Blocking by fixed and variable stimuli: Effects of stimulus distribution on blocking

Dómhnall J. Jenningsa and Charlotte Bonardib

aInstitute of Neuroscience, Newcastle University, Newcastle upon Tyne, UK; bSchool of Psychology, University of Nottingham, Nottingham, UK

ABSTRACT
An experiment with rats compared the ability of fixed and variable duration cues to produce blocking. Rats in group B (Blocking) were trained that both fixed- (F) and variable- (V) duration cues would be followed by food delivery. In a subsequent training stage F and V continued to be reinforced, but F was accompanied by X, and V by Y. In the test phase responding to X and Y was examined. Control group O (Overshadowing) received identical treatment, except that F and V were nonreinforced in the first training stage. In group B there was evidence for blocking, but only of X, which had been conditioned in compound with the fixed-duration F; there was no evidence for blocking of Y, which had been conditioned in compound with the variable-duration V. It is suggested that this result may occur because fixed cues reach a higher, more stable asymptote of associative strength than do their variable equivalents.

ARTICLE HISTORY
Received 15 April 2016
Accepted 13 July 2016
First Published Online 10 August 2016

KEYWORDS
Associative learning; Blocking; Rats; Stimulus distribution form; Timing

In a typical classical conditioning task a neutral conditioned stimulus (CS) of a fixed duration is followed by delivery of a motivationally significant outcome, the unconditioned stimulus (US). Thus the CS comes to act both as a signal that the US will occur and when it will occur (conditioning and timing, respectively). Despite the intimate relation between these two types of learning, theories of conditioning and timing have developed relatively independently, and only recently has the relationship between temporal features of the CS and conditioning been explored. For example, in a series of experiments in rats, Jennings, Alonso, Mondragón, Franssen, and Bonardi (2013) compared levels of conditioned responding to fixed-duration CSs with responding to CSs whose duration varied from trial to trial. Even under equivalent testing conditions, fixed-duration cues elicited higher levels of responding than did variable-duration cues of the same mean duration. In a later study Bonardi, Mondragón, Brilot, and Jennings (2015) found that overshadowing was more profound when the overshadowing cue was of fixed duration than when it was variable.

They interpreted these findings in terms of a real-time model of conditioning, temporal difference (TD) learning (Sutton & Barto, 1987, 1990). Real-time models differ from other associative models by conceptualizing the CS as a series of temporally ordered elements that independently acquire associative strength (e.g., Sutton & Barto, 1990; Vogel, Brandon, & Wagner, 2003). A recent version of the TD account has been developed which allows it to compute learning for both serial and simultaneous stimulus compounds (Mondragón, Gray, Alonso, Bonardi, & Jennings, 2014). This SCC TD model was able to simulate our (2014) findings, predicting that fixed-duration stimuli acquire a higher, more stable asymptote of associative strength than their variable counterparts (Jennings et al., 2013; Mondragón et al., 2014). This is because each of the time-linked elements of a fixed CS can reach asymptote; in contrast, even though a variable CS comprises the same average number of temporal elements as the fixed one, many of these will be contiguous with the US on some trials and gain associative strength but distant
from the US on other trials, and therefore lose it. This constant fluctuation ensures that variable stimulus elements never reach a stable asymptote. As associative strength is assumed to be monotonically related to performance on a conditioning task, this would explain why fixed-duration CSs elicited more conditioned responding. It could also make such cues better competitors for associative strength when they are conditioned in compound with another stimulus. For example, according to two influential accounts of conditioning (Pearce & Hall, 1980; Rescorla & Wagner, 1972), a given US can support only a limited amount of associative strength. Thus when conditioning compound \(AX\), the more strength that is acquired by \(A\), the less is available for \(X\) (see also Mackintosh, 1975; Wagner, 1981).

This study tested another implication of this account: that fixed-duration CSs produce better blocking than do variable CSs. Two groups of rats were trained with fixed- and variable-duration cues, \(F\) and \(V\); for group B (blocking) \(F\) and \(V\) were reinforced, whereas for group O (overshadowing) they were not (see Table 1). To equate the groups’ experience, they were also trained with a second pair of cues, \(F_c\) and \(V_c\). Again, \(F_c\) was fixed and \(V_c\) variable; they were both reinforced in group O but nonreinforced in group B (cf., Rescorla, 1999). Then all rats experienced two reinforced stimulus compounds, of \(F\) with \(X\), and of \(V\) with \(Y\), and, finally, performance to \(X\) and \(Y\) was examined. In Stage 2 \(F\) and \(V\) had the opportunity to block acquisition of associative strength by \(X\) and \(Y\). If fixed CSs reach a higher, more stable asymptote than variable cues they should produce better blocking, and \(X\) should support less conditioned responding than \(Y\) at test.

