

Positive and negative mediation as a function of whether the absent cue was previously associated with the outcome

Leyre Castro

University of Iowa, Iowa City, IA, USA

Helena Matute

Universidad de Deusto, Bilbao, Spain

After presenting two cues, A and B, together, later pairings of one of the cues alone with an outcome can generate changes in the associative value of the absent cue. These changes can be in the same direction as the present cue (i.e., positive mediation) or in the opposite direction to the present cue (i.e., negative mediation). We found both mediational effects in a human contingency task. In addition, we found that the direction of the change was determined by the existence of a prior association between the absent cue and the outcome. When a prior association exists, the absent cue tends to change its value in the opposite direction to the present cue, whereas when there is no prior association, the absent cue tends to change its value in the same direction as the present cue. Recent associative models (Stout & Miller, 2007) can explain our results.

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There are many occasions in which absent events become the content of learning. If two events always occur together but then one of them is absent we might nonetheless learn something about the absent cue. For example, if we always add milk to our coffee but one day we run out of milk, and we enjoy the coffee even more, our opinion of the goodness of milk in coffee will surely change. If Sarah and Mike always agree with each other, and one day Sarah enthusiastically

praises our new canvas, we will probably think that absent Mike will like it too. These two examples show that we can learn about absent events and, also, that what we learn about the absent event can be opposite or similar to what we learn about the event that is actually present.

In an experimental situation, if two stimuli, Cue A and Cue B, are always presented together, an associative link will be established between their mental representations; once this connection

Correspondence should be addressed to Leyre Castro, Department of Psychology, E11 Seashore Hall, The University of Iowa, Iowa City, IA-52242, USA. E-mail: leyre-castroruiz@uiowa.edu

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has been formed, the representation of one cue can be activated by the associative link it has forged with the other cue. When Cue A is later presented alone, the representation of Cue B will be activated by means of this associative link; because its representation is activated, the absent cue can engage in the learning process (e.g., Dickinson & Burke, 1996; Van Hamme & Wasserman, 1994).

But, what do we learn about that absent event? The rules that govern the formation of associations involving absent events are far from being fully understood. As shown by the examples above, two possibilities are: (a) The representation of an absent event enters into associations in the *same* way as that of a present event, and (b) the representation of an absent event enters into associations in the *opposite* way to that of a present event. Evidence from animal and human associative learning studies suggests that both results are possible. We refer to the former case as positive mediation and to the latter case as negative mediation.

For example, human retrospective revaluation studies have consistently found that, after pairing a compound cue (e.g., a pair of foods, such as chicken and strawberries) with an outcome (e.g., a fictitious allergy), subsequent pairings of one of these cues alone with the outcome (e.g., chicken with allergy) leads to weakened causal attribution to the absent cue (e.g., strawberries). Presumably, the single cue presented in the second stage (chicken) activates a representation of the absent cue (strawberries), and the value of the absent cue changes in the *opposite* direction to that of the presented cue (see Chapman, 1991; Dickinson & Burke, 1996; Wasserman & Berglan, 1998; Williams, Sagness, & McPhee,

1994). Similar results have been obtained with animals (see Harris & Westbrook, 1998; Kaufman & Bolles, 1981; Miller & Matute, 1996).

The case in which responding to an absent cue changes in the same direction as that to a present cue has been reported mostly in animal studies. For example, Brogden (1939) presented dogs with a bell and a light together several times; later, the bell alone was paired with shock. Although the light had never been paired with shock, it elicited a conditioned response at testing; that is, the bell–shock pairings led the animals to change their responding to the absent cue—the light (this is the so-called sensory preconditioning effect). Presumably, during the bell–shock pairings, the bell activated the representation of its associate, the light, so that the light became associated with the shock as well.¹ Very little evidence for this mediational effect has been reported with human participants, except for studies using electrodermal conditioning paradigms (Brogden, 1947, and, more recently, Vansteenwegen, Crombez, Baeyens, Hermans, & Eelen, 2000). To our knowledge, there is no evidence for this effect in human contingency learning, a domain in which retrospective revaluation is often observed.

Thus, in sensory preconditioning, the associative strength of the absent cue changes in the same direction as that of the present cue that is activating its representation—*positive mediation*. But, in the case of retrospective revaluation, the associative strength of the absent cue changes in the opposite direction to that of the present cue that is activating its representation—*negative mediation*. One possible crucial factor is the presence of the outcome during compound training.

¹ Brogden (1939) presented the bell and the light (let us call them Cue A and Cue B, respectively) simultaneously. If the cues are presented sequentially (that is, $A \rightarrow B$), the standard explanation is that, at the time of testing, Cue B is able to elicit a conditioned response by means of the associative chain $A \rightarrow B \rightarrow \text{outcome}$. Although this explanation seems quite straightforward when A and B are presented sequentially, it has problems in accounting for the case in which A and B are presented simultaneously (as in Brogden's study and as it is in our experiment) or using a backward sensory preconditioning procedure (see Hall, 1996, for a complete explanation of these different cases). So, even when the associative chain might have a role at the time of testing in the case of sequential forward sensory preconditioning, it also seems likely that the associatively activated representation of a cue can acquire associative strength during training in the simultaneous and backward procedures. Because we present the cues in our compounds simultaneously, we assume, for the time being, that learning processes are taking place when the cues are absent during training.

Presence of the outcome during compound training

In the studies that show positive mediation, the outcome is never presented during the compound training phase (e.g., Brogden, 1939), whereas the outcome is presented during the compound training phase in the studies that show negative mediation (e.g., Chapman, 1991; Dickinson & Burke, 1996; Wasserman & Berglan, 1998). It thus seems plausible to consider the occurrence or nonoccurrence of the outcome when the compound cue is presented as a relevant factor.

When the outcome is present during the compound phase (as is the case when retrospective reevaluation is observed), the target cue forms a within-compound association with the other cue as well as an association with the outcome. When later its associate cue is paired alone with the outcome, the absent target cue would be retrieved in memory by means of the within-compound association with the present cue. In this way, the association of the absent cue with the outcome has the opportunity to be modified. Learning about the absent cue in this case involves the *modification* of a previously established association. On the other hand, when the outcome is absent during the compound phase, the target cue cannot enter into an association with the outcome. When later its associate cue is paired with the outcome, the absent target cue would be retrieved in memory and will have the opportunity to develop an association with the outcome. In this case, learning entails the *formation* of a new association. Thus, it seems plausible that, when a prior association exists, the absent cue tends to change its value in the opposite direction to that of the present cue—negative mediation—whereas, when there is no prior association, the absent cue forms a new association that is in the same direction as that of the present cue—positive mediation.

Cue–no–outcome association versus no association

At this point, we should clarify what it means to say that a cue has no association with the outcome.

Although it seems a simple matter, the answer is far from being straightforward, especially in human studies, where the cue can be presented without any mention of the outcome or the cue can be explicitly paired with the absence of the outcome.

