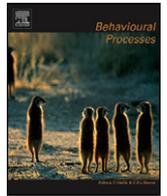




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Interference between cues of the same outcome in a non-causally framed scenario

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ABSTRACT

Retroactive interference between cues of the same outcome (i.e., IbC) occurs when the behavioral expression of an association between a cue and an outcome (e.g., $A \rightarrow O1$) is reduced due to the later acquisition of an association between a different cue and the same outcome (e.g., $B \rightarrow O1$). Though this interference effect has been traditionally explained within an associative framework, there is recent evidence showing that IbC effect may be better understood in terms of the operation of higher order causal reasoning processes. The results from Experiments 1 and 2 showed an IbC effect in a learning task within a game scenario suggesting non-causal relationships between events. Thus, these results showed that IbC may have a diverse origin, one of them being of an associative nature.

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The comprehension of associative learning has greatly benefited from the study of empirical phenomena such as interference (Bouton, 1993). Generally, interference refers to a difficulty in the recall of specific information due to the acquisition, at a different moment, of some other information. Among interference phenomena, retroactive interference between cues of the same outcome (IbC hereafter) occurs when the behavioral expression of an association between a cue and an outcome (e.g., $A \rightarrow O1$) is reduced due to the later acquisition of an association between a different cue and the same outcome ($B \rightarrow O1$; see Table 1). IbC represents a general learning phenomenon as it has been shown both in animal as well as in human learning preparations and using analogous treatments (see Escobar et al., 2001; Escobar and Miller, 2003; in the animal conditioning literature; and Escobar et al., 2002; Matute and Pineño, 1998a; Pineño et al., 2000; Vadillo et al., 2008; in the human contingency learning literature). Despite its simplicity, at least regarding the number of cues and outcomes involved, standard associative models (e.g., Bouton, 1993; Rescorla and Wagner, 1972; Stout and Miller, 2007) cannot offer an explanation of IbC. Probably, the most cited explanation of IbC, still within an associative framework, comes from an extension of Bouton's (1993) theory (e.g., see Escobar et al., 2001; Matute and Pineño, 1998a; Miller and Escobar, 2002). This explanation capitalizes on the similarities between IbC and interference between outcomes (i.e., the interference produced when a single cue is associated with different outcomes); especially, those similarities concerning the existence

of equivalent contextual effects in both phenomena. The explanation builds 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), in which case such associations are learned separately. Second, the context in which a given association is acquired has the capacity of priming such association in detriment of others. Consequently, if two associations have a common cue or outcome, our cognitive system tends to diminish the retrievability of each association in the context in which the other was learned. According to these assertions, interference between cues occurs because: (1) the associations learned in the first and the second training phases have the outcome as a common element; (2) the context for the test is the same (or almost the same) as for the second training phase.

However, more recently, Cobos et al. (2007) and Luque et al. (2008) provided evidence showing that IbC can be produced by non-associative causal reasoning processes provided that participants interpret the learning task according to a diagnostic causal situation, i.e., a situation in which cues and outcomes play the role of effects and causes, respectively. Additionally, they argued that previous demonstrations of IbC in human contingency learning are based on the use of cover stories that might have strongly induced non-arbitrary diagnostic causal interpretations of the learning tasks. For instance, in the spy radio task (the most successfully used), participants have to rescue as many refugees as possible in a war zone plagued with hidden mines by repeatedly pressing the space bar to place the refugees in a series of trucks. The colored lights of a spy radio, which play the role of cues, tell the state of the road (free of mines, mined, or closed) on each trial. The outcome consists of a message which includes information about the actual

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Table 1
Experimental design for interference between different cues of the same outcome.

Conditions	Training phases		
	Phase 1	Phase 2	Test phase
	Context X	Context Y	Context Y
Experimental	A → O1	B → O1	A?
	C → O3	C → O3	
Control	A → O1	B → O2	A?
	C → O3	C → O3	

Note: Letters A–C stand for cues and O1–O3 refer to the different outcomes; letters X and Y stand for the different contexts.

state of the road. In this case, the cover story is very likely to induce participants to interpret the colored lights of the spy radio as effects of the state of the road on some kind of detector device. After all, people are frequently exposed to lights and electronic indicators which provide diagnostic information about devices or about the state of things. A very similar analysis can be made in the case of the Martians and the airplane task. In both cases the cues played the role of indicators of some events (the outcomes) that could be easily thought of as having produced the changes of state in these cues.