A second aim was to evaluate an alternative interpretation of Bonardi et al.’s (2015) results. Many trial-based theories of conditioning assume that any stimulus has a level of associability, which determines the speed with which it conditions (e.g., Mackintosh, 1975; Pearce & Hall, 1980). If for some reason fixed-duration cues were to have higher associability than variable CSs, it could explain their greater ability to compete for associative strength, and produce overshadowing. However, associability differences would be less likely to affect blocking, because here (unlike overshadowing) \(F\) and \(V\) must be conditioned before their ability to compete for associative strength is assessed. It is usually assumed that associability changes over the course of conditioning, as the outcome is fully predicted (Mackintosh, 1975; Pearce & Hall, 1980); thus any difference in associability between fixed and variable cues will have diminished before their ability to produce blocking is evaluated. Thus if fixed-duration cues still produce more effective blocking, this would be more difficult to interpret in terms of associability differences. To strengthen this logic, Stage 1 training was continued until levels of responding to \(F\) and \(V\) in group B had asymptoted. If conditioned responding is an index of associative strength, this is consistent with the associative strength of the fixed and variable cues having reached asymptote, further minimizing differences in their associability before the start of compound training. Thus if Bonardi et al.’s results (2015) arose because fixed cues have higher associability, they would not necessarily produce better blocking in this study.

### Experimental study

#### Method

**Subjects**

Subjects were 32 male Lister hooded rats (Charles River UK) with a mean free-feeding weight of 308 g (range: 275–330 g). They were weighed daily and their daily food ration restricted so they gradually reduced to 85% of their free-feeding weights before the start of training. They were maintained at this level, 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. The holding room was on a 12-hour light/dark cycle, with lights being turned on at 7 am, and temperature was maintained at 21°C (±1); the humidity was 60% (±10%). There were 16 animals per group.

**Apparatus**

All testing was conducted in eight identical chambers (20 × 24 × 30 cm), each situated in a ventilated, noise-attenuating shell (74 × 38 × 60 cm; MED Associates).

### Table 1. Design of experiment.

| Group | Stage 1 | Stage 2 | Test |
|-------|---------|---------|------|
| B     | \(F^+\) | \(V^+\) | \(FX^+\) | \(VY^+\) | \(?\) | \(?\) |
| \(F_c^−\) | \(V_c^−\) |         |       |
| O     | \(F^−\) | \(V^−\) | \(FX^+\) | \(VY^+\) | \(?\) | \(?\) |
| \(F_c^+\) | \(V_c^+\) |         |       |

Note: \(F\) and \(F_c\) were auditory stimuli of a fixed 10-s duration; \(V\) and \(V_c\) were variable-duration auditory stimuli with a mean duration of 10 s; \(X\) and \(Y\) were visual stimuli either of fixed or of variable duration. \(B = \) Block, \(O = \) Overshadow; + denotes reinforcement, − nonreinforcement.
Each chamber was equipped with a food cup, into which 45-mg Noyes (Improved Formula A) food pellets could be delivered by a pellet dispenser (Model ENV-203). Each head entry into the food cup was recorded, by the breaking of an infra-red photobeam, as a response. Each chamber had a 2.8-W houselight 11 cm above the food cup, the bottom half of which was shielded, that was illuminated throughout the experimental session, and a speaker, on the right side of the back wall of the chamber opposite the food cup, which could deliver a white noise, a 10-Hz click, a 2-kHz tone, and a 4-kHz tone, all approximately 75 dB (Scale A, measured near food cup). There were two 2.8-W jewel lights, one 2.5 cm to each side of the food cup; the left light was kept on throughout its scheduled presentations, and the right light was always pulsed (.33 s on and .33 s off). Med-PC for Windows (Tatham & Zurn, 1989) controlled experimental events.

Procedure

Stage 1. Each of the 12 sessions in this stage comprised 10 presentations each of F, V, Fc, and Vc, intermixed in a semi-random order (see Table 1). F and Fc were of a fixed 10-s duration, but the durations of V and Vc varied from trial to trial, with a mean of 10 s (all variable durations were drawn from an exponential distribution). The mean duration of V and Vc over all sessions of this stage was 9.34 s and 10.46 s, respectively, for group B, and 9.77 and 10.98 s for group O. For group B F and V were each followed by delivery of a food pellet, while Fc and Vc were not; the reverse was true for group O. For half of each group F was click and V noise, and Fc high tone and Vc low tone; for the remainder F was noise and V click, and Fc low tone and Vc high tone. The intertrial interval (ITI) comprised a fixed 80-s interval plus a variable component with a mean of 60 s. The 20-s portion of the ITI that immediately preceded each stimulus presentation served as the pre-CS period.

