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Spontaneous recovery from interference between cues but not from backward blocking

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ABSTRACT

In the present study, we examined the differential effect on backward blocking (BB) and on interference between cues (IbC) of including a delay right before the test phase vs. between training phases 1 and 2 in humans. While models of IbC predict a spontaneous recovery (SR) of responding if the delay is placed immediately before the test instead of between phases 1 and 2, BB models predict that no difference should be observed due to the position of the delay. In our experiment, we obtained the SR from IbC but not from BB. These results suggest that backward blocking and interference between cues are likely to be the result of different processes.

1. Introduction

Backward blocking (BB) and interference between cues (IbC) are two learning and memory phenomena with many similarities regarding the standard experimental conditions in which they are observed. In a BB design, a compound of two cues, A and B, is first paired with an outcome. Later on, one of the elements of the compound, B, is presented repeatedly with the same outcome (i.e., AB → O1 followed by B → O1). When the other element of the compound, A, is subsequently tested, it elicits a lower response than if participants had not been exposed to B → O1 pairings (e.g., Shanks, 1985). Similarly, in an IbC design, a cue, A, is paired with an outcome, and, later on, another cue, B, is trained with the same outcome (i.e., A → O1 followed by B → O1). As in the case of BB, when Cue A is then presented at test, participants' responses are weaker than in a control group not exposed to B → O1 pairings (e.g., Matute and Pineño, 1998a). Thus, in both phenomena the B → O1 relationship learned during the second stage reduces the expression of the previously learned relationship between the absent cue, A, and the outcome. Thus, at the empirical level, the only difference between them is whether the two cues receive compound training during stage 1.

But despite the resemblances of the conditions in which these phenomena have been observed, BB and IbC have generally been assumed to involve very different mechanisms, and have received very different theoretical explanations. In the case of BB, associative models propose that a within-compound association between Cues A and B develops as they are jointly paired with the outcome during the first training stage. This within-compound association mediates a cue interaction process that may take place through different mechanisms proposed by different theories. For example, according to Van Hamme and Wasserman (1994) and Dickinson and Burke (1996), the presentation of Cue B on B → O1 trials during the second phase activates the representation of Cue A through the within-compound association. The temporal overlap between such representation and the presence of the outcome causes a decrease in the associative strength between the representation of Cue A and O1. Specifically, according to Van Hamme and Wasserman (1994), the activation of the representation of absent stimuli makes the alpha parameter for such stimuli to take a negative value and, hence, the Cue A → outcome association decreases in phase 2. Alternatively, according to Dickinson and Burke (1996), the activation of the representation of absent stimuli produces an inhibitory association between such stimuli and the outcome and, hence, the Cue A has a reduced net excitation when tested.

According to the comparator hypothesis (see Stout and Miller, 2007) when Cue A is presented at test, it activates the representation of Cue B through the within-compound association. The degree of activation of the representation of O1 will depend on the comparison between the associative strength of Cue A and the associative strength of Cue B with the outcome. The BB effect is found because the second learning phase renders Cue B more strongly associated with O1 than Cue A.

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Importantly, for all these explanations the decrease in responses to Cue A is not the result of a retrieval difficulty but the result of the interaction of Cues A and B via their within-compound association.

The theoretical explanations of IbC are based on very different models, focused on retrieval processes rather than on acquisition or response mechanisms (Matute and Pineño, 1998b; Miller and Escobar, 2002). Without going into specific details concerning these explanations, overall, they build on the following two main ideas. First, interference occurs if two learned associations have a common element in a common temporal location (whether the cue or the outcome location). Second, the (physical or temporal) context in which a given association is acquired has the capacity of priming such association in detriment of other associations that share a common element with it. According to these assertions, IbC occurs because: (1) the associations learned in the first and the second training stages share the same outcome; (2) the context used for the test phase is the same (or almost the same) as for the second training stage; thus it primes the most recently learned association in detriment of the oldest one.

In summary, whereas BB is thought to be the result of an interaction between cues mediated by within-compound associations, IbC is thought to be the result of a retrieval conflict between associations modulated by the physical or temporal context.