Thus, it would be interesting to know whether IbC in human learning can also be found when cues and outcomes are not framed within scenarios that fit familiar diagnostic causal schemes so well. This would give some support to the idea that IbC can be generated not only by causal reasoning processes, but also, by associative mechanisms. The objective of the experiments reported here was to find IbC in a task in which the instructions did not suggest the existence of causal relationships fitting some familiar causal scheme. This was achieved by framing the learning task within a game scenario suggesting arbitrary relationships between events.

1. Experiment 1

1.1. Method

1.1.1. Participants

The participants were 31 undergraduate Psychology students from the University of Málaga who took part in the experiment for course credits.

1.1.2. Apparatus

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

1.1.3. Procedure

Participants started by reading the instructions on the computer screen. The instructions contained a detailed description of the task described here. Once they had finished and all possible doubts were clarified, the experimental task started straight away.

Throughout the task, participants had to learn the set of relationships between the different cues and outcomes shown in Table 1. Cues A–C were represented by different colored rectangles (i.e., brown, yellow, and blue; counterbalanced) and outcomes O1–O3 were pictures of three tropical plants labeled with fictitious names (i.e., “Dobe”, “Yamma”, and “Kollin”; both pictures and picture names’ assignments to the different outcomes were fully counterbalanced and the same as in Cobos et al., 2007). Participants were instructed that each color predicted the correct plant on each trial. Thus, after seeing the cue present on a particular trial, participants had to press the key from the computer keyboard corresponding to the outcome with which this cue was related.

A system of gaining (or losing) points was established to facilitate the learning of these relationships. Specifically, on each trial,

participants could gain (or lose) points by pressing the key for the correct (or incorrect) outcome. The actual number of points won (or lost) depended on the number of key-presses that had been made on the correct (or incorrect) outcome. In addition, keeping the key pressed was also allowed for a more rapid winning (or losing) of points.

Thus, on each trial, participants could first see the three possible outcomes at the bottom of the screen (i.e., right, centre, or left). These positions were randomized for each trial. Two seconds later, the cue was present at the upper centre of the screen and, only then, participants’ responses were allowed to the right, centre, or left plant (i.e., keys 1–3 from the keyboard, respectively) with which they thought the cue was related. The cue was present for 2.5 s and responses were allowed during that period of time. The number of key responses (the number of points that could be won) was indicated graphically by a horizontal scroll bar moving from left to right as response rate increased, and numerically by a small text box on top of the plant picture. No more than 100 responses were allowed on each trial though the time allotted hardly allowed for more than 60 responses.

Once participants had made their responses, the correct outcome was indicated. Specifically, the two incorrect outcomes disappeared from the screen. This way, participants could learn the different cue–outcome relationships programmed. In addition, the number of points won (or lost) in the trial was indicated numerically towards the centre of the screen. Finally, the cumulative number of points that had been won or lost throughout trials could be seen in a text box at the upper right corner of the screen. Participants had to press “X” for the next trial.

For both conditions, during Phase 1, all participants were exposed to 10 A–O1 trials intermixed with 10 C–O3 trials. Then, in Phase 2 participants in the experimental condition received 10 B–O1 trials intermixed with 10 C–O3 trials, whereas participants in the control condition received 10 B–O2 trials intermixed with 10 C–O3. The order of trials was randomized, with the constraint that no more than two consecutive presentations of the same trial type were allowed. At the end of the second phase a test trial was included with no interruption. Participants were not told about this test trial during the instructions; for participants, the test trial was an additional training trial in which cue A was presented. The test trial only differed from the training trials in that the presentation time for the cue and, therefore, the time in which responses were allowed, was 5 s instead of 2.5 s. Additionally, no feedback was provided at test. It is important to note that test trial was exactly the same in both conditions.

Finally, Phases 1 and 2 were signaled to participants by different contextual cues (X or Y) consisting of a big colored rectangle as wide as the screen width at the centre of the screen. The rectangle was either green or red and the assignment of each color to each phase was counterbalanced across participants. This contextual cue was included here for comparability to other procedures used in the literature (e.g., Cobos et al., 2007). The same colored rectangle used for Phase 2 training trials was used for the test trial.