Stage 2. Each of the 4 sessions in this stage comprised trials on which F was accompanied by X, and V by Y. X and Y were visual: illumination of one of the two lights. For half the rats in each group, X and Y were a fixed 10 s, and for the rest they were variable with a mean of 10 s. These 4 subgroups were counterbalanced for stimulus identity: for half the rats in each subgroup F was click and V noise, and Fc high tone and Vc low tone, etc. (see above). The click was always presented with the left light, and the noise with the right light; thus X and Y were fully counterbalanced for stimulus identity. When both stimuli were fixed or both variable, they started and ended simultaneously; when one was fixed and the other variable, they terminated together. All stimulus presentations were followed by the delivery of a food pellet. These sessions were otherwise identical to those of Stage 1.

Test. The three test sessions were identical to those of Stage 2, except that 20 trials—10 with FX and 10 with VY—were replaced by test trials with X or Y alone, 10 of each. All test trials were a fixed 10 s in duration, and 4 X and 4 Y trials were followed by the delivery of a food pellet; the remaining 6 X and 6 Y trials were nonreinforced.

Data treatment

The mean response rates during each trial type 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 was indexed by a difference score—mean response rate during each CS type after subtraction of the rate during the preCS period.

Number of responses in successive 1-s time bins of the reinforced CSs in Stage 1 was used to calculate the response rate in each bin for each rat, pooled over 2 6-session bins. For the variable CS computation of response rate took into account the number of trials on which the CS was present in each bin. The response rate functions were normalized, a linear function fitted to each normalized response rate function, and the slope determined from the best-fitting linear curve for each rat (Bonardi et al., 2015). The temporal slopes were compared against a mean of zero with one-sample t-tests, using the Bonferroni correction.

Data were subject to mixed ANOVA; significant two-way interactions were explored with simple main effects using the pooled error term. Partial $\eta^2$ is given as a measure of effect size for significant main effects and interactions.

Results

Stage 1

Difference scores for Stage 1 are shown in Figure 1. Responding increased to the reinforced cues (F and V in group B, Fc and Vc in group O) but remained low to the nonreinforced cues (Fc and Vc in group B,
Responding seemed higher in group B than in group O during later sessions, and higher to the fixed F and Fc than to the variable V and Vc on reinforced trials. To assess whether asymptote had been reached, data were analysed in 3 four-session blocks, with session as a factor within each block. ANOVA with group (B/O), reinforcement, fixed/variable (F/Fc versus V/Vc), block (1–3) and session (1–4) as factors revealed significant effects of block, $F(2, 60) = 69.45$, $p < .001$, $MSE = 210.81$, $\eta^2_p = .70$, session, $F(3, 90) = 86.66$, $p < .001$, $MSE = 29.89$, $\eta^2_p = .74$, fixed/variable, $F(1, 30) = 11.58$, $p = .002$, $MSE = 54.33$, $\eta^2_p = .28$, and reinforcement, $F(1, 30) = 305.22$, $p < .001$ $MSE = 599.46$, $\eta^2_p = .91$, two-way interactions of block and session, $F(6, 180) = 25.78$, $p < .001$, $MSE = 59.72$, $\eta^2_p = .46$, block and reinforcement, $F(2, 60) = 111.91$, $p < .001$, $MSE = 199.10$, $\eta^2_p = .79$, session and reinforcement, $F(3, 90) = 46.87$, $p < .001$, $MSE = 31.83$, $\eta^2_p = .61$, and a three-way interaction between these three factors, $F(6, 180) = 7.78$, $p < .001$, $MSE = 57.77$, $\eta^2_p = .21$; the fixed/variable $\times$ reinforcement interaction was also significant, $F(1, 30) = 7.79$, $p = .009$, $MSE = 71.63$, $\eta^2_p = .21$. Nothing else was significant, largest $F(1, 30) = 3.02$, $p = .09$, $MSE = 599.46$.

