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The influence of partner cues on the extinction of causal judgments in people

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Abstract Studies in laboratory animals have shown that the extinction of a conditioned stimulus, A, is regulated by the associative history of a second stimulus, X, when the two are extinguished in simultaneous compound: An inhibitory X protects A from extinction (Rescorla *Learning & Behavior*, *31*, 124–132, 2003), whereas an excitatory X facilitates, and under some circumstances deepens, the extinction of A (Rescorla *Journal of Experimental Psychology: Animal Behavior Processes*, *26*, 251–260, 2000, *Journal of Experimental Psychology: Animal Behavior Processes*, *32*, 135–144, 2006). In the present study, we used the allergist task to examine whether the extinction of causal judgments in people is similarly regulated by the causal status of co-present stimuli. Experiment 1 showed that a cue trained as a conditioned inhibitor protected a target cue from extinction: The target extinguished in compound with the inhibitor was rated as being more causal of the outcome than was a target extinguished in compound with a control cue lacking inhibitory properties. In contrast, the remaining experiments showed that the extinction of a target cue was regulated by the presence, but not the causal status, of a partner cue: Target cues extinguished in compound were protected from extinction, and no evidence showed that an already extinguished partner conferred more protection (Exp. 2), or that an excitatory partner conferred any less protection (Exps. 2 and 3), or that an excitatory partner deepened the extinction of its already extinguished target. These findings are inconsistent with elemental models that rely on a common error term to explain associative changes in extinction. They are largely, but not completely, consistent with the configural model proposed by Pearce (*Psychological Review*, *94*, 61–73, 1987), which predicts an ordering of levels of protection that was not observed.

Keywords Human causal learning · Extinction · Associative learning

Extinction occurs when the signaling relation between a conditioned stimulus (CS) and an unconditioned stimulus (US) is broken by presentations of the former in the absence of the latter. The responding produced by the signaling relation declines across the CS-alone presentations and eventually ceases. Responding is said to be extinguished. Theories that identify learning with a single construct, such as the strength of an association (Bush & Mosteller, 1953; Estes, 1950; Rescorla & Wagner, 1972) or connection weights (Rumelhart, Hinton, & McClelland, 1986), explain extinction as the gradual weakening of the association, or restoration of the original connection weights between the CS and US. However, it is now clear that such theories are incomplete or wrong. For example, the responding that has been eliminated by extinction spontaneously recovers with the lapse of time (Pavlov, 1927), is renewed when the CS is tested outside the context in which extinction has occurred (Bouton & Bolles, 1979), and is reinstated when presentations of the US are interpolated between extinction and test of the CS (Rescorla & Heth, 1975). These phenomena show that some of the learning produced by pairings of the CS and US survives the CS-alone presentations that extinguish conditioned responding. Moreover, an extinguished CS is just as able to control the selection of an instrumental response with which it shared the same outcome (Delamater, 1996), and just as sensitive to changes in the value of its associated outcome, as is a nonextinguished CS (Rescorla, 1993, 1997). These results show that much, if not all, of the original learning remains intact, in spite of the fact that the extinguished CS

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fails to elicit responding. They imply that extinction involves new learning that interferes with the retrieval of the original learning and/or with its expression in responding (for reviews, see Bouton, Westbrook, Corcoran, & Maren, 2006; Delamater & Westbrook, 2014).

Although the nature of this new learning continues to be debated, several studies have demonstrated that responding to a target CS, A, is influenced by the presence of other stimuli at the time of its extinction. For example, relative to the extinction of a control CS presented alone, the extinction of A is attenuated when it is extinguished in simultaneous compound with a neutral stimulus X. Under such conditiong, responding controlled by A is suppressed across nonreinforced presentations of the AX compound, but re-emerges when A is tested in the absence of X (Pearce & Wilson, 1991; see also Laborda, Witnauer, & Miller, 2011; Taylor & Boakes, 2002; Urcelay, Lipatova, & Miller, 2009; Wilson & Pearce, 1992).