1.2. Results and discussion

The data from two participants whose level of responding was not above chance were excluded from the analysis (i.e., they scored a negative number of total points at the end of the task; see Luque et al., 2008 for a similar selection criterion). In addition, the data from 13 participants could not be analyzed as they seemed to have misunderstood the task instructions concerning how to respond on the response key. Specifically, they only responded once per training and test trials, that is, they did not repeatedly pressed the response key (or left it pressed) during the time allotted to respond. As a result, 16 participants from the initial sample were included

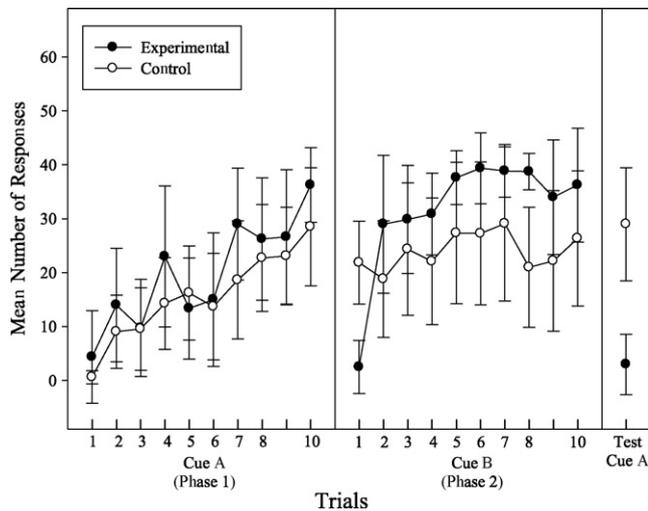


Fig. 1. Experiment 1 mean number of responses for cue A (i.e., Phase 1) and cue B (i.e., Phase 2; see Table 1) during training trials and the test trial. Error bars represent 95% confidence interval.

in the analysis: 8 from each of both the experimental and the control group. Fig. 1 displays the mean number of responses on each trial with cue A (from Phase 1) and cue B (from Phase 2). As it can be seen, no difference in acquisition was found during Phase 1 (when the target A–O1 relationship was acquired) between the experimental and control conditions. To evaluate the effect of conditions in the acquisition of A–O1 associations, a repeated measure ANOVA was performed with Condition (experimental vs. control) as a between-subject factor and Trials (1–10) as a within-subject factor. A significant main effect of Trial was found $F(9, 126) = 9.92$, $p < .001$, showing a learning effect across trials. All F 's involving the Condition factor were < 1 . Thus, the target cue–outcome relationship A–O1 was learnt in a similar way in both conditions. Although cue A was presented for 5 s at test, only responses during the first 2.5 s were analyzed for comparison purposes with the training trials. As shown in Fig. 1, this number of responses was lower in the experimental than in the control condition. This impression was confirmed by a Student's t -test for independent measures $t(14) = -4.28$; $p < .001$ (two tailed).¹

Thus, for the first time, an IbC effect was found in a task where cues and outcomes were not framed in a scenario susceptible of a causal interpretation. Hence, a causal reasoning account is hardly applicable to the present results.

However, there was a procedural difference between both conditions that may speak of a more strategic rather than of a genuine associative IbC effect. Specifically, in the experimental condition, the response option associated to outcome O2 was always present despite that such outcome never occurred. Thus, participants may have responded to this outcome at test simply because, as it had never occurred before, they might have reasoned that it was appropriate for this new situation (i.e., a situation in which cue A was present in Context Y for the first time). Responding with the outcome O2 option to cue A could, in turn, have reduced the number of outcome O1 responses. In fact, the mean number of outcome O2 responses at test was 19.75 and 0.12 in the experimental and the control condition, respectively. It should be noted that other demonstrations of IbC are also potentially affected by this procedu-

ral artifact (e.g., Pineño et al., 2000). Experiment 2 served to control for this alternative explanation of the effect. In addition, to improve participants' understanding of the task instructions, specific verbal instructions were added highlighting the fact that to obtain a greater number of points per trial, the key corresponding to the correct outcome should be pressed repeatedly or be kept pressed.

2. Experiment 2

The aim of Experiment 2 was to replicate the results obtained in Experiment 1 improving the procedure to overcome the shortcomings mentioned above. First, outcome O2 was not included in the experimental condition of Experiment 2 to avoid the strategic response mentioned. Second, verbal instructions emphasized to participants that in order to do the task correctly and obtain more points it was necessary to respond repeatedly or to keep the key pressed in each trial while the cue was present.