The three-way interaction between block, session, and reinforcement was explored with 2-way ANOVAs, with block and session as factors, on the data from reinforced and nonreinforced trials. For reinforced trials this yielded a significant interaction, $F(6, 186) = 15.70$, $p < .001$, $MSE = 108.2$, $\eta^2_p = .34$, and an effect of session on Block 1, $F(3, 93) = 128.69$, $p < .001$, $MSE = 53.03$, and Block 2, $F(3, 93) = 8.32$, $p < .001$, $MSE = 53.03$, but not Block 3, $F(3, 93) = 1.85$, $p = .14$, $MSE = 53.03$. A parallel ANOVA on nonreinforced trials also revealed a significant interaction, $F(6, 186) = 23.77$, $p < .001$, $MSE = 12.22$, $\eta^2_p = .43$, and an effect of session on Block 1, $F(3, 93) = 74.9$, $p < .001$, $MSE = 8.32$, and Block 2, $F(3, 93) = 8.22$, $p < .001$, $MSE = 53.03$, but not Block 3, $F < 1$. Thus responding to both reinforced and nonreinforced CSs was steady over the last block of training, suggesting that conditioning responding had reached asymptote.

Exploration of the Fixed/variable $\times$ Reinforcement interaction confirmed that on reinforced trials response rates were higher during the fixed stimuli, $F(1, 60) = 18.83$, $p = .001$ $MSE = 62.98$. This was not the case on the nonreinforced trials, $F < 1$. This is consistent with the hypothesis that fixed CSs gain more associative strength than do variable CSs.

To lend further weight to the implication of the previous analysis that conditioned responding had reached asymptote, we conducted a Bayesian analogue of the paired t-test (Rouder, Speckman, Sun, Morey, & Iverson, 2009; http://pcl.missouri.edu/bayesfactor). This allows quantification of evidence for the null hypothesis (Wagenmakers, 2007); it assumes effect sizes that equal zero under the null hypothesis but differ from zero under the alternative hypothesis, and it yields a Bayes factor indicating how much more likely the null hypothesis is than the alternative. We employed the $F$ values relating to the change in responding on reinforced, and nonreinforced, trials between the first and last two sessions of the final test block. This yielded scaled Bayes factors of 4.03 and 5.27 for reinforced and nonreinforced trials respectively; as values of $\geq 3$ may be taken as evidence for the null hypothesis (Jeffreys, 1961), we take this as further evidence that conditioning had reached asymptotic levels on both types of trial.

Group mean rates of preCS responding (over all trial types) in each session were 3.58, 4.73, 3.54, 2.07,
1.20, 1.11, 0.96, 0.70, 0.97, 1.20, 0.94, and 0.86 rpm for group B, and 4.79, 6.52, 4.10, 2.73, 1.78, 1.53, 1.40, 1.29, 1.28, 1.17, 1.13, and 1.10 rpm for group O. ANOVA with group and sessions as factors revealed an effect of group, $F(1, 30) = 4.22$, $p = .049$, $MSE = 8.35$, $\eta^2_p = .03$ reflecting a slightly higher rate of responding in group B. There was also an effect of block, $F(2, 60) = 93.76$, $p < .001$, $MSE = 14.75$, $\eta^2_p = .76$, sessions, $F(3, 90) = 13.45$, $p < .001$, $MSE = 7.78$, $\eta^2_p = .31$, and a Block x Sessions interaction, $F(6, 180) = 9.72$, $p < .001$, $MSE = 6.49$, $\eta^2_p = .24$; there was an effect of sessions on Block 1, $F(3, 270) = 32.38$, $p < .001$, $MSE = 6.92$, but not on Blocks 2 and 3, $Fs < 1$.

We also anticipated that the distribution of responding should differ across the fixed and variable CSs, increasing over the course of the fixed CS but remaining steady over the variable CS. This would result in higher slopes in the fixed CS—but we did not find strong evidence for this. These data were computed for the first and second half of the training stage, to reduce noise (timing data are routinely collected over a relatively large number of trials). Mean slopes for fixed and variable cues in group B were 0.24 and 0.28 in the first half, and 0.95 and 1.01 in the second half; corresponding values for group O were 0.57 and 0.18, and 0.43 and 0.51. ANOVA with group (B/O), fixed-variable, and block as factors revealed a main effect of sessions, $F(1, 29) = 2.57$, $p = .12$, $MSE = 144.41$. Responding was higher in group B on session 13, $F(1, 116) = 4.27$, $p = .041$, $MSE = 312.11$, but not on any other session, $Fs < 1$.