Although these quite similar phenomena have received very different explanations, very little research based on systematic comparisons between BB and IbC has been done to determine whether they are produced by so different mechanisms or not. Given the impact of these phenomena on learning theories and models, such an issue deserves more effort and attention. As an exception, we should mention the experiments reported by Escobar et al. (2002) and Vadillo et al. (2008), in which BB and IbC conditions were directly compared with the same control condition in the same experiment. However, these previous studies have two shortcomings. First, the strategy followed was restricted to a comparison of the magnitude of BB and IbC effects in the same experiment, and thus did not allow a conclusion concerning their underlying mechanisms. Following this strategy, even if a difference in magnitude between both effects was found, it would not necessarily mean that each effect is caused by a different mechanism. The difference in magnitude could be caused by a single mechanism which is sensitive to specific differences between the procedures used to obtain BB and IbC.

Second, it seems that this strategy has not provided a clear pattern of results since whereas Escobar et al. (2002, Experiment 2) found similar IbC and BB effects, Vadillo et al. (2008) found BB to be larger than IbC.

The aim of the present experiment is to provide more compelling evidence that BB and IbC are produced by different mechanisms. To achieve this aim we introduced a manipulation that, theoretically, should only affect IbC but not BB because only the putative mechanism for IbC is thought to be sensitive to such manipulation. The introduction of a manipulation, instead of the direct comparison of effect sizes, is a common strategy in cognitive psychology to provide evidence that two phenomena are produced by different mechanisms (e.g., see Baddeley, 1997, for the use of this strategy in the memory field). Specifically, we examined the differential effect of including a delay right before the test phase on IbC and BB (see Table 1). It is well known that this variable affects interference mechanisms dramatically. Numerous experiments with this manipulation have shown spontaneous recovery (SR) of an extinguished response when the delay is placed right before the test phase (see Bouton, 1993). Importantly, there is also evidence showing the SR effect in IbC in humans: When a delay is inserted between the second learning phase and the test phase, IbC tends to disappear and a SR of responses to the interfered cue, A, is found (Pineño et al., 2000). The explanation of SR is very similar for extinction and for IbC effects. The time interval between the second learning phase and the test phase is thought to increase the difference between the temporal contexts of both phases. Thus, the context of the test phase loses its capacity of priming the association learned in the second phase over the association learned in the first phase, and, as a consequence, the response of the first phase is retrieval correctly. If, alternatively, the same delay is introduced between the first and the second learning phase (to hold constant the temporal distance between the first learning phase and the test phase), the IbC effect should remain unaffected (see Pineño et al., 2000).

On the other hand, theoretical models of BB do not make specific predictions for time passage, and it is supposed that the cue interaction mechanisms underlying BB, which only depends on the strength of within-compound associations, should not be affected by when delay occurs. Indeed, maybe because SR is not expected in BB, no such experiments have been made with humans as participants that include the manipulation of time delay.

In sum, we expected, first, to obtain SR from IbC, replicating the result of Pineño et al. (2000), i.e., a larger effect of IbC when the delay was placed between phase 1 and phase 2 than when the delay was placed between phase 2 and the test phase. Second, we expected BB to be unaffected by where the delay was placed. This pattern of results would indicate that both phenomena are caused by different processes, at odd with the conclusions drawn by Escobar et al. (2002). It is important to note that, to correctly determine the finding of an IbC as well as a BB effect, it is crucial to compare the responses to cues under the IbC and BB treatments with the responses to control cues. In this case, the appropriate control condition for both treatments is the overshadowing condition. This is the reason for including the EF → O3 trials in the first training phase as shown in Table 1. Thus, an IbC effect would be found if responses to the correct outcome are less frequent when Cue C (the cue under the IbC treatment) is presented at test than when either Cue E or Cue F is presented at test. Likewise, a BB effect would be found if responses to the correct outcome are less frequent when Cue A (the cue under the BB treatment) is presented at test than when Cue E or Cue F is presented at test. Hence, the simple assessment of the

Table 1
Experimental design for backward blocking, interference between different cues of the same outcome and their overshadowing control.

Group		Phase 1		Phase 2		Test phase
Delay:1-2	BB	AB – O1	Delay	B – O1		A?
	IbC	CD – O2		G – O2		C?
	Overshad.	EF – O3				E?
Delay:2-Test	BB	AB – O1		B – O1	Delay	A?
	IbC	CD – O2		G – O2		C?
	Overshad.	EF – O3				E?

Note: Letters A–C stand for cues and O1–O3 refer to the different outcomes. In the Delay:1-2 group a delay of 225 s was introduced between phase 1 and phase 2. In the Delay:2-Test group a delay of the same duration was included between the phase 2 and the test. BB, IbC and Overshad. indicate 'backward blocking', 'interference between cues' and 'overshadowing' conditions, respectively.

effect of delay on responses to Cues A and C would tell us little about how the delay modulates the BB and IbC effects.