When no information at all is provided about the occurrence or nonoccurrence of the outcome, the cue is likely to remain “neutral”, in the sense that nothing can be learned about its value as a predictor of the occurrence or nonoccurrence of the outcome. In the case of explicit pairing of the cue with no outcome, the cue is likely to become a predictor of the absence of the outcome; so, it does not remain neutral. For example, Karazinov and Boakes (2004) reported that a cue paired with no outcome yielded ratings as negative as those for a cue involved in conditioned inhibition training. The reason for this result seems to be the knowledge that participants have about the existence of the outcome. The mere expectation of the occurrence of the outcome might endow the context with some excitatory value that might cause the cues paired with no outcome to become inhibitory. Indeed, when inhibition studies are conducted with animals, the target (inhibitory) cue needs be presented in an excitatory context (Miller, Hallam, Hong, & Dufore, 1991).

Let us examine now the case of backward conditioned inhibition. In human contingency studies, pairings of the compound AB with no outcome in a first phase, followed by pairings of Cue A with the outcome, leads to a decrease in ratings of Cue B—backward conditioned inhibition, another negative mediation effect (e.g., Chapman, 1991; Larkin, Aitken, & Dickinson, 1998; Melchers, Lachnit, & Shanks, 2004; Wasserman, Kao, Van Hamme, Katagiri, & Young, 1996; Williams & Docking, 1995).

In a typical backward conditioned inhibition experiment, participants are informed about the absence of the outcome during the compound phase by explicitly pairing the cues with no outcome. Thus, at the end of the compound phase, Cue B will have an association with the outcome—in this case, an inhibitory association. Changes to Cue B in Phase 2 will therefore

imply a *modification* of its prior association. As we hypothesized before, when a prior association exists the absent cue tends to change its value in the opposite direction to that of the present cue. This is exactly what it is observed in backward conditioned inhibition: When Cue A is paired with the outcome in Phase 2, and its value consequently increases, the value of Cue B decreases even more, compared to Phase 1.

For positive mediation to occur, it is probably necessary that the target cue remains in a neutral state at the end of the first (compound) phase; that is, it may be necessary that the outcome has been absent, and no information about it has been provided, so that the target cue does not develop any association with the outcome, instead of developing an inhibitory association with the outcome—or an association with no outcome (Konorski, 1967).

Thus, we hypothesize that, when a cue has already developed an association with the outcome, be this excitatory or inhibitory, the presentation of its associate paired with the outcome (or with no outcome) will lead to changes in the value of the absent cue in the opposite direction as the present cue—negative mediation—whereas when the cue has no prior association with the outcome, the presentation of its associate paired with the outcome (or with no outcome) will lead to an association of the absent cue with the outcome in the same direction as the present cue—positive mediation. In order to test this hypothesis and to illuminate the difference between absence of the outcome and explicit presence of no outcome, in the present study we compared three different compound training possibilities—namely, compounds paired with outcome, compounds paired with no outcome, and simple exposure to the compounds.

EXPERIMENT

In our experiment, we presented participants with three compounds, AB, CD, and EF, in the first training phase, and then, in Phase 2, we presented Cue A alone followed by the outcome and Cue E

alone followed by no outcome (fillers GH and G were presented as well). We studied the influence of presenting the compound cues in Phase 1 paired with: (a) presence of the outcome, (b) presence of no outcome, and (c) absence of the outcome (mere exposure to the cues). We wanted to see whether later, when one of the cues of the compound is paired with the outcome (or no outcome), the value of the absent cue changes in the same or in the opposite direction to that of the present cue depending on the type of training given in Phase 1.

In addition, we thought that the specific cover story used could be critical (see Melchers, Üngör, & Lachnit, 2005b, for different results depending on the experimental task). Some authors have classified predictive relationships into two types: causal and structural (see, for example, J. H. Holland, Holyoak, Nisbett, & Thagard, 1986; Shanks, 1995). When a causal relationship exists, organisms learn that an event generates another event after a temporal interval, whereas when a structural relationship exists, organisms learn to predict a feature of an event from other features that occur at the same time (as in category learning, for example).

Common scenarios in retrospective revaluation studies, like the food–allergy task (e.g., Wasserman, 1990), entail a causal relationship. In this case, people might be trying to find which event is *the* cause of the effect, so that the causal value of one cue is discounted in favour of a stronger cue, a process that leads to negative mediation. But other scenarios, like the symptoms–disease task (e.g., Shanks, 1991) might reflect a structural relationship. That is, the symptoms can be considered to be the constituents of the disease, so that when one of the symptoms becomes associated with the disease, the others may be assumed to become associated with the disease as well, a process that leads to positive mediation. If the task scenario plays a role, then a structural cover story should help promote positive mediation, whereas a causal cover story should promote negative mediation.

At the same time, the distinction between the food–allergy task and the symptoms–disease

task could be conceptualized as a distinction between a predictive and a diagnostic task. In a predictive task, participants are shown a cause (e.g., food) and have to predict the occurrence of the effect (e.g., allergy). In a diagnostic task, the presentation of the causal information is reversed, so that participants are shown an effect (e.g., symptom) and have to predict the occurrence of the cause (e.g., disease). Although many researchers have found no differences between predictive and diagnostic scenarios (e.g., Arcediano, Matute, Escobar, & Miller, 2005; Baker, Murphy, Mehta, & Baetu, 2005; Cobos, López, Caño, Almaraz, & Shanks, 2002; Matute, Arcediano, & Miller, 1996; Price & Yates, 1995; Shanks & López, 1996), others have found negative mediation with predictive scenarios but not with diagnostic scenarios (e.g., Tangen & Allan, 2004; Waldmann, 2000; Waldmann & Holyoak, 1992). Thus, regardless of the symptoms–disease task being understood as a structural or as a diagnostic task, the current evidence suggests that it is more likely to obtain negative mediation with the food–allergy task; the symptoms–disease task might be more likely to yield either no effect (if it is understood as a diagnostic task) or positive mediation (if it is understood as a structural task).

In the present experiment, we used a task that permits identical materials to be described in different ways. All of the participants had to learn the relationship between different substances in the blood and Hamkaoman disease—cues and outcome, respectively. Half of the participants (causal groups) were told that the substances in the blood may be the cause of Hamkaoman disease, so that a causal relationship between the cues and the outcome was described. The other half (structural groups) were told that the substances in the blood may be constituents of Hamkaoman disease, so that a structural relationship between the cues and the outcome was described.

We expected to find negative mediation when either outcome or no outcome was presented in Phase 1 (O and no-O groups) and to find positive mediation when there was no information about the outcome (exposure groups). In addition, we

expected the structural task to facilitate positive mediation and the causal task to facilitate negative mediation. Thus, we expected to find the largest negative mediation effect in the O and no-O groups with a causal cover story and the largest positive mediation effect in the exposure group with a structural cover story.

Method

Participants and apparatus

A total of 150 students at the University of Iowa took part in this experiment and received course credit for their participation. Participants were randomly assigned to six groups: O–causal, no-O–causal, exposure–causal, O–structural, no-O–structural, and exposure–structural, yielding 25 participants in each of the groups. Between 1 and 4 participants were studied concurrently on each of four identically configured computer workstations.