Mean rates of preCS responding in each session were 1.56, 1.46, 1.37, and 1.50 rpm for group B, and 4.60, 2.77, 2.49, and 2.73 rpm for group O—marginally higher in group O. ANOVA with group and sessions as factors revealed main effects of group, $F(1, 29) = 21.64$, $p < .001$, $MSE = 4.01$, $\eta^2_p = .43$, and session, $F(3, 87) = 6.25$, $p = .001$, $MSE = 1.34$, $\eta^2_p = .18$, and an interaction between these factors, $F(3, 87) = 4.84$, $p = .004$, $MSE = 1.34$, $\eta^2_p = .14$; preCS responding was lower in group B on all four sessions, smallest $F(1, 116) = 4.79$, $p = .031$, $MSE = 2.01$. This could be due to the pretrained $F$ and $V$ blocking the context in group B. In group O the previously nonreinforced $F$ and $V$ were now for the first time followed by food, but in group B unexpected delivery of food after $X$ and $Y$ would be predicted by $F$ and $V$, which could block any context conditioning occurring on these trials, thus reducing preCS responding in these animals.

### Test

Responding to $X$ and $Y$ is shown in Figure 1 (lower panel; see Table 2 for the same data separated according to whether $X$ and $Y$ were fixed or variable). Responding to $X$ in group B appeared lower than in the other conditions, which seemed not to differ. ANOVA with group (B/O), target ($XY$ fixed or variable), ($XY$), and session as factors revealed an effect of $XY$, $F(1, 27) = 7.86$, $p = .009$, $MSE = 32.85$, $\eta^2_p = .22$, which interacted with group, $F(1, 27) = 5.95$, $p = .022$, $MSE = 32.85$, $\eta^2_p = .18$. The effect of $XY$ was significant in group B, $F(1, 27) = 14.23$, $p = .001$, $MSE = 32.85$, but

### Table 2. Group mean response rates to $X$ and $Y$ in group B and O, in subgroups for which $X$ and $Y$ were fixed or variable, in each of the three test sessions.

| Subgroup | 1     | 2     | 3     |
|----------|-------|-------|-------|
|          | $X$   | $Y$   | $X$   | $Y$   | $X$   | $Y$   |
| B (F)    | 16.01 | 21.26 | 13.91 | 19.01 | 11.25 | 17.62 |
| B (V)    | 11.66 | 15.68 | 12.94 | 16.65 | 17.40 | 19.43 |
| O (F)    | 16.20 | 22.50 | 14.44 | 15.00 | 12.00 | 15.99 |
| O (V)    | 25.80 | 22.09 | 20.48 | 18.90 | 24.86 | 21.15 |

Note: $F$ = fixed subgroup; $V$ = variable subgroup.
not in group O, $F < 1$, and responding was lower in group B than in group O during X, $F(1, 54) = 5.68, p = .021$, $MSE = 119.93$, but not during Y, $F < 1$. There were also significant interactions between target and session, $F(2, 54) = 4.59, p = .014$, $MSE = 40.08$, $\eta^2_p = .15$, and target and X/Y, $F(1, 27) = 7.05, p = .013$, $MSE = 32.85$, $\eta^2_p = .21$; critically, neither of the interactions involving target, group, and X/Y were significant, largest $F(1, 27) = 1.62, p = .21$, $MSE = 32.85$. This confirmed that the critical finding—greater blocking of X than of Y in group B—was not influenced by whether X and Y were fixed or variable. Nothing else was significant, largest $F(1, 27) = 2.80, p = .11$, $MSE = 207.02$. Group mean rates of preCS responding in the three sessions were, for group B, 0.96, 1.00, and 1.05 rpm for those trained with a fixed-duration cue, and 2.79, 1.93, and 2.46 rpm for those trained with a variable X and Y. The corresponding means for group O were 0.89, 1.00, and 1.29 rpm, and 2.48, 2.33, and 2.57 rpm. ANOVA with group sessions and target as factors revealed only a main effect of target, $F(1, 27) = 11.04, p < .001$, $MSE = 4.08$, $\eta^2_p = .29$, reflecting the generally higher preCS rates when X and Y were variable; nothing else was significant, $F_s < 1$. This confirms that the rates of baseline responding from which the difference scores were derived did not differ systematically among the critical factors of XY and group.