2. Methods

2.1. Participants

The participants were 164 undergraduate Psychology students from the University of Málaga who took part in the experiment for course credits. They were randomly assigned to one of the two between-subjects experimental conditions shown in Table 1.

2.2. Apparatus and stimuli

2.2.1. Apparatus

The task was performed in semi-isolated cubicles on 10 PC computers running software developed in Visual Basic.

2.2.2. Stimuli

Coloured rectangles stood for cues. These coloured rectangles were blue, brown, yellow, orange, red, green, and pink, and their role as the abstract cues shown in Table 1 were counterbalanced. The rectangle or rectangles (on compound-cue trials as AB-1) that stood for cues on each trial were placed at the middle top of the screen. When two cues were displayed on the same trial, they were placed one beside the other, the specific location for each one being counterbalanced on a trial-by-trial basis. Stimuli used as outcomes were pictures of fictitious plants labelled as Kollin, Dobe, and Yamma, and their role as the abstract outcomes shown in Table 1 were also counterbalanced. The different possible outcomes on each trial were randomly placed at the bottom of the screen in different locations (left, centre, and right) on a trial-by-trial basis. Beneath each possible outcome, a horizontal scroll bar was displayed for participants to make responses. A small text box at the right of each scroll bar was used to provide numerical information of participants' responses.

2.3. Design and procedure

2.3.1. Design

The design of the experiment is shown in Table 1. During phase 1, all the participants saw three compound cues (AB, CD and EF) that were paired with three outcomes (O1, O2 and O3, respectively). Each type of trial in phase 1 was presented 20 times in pseudorandom order (i.e., avoiding more than two consecutive trials of the same type). During phase 2, the participants were exposed to 15 B – O1 trials pseudorandomly intermixed with 15 G – O2 trials. BB of Cue A should be observed because, during the first phase, A was always compounded with a competing cue, B, which, during the second phase, was followed by the same outcome (O1). G was a new cue that was presented only during phase 2 and which shared the outcome with the compound-cue CD, as in an IbC design. Therefore, IbC should be observed when testing either C or D because none of these cues had been trained in compound with G. The compound cues E and F, and the corresponding outcome, O3, were not presented in phase 2 and therefore both E and F served as overshadowing controls. In the test phase, Cues A, C, and E were presented only once per participant in counterbalanced order.

As can be seen in Table 1, the delay factor was treated as a between-subjects manipulation. Thus, in the Delay:2-Test group, a delay was introduced between phase 2 and the test phase. This delay was equivalent to the duration of the second phase (i.e., 225 s). On the other hand, in the Delay:1-2 group, a delay of the same duration (i.e., 225 s) was introduced between the first and the second phase.

2.3.2. Procedure

The procedure of the experiment was similar to that used in Luque et al.'s (2009) study. First of all, participants read the instructions and had the opportunity to clarify any doubts. The participants could earn points by responding on each trial. In order to do so, they had to learn the relationships between some coloured rectangles and some fictitious plants which played the role of cues and outcomes, respectively.

The first thing to occur immediately before each trial was the randomly ordered display of the three possible response options, one per outcome, consisting of the labelled plant photos. This was done so that participants could know, before the current trial started, which key should be pressed during that trial for each of the three possible outcomes. Under each response option, a scroll bar, together with a text box, was displayed. Then, the cue or cues appeared for 2.5 s during which the participants had to respond which of the three plants they thought was related with the cue (or cues) present on the screen on that trial. Once the 2.5 s time had passed, the cue (or cues) disappeared, which was indicated by the rectangle (or rectangles) taking on the grey colour.

To respond, the participants responded by pressing either Key "1", or Key "2", or Key "3" for the plant (i.e., the response option) placed at the left, middle, or right bottom of the screen, respectively. While a given response key was kept pressed, the participants 'bet' points for the corresponding option. The number of responses for each option was indicated numerically from 0 to 100 in the corresponding text box, as well as analogically by the movement of the scroll-bar face from left to right. Once the cue had disappeared, pressing any of the response keys had no effect. On each trial, participants earned as many points as those bet for the correct outcome, and lost as many points as those bet for an incorrect outcome.

After each bet, feedback was given consisting of (a) the correct plant, which was indicated by keeping it visible, and removing the remaining ones from the screen, (b) the amount of points earned on a given trial, which was indicated in a text box placed at the centre of the screen over the labelled plant photos, and (c) the total points gained throughout the training trials, which was indicated in a text box at the right top of the screen. The correct plant and the amount of points earned in a given trial were presented for 0.5 s. After the feedback, the next trial started after an inter-trial interval (ITI). The ITI duration was pseudorandom with a range between 0.5 and 1.5 s, and a mean of 1 s.