2.1. Method

2.1.1. Participants

The participants were 26 undergraduate Psychology students from the University of Málaga who took part in the experiment for course credits.

2.1.2. Apparatus

The same apparatus was used as in Experiment 1.

2.1.3. Procedure

The procedure was as in Experiment 1 except for the following variations. First, outcome O2 did not occur in the experimental condition. Thus, only two plant photos and theirs corresponding names were included in this experimental condition (see Section 1.1.3). Hence, response options were reduced to two responses, keys 1 and 2 for the left and right plant, respectively. In addition, initial instructions were modified accordingly. The control condition was as in Experiment 1.

Once participants had read the written instructions, they all received specific verbal instructions indicating that, for a good performance in the task, they should try to obtain as many points as possible on each trial. To achieve this, they should press the correct key as many times as possible or leave the key pressed while the cue was present.

2.2. Results and discussion

As in Experiment 1, the data from two participants who had scored a negative number of points at the end of the training stage were excluded from the analysis. Despite the attempts made to improve participants understanding of the instructions, the responses of four participants could not be included in the analysis for the same reasons as in Experiment 1. Thus, a total of 20 participants were included in the data analysis: 11 from the experimental condition and 9 from the control condition. Nevertheless, the verbal instructions appeared to be partially effective in reducing the number of participants' data that could not be analyzed (from 45% in Experiment 1 to 15% in this experiment).²

¹ If the mean number of responses is analyzed at the end of the trial test (5 s), there was also a lower mean number of responses in the experimental than in the control condition (Experimental: $M = 11.88$, $MSE = 11.73$, Control: $M = 83$, $MSE = 27.47$). This impression was confirmed by a Student's t -test for independent measures $t(14) = -4.67$; $p < .001$ (two tailed).

² The number of participants whose data could not be analyzed in both experiments deserves some comments. Though they only gave a single response, their performance was in line with the IbC effect found. Specifically, in Experiment 1, 7 and 6 participants were excluded from the control and experimental groups, respectively. During test, all those participants excluded from the control group responded to target cue A with one press of the key corresponding to outcome O1 (i.e., the correct outcome). On the other hand, in the experimental group, only two of them chose the key corresponding to outcome O1 and the other four, then, chose the key

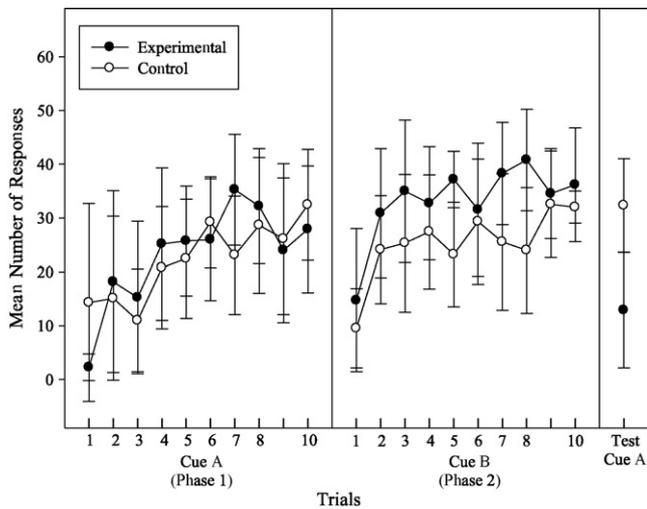


Fig. 2. Experiment 2 mean number of responses for cue A (i.e., Phase 1) and cue B (i.e., Phase 2; see Table 1) during training trials and the test trial. Error bars represent 95% confidence interval.

