.50	*8mlBX*	*8mlAX*													11.50	7.00	4.00	6.50
5	Long	*20min*	12.00	10.50	*8mlBX*	*8mlAX*													12.00	2.50	3.00	3.50
6	Long	*20min*	14.00	10.75	*8mlBX*	*8mlAX*													13.50	6.00	5.00	8.50
7	Long	*20min*	12.00	11.00	*8mlBX*	*8mlAX*													11.50	4.00	4.50	7.00
8	Long	*20min*	9.50	9.00	*8mlBX*	*8mlAX*													12.00	7.00	4.00	7.50
9	LongC	*20min*	11.00	9.50	*8mlW*	*8mlAX*													10.00	2.00	4.00	6.00
10	LongC	*20min*	8.50	10.50	*8mlW*	*8mlAX*													10.00	2.00	2.00	4.00
11	LongC	*20min*	8.50	9.00	*8mlW*	*8mlAX*													11.00	2.00	3.50	4.50
12	LongC	*20min*	10.00	9.00	*8mlW*	*8mlAX*													12.00	3.00	3.25	6.00
13	LongC	*20min*	12.00	12.00	*8mlW*	*8mlAX*													13.00	3.00	5.00	7.50
14	LongC	*20min*	9.50	10.00	*8mlW*	*8mlAX*													9.75	0.50	3.00	4.00
15	LongC	*20min*	8.00	9.00	*8mlW*	*8mlAX*													12.00	4.00	5.25	7.50
16	LongC	*20min*	10.50	10.00	*8mlW*	*8mlAX*													10.50	4.00	4.50	7.50
17	Short												*20min*	8.00	12.50	*8mlBX*	*8mlAX*		13.50	5.00	3.50	5.50
18	Short												*20min*	10.00	12.00	*8mlBX*	*8mlAX*		14.00	8.00	4.50	6.00
19	Short												*20min*	9.50	12.00	*8mlBX*	*8mlAX*		11.50	9.50	4.25	6.00
20	Short												*20min*	12.50	11.50	*8mlBX*	*8mlAX*		13.00	6.50	3.50	6.00
21	Short												*20min*	10.50	10.00	*8mlBX*	*8mlAX*		12.50	7.50	3.00	4.00
22	Short												*20min*	10.00	12.00	*8mlBX*	*8mlAX*		12.50	6.00	4.50	6.00
23	Short												*20min*	12.50	12.50	*8mlBX*	*8mlAX*		14.50	7.00	3.50	5.00
24	Short												*20min*	9.50	12.00	*8mlBX*	*8mlAX*		13.50	7.50	7.00	8.50
25	ShortC												*20min*	10.50	10.00	*8mlW*	*8mlAX*		12.50	4.00	3.50	5.00
26	ShortC												*20min*	9.50	9.00	*8mlW*	*8mlAX*		12.00	2.00	2.50	3.00
27	ShortC												*20min*	10.00	9.50	*8mlW*	*8mlAX*		13.00	3.00	3.25	4.00
28	ShortC												*20min*	9.50	10.00	*8mlW*	*8mlAX*		10.00	3.00	5.00	6.50
29	ShortC												*20min*	11.50	12.50	*8mlW*	*8mlAX*		12.00	5.00	6.50	7.00
30	ShortC												*20min*	10.50	10.00	*8mlW*	*8mlAX*		12.00	4.50	4.75	5.25
31	ShortC												*20min*	9.50	10.00	*8mlW*	*8mlAX*		11.00	4.50	4.25	5.00
32	ShortC												*20min*	12.00	12.00	*8mlW*	*8mlAX*		13.00	4.00	4.50	7.50

†Results omitted from analysis on statistical grounds, see *Results* section.