**General discussion**

In the present experiment only the fixed-duration cue F produced a reliable blocking effect. We did not, however, find that F also produced better overshadowing in group O—responding to X and Y did not differ in group O (although it should be acknowledged that we did not include an overshadowing control group in this experiment). This would contrast with our previous findings (Bonardi et al., 2015); however, the experiments differed in a number of ways. In particular, in this study rats had extensive experience of the blocking/overshadowing cues F and V before the compound phase, which would be expected to reduce their associability (Mackintosh, 1975; Pearce & Hall, 1980) and thus reduce the degree to which they could acquire associative strength and produce overshadowing. In contrast, in the experiment reported by Bonardi et al. (2015) the overshadowing cues were completely novel at the start of compound training. The Stage 1 preexposure of F and V in group O could have other effects; for example, such differential training might perhaps result in F and V becoming net conditioned inhibitors and thus being able to produce superconditioning of X and Y in Stage 2—thus exaggerating the size of the observed blocking effect. However, inhibitory conditioning after differential conditioning is not anticipated by standard associative models; for example, Rescorla and Wagner (1972) predict that any inhibitory learning to F and V would stem from the nonreinforcement of these stimuli in an excitatory context, which would result in a neutral compound of F (or V) and the context. Thus F and V could only produce superconditioning in Stage 2 if it were conducted in a different and less excitatory context. Moreover, theory aside, there is little evidence in the literature for such a differential inhibition effect; although Williams, Travis, and Overmier (1986) did find evidence for inhibition after such differential conditioning, this was in a shock conditioning procedure in which the initial conditioning stage was conducted off baseline. This could produce precisely the type of context change that we have argued would be required to reveal such a differential inhibition effect.

Another potential issue relates to the fact that compounding fixed- and variable duration stimuli necessarily introduces an asymmetry into the stimulus compounds in which X and Y are conditioned. For example, when X and Y are fixed, in Stage 2 the FX compound comprises two coextensive stimulus elements, whereas the VY compound does not. This could produce a differential effect on test responding to X and Y; never having been experienced alone before the test, X might suffer more generalization decrement from Stage 2 training, and this alone could result in it supporting less conditioned responding than Y at test. However, when X and Y are variable, the reverse is the case: here the coextensive compound is VY—meaning that this generalization decrement mechanism would result in less responding to Y than to X. Thus it is not clear how this mechanism could explain the pattern of results we observed: less responding to X than to Y regardless of whether X and Y were fixed or variable. Moreover, this effect was confined to group B—despite the fact that group O were conditioned with formally identical stimulus compounds during the compound conditioning stage.

Finding that a fixed-duration CS produces better blocking than does a variable CS is consistent with our proposal that fixed-duration cues reach a higher, more stable asymptotic level of associative strength. This conclusion does, of course, rely on the
assumption that ability to produce blocking is a measure of associative strength—and there are alternative interpretations (cf. Ward et al., 2012; see further discussion below). Our results are less consistent with the alternative hypothesis, that fixed-duration stimuli produce more overshadowing or blocking because they have higher associability. As both fixed and variable cues receive conditioning before their ability to produce blocking is assessed, any associability differences should be relatively small (Mackintosh, 1975; Pearce & Hall, 1980). We attempted to reduce such potential associability differences even further by conditioning both X and Y to asymptote before the start of the compound phase. However, although conditioned responding is thought to be monotonically related to associative strength, it is still possible that associative strength was increasing, even though conditioned responding had asymptoted. Thus we have no direct evidence that asymptotic levels of associative strength had been reached (and it is difficult to see how such evidence could be obtained). Nonetheless, we would argue that these considerations make an explanation of our results in terms of associability less plausible.

These findings add to a growing body of literature illustrating the effects of temporal variables on associative learning. But although many theories from both conditioning and timing traditions have been proposed to explain such learning, most of them cannot explain our results. Kirkpatrick (2014) classified these models into three broad classes.

**Time-based hybrid models.** Some accounts of this type allow time of reinforcement to be accurately encoded by assuming that the CS is represented in a form that varies as a function of time. This may be because CS onset initiates activity in a series of oscillators with different periods (Church & Broadbent, 1990) or activates a series of memory traces (Machado, 1997), or provokes some other time-based process that uniquely defines different portions of the CS (cf. Grossberg & Schmajuk, 1989). Another set of models in this class supposes that timing arises through some computation based on processing of a previously experienced reinforcer (e.g., Kirkpatrick, 2002; Staddon & Higa, 1996). However, the majority of these models cannot explain cue competition effects such as blocking (although see e.g., Guilhardi, Yi, & Church, 2007), and those that can do not allow it to be influenced by temporal variables (Kirkpatrick, 2014).