Stimuli

Eight different pictures of chemical substances of different colours (*red, orange, lilac, green, blue, yellow, pink, and white*) served as cues, and Hamkaoman disease served as the outcome. The cues were counterbalanced following a partial Latin square design, which ensured that every substance was equally often assigned to each cue role. When two cues were presented, the picture of each specific substance appeared half of the trials on the left side of the screen and half of the trials on the right side of the screen. When only one cue was presented, the picture of the substance appeared centred on the screen.

Procedure

Participants sat in front of a workstation and were introduced to a scenario in which they played the role of a doctor trying to discover the relation between a number of substances in blood and Hamkaoman disease.

We included two between-group factors: training in Phase 1 (outcome vs. no outcome vs. exposure), and causal versus structural scenario. The combination of these two factors yielded an

experimental design with six groups. The design of the experiment is summarized in Table 1.

During Phase 1, all groups observed four compounds, consisting of two substances each: AB, CD, EF, and GH. Each of the compound cues was presented 20 times in random order. In this first phase, the two exposure groups were presented with just the compound cues, and they were not informed about the occurrence or nonoccurrence of the outcome during this phase. The following instructions appeared on the screen at the beginning of the first phase for exposure groups:

We would like you to imagine that you are a specialist who tries to discover the relationship between different substances in the blood and diseases. In an attempt to accomplish this task, you will first observe different patients' files which show different substances in the blood. You will see a separate screen for each patient. First, you will see files with the substances that each patient has, but you will not know yet whether or not the patients have Hamkaoman disease. Nonetheless, to familiarize yourself with the substances at this point will help you to decide later on. In order to help you to remember the substances, you will be asked to press the initials of each substance when it appears on the screen.

The sentence "you will not know yet whether or not the patients have Hamkaoman disease," adapted from Graham (1999), intended to maintain the cues in a neutral or ambiguous state. Participants were asked to press the initials of each of the substances in the order that they appeared on the screen; this was done to make

sure they were reading the screen and paying attention to the task. After entering the correct initials, the participants could proceed to the next trial. On completion of Phase 1, the following instructions appeared on the screen:

Now you will be presented with patients' files in which some of them will have Hamkaoman disease, and some of them will not. You will be shown the name of only one substance and will be asked to indicate whether or not you believe the patient will have Hamkaoman disease. After you make your prediction, the computer will inform you of the correct answer.

On each trial of Phase 2, participants were required to answer whether or not Hamkaoman disease would appear. Once participants clicked on the "Yes" or "No" button, the actual outcome (Hamkaoman disease or no Hamkaoman disease) was presented. Then, participants could proceed to the next trial.

The instructions for the rest of the groups: O-causal, O-structural, no-O-causal, and no-O-structural, were similar, except that they were told, from the beginning of Phase 1, that they would see patients who could have or not have Hamkaoman disease, and they would have to predict on every trial whether or not each of the patients had the disease.

In groups O-causal and O-structural, compounds AB, CD, and EF were always followed by Hamkaoman disease, whereas the filler compound, GH, was always followed by no Hamkaoman disease. We included compound

Table 1. *Experimental design*

Group	Training		Testing
	Phase 1	Phase 2	
Exposure-structural Exposure-causal	AB, CD, EF, <i>GH</i>		
O-structural O-causal	AB→O, CD→O, EF→O, <i>GH→no-O</i>	A→O, E→no-O, <i>G→no-O</i>	B, C/D, F
No-O-structural No-O-causal	AB→no-O, D→no-O, EF→no-O, <i>GH→O</i>		

Note: Cues B, C/D, and F are the target cues, and Cues A and E are their associates. Cues G and H are filler cues (shown in italic). "O" indicates that the outcome was presented; "no-O" indicates that no outcome was presented.

GH in order to prevent participants from expecting the outcome to follow all of the compound cues. In groups no-O-causal and no-O-structural, compounds AB, CD, and EF were always followed by no Hamkaoman disease, whereas the filler compound GH was always followed by Hamkaoman disease; here, the inclusion of compound GH was intended to prevent participants from expecting no outcome to follow all of the compound cues.

With regard to the description of the relationship between the cues and the outcome, the causal groups were told in the instructions at the beginning that they had to “discover to what extent certain new types of substances in the blood cause Hamkaoman disease”, and that they would have “to rate the likelihood that each substance is cause of Hamkaoman disease”. In addition, on each trial they had to answer the question “Do you think this patient will have Hamkaoman disease?”

On the other hand, the structural groups were told that they had to “discover to what extent certain new types of substances in the blood are related to Hamkaoman disease”, and that they would have “to rate the likelihood that each substance is indicative of Hamkaoman disease”. In addition, on each trial they had to answer the question “Do you think this patient has Hamkaoman disease?”

In Phase 2, Cues A, E, and G, were presented. In all six groups, Cue A was always followed by the outcome, whereas Cues E and G were always followed by no outcome. Each of the cues was presented 20 times each in random order. Once Phase 2 was completed, all groups proceeded to the testing phase.

In testing, the eight chemical substances were presented one by one, in a randomized order. Each of the testing screens was headed by: *Please indicate to what extent this substance is indicative (or cause) of Hamkaoman disease.* Each of the eight substances was presented along with a rating scale ranging from 0 (*definitively no*) to 8 (*definitively yes*). The middle point of the scale, 4, was labelled *possibly*. Participants had to give their ratings by moving a slider along this scale. Once the rating for one cue was entered,

participants advanced to the next screen, so that they did not have the opportunity to alter their prior ratings.

Preanalysis of the data

It would make no sense to expect any effect in those participants who had not learned the contingencies between the cues and the outcome during Phase 2. Therefore, we eliminated from the analysis the data of participants who did not give a correct answer on the last trial of Cue A and on the last trial of Cue E in Phase 2. Using this criterion, the data from 1 participant in group O-causal was excluded. An alpha level of .05 was adopted for tests of statistical significance. In addition, 95% confidence intervals (CIs) are reported for the planned comparisons between individual means.

Results and discussion

Ratings of all of the cues in the six groups are shown in Table 2. Because Cues C and D received the same training, their ratings were averaged for statistical analyses. We refer to this average cue as C/D. Ratings of Cue A, which had been presented followed by the outcome, were high; ratings of Cues E and G, which had been presented followed by the occurrence of no outcome, were low. The critical results, ratings of the target cues B, C/D, and F, are displayed in Figure 1 as well. The order of the cues, $B > C/D > F$, suggests positive mediation in groups exposure-structural and exposure-causal. In contrast, the order of the cues in groups O-structural and O-causal, $B \leq C/D < F$, suggests negative mediation. In groups no-O, a slight tendency to negative mediation can be observed in group no-O-causal and no effect in group no-O-structural.