The extinction of responding to a target CS, A, is not only influenced by the presence of another stimulus X. It is also regulated by the associative history of that stimulus. For example, Rescorla (2003) showed that a target CS A, extinguished in compound with an inhibitor X, undergoes less extinction than does a control CS, B, extinguished in compound with a neutral stimulus, Y (Rescorla, 2003, Exps. 2 and 3): AX evoked less magazine approach than did BY across their extinctions, but A evoked more magazine approach than did B when these CSs were tested alone (see also McConnell & Miller, 2010; Soltysik, Wolfe, Nicholas, Wilson, & Garcia-Sanchez, 1983). Other evidence suggests that an already extinguished CS also functions like a conditioned inhibitor in protecting a target CS, A, from the effects of nonreinforcement. Specifically, Calton, Mitchell, and Schachtman (1996) reported that, following extinction of a conditioned aversion to saccharine, the presence of saccharine attenuated the extinction of an aversion to another taste CS (see also Pineño, 2007). Hence, in both appetitive and aversive conditioning paradigms, an inhibitory or extinguished CS protects an excitatory target CS from extinction.

However, the extinction of responding to a target CS, A, is not always protected by the presence of another stimulus, X. Rescorla (2000) reported that a compound composed of A and an excitatory X elicits more responding than does an excitatory CS, B, presented alone (i.e., summation)—but critically, that the extinction of AX facilitates (rather than impairs) the extinction of A, as compared to B extinguished in isolation (see also Wagner, 1969; McConnell, Miguez, & Miller, 2013). Rescorla (2000) also reported that a compound composed of A and an excitatory X elicits more responding than does a compound composed of an excitatory CS, B, and a neutral CS, Y, and that nonreinforcement of these compounds, AX– and BY–, more effectively extinguishes A than B. Taken together, these results show that learning about a target CS is regulated by the associative value of another stimulus present at the time of its extinction, a stimulus that signals the absence of the US protects, whereas a conditioned excitor facilitates long-term extinction of responding to the target CS.

Studies with human participants have also shown that a conditioned inhibitor can protect a target CS from extinction. Lovibond, Davis, and O'Flaherty (2000) established two cues, A and B, as predictors of shock (A+ and B+). Both cues were then extinguished: one alone (B–), and the other in compound with a putative conditioned inhibitor, X (AX–). The test results showed that X protected its within-compound associate, A, from extinction: A elicited higher shock expectancy ratings and skin conductance responses than did the cue that had been extinguished alone, B. However, in contrast to the animal studies, Lovibond et al. additionally reported that the level of protection conferred by a conditioned inhibitor, X, was equivalent to that conferred by a cue Y that not only lacked any such inhibitory properties, but was in fact excitatory. O. Griffiths and Westbrook (2012) reported similar results in a study of judgments of causality in people. In these experiments, participants were trained in Stage 1 to expect an allergy following the presentation of any one of four cues (A+, X+, B+, and Y+). In Stage 2, they were extinguished to one of these cues (X–). In Stage 3, two compounds were then extinguished: one composed of the already extinguished X and an excitor, A (AX–), and the other composed of two excitors (BY–). On the basis of animal studies, it was anticipated that A would undergo relatively little change, because it was extinguished in compound with the already extinguished X (protection from extinction), whereas B would undergo substantial change, because it was extinguished in compound with the excitatory Y. Testing confirmed that X, extinguished in both Stages 2 and 3, was rated as being less causal of the outcome than Y, just extinguished in Stage 3. However, rather than rating A as being more causally effective than B, participants rated A as being just as effective as B.

To the best of our knowledge, there is no evidence in human studies that the extinction of a target cue is regulated by the associative history of co-present stimuli. However, it is typically problematic to accept the null hypothesis, and it remains possible that in previous studies with people, the extinction of a target cue was sensitive to the associative value of co-present stimuli, but simply not detected for some other reason. Accordingly, the present experiments constitute a further examination of how people change their judgments about the causal effectiveness of a target allergenic cue extinguished in compound with cues that differed in their allergenic history. In Experiment 1, we examined whether the causal effectiveness of a target allergenic cue was protected when it was extinguished in compound with a cue that had previously signaled the absence of an otherwise expected outcome (a putative conditioned inhibitor, or preventative cause; see McConnell & Miller, 2010;