**Information processing models.** These models assume sensitivity to temporal information in the conditioning episode, allowing computation of the rate of reinforcement during environmental events. If reinforcement rate during the CS exceeds that in the background, the CR is produced (Gibbon & Balsam, 1981; cf. Rate Expectancy Theory RET Gallistel & Gibbon, 2000). Some of these accounts can also explain cue competition within this alternative theoretical framework; for example, RET assumes that when the novel cue is added during a blocking task, the rate of reinforcement is attributed solely to the blocking cue (cf., Balsam & Gallistel, 2009; Kirkpatrick & Church, 2003), producing a blocking effect. Most of these accounts assume that temporal information is summed across trials, such that fixed- and variable-duration cues would be treated as equivalent if their overall duration is matched, meaning they could not explain the present findings. However, in one variant of RET, Balsam and Gallistel (2009) suggested that the CS’s information value influences conditioned responding: as fixed CSs are more informative than variable CSs, they should condition more quickly (Balsam & Gallistel, 2009; Ward et al., 2012). In fact attempts to verify this prediction have not supported this model: Ward et al. (2012) found no difference in the speed of acquisition by fixed and variable CSs, whereas Jennings et al. (2013) reported that variable cues developed conditioned responding faster than fixed CSs—the opposite of what this account predicts. But as a difference in a CS’s information value might also influence its ability to produce blocking, our results might provide an alternative means of evaluating this theory. For example, if the blocking cue were fixed, addition of a target cue would add little further information, regardless of whether it was fixed or variable. But if the blocking/overshadowing cue were variable, addition of a fixed target could add further information and so therefore elicit higher levels of conditioned responding than a variable target cue. This does stretch the model to an extent—if blocking is based on a decision to attribute the CR to the CS that provides the most information, then the pre-conditioned blocking stimulus will always be more informative than the unconditioned target stimulus, and variations in the degree to which this is the case should not affect the outcome of the decision.
process. Nonetheless, as we did include this factor in our analysis, it is possible to investigate this possibility—and, as noted above, we did obtain a significant interaction between whether the blocking/overshadowing cues and the target cues were fixed or variable, although this did not interact with group (Table 2). Exploration of this interaction reflected lower responding to the fixed than to the variable target when the blocking/overshadowing cue was fixed (13.95 and 18.61 rpm for X and Y, respectively, $F(1, 27) = 4.66, p = .035$, $MSE = 119.32$), not when it was variable (18.86 and 18.98 rpm for X and Y, respectively, $F < 1$)—the opposite of what this theory predicts.

**Prediction error-based hybrid models.** Although most conditioning models do not consider the temporal structure of the CS, there are exceptions. The temporal coding hypothesis (Matzel, Held, & Miller, 1988) assumes that associations incorporate temporal information about the CS–US pairing (e.g., Barnet, Grahame, & Miller, 1993). But this model predicts that blocking will be maximal if the temporal information in the blocking cue matches that of the blocked cue. In our study this predicts that $F$ will produce more blocking than $V$ when $X$ was fixed, but less when $X$ was variable. As in our study half the animals had a fixed-duration $X$ and $Y$ and the other half a variable-duration $X$ and $Y$, this would predict no overall difference in blocking—which was not what we observed.

There is also, as we mentioned in the introduction, a class of time-based models of conditioning that base learning on prediction error (e.g., Mondragón et al., 2014; Sutton & Barto, 1990; Vogel et al., 2003). Models of this type can anticipate the pattern of results reported here and may represent the best chance for associative theories to accommodate time-based characteristics of the conditioning process.

In summary, the present results provide further support for the proposal that associative learning can be influenced by the temporal distribution of the CS. Fixed-duration CSs support a higher rate of conditioned responding than variable cues matched in mean duration (Jennings et al., 2013); they also produce better overshadowing (Bonardi et al., 2015) and in the present study better blocking. The models of conditioning and timing best equipped to explain these findings come from the class of hybrid models based on prediction-error learning in real time, such as the SCCC TD model proposed by Mondragón et al. (2014).

**Disclosure Statement**

No potential conflict of interest was reported by the authors.

**Funding**

This work was funded by the Biotechnology and Biological Sciences Research Council (BBSRC) [BB/H008160/1].

**References**

Balsam, P. D., & Gallistel, C. R. (2009). Temporal maps and informativeness in associative learning. *Trends in Neurosciences*, 32, 73–78.

Barnet, R. C., Grahame, N. J., & Miller, R. R. (1993). Temporal encoding as a determinant of blocking. *Journal of Experimental Psychology: Animal Behavior Processes*, 19, 215–230.

Bonardi, C., Mondragón, E., Brilot, B., & Jennings, D. J. (2015). Overshadowing by fixed and variable duration stimuli. *The Quarterly Journal of Experimental Psychology*, 68, 523–542.

Church, R. M., & Broadbent, H. A. (1990). Alternative representations of time, number and rate. *Cognition*, 37, 55–81.

Gallistel, C. R., & Gibbon, J. (2000). Time, rate and conditioning. *Psychological Review*, 107, 289–344.