A 3 (outcome in Phase 1: exposure vs. outcome vs. no outcome) \times 2 (scenario: structural vs. causal) \times 3 (target cue: B vs. C/D vs. F) analysis of variance (ANOVA) revealed a significant main effect of outcome, $F(2, 143) = 158.44$, $MSE = 4.41$, $p < .001$, a significant main effect of scenario, $F(1, 143) = 5.36$, $MSE = 4.41$, $p = .02$, and a significant main effect

Table 2. Mean ratings for all cues in all groups

Cue	Group					
	Exposure		Outcome		No outcome	
	Structural	Causal	Structural	Causal	Structural	Causal
A	7.96 (0.04)	7.64 (0.32)	7.76 (0.17)	7.54 (0.21)	6.76 (0.33)	5.56 (0.37)
B	5.32 (0.32)	3.96 (0.49)	5.04 (0.35)	3.91 (0.32)	1.12 (0.34)	0.16 (0.09)
C	3.48 (0.39)	2.96 (0.36)	5.00 (0.41)	4.54 (0.31)	0.84 (0.25)	0.56 (0.23)
D	3.52 (0.33)	2.84 (0.33)	4.36 (0.39)	4.66 (0.29)	1.08 (0.35)	0.44 (0.19)
E	0.68 (0.23)	0.64 (0.22)	1.64 (0.44)	0.62 (0.30)	0.76 (0.33)	0.52 (0.30)
F	2.60 (0.39)	2.52 (0.50)	5.88 (0.52)	6.29 (0.33)	1.00 (0.35)	0.96 (0.35)
G	0.04 (0.08)	0.04 (0.04)	0.76 (0.44)	0.04 (0.04)	2.96 (0.54)	2.24 (0.50)
H	3.60 (0.46)	2.28 (0.48)	0.60 (0.35)	0.54 (0.26)	5.76 (0.42)	6.84 (0.39)

Note: Means; standard errors in parentheses. Ratings of the target cues are shown in italics.

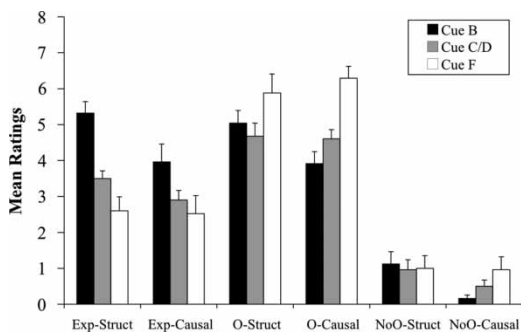


Figure 1. Mean final ratings of target cues B, C/D, and F for the six groups. Error bars indicate the standard error of the means. Exp = exposure. Struct = structural. O = outcome.

of cue, $F(2, 286) = 3.20$, $MSE = 2.21$, $p = .04$. Importantly, the Outcome \times Target Cue interaction was significant, $F(2, 286) = 20.00$, $MSE = 2.21$, $p < .001$, revealing that ratings of the target cues changed differently depending on the status of the outcome during Phase 1. The Scenario \times Target Cue interaction was significant as well, $F(2, 286) = 5.12$, $MSE = 2.21$, $p < .01$, revealing that task scenario also affected ratings of the target cues. The triple interaction was not significant.

In group exposure-structural, the order of the target cues, $B > C/D > F$, suggested positive mediation. Planned comparisons confirmed that ratings of Cue B were higher than ratings of Cue F, $F(1, 143) = 26.46$, $MSE = 3.49$, $p < .001$,

CI 1.[69, 3.74]. In order to explore whether this positive mediation was due to an increase in Cue B or to a decrease in Cue F, we performed additional comparisons. Ratings of Cue B were higher than ratings of control cue C/D, $F(1, 143) = 30.30$, $MSE = 1.36$, $p < .001$, CI [1.03, 2.60], and ratings of Cue F were lower than ratings of control cue C/D, $F(1, 143) = 5.67$, $MSE = 1.78$, $p = .01$, CI [0.08, 1.71]. Thus, both an increase in the ratings of Cue B and a decrease in the ratings of Cue F took place.

In group exposure-causal, the order of the target cues, $B > C/D > F$, suggested positive mediation as well. Planned comparisons confirmed that ratings of Cue B were higher than ratings of Cue F, $F(1, 143) = 7.41$, $MSE = 3.49$, $p < .001$, CI [0.18, 3.06], so that positive mediation did take place. As in group exposure-structural, ratings of Cue B were higher than ratings of control cue C/D, $F(1, 143) = 10.27$, $MSE = 1.36$, $p = .001$, CI [0.95, 2.02], indicating an increase in the value of Cue B. Ratings of Cue F were not significantly lower than ratings of control cue C/D, $F(1, 143) = 1.01$, $MSE = 1.78$, $p = .3$, CI [-0.62, 1.38].

Both groups exposure-structural and exposure-causal exhibited positive mediation. The specific scenario did not reverse the ordering of the target cues, but it seems to have weakened the effect in group exposure-causal.

The direction of the changes was different for groups O-structural and O-causal. In group O-structural, the target cues were ordered $B = C/D < F$. Planned comparisons revealed that the difference between ratings of Cue B and Cue F was nearly significant, $F(1, 143) = 3.72$, $MSE = 3.49$, $p = .05$, $CI [-2.18, 0.50]$. The difference between ratings of Cue B and Cue C/D was not significant, $F(1, 143) = 0.54$, $MSE = 1.36$, $p = .45$, $CI [-0.33, 1.05]$, but the difference between ratings of Cue C/D and Cue F was significant, $F(1, 143) = 11.21$, $MSE = 1.78$, $p = .001$, $CI [-2.23, -0.16]$.

In group O-causal, the order of the target cues, $B < C/D < F$, suggested the existence of negative mediation as well. Planned comparisons confirmed that ratings of Cue B were lower than ratings of Cue F, $F(1, 143) = 16.07$, $MSE = 3.49$, $p < .001$, $CI [-3.18, -1.56]$, so negative mediation took place. The difference between ratings of Cue B and Cue C/D, although in the right direction, was not significant, $F(1, 143) = 2.66$, $MSE = 1.36$, $p = .1$, $CI [-1.33, -0.44]$. The difference between ratings of Cue C/D and Cue F was significant, $F(1, 143) = 17.48$, $MSE = 1.78$, $p < .001$, $CI [-2.35, -1.02]$.

Both groups O-structural and O-causal exhibited negative mediation. Again, the specific scenario did not reverse the ordering of the target cues, but it seems to have weakened the effect in group O-structural.

In groups no-O-structural and no-O-causal, the target cues, B, C/D, and F were all given very low ratings. We found a single effect of cue in group no-O-causal, $F(2, 23) = 2.89$, $MSE = 1.39$, $p < .05$, suggesting backward conditioned inhibition, but none of the planned comparisons were significant.