During the delay, the screen showed a message indicating that participants were in a rest time. The message remarked to the participants that they had to keep watching the screen during all the rest time. Indeed, when 10 s remained to the end of the delay, a new message within a red square appeared intermittently at the centre of the screen warning for the end of the rest time.

The test phase consisted of additional trials which only differed from the previous training trials in two respects: cues were presented for 5 s, and the participants received no feedback after their bets. The amount of the bet made for the correct outcome on each test trial was taken as the dependent variable used in the data analysis (see Luque et al., 2009 or Pineño et al., 2000 for a similar dependent variable).

3. Results

Data were excluded from two participants whose total points earned at the end of the two learning phases were two standard deviations away from the mean of the whole sample (Final $N = 162$: 82 in the Delay:2-Test group, and 80 in the Delay:1-2 group). Fig. 1 displays the mean number of responses on each test trial, i.e., the mean of the points bet on the correct outcome in trials testing for Cues A, C, and E.

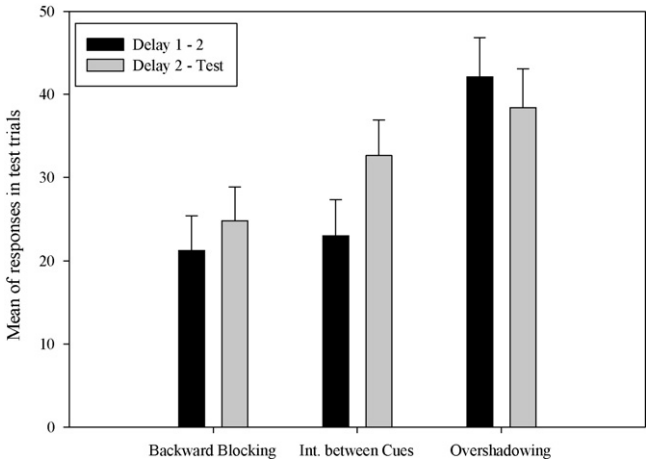

Fig. 1. Mean number of responses (mean points bet) for target cues during test trial in Delay:1-2 and Delay:2-Test groups. Error bars represent standard error.

In order to confirm the predictions established in the introduction, we firstly assessed whether the interaction between delay and IbC was significant. Secondly, we repeated the same analysis for BB. Because the two ANOVAs needed to test SR on BB and IbC, we used the FREGW method to prevent an alpha inflation as presented in Hsu (1996).

Thus, first, we performed a mixed 2×2 ANOVA with participants' responses at test as the dependent variable, and IbC (IbC: interfered cue C vs. overshadowed cue E), and Delay (Delay:1-2 group vs. Delay:2-Test group) as within-subjects and between-subjects factors, respectively, revealing a significant main effect of IbC, $F(1, 160) = 15.93$, MSE = 12559.44, $p < .001$, $\eta^2 = .091$, and a significant IbC $\times$ Delay interaction, $F(1, 160) = 4.6$, MSE = 3632.23, $p = .033$, $\eta^2 = .028$. There was no significant effect of Delay, $F(1, 160) < 1$. The significant interaction effect motivated a *t*-test for related measures in order to analyze the IbC effect within each level of the variable Delay. The analyses performed showed that the number of responses to the interfered cue, Cue C, were significantly lower than the number of responses to the overshadowed cue, Cue E, in the Delay:1-2 group $t(79) = 3.72$, $p < 0.001$ but not in the Delay:2-Test group $t(81) = 1.61$, $p = 0.111$. These results confirmed our prediction and replicated Pineño et al.'s (2000) results: IbC was affected by the delay. Specifically, we found a spontaneous recovery (SR) from interference when the delay was introduced between the second training phase and the test phase, i.e., IbC was only significant when the delay was placed between the first and the second phase.

Indeed, this interpretation of the interaction is, to a great extent, corroborated by the analysis of the differential effect of the delay on participants' responses to the interfered and the overshadowed cues (Cues C and E, respectively). A look at the mean responses to Cue E in Groups Delay:1-2 and Delay:2-Test (42.1 and 38.4, respectively) reveals that participants in both groups tended to respond in a rather similar way [$t(160) = 0.56$, $p = .576$]. However, participants' responses to Cue C in Group Delay:2-Test tended to be clearly higher than in Group Delay:1-2 (means were 32.6 and 22.9, respectively). Unfortunately, though this trend was quite consistent with our interpretation of the interaction, it was not significant [$t(160) = 1.583$, $p = .115$]. Probably the fact that these were between-subjects comparisons rendered the statistical tests less sensitive to differences between means.