Gibbon, J., & Balsam, P. (1981). Spreading association in time. In L. C. Locurto, H. S. Terrace, & J. Gibbon (Eds.), *Autoshaping and conditioning theory* (pp. 219–253). New York: Academic Press.

Grossberg, S., & Schmajuk, N. A. (1989). Neural dynamics of adaptive timing and temporal discrimination during associative learning. *Neural Networks*, 2, 79–102.

Guilhardi, P., Yi, L., & Church, R. M. (2007). A modular theory of learning and performance. *Psychonomic Bulletin and Review*, 14, 543–559.

Jeffreys, H. (1961). *Theory of probability* (3rd ed.). Oxford: Oxford University Press, Clarendon Press.

Jennings, D., Alonso, E., Mondragón, E., Franssen, M., & Bonardi, C. (2013). The effect of stimulus distribution form on the acquisition and rate of conditioned responding: Implications for theory. *Journal of Experimental Psychology: Animal Behavior Processes*, 39, 233–248.

Kirkpatrick, K. (2002). Packet theory of conditioning and timing. *Behavioural Processes*, 57, 89–106.

Kirkpatrick, K. (2014). Interactions of timing and prediction error learning. *Behavioural Processes*, 101, 135–145.

Kirkpatrick, K., & Church, R. M. (2003). Tracking of the expected time to reinforcement in temporal conditioning procedures. *Animal Learning and Behavior*, 31, 3–21.

Machado, A. (1997). Learning the temporal dynamics of behaviour. *Psychological Review*, 104, 241–265.

Mackintosh, N. J. (1975). A theory of attention: Variation in the associability of stimuli with reinforcement. *Psychological Review*, 82, 276–298.

Matzel, L. D., Held, F. P., & Miller, R. R. (1988). Information and expression of simultaneous and backward associations: Implications for contiguity theory. *Learning and Motivation*, 19, 317–344.

Mondragón, E., Gray, J., Alonso, E., Bonardi, C., & Jennings, D. J. (2014). SCCC TD: A serial and simultaneous configural-cue...
compound stimuli representation for temporal difference learning. *PloSOne*, 9, e0102469.

Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, 87, 532–552.

Rescorla, R. A. (1999). Learning about qualitatively different outcomes during a blocking procedure. *Animal Learning & Behavior*, 27, 140–151.

Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: The effectiveness of reinforcement and non-reinforcement. In A. H. Black, & W. F. Prokasy (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). New York: Appleton-Century-Crofts.

Rouder, J. N., Speckman, P. L., Sun, D., Morey, R. D., & Iverson, G. (2009). Bayesian t-tests for accepting and rejecting the null hypothesis. *Psychonomic Bulletin & Review*, 16, 225–237.

Staddon, J. E. R., & Higa, J. J. (1996). Multiple time scales in simple habituation. *Psychological Review*, 103, 720–733.

Sutton, R. S., & Barto, A. G. (1987). A temporal difference model of classical conditioning (Technical report TR 87-509.2). Waltham, MA: GTE Lab.

Sutton, R. S., & Barto, A. G. (1990). Time derivative models of Pavlovian reinforcement. In M. R. Gabriel, & J. W. Moore (Eds.), *Learning and computational neuroscience: Foundations of adaptive networks* (pp. 497–537). Cambridge (MA): MIT Press.

Tatham, T. A., & Zurn, K. R. (1989). The Med-PC experimental apparatus programming system. *Behavioral Research Methods, Instruments, and Computers*, 21, 294–302.

Vogel, E. H., Brandon, S. E., & Wagner, A. R. (2003). Stimulus representation in SOP: II. An application to inhibition of delay. *Behavioural Processes*, 62, 27–48.

Wagenmakers, E. J. (2007). A practical solution to the pervasive problems of p values. *Psychonomic Bulletin & Review*, 14, 779–804.

Wagner, A. R. (1981). SOP: A model of automatic memory processing in animals. In N. E. Miller & R. R. Spear (Eds.), *Information processes in animals: Memory mechanisms* (pp. 95–128). Hillsdale (NJ): Erlbaum.

Ward, R. D., Gallistel, C. R., Jensen, G., Richards, V. L., Fairhurst, S., & Balsam, P. D. (2012). Conditioned stimulus informativeness governs conditioned stimulus–unconditioned stimulus associability. *Journal of Experimental Psychology: Animal Behavior Processes*, 38, 217–232.

Williams, D. A., Travis, G. M., & Overmier, J. B. (1986). Within-compound associations modulate the relative effectiveness of differential and Pavlovian conditioning inhibition procedures. *Journal of Experimental Psychology: Animal Behavior Processes*, 12, 351–362.