As we predicted, the mere exposure to the cues without the outcome (and with instructions that maintain the neutral relationship between the cues and the outcome) generates very different results compared to pairing the cues with no outcome. On the one hand, the ratings of target cues in groups no-O were much lower than the ratings of target cues in groups exposure, suggesting that pairing the cues with no outcome

leads participants to consider them as predictors of the nonoccurrence of the outcome, instead of considering them as not having an association with the outcome. On the other hand, we observed negative mediation—albeit small—in group no-O-causal, but positive mediation in group exposure-causal, suggesting that only when the target cues are maintained in a neutral or ambiguous state does training of their associates lead to a change in the value of the target cues in the same direction as the change of their associates. If the target cues are predictors of the nonoccurrence of the outcome, training of their associates leads to a change in the value of the target cues in the opposite direction to the change of their associates. Thus, it seems that what is crucial to generate positive mediation is the lack of an association between the cues and the outcome; if an association has been previously established, then negative mediation is the most likely result.

GENERAL DISCUSSION

We investigated whether the value of absent cues changes in the same (e.g., positive mediation) or in the opposite direction (e.g., negative mediation) as the value of present cues. We found both positive and negative mediation effects. After presenting Cues A and B together, later pairings of Cue A alone with the outcome generated high ratings, not only of the A-outcome association, but of the B-outcome association as well; and, after presenting Cues E and F together, later presentations of Cue E alone with no outcome generated low ratings, not only of the E-outcome association, but of the F-outcome association as well. Thus, the value of the absent cues B and F changed in the *same* direction as the value of the present cues A and E that were activating their representation—positive mediation. This effect has never been previously reported in a human contingency study.

We also found negative mediation, a result frequently reported (e.g., Chapman, 1991; Dickinson & Burke, 1996; Wasserman & Berglan, 1998). In this case, after presentation of Cues A and B

together followed by the outcome, later pairings of Cue A alone with the outcome generated low ratings of the B–outcome association; and, after presentation of Cues E and F together followed by the outcome, later presentations of Cue E alone with no outcome generated high ratings of the F–outcome association. In this case, the value of the absent cues B and F changed in the *opposite* direction to the value of the present cues A and E that were activating their representation.

Most important, we found that the status of the outcome during early compound training influenced later changes in the value of the absent cues: Explicit information about the occurrence or nonoccurrence of the outcome led to negative mediation, whereas no information led to positive mediation. This pattern of results suggests that, when a prior association exists between the cue and the outcome—either the cue as predictor of outcome or the cue as predictor of no outcome—the value of the absent cue changes in the opposite direction to the value of the present cue, and, when no prior association exists, the value of the absent cue changes in the same direction as the value of the present cue.

Excitatory association versus inhibitory association versus no association

After developing an excitatory association with the outcome during the earlier compound phase, the value of the absent cue could still, at least in principle, continue increasing during Phase 2, as it did not reach asymptotic value during Phase 1 (in fact, the value of its companion does increase during Phase 2); however, no studies in human contingency learning or in animal conditioning have found an increase in the value of the absent cue when the associate cue is trained with the outcome after earlier training of the compound cue with the outcome. This fact suggests that, once a cue has acquired an excitatory association with an outcome, it is unlikely that further training of the associate cue alone will produce a change in the absent cue that mimics the change in the associate cue; that is, positive mediation is unlikely under these conditions.

The case in which the compound is initially followed by the absence of the outcome turns out to be more complex because, now, either an inhibitory association or no association might develop between the cues and the outcome. Finding one or the other result will depend on the extent to which the occurrence of the outcome is expected in that context (e.g., Miller et al., 1991). Although controlling for the expectation of the outcome is quite straightforward with animals, it is far from simple with humans, because the presentation of cues followed by no outcome, the most usual procedure, can generate an explicit expectation for the outcome in the training context and thus an inhibitory association between the target cue and the outcome (e.g., Karazinov & Boakes, 2004) even when, in theory, no association between the cue and the outcome should be formed.

Our results show that explicit pairing of the cues with no outcome generates a very different result from that obtained by simply exposing the cues and maintaining them in a neutral state in relation to the outcome. We observed negative mediation in group no–O–causal, but positive mediation in group exposure–causal. This pattern of results suggests that the target cue should have no association with the outcome—that is, it should neither predict the presence of the outcome nor predict the absence of the outcome, in order to later change its value in the same direction as its associate cue. Once a cue has acquired positive or negative value, it seems that it will change in the opposite direction to its associate cue.

The absence of reports of backward conditioned inhibition with animals supports our view. Only when another cue or the context elicits an expectation of the occurrence of the outcome will a cue that is presented without the outcome become inhibitory (e.g., Miller et al., 1991). Therefore, it seems that animals exhibit positive mediation to Cue B after presentations of the compound AB in Phase 1 followed by pairings of Cue A with the outcome in Phase 2 (e.g., sensory preconditioning), because during Phase 1 there is nothing that can elicit the expectation of

the occurrence of the outcome. Without an expectation of the outcome in Phase 1, no backward conditioned inhibition occurs.

In most backward conditioned inhibition studies with humans, the absence of the outcome is made explicit (the cues are paired with no outcome) or the possibility of occurrence of the outcome is mentioned in the initial instructions (thus creating an excitatory context). These procedures lead to the cues becoming inhibitory after compound training in Phase 1. The only exception seems to be Urcelay, Perelmuter, and Miller's (2008) study, who did not give any information about the outcome until the beginning of the second training phase. Urcelay et al. were concerned about the common practice of pairing the cues with no outcome in backward conditioned inhibition studies and, in general, about the effect of informing the participants, before the start of training, about the outcome. So, Urcelay et al. presented their participants with a contingency task in which participants were told simply to observe the events appearing on the screen. In the first phase, two geometrical shapes of different colour, A and X, appeared side by side; in the second phase, only one of these shapes, A, was presented, and immediately after its disappearance, a cross-eyed human baby (the outcome) appeared. Later, the results showed that X had become a conditioned inhibitor. That is, its value had changed in the direction opposite to that of the present cue. According to our argument, X should not develop any association with the outcome in Phase 1 (and thus, should not become an inhibitor later on), because the cues are presented on their own, and no information is provided about the nonoccurrence of the outcome.

However, we believe that the finding by Urcelay et al. (2008) can possibly be understood in terms of the participants' later rehearsal (and revaluation) of the compound trials presented in Phase 1. Participants could mentally present themselves with AX and A–outcome episodes while they actually experienced only the A–outcome trials of Phase 2, so that their training would be close to real training with AX and A–outcome trials

intermixed (Chapman, 1991; see also Melchers et al., 2004). Urcelay et al.'s simple design (training consisted of only three types of trial) may have rendered rehearsing particularly easy. This process may thus have prompted participants to infer during Phase 2 that it must have been the target cue X that was preventing the outcome from happening during Phase 1; thus, the formation of an inhibitory association between X and the outcome could be readily explained.