Fig. 2 displays the mean number of responses for each condition on each trial with cue A (from Phase 1) and cue B (from Phase 2). As it can be seen, no difference in acquisition was found during Phase 1 between the experimental and control conditions. This impression was confirmed by a repeated measure ANOVA with Condition (experimental vs. control) as a between-subject factor and Trials (1–10) as a within-subject factor. A significant main effect of Trial was found $F(9, 162) = 4.65, p < .001$. All F s involving the Condition factor were < 1 . Thus, the two groups can be considered as equivalent regarding the acquisition of the target A–O1 cue–outcome relationship. As in Experiment 1, the mean number of responses made during the first 2.5 s in which cue A was present at test was analyzed. A Student’s t -test for independent measures showed that the number of responses was significantly lower in the experimental than in the control condition, $t(18) = -2.66; p = .016$ (two tailed).³

The IbC effect found in Experiment 1 was replicated here. Thus, the effect found in the previous experiment was not due to the strategy described above concerning the fact that outcome O2 had never occurred during the training phase in the experimental condition. This factor was controlled by having two and three response options in the experimental and control condition, respectively. Note that this asymmetry, on its own, should only have benefited the number of outcome O1 responses at test in the experimental over the control group, which runs against the IbC observed.

3. General discussion

Overall, the two experiments reported here showed that IbC may be found in tasks in which the cue–outcome relationships

corresponding to outcome O2. In Experiment 2, all participants excluded were from the control condition but, then again, responded to target cue A with one press to the correct key. Thus, it may be argued that the specific distribution of errors described within groups was in agreement with the idea that the behavioral expression of the A–O1 association during test was higher in the control than in the experimental group, and this is in accordance with the IbC reported.

³ If we analyzed the mean number of responses at the end of the trial test (5 s), there was also a lower mean number of responses in the experimental than in the control condition (Experimental: $M = 41.27, MSE = 14.36$, Control: $M = 94.44, MSE = 3.46$). A Levene test for homogeneity of variance showed that both distributions had dissimilar variances, so a Brown-Forsythe test was used. This test showed that the differences between the experimental and control conditions was significant $F(1, 18) = 10.60; p = .004$.

programmed are not framed in scenarios that could be causally interpreted. This would give support to the idea that IbC can be generated not only by causal reasoning processes, but also by associative mechanisms. Causal reasoning processes should only apply in situations where the relationships to be learnt are of a causal nature (i.e., the instructions suggest a causal interpretation for the role of cues and outcomes). Specifically, the causal reasoning account described in Cobos et al. (2007) would only be applicable in learning situations where cues A and B are interpreted as effects of outcome O1, the common cause.

In addition, the IbC found in Experiment 2 could not be attributed to the strategic factor (see above) that potentially affected the results from Experiment 1 and other results in the literature. Thus, the effect found is more likely to be caused by associative processes than in previous experiments. Nevertheless, within the associative framework, the explanation of IbC is not a solved question at all. In addition to the extension of Bouton’s (1993) theory described in the introduction, there are a few other modifications of extant associative theories that have been proposed. For example, Matute and Pineño (1998b), among others, have argued that Van Hamme & Wasserman’s model (Van Hamme and Wasserman, 1994) may account for the effect if it is assumed that backward associations (i.e., outcome–cue associations) are also formed during training. This way, during Phase 2 in the experimental condition, O1 activates a representation of cue A (due to prior O1–A associations formed during Phase 1). Thus, the O1–A association is weakened during Phase 2, as O1 appears in a situation where cue A is absent though expected (i.e., in these cases, the model assumes that the attentional parameter for a cue is negative; hence the decrement in the A–O1 association). In the control condition, the O1–A association is not altered during Phase 2 and hence, the IbC effect. A similar account may be derived from Dickinson & Burke’s revised SOP model (Dickinson and Burke, 1996).

Finally, it should be acknowledged that previous attempts in our laboratory to find IbC in non-causal tasks were unsuccessful (Luque et al., 2008). In addition to Luque et al. (2008), other results show that IbC seems to be somewhat elusive (Lipp and Dal Santo, 2002). Thus, failures in observing IbC are not rare, and the reasons for such failures remain unknown. The experiments reported here are based on a new procedure which includes numerous differences compared to that used by Luque et al. (2008). Thus, it is not clear which of these differences is responsible for the different results obtained. As a tentative hypothesis, it may be argued that IbC might have been facilitated by the assignment of the different outcomes to responses on different keys from the keyboard. At variance with this, in Luque et al.’s (2008) procedure, different outcomes were assigned to different response options but all of them involved the same response key from the keyboard. In fact, this multi-response procedure is closer to those used in paired associates paradigms, in which IbC have been shown in several studies (e.g., Keppel et al., 1971). Future research should shed light whether or not this specific factor, or others, modulate IbC in non-causal tasks.

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