Rescorla, 2003). Experiment 2 was based on a design similar to that used by O. Griffiths and Westbrook (2012) to examine whether an already extinguished cue functions like a conditioned inhibitor in protecting a target cue from the effects of extinction (see Calton et al., 1996), and/or whether the presence of a second allergenic/excitatory cue facilitates the extinction of causal judgments about the target cue (see Rescorla, 2000). Finally, in Experiment 3 we examined whether facilitated or deepened extinction of causal judgment could be observed using a design that is potentially more sensitive to such an effect (see Hendry, 1982; Leung, Reeks, & Westbrook, 2012; Rescorla, 2006).

Experiment 1

In this experiment, we assessed whether people use the signal value of a conditioned inhibitor to regulate change to its excitatory associate in an extinguished compound. The design is shown in Table 1. The critical component of the design involved the extinction of two compounds: one of which, AX, contained an excitor, A, and a conditioned inhibitor (or preventative cause) of the allergy, X, whereas the other, BY, contained an excitor, B, and a cue, Y, that was equally familiar as X but that lacked inhibitory properties. On the basis of animal studies showing that the presence of a conditioned inhibitor protects a target CS from extinction, we anticipated that the presence of X would protect A from extinction across the AX– trials, and therefore, that participants would ultimately rate A as being more allergenic than B.

Method

Participants A group of 103 second-year psychology students participated in partial fulfillment of course requirements. Their mean age was 21 years, and 74 were female.

Table 1 Design of Experiment 1

Stage 1	Stage 2	Stage 3	Test	Comment
A+		AX–	A vs. B	A: extinguished with conditioned inhibitor
B+		BY–	NX vs. MY	B: extinguished with neutral partner
X–	X–			X: conditioned inhibitor (CI)
Y–	Y–			Y: control for conditioned inhibitor
C+	C+			
D+	D+			
E–	E–			
F–	F–			
	CX–			CI trials
	DX–			
	LX–			
	EY–			Controls for CI trials
	FY–			
	PY–			
Controls				
G+		G–		G: extinguished alone
H–		H–		H: safe cue
I–		IJ–		I: compound safe
J–				J: compound safe (2)
K+				K: trained but not extinguished
Fillers				
L+	L+	L+		
M+	MN+	MN+		
N+		N+		
	OQ+	OQ+		
P–	P–	P–		
Q–				

The letters A–Q, X and Y denote cues (foods). The "+" symbol denotes the presence of the allergy outcome, the "–" represents a trial in which feedback was provided about the absence of the outcome, and the "?" symbol indicates a trial on which no feedback was provided.

Design The experiment involved three stages, followed by test. The design is shown in Table 1. In Stage 1, several cues were each paired with an allergic reaction (A+, B+, and G+). Then, in Stage 2, three of these cues continued to signal an allergic reaction when presented in isolation, but they did not signal that reaction when compounded with X (i.e., C+, CX–, D+, DX–, L+, LX–). X was trained in this fashion to increase the chance of its acquiring the properties of a conditioned inhibitor. Y was designed as a control for the inhibitory cue X. This was done by showing Y as often as X, presenting Y in as many different stimulus compounds as X (EY–, FY–, PY– trials), and arranging that it was never causal of allergy. The critical difference between the inhibitor, X, and its control, Y, was that, in Stage 1, the C, D, and L associates of X did, but the E, F, and P associates of Y did not, signal an allergy. This should result in X, but not Y, becoming a conditioned inhibitor or preventative cause of the allergy, despite their otherwise equivalent training. Critically, in Stage 3, A was extinguished in compound with X (AX–), B in compound with Y (BY–), and cue G was extinguished in isolation (G–). The control cues were selected so as to match A and B with respect to extinction training (elementally extinguished cue G), overall exposure (safe cue H), type of exposure (elemental then compound presented cues I and J), and total reinforcement (elementally trained cue K). The filler cues were intended to exclude the use of rules, such as "all compounds do or do not cause an allergic reaction"; to equate the number of cues followed or not followed by the allergic reaction in each stage; and to control for the number of cues that changed their relation to the allergic reaction between stages, as well as the number of cues presented in isolation or in compound.