Then the effect of the delay over BB was analyzed. For this, we performed another mixed 2×2 ANOVA with participants' responses at test as the dependent variable, and BB (BB: blocked cue A vs. overshadowed cue E) and Delay (Delay:1-2 group vs. Delay:2-Test group) as within-subjects and between-subjects factors, respectively. This analysis revealed a significant main effect of BB, $F(1, 160) = 30.35$, MSE = 24112.09, $p < .001$, $\eta^2 = .159$ and no other significant effect [Delay effect: $F(1, 160) < 1$, BB $\times$ Delay interaction: $F(1, 160) = 1.34$]. These results indicate an effect of BB that was unaffected by the delay. Thus, we did not find any evidence of SR from BB.

4. Discussion

The objective of the present study was to assess whether BB and IbC in humans are produced by different mechanisms, as suggested by the most popular theories of BB (e.g. Dickinson and Burke, 1996) and IbC (Matute and Pineño, 1998b; Miller and Escobar, 2002). The strategy followed was to analyze the differential effect of time delay on BB and IbC. IbC has been shown to be highly sensitive to the inclusion of a short delay between the second training phase and test trials in previous studies (Pineño et al., 2000). As for BB, though the effect of such a delay has not been assessed in humans so far, SR is not expected to be found according to the dominating theories in the field. If BB and IbC were produced by the same mechanism, the manipulation of delay performed in the present experiment should produce the same changes in both effects.

The results of the current experiment showed the SR effect in the case of IbC, but not in the case of BB. First, as it was expected, we obtained a reliable SR from IbC. When the delay was placed between training phases 1 and 2, we found IbC, replicating previous results of IbC in humans (e.g., Luque et al., 2009). When the delay was moved just before the test, the IbC effect decreased and became non significant. It is important to recall that the IbC effect was assessed by comparing participants' responses to the interfered cue (Cue C) with participants' responses to the control (overshadowed) cue (Cue E). Otherwise, we could not strictly speak of an IbC effect. Thus, we replicated the only study of SR from IbC, i.e., Pineño et al. (2000). As it was explained in the introduction, this result is favorable to the explanation of IbC in terms of retrieval interference mediated by context.

On the other hand, the BB effect was unaffected by where the delay was placed (between learning phases or prior to the test) and, hence, we obtained a significant main effect of BB that did not interact with the factor Delay. As in the previous case, it is again important to recall that, to strictly assess a BB effect, we had to compare participants' responses to Cue A (the cue under the BB treatment) with participants' responses to the control cue (Cue E).

Considering both results it becomes unlikely that both effects were caused by the same mechanism, as it was proposed by Escobar et al. (2002). It seems more plausible that IbC relied on retrieval mechanisms dependent on the (temporal/physical) context present at test, while BB was caused by mechanisms of cue competition for which the role of context is immaterial (Dickinson and Burke, 1996; Miller and Matzel, 1988; Van Hamme and Wasserman, 1994).

Despite all of the above, we would like to make clear that we are not claiming that BB should not be affected by the passage of time in any case. The delay manipulation used in our experiment is a specific manipulation that has been shown to affect IbC in humans. But other delay manipulations could also affect BB. For example, Pineño et al. (2005) found SR from BB in rats with a very long delay, 20 days, between phase 2 and test trials, but using a control group without any delay. They explained this result by claiming that the delay might have weakened the A–B within-compound association learned in training phase 1, which, according to the comparator hypothesis (Miller and Matzel, 1988), is a necessary condition for BB. Thus, the apparent conflict between this result and ours vanishes because the amount of time between the end of phase 1 (in which all within-compound associations should have been learned)

and the test phase in our experiment was the same for both the Delay:1-2 and the Delay:2-Test groups. Thus, according to Pineño et al.'s (2005) account, both groups should show the same level of BB. The lack of another control group without any delay introduced prevents us from drawing any further conclusion about this.

In sum, the present experiment shows that, although BB and IbC are very similar regarding the experimental design and the magnitude, they are likely to be the outcome of very different mechanisms. Further research is needed to specify the characteristics of these mechanisms, how they interact, and the circumstances under which they do so.

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