Structural versus causal and elemental versus configural

Although we did not find that our cover story could switch ratings of the target cues from positive mediation to negative mediation, we observed that the positive mediation effect was largest when the structural task was used, whereas negative mediation was largest when the causal task was used. Although, as we mentioned in the Introduction, the symptoms–disease task could be understood as a diagnostic task, this possibility seems unlikely in our study. On the one hand, there are no reports in the contingency judgement literature that suggest the possibility of positive mediation in a diagnostic setting (just failures or difficulties to obtain negative mediation; Tangen & Allan, 2004; Waldmann, 2000; Waldmann & Holyoak, 1992). On the other hand, our instructions emphasized a structural relationship between the cues and the outcome. Participants had to learn how the cues and the outcome were “related” and to rate the likelihood of each cue being “indicative” of the outcome; cause–effect terms were never used. Thus, under these circumstances, it seems unlikely that the task was understood as a diagnostic task; the structural conceptualization seems to be more adequate.

In a structural relationship, because all of the events are part of the same entity, it makes sense to assume that what happens to one element of the entity can be extended to the other elements; if that is the case, then positive mediation should be more likely to be observed. On the other hand, when the scenario entails a causal relationship, several authors have pointed out that

people normally select one among many potential causes as *the* cause of the event (Hesslow, 1983; Hilton & Slugoski, 1986; Mackie, 1974; McGill, 1989). Discounting of the causal value of one cue in favour of a stronger cue leads to negative mediation. But, this process can happen only if cues within the compound are processed as individual and independent elements.

Actually, although negative mediation effects have been repeatedly observed in human contingency judgements (i.e., Chapman, 1991; Dickinson & Burke, 1996; Larkin et al., 1998; Melchers et al., 2004; Shanks, 1985; Wasserman & Berglan, 1998; Wasserman & Castro, 2005; Wasserman et al., 1996), there are several studies that have failed to show this effect (i.e., De Houwer, Beckers, & Glautier, 2002; Waldmann, 2000; Waldmann & Holyoak, 1992; Williams et al., 1994). One of the explanations for these diverse results is that, sometimes, compound cues can be processed as elements and, other times, as configurations. Compound cues that are processed configurally are less subject to negative mediation than compound cues that are processed elementally (Williams et al., 1994). In order to find negative mediation, elemental processing is necessary, because if the cues are perceived as part of a configuration, then their values might not be assumed to change independently of each other.²

Several factors can influence whether two cues that are presented in compound will be encoded as two elements or as a unique configuration (for a review, see Melchers, Shanks, & Lachnit, 2008). If participants are pretrained with elementally solvable tasks (Mehta & Williams, 2002; Melchers, Lachnit, Üngör, & Shanks, 2005a; Williams et al., 1994) or are given instructions that encourage them to encode the cues as separate entities (Williams et al., 1994), elemental encoding, and therefore negative mediation, is more likely. Grouping and spatial location of the cues can generate a difference as well. Glautier (2002) used colours and symbols on cards as cues and

found negative mediation only when the cues were spatially separated and ungrouped, but not if they were spatially close together (see also Livesey & Boakes, 2004). All of these studies found that an elemental strategy encourages negative mediation, but none of them reported any sign of positive mediation when participants were using a configural strategy. However, a more recent study by Liljeholm and Balleine (2009) did report opposite effects. They found that high similarity and no spatial separation of the elements of the compound promoted generalization between them, so positive mediation was observed; on the other hand, low similarity and spatial separation of the elements of the compound led to negative mediation.

In relation to our study, it is likely that this difference between elemental and configural encoding could underlie the effect that the use of a structural and a causal scenario had: The structural scenario should probably prompt participants to process cues configurally whereas the causal scenario should possibly favour an elemental strategy.

Theoretical explanations

Opposite changes in the value of absent cues pose a critical challenge to learning theories because, in general, models that can explain one directional effect cannot explain the opposite one. By way of example, consider Wagner's (1981) SOP model. According to Wagner's model, cues and outcomes are represented by nodes that consist of several elements. The elements in a node can be in an inactive state (I) or in one of two possible activation states: a primary active state (A1), when a stimulus is present, or a secondary active state (A2), when a stimulus is not present but its representation is activated by means of another stimulus with which it was previously paired. An excitatory association between a cue and an outcome will be formed when both are present—that is, when their elements are in the A1 state.

² It has to be noted that Pearce's (1987, 1994) configural theory does predict negative mediation but, even when this theory assumes that stimuli are processed configurally, it uses elemental information to determine the level of generalization between two configurations.

But, a stimulus in the A2 state cannot enter into an association with another stimulus. Thus, an absent cue activated in the A2 state is not allowed to change its associative strength.

Dickinson and Burke (1996) extended Wagner's (1981) SOP model so that it could explain negative mediation effects. According to Dickinson and Burke, an excitatory association may develop from a stimulus in A1 or A2 to another concurrent stimulus in the same activation state, and an inhibitory association may develop when two concurrent stimuli are in different states (see also Aitken & Dickinson, 2005). However, sometimes some elements of a stimulus may be in A1 whereas other elements are in A2, in which case whether an excitatory or an inhibitory association develops will depend on which of the processes is stronger. In our case, after pairing the AB compound with the outcome, when Cue A is presented in Phase 2 paired with the outcome as well, it should activate both Cue B and the outcome in A2. Because the outcome is present, some elements of its representation will be also in A1. Then, absent Cue B will be involved in two learning processes: one excitatory, consequence of Cue B's activation in A2 and the activation in A2 of some elements of the outcome, and another inhibitory, consequence of Cue B's activation in A2 and the activation in A1 of some elements of the outcome. Thus, according to this model, a weak negative mediation effect may or may not occur (and, potentially, a positive mediation effect as well) depending on which of these two processes is stronger (Aitken & Dickinson, 2005; Larkin et al., 1998). Indeed, we found a weak backward blocking effect (ratings to Cue B were lower than ratings to Cue C/D in group O-causal, although this difference fell short of statistical significance), a result that supports this view, although robust backward blocking has been reported as well (e.g., Shanks, 1985; Wasserman & Berglan, 1998; Wasserman & Castro, 2005).

But, Dickinson and Burke's (1996) modified SOP cannot predict the formation of an excitatory association between Cue B and the outcome when Cues A and B have been paired together without the outcome. In this case, when Cue A is paired

with the outcome in Phase 2, Cue A will activate the representation of Cue B in A2; because all of the elements of the outcome are in A1, an inhibitory association between B and the outcome should form. So, Dickinson and Burke's modified SOP cannot explain positive mediation under these conditions.

P. C. Holland (1983) proposed a different modification of the SOP model. According to Holland, when the elements of a representation are in A2, an excitatory association should form with another representation in A1; on the other hand, when two concurrent representations are in A2, an inhibitory association should develop. This modification can readily explain the opposite effects due to the presence or absence of the outcome in the first phase. When Cues A and B have been paired together without the outcome, and Cue A is later paired with the outcome, Cue A will activate the representation of Cue B in A2; because all of the elements of the outcome are in A1, an excitatory B-outcome association will be formed (albeit weaker than if the elements of Cue B were also in A1). Thus, positive mediation should be observed. When Cues A and B have been paired together with the outcome, later presentations of Cue A alone will activate the representation of Cue B in A2, but also the representation of the outcome in A2. In this case, the associative value of Cue B could change in two ways because the outcome will have elements in both A2 and A1. Parameters of the task will then determine which association will be stronger.