Procedure The experiment was conducted in tutorial classes of approximately 20 students. Participants engaged in a computer-based causal learning task. They were first instructed to assume the role of an allergist who had to learn which foods made a new patient (Mr. X) feel ill and those that were safe for him to consume. They received two practice trials with a second patient, Mr. Y, who ate different foods from Mr. X, and then proceeded to Stage 1. On each trial, participants were shown either a single food (e.g., the word "carrots") or two foods (e.g., "beans and broccoli"), and they were asked to predict whether Mr. X would feel ill. Foods were randomly assigned to cue types for each participant. Two response keys were available, one labeled "Sick" and the other "Healthy." The selection of a response key resulted in a picture of the patient feeling sick (+) or healthy (–). If an incorrect prediction was made, the word "incorrect" was shown in red, a beep tone sounded, and the correct answer was shown for 2 s. If a correct prediction was made, the word "correct" was shown in green and no tone or time penalty occurred. The order of the trials in each stage was randomized, with the constraint that all trial types were shown once before any trial type was shown a second time. Eight instances of each trial type were presented in each stage. The transition between stages was not signaled.

Upon completing Stage 3, participants were tested. This started with two forced choice tests in which they had to select which of two cues was more likely to result in Mr. X experiencing an allergic reaction. One was essentially a summation test for conditioned inhibition (Rescorla, 1969), consisting in participants choosing which of two compounds, NX versus MY, was more likely to cause an allergic reaction. Each compound contained an allergenic cue, M and N, and either the putative inhibitor, X, or its matched control, Y. If X had acquired inhibition or become a preventative cause of the allergy, participants should choose NY as being more causal than MX. The other forced choice test consisted in participants choosing which of the two target cues, A versus B, was more likely to cause an allergic reaction. If X had protected A from extinction, participants should choose A as being more likely to cause an allergic reaction than B. The screen positions (left or right) were randomly determined for each trial, and the trials were shown in a random order. Participants then rated each of the 17 cues that had been presented. Each cue was accompanied by an image of Mr. X feeling sick, and participants indicated on a scale from 0 (*very unlikely*) to 100 (*very likely*) the likelihood of that cue causing an allergic reaction.

Results

Exclusion criteria Of the total sample of 103 participants, eight were excluded from the analysis for failing to show any evidence of learning the training contingencies. Specifically, four participants did not satisfy the Stage 3 extinction criterion of consecutive "no allergy" responses for at least one of the target trial types AX, BY, or G; another four participants had an average rating greater than 50 to cues that were never predictive of allergy, or less than 50 to cues that were always predictive of allergy. This yielded a final sample of 95 participants.

Training data Figure 1 shows the responses to the critical cues across Stages 1, 2, and 3. Cues A, B, and G were rapidly associated with the allergic reaction during Stage 1, as were the cues C, D, and L, used to establish X as a conditioned inhibitor in Stage 2 (left). In Stage 2, C, D, and L signaled an allergic reaction in isolation, but not when each was compounded with X. Figure 1 (center) shows that participants discriminated between the cues by the end of Stage 2, indicating expectancies of allergic reactions to C, D, and L when they were presented alone, but not when they were accompanied by X. Inspection of the data from Stage 3 (right) suggests that the proportions of participants who responded with an allergic reaction to AX, BY, and G differed on the first trial. McNemar's chi-squared test of dependent proportions

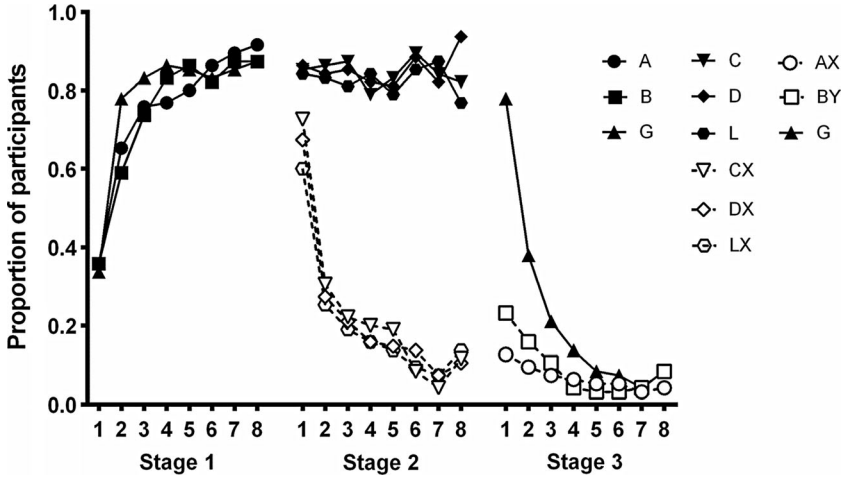

Fig. 1 Proportions of participants who responded "allergy" to target cues and compounds across trials of Stages 1 to 3 in Experiment 1. During Stage 3, A was extinguished in compound with the putative preventative cue X, whereas B was extinguished in compound with the nominally neutral control cue Y

revealed that more participants predicted that the outcome would occur on the initial G– trial than on the initial AX– or BY– trials, minimum $\chi^2(1, .05) = 46.62$, $p < .001$. An odds ratio (OR) for each of the two comparisons, G versus AX and G versus BY, was calculated as the number of participants who responded "allergy" to G but not to AX, divided by the number who responded "allergy" to AX but not to G, and the number of participants who responded "allergy" to G but not to BY, divided by the number who responded "allergy" to BY but not to G. The smaller of these odds ratios was 18.33. Differences between G versus AX and G versus BY persisted to the second trial, minimum $\chi^2(1, .05) = 10.26$, $p < .001$, smaller OR = 2.91. It appeared as though more participants expected an allergic reaction when first presented with BY than with AX, consistent with inhibition by X. However, this difference on what is effectively a summation test for conditioned inhibition to X was not significant, $\chi^2(1, .05) = 3.57$, $p = .06$. We found no statistically significant differences in the proportions of participants who responded "allergy" to AX, BY, and G on the final trial of Stage 3, $\chi^2(1, .05) < 1$, indicating equivalent levels of extinction to these cues

Forced choice data Participants' responses to each of the two-answer forced choice test items (e.g., MY vs. NX and A vs. B) were tallied and compared against chance responding using a binomial test. The number of participants who chose compound NX as being less allergenic than compound MY (57 participants, 60%) was not significantly different from chance, CI = .50–.69; and the number who chose A as being more allergenic than B (53 participants, 55.8%) was also not significantly different from chance, CI = .46–.69.

The relationships between responses to the forced choice questions were also examined by comparing the likelihood of A> B among participants who chose either MY> NX or MY < NX. Reassuringly, participants' responses to the two forced choice questions were related; that is, the distributions of responses to NX versus MY and A versus B were significantly different from the ones that would be expected according to chance levels of responding, $\chi^2(1, .05) = 5.77$, $p < .05$, OR = 2.25. In fact, the distributions were related in a manner consistent with a conditioned inhibitor, X, having protected its partner, A, from extinction, at least among some participants: Specifically, among the participants who chose MY as being more causal than NX (i.e., the ones that showed evidence of inhibition to X), 68% chose A as being more causal than B; in contrast, among the participants who chose MY as being less causal than NX, 47% chose A as being more causal than B. The difference between these proportions was significant, $z = 2.02$, $p < .05$, CI = .01–.39.

Individual cue ratings Figure 2 shows the causal ratings for all of the cues. These data were analyzed using a set of three

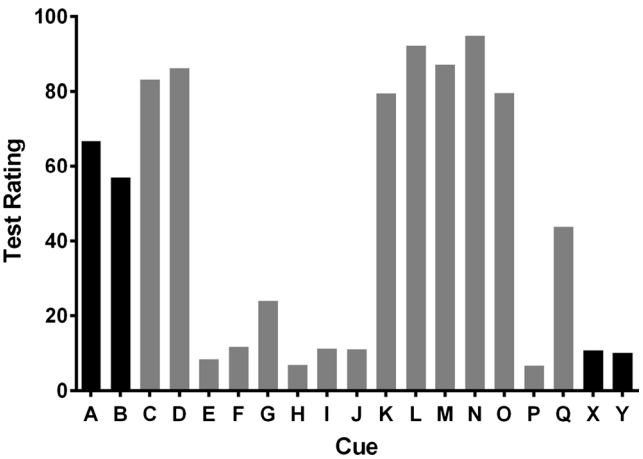

Fig. 2 Mean causal ratings of each cue in Experiment 1. Target cues are in black: A had been extinguished in compound with the putative preventative cue X, whereas B had been extinguished in compound with the nominally neutral control cue Y

planned, orthogonal contrasts. The first contrast tested whether extinction was maintained on testing through comparison of the average ratings of cues subjected to extinction, A, B, and G, with the cue trained but not extinguished, K (i.e., A, B, and G vs. K). We found that ratings of the extinguished target cues, A, B, and G, were less than those of K, which was equivalently reinforced but not extinguished, $F(1, 94) = 102.40$, $p < .001$, CI = 0.75–1.12. The second contrast of interest compared the cues extinguished in compound, A and B, with the cue extinguished in isolation, G (i.e., A and B vs. G). The elementally extinguished cue, G, was rated as being significantly less allergenic than the compound extinguished cues (A and B), $F(1, 94) = 91.42$, $p < .001$, CI = 0.93–1.42. The third contrast compared cue A, extinguished in compound with the preventative cause X, with cue B, extinguished in compound with the neutral Y (A vs. B). Cue A was rated as being more allergenic than B, $F(1, 94) = 4.12$, $p < .05$, CI = 0.01–0.60, showing that A had been protected from extinction by its inhibitory associate, X, more than B had been protected by its safe associate, Y. Post-hoc tests assessed whether the differences in ratings of A and B were the same or different among participants who selected either MY or NX as being more causal in the forced choice test. These tests showed that those who selected MY> NX rated A>B, $F(1, 56) = 18.60$, $p < .05$, whereas those who selected NX> MY rated A and B equivalently, $F < 1$. A final post-hoc test showed that participants did not differ in their ratings of X and Y, $F < 1$, perhaps due to floor effects.

Discussion

Participants learned that some cues caused allergy and others did not. In this experiment, X was trained in a manner intended to establish it as a conditioned inhibitor or preventative cause of the allergic reaction. Y was matched with X for exposure in compounds, but in ones that did not contain an allergenic component. The critical finding was that X and Y differed in their effects on responding controlled by other cues, as well as on the extinction of those cues. When X and Y were compounded with allergenic foods A and B, respectively, in Stage 3, more participants expected an allergic reaction to BY than to AX, though this difference only approached significance. Moreover, when X and Y were compounded with allergenic foods N and M, respectively, in the forced choice test, more participants selected MY as being more likely to cause an allergic reaction than NX, although again this difference failed to reach a conventional level of significance. However, critically, A was rated as being more causal than B in the final test, and this difference was greater among those who in fact selected MY as being more causal than NX. Taken together, these findings imply that, at least in many participants, X acquired inhibition, suppressed responding to compounds in which it was present, and protected its partners from extinction.

Experiment 2

In this experiment, we examined whether an already extinguished cue functions like a conditioned inhibitor in protecting a within-compound associate from the effects of extinction. This effect has been seen previously in animal conditioning studies (e.g., Calton et al., 1996), but was not found in a prior study of human causal learning (O. Griffiths & Westbrook, 2012). One possible explanation as to why O. Griffiths and Westbrook did not observe error-driven modulation of extinction in human learning was that the compound extinction trials used in that procedure may have only produced weak extinction learning, and consequently, their procedure may have been insensitive to modulations of that (possibly weak) extinction learning. Their controls did not afford a good measure of the magnitude of extinction learning. In the present experiment we addressed this concern by including the more robust set of control cues used in Experiment 1 (elemental conditioned cues, elementally extinguished cues, etc.). Second, because Experiment 1 had detected differential extinction of target cues due to their partner cues, we can be confident that the present procedure is sufficiently sensitive to detect differences in extinction learning.

The design of Experiment 2 is shown in Table 2. In Stage 1, participants were trained on a discrimination in which each of several foods (A, B, C, etc.) led to an allergic reaction (+)—notably, A+, B+, C+, D+, F+, and G+—whereas others did not lead to that reaction (–)—notably, E–. In Stage 2, they were exposed repeatedly to one of the dangerous foods, but now in the absence of the allergic reaction, A–. In Stage 3, participants were exposed to another dangerous food also in the absence of the allergic reaction, G–. They were also exposed to three compounds, none of which was followed by the allergy. One of these compounds contained the already extinguished A and the dangerous B (AB–), the second consisted in two dangerous foods (CD–), whereas the third compound was composed of the safe E and the dangerous F (EF–). Finally, participants were asked to rate each food with regard to its effectiveness in producing an allergic reaction. The foods of major interest were G, extinguished in isolation; B, extinguished in compound with the already extinguished A; F, extinguished in compound with the safe E; and, finally, the dangerous C and D, extinguished in compound with each other. On the basis of findings reported in animal studies, we anticipated that B and F would be protected by their partners A and E, respectively,

Table 2 Design of Experiment 2

Stage 1	Stage 2	Stage 3	Test	Comment
A+	A–	AB–	B vs. D	A: already extinguished partner for B
B+				B: extinguished with already extinguished partner
C+		CD–	D vs. F	C: excitatory partner for D
D+				D: extinguished with excitatory partner
E–	E–	EF–	F vs. B	E: safe partner for F
F+				F: extinguished with safe partner
Controls				
G+		G–		G: extinguished alone
H–		H–		H: safe cue
I–		IJ–		I: compound safe cue
J–				J: compound safe cue (2)
K+				K: trained but not extinguished
Fillers				
L+	L+	LM+		
M+	M+			
		N+		
		O+		
P–		PQ+		
Q–				

The letters A–Q denote cues (foods). The "+" symbol denotes the presence of the allergy outcome, the "–" represents a trial in which feedback was provided about the absence of the outcome, and the "?" symbol indicates a trial on which no feedback was provided.

but that C and D could have undergone superextinction. Hence, B and F should be rated as being more allergenic than G, which, in turn, should be rated as being more allergenic than either C or D, depending on whether extinction had reached asymptote.

Method

Participants The participants were 125 undergraduates who participated in partial fulfillment of a course requirement. Their mean age was 21 years, and 92 were female.

Design and procedure The design (see Table 2) consisted in the target cues B, extinguished in compound with the already extinguished A; D, extinguished in compound with the allergenic C; and F, extinguished in compound with the safe E; control cues G–K; and filler cues L–O. Control and filler cues were selected as per the details of Experiment 1. Upon completing Stage 3, participants were tested. This started with a forced choice in which they had to select which of two cues was more likely to result in Mr. X experiencing an allergic reaction. On three separate trials, participants chose between B and D, B and F, and D and F. As we described previously, the screen positions (left or right) were randomly determined for each trial, and the trials were shown in a random order. Participants then rated each of the 17 cues that had been presented, in the manner described previously.

Results

Exclusion criteria Of the total sample of 125 participants, 28 of the participants did not satisfy the extinction criterion (consecutive "no allergy" responses) for either AB, CD, EF, or G in Stage 3, and thus were excluded from the statistical analysis. This yielded a final sample of 97 participants.

Training data Figure 3 (left panel) shows that participants learned that Mr. X suffered an allergic reaction after eating any of the target foods A+, B+, C+, D+, F+, and G+, but not after eating other foods, notably the target safe food E–. Participants then learned that one of the dangerous foods, A–, was no longer followed by the allergic reaction, and that E– continued to be a safe food (center panel). Finally, in Stage 3, they learned that various meals were no longer dangerous: specifically, a meal composed of one food, G–, or various meals composed of two foods, either two dangerous foods, CD–, or an extinguished and a dangerous food, AB–, or a safe and a dangerous food, EF– (right panel).

Forced choice data Participants' responses to the three binary forced choice responses were tallied and analyzed using a binomial test that was used to compare the proportions of participants who selected either of the two cues as being more allergenic (B vs. D; B vs. F; D vs. F). This analysis failed to reveal any differences in judgments of the effectiveness of B,