However, P. C. Holland's (1983) revision of the SOP model fails to explain why Cue F increases its associative value when the EF compound is paired with the outcome, and, later, Cue E is presented without the outcome. In this condition, Cue E alone is presented in Phase 2, and both Cue F and the outcome will be activated in A2. According to Holland's revision, an inhibitory association will always develop, because the outcome is not present, and its elements do not move into the A1 state. An increment in the value of Cue F (as we observed in O-causal and O-structural groups) would never be expected.

Thus, neither the original SOP nor any of its revisions can explain our entire pattern of results.

More promising turns out to be the sometimes-competing retrieval (SOCR) model recently proposed by Stout and Miller (2007), a formalized version of Miller and his colleagues' comparator hypothesis (Denniston, Savastano, & Miller, 2001; Miller & Matzel, 1988). According to the SOCR model, cues can activate the outcome directly (when they have been paired with the outcome) or indirectly (when they activate other associate cues, which in turn do activate the outcome directly). Organisms need to learn to differentiate these two types of activation. If the target cue B has at some point been paired with the outcome, then participants can readily distinguish that the representation of the outcome is being directly activated by Cue B. But, if the target cue B has never been paired with the outcome, and its associate Cue A has, then participants might not distinguish whether Cue B activates the outcome directly or indirectly via the association of its associate Cue A with the outcome. This situation is more likely to happen when participants have little experience with Cue B. As training proceeds, participants learn when the representation of the outcome is being directly and indirectly activated.

In the case of our experiments, when Cues A and B are paired together with the outcome, and later Cue A is paired with the outcome, the SOCR model predicts that Cue B will activate the outcome directly when presented later at testing. Because the direct association between B and the outcome is not as strong as the association between A and the outcome (that has been paired alone with the outcome), low responding to B (that is, negative mediation) will be observed at testing. In the case of Cues A and B being paired together without the outcome, and later Cue A being paired with the outcome, Cue B has never been paired with the outcome; thus, when Cue B is presented at testing it can activate the representation of the outcome only indirectly (because of its association with Cue A). Because of the lack of experience with Cue B alone, participants cannot distinguish what exactly is causing the

activation of the outcome, and high responding to B (that is, positive mediation) will be observed at testing. In short, if the target cue has been previously paired with the outcome, then negative mediation should be observed; if the target cue has never been paired with the outcome, then either negative mediation or positive mediation may be observed, depending on the amount of experience with the target cue alone.

Interestingly, the SOCR's means of explaining opposite effects may even embrace the differences generated by elemental and configural processing. At the beginning of training, organisms might tend to perceive the stimuli presented in compound as a unique entity because they do not have much experience with the components that combine to create the compound, so cue facilitation should be favoured, but, with further training, organisms can learn to discriminate the elements within the compound, so that negative mediation could be favoured.

The SOCR model is a performance model; it focuses on information processing at the testing stage, as compared to the SOP model and its modifications (and models like Mackintosh, 1975; Rescorla & Wagner, 1972; Van Hamme & Wasserman, 1994), which emphasize information processing during the learning stages. The fact that the SOCR seems to be the most adequate model to explain our results suggests, therefore, that maybe the positive and negative mediation effects are better understood as performance rather than acquisition effects. If that is the case, we should conclude that the response to absent cues can change both in the same and in the opposite direction as the response to present cues, and the direction of this change seems to depend on whether or not the absent cue has a prior association with the outcome. Our experiment did not try to address whether the effect takes place during learning or subsequent responding, but it will be something certainly worth studying in the future.

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REFERENCES

- Aitken, M. R. F., & Dickinson, A. (2005). Simulations of a modified SOP model applied to retrospective reevaluation of human causal learning. *Learning & Behavior, 33*, 147–159.
- Arcediano, F., Matute, H., Escobar, M., & Miller, R. R. (2005). Competition between antecedent and subsequent stimuli. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 31*, 228–237.
- Baker, A. G., Murphy, R. A., Mehta, R., & Baetu, I. (2005). Mental models of causation: A comparative view. In A. J. Wills (Ed.), *New directions in human associative learning* (pp. 11–40). Mahwah, NJ: Lawrence Erlbaum Associates.
- Brogden, W. J. (1939). Sensory pre-conditioning. *Journal of Experimental Psychology, 25*, 323–332.
- Brogden, W. J. (1947). Sensory preconditioning of human subjects. *Journal of Experimental Psychology, 37*, 527–539.
- Chapman, G. B. (1991). Trial order affects cue interaction in contingency judgment. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17*, 837–854.
- Cobos, P. L., López, F. J., Caño, A., Almaraz, J., & Shanks, D. R. (2002). Mechanisms of predictive and diagnostic causal induction. *Journal of Experimental Psychology: Animal Behavior Processes, 28*, 331–346.
- De Houwer, J., Beckers, T., & Glautier, S. (2002). Outcome and cue properties modulate blocking. *Quarterly Journal of Experimental Psychology, 55A*, 965–985.
- Denniston, J. C., Savastano, H. I., & Miller, R. R. (2001). The extended comparator hypothesis: Learning by contiguity, responding by relative strength. In R. R. Mowrer & S. B. Klein (Eds.), *Handbook of contemporary learning theories* (pp. 65–117). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Dickinson, A., & Burke, J. (1996). Within-compound associations mediate the retrospective reevaluation of causality judgements. *Quarterly Journal of Experimental Psychology, 49B*, 60–80.
- Glautier, S. (2002). Spatial separation of target and competitor cues enhances blocking of human causality judgements. *Quarterly Journal of Experimental Psychology, 55B*, 121–135.
- Graham, S. (1999). Retrospective reevaluation and inhibitory associations: Does perceptual learning modulate our perception of the contingencies between events? *Quarterly Journal of Experimental Psychology, 52B*, 159–185.
- Hall, G. (1996). Learning about associatively activated stimulus representations: Implications for acquired equivalence and perceptual learning. *Animal Learning & Behavior, 24*, 233–255.
- Harris, J. A., & Westbrook, R. F. (1998). Retroactive reevaluation of an odor–taste association. *Animal Learning and Behavior, 26*, 326–335.
- Hesslow, G. (1983). Explaining differences and weighting causes. *Theoria, 49*, 87–111.
- Hilton, D. J., & Slugoski, B. R. (1986). Knowledge-based causal attributions: The abnormal conditions focus model. *Psychological Review, 93*, 75–88.
- Holland, J. H., Holyoak, K., Nisbett, R. E., & Thagard, P. R. (1986). *Induction: Processes of inference, learning, and discovery*. Cambridge, MA: MIT Press.
- Holland, P. C. (1983). Representation mediated overshadowing and potentiation of conditioned aversions. *Journal of Experimental Psychology: Animal Behavior Processes, 9*, 1–13.
- Karazinov, D. M., & Boakes, R. A. (2004). Learning about cues that prevent an outcome: Conditioned inhibition and differential inhibition in human predictive learning. *Quarterly Journal of Experimental Psychology, 57B*, 153–178.
- Kaufman, M. A., & Bolles, R. C. (1981). A nonassociative aspect of overshadowing. *Bulletin of the Psychonomic Society, 18*, 318–320.
- Konorski, J. (1967). *Integrative activity of the brain: An interdisciplinary approach*. Chicago: University of Chicago Press.
- Larkin, M. J. W., Aitken, M. R. F., & Dickinson, A. (1998). Retrospective reevaluation under positive and negative contingencies. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 24*, 1331–1352.
- Liljeholm, M., & Balleine, B. W. (2009). Mediated conditioning vs. retrospective reevaluation in humans: The influence of physical and functional similarity of cues. *Quarterly Journal of Experimental Psychology, 62*, 470–482.
- Livesey, E. J., & Boakes, R. A. (2004). Outcome additivity, elemental processing and blocking in human causality judgements. *Quarterly Journal of Experimental Psychology, 57B*, 361–379.
- Mackie, J. L. (1974). *The cement of the universe: A study of causation*. Oxford, UK: Clarendon Press.
- Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review, 82*, 276–298.

- Matute, H., Arcediano, F., & Miller, R. R. (1996). Test question modulates cue competition between causes and between effects. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *22*, 182–196.
- McGill, A. L. (1989). Context effects in judgments of causation. *Journal of Personality & Social Psychology*, *57*, 189–200.
- Mehta, R., & Williams, D. A. (2002). Elemental and configural processing of novel cues in deterministic and probabilistic tasks. *Learning and Motivation*, *33*, 456–484.
- Melchers, K. G., Lachnit, H., & Shanks, D. R. (2004). Within-compound associations in retrospective reevaluation and in direct learning: A challenge for comparator theory. *Quarterly Journal of Experimental Psychology*, *57B*, 25–53.
- Melchers, K. G., Lachnit, H., Üngör, M., & Shanks, D. R. (2005a). Prior experience can influence whether the whole is different from the sum of its parts. *Learning and Motivation*, *36*, 20–41.
- Melchers, K. G., Shanks, D. R., & Lachnit, H. (2008). Stimulus coding in human associative learning: Flexible representations of parts and wholes. *Behavioural Processes*, *77*, 413–427.
- Melchers, K. G., Üngör, M., & Lachnit, H. (2005b). The experimental task influences cue competition in human causal learning. *Journal of Experimental Psychology: Animal Behavior Processes*, *31*, 477–483.
- Miller, R. R., Hallam, S. C., Hong, J. Y., & Dufore, D. S. (1991). Associative structure of differential inhibition: Implications for models of conditioned inhibition. *Journal of Experimental Psychology: Animal Behavior Processes*, *17*, 141–150.
- Miller, R. R., & Matute, H. (1996). Biological significance in forward and backward blocking: Resolution of a discrepancy between animal conditioning and human causal judgment. *Journal of Experimental Psychology: General*, *125*, 370–386.
- Miller, R. R., & Matzel, L. D. (1988). The comparator hypothesis: A response rule for the expression of associations. In G. Bower H. (Ed.), *The psychology of learning and motivation* (Vol. 22, pp. 51–92). San Diego, CA: Academic Press.
- Pearce, J. M. (1987). A model for stimulus generalization in Pavlovian conditioning. *Psychological Review*, *94*, 61–73.
- Pearce, J. M. (1994). Similarity and discrimination: A selective review and a connectionist model. *Psychological Review*, *101*, 587–607.
- Price, P. C., & Yates, J. F. (1995). Associative and rule-based accounts of cue interaction in contingency judgment. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *21*, 1639–1655.
- Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. Prokasy F. (Eds.), *Classical conditioning II: Current research and theory* (pp. 64–99). New York: Appleton-Century-Crofts.
- Shanks, D. R. (1985). Forward and backward blocking in human contingency judgement. *Quarterly Journal of Experimental Psychology*, *37B*, 1–21.
- Shanks, D. R. (1991). Categorization by a connectionist network. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *17*, 433–443.
- Shanks, D. R. (1995). *The psychology of associative learning*. Cambridge, UK: Cambridge University Press.
- Shanks, D. R., & López, F. J. (1996). Causal order does not affect cue selection in human associative learning. *Memory & Cognition*, *24*, 511–522.
- Stout, S. C., & Miller, R. R. (2007). Sometimes-competing retrieval (SOCR): A formalization of the comparator hypothesis. *Psychological Review*, *114*, 759–783.
- Tangen, J. M., & Allan, L. G. (2004). Cue interaction and judgments of causality: Contributions of causal and associative processes. *Memory & Cognition*, *32*, 107–124.
- Urcelay, G. P., Perelmuter, O., & Miller, R. R. (2008). Pavlovian backward conditioned inhibition in humans: Summation and retardation tests. *Behavioural Processes*, *77*, 299–305.
- Van Hamme, L. J., & Wasserman, E. A. (1994). Cue competition in causality judgments: The role of nonpresentation of compound stimulus elements. *Learning and Motivation*, *25*, 127–151.
- Vansteenwegen, D., Crombez, G., Baeyens, F., Hermans, D., & Eelen, P. (2000). Pre-extinction of sensory preconditioned electrodermal activity. *Quarterly Journal of Experimental Psychology*, *53B*, 359–371.
- Wagner, A. R. (1981). SOP: A model of automatic memory processing in animal behavior. In N. E. Spear & R. Miller R. (Eds.), *Information processing in animals: Memory mechanisms* (pp. 5–47). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Waldmann, M. R. (2000). Competition among causes but not effects in predictive and diagnostic learning.

- Journal of Experimental Psychology: Learning, Memory, and Cognition*, 26, 53–76.
- Waldmann, M. R., & Holyoak, K. J. (1992). Predictive and diagnostic learning within causal models: Asymmetries in cue competition. *Journal of Experimental Psychology: General*, 121, 222–236.
- Wasserman, E. A. (1990). Attribution of causality to common and distinctive elements of compound stimuli. *Psychological Science*, 1, 298–302.
- Wasserman, E. A., & Berglan, L. R. (1998). Backward blocking and recovery from overshadowing in human causal judgment: The role of within-compound associations. *Quarterly Journal of Experimental Psychology*, 51B, 121–138.
- Wasserman, E. A., & Castro, L. (2005). Surprise and change: Variations in the strength of present and absent cues in causal learning. *Learning & Behavior*, 33, 131–146.
- Wasserman, E. A., Kao, S.-F., Van Hamme, L. J., Katagiri, M., & Young, M. E. (1996). Causation and association. In D. R. Shanks, K. J. Holyoak, & D. Medin L. (Eds.), *The psychology of learning and motivation* (Vol. 34, pp. 207–264). San Diego, CA: Academic Press.
- Williams, D. A., & Docking, G. L. (1995). Associative and normative accounts of negative transfer. *Quarterly Journal of Experimental Psychology*, 48A, 976–998.
- Williams, D. A., Sagness, K. E., & McPhee, J. E. (1994). Configural and elemental strategies in predictive learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 20, 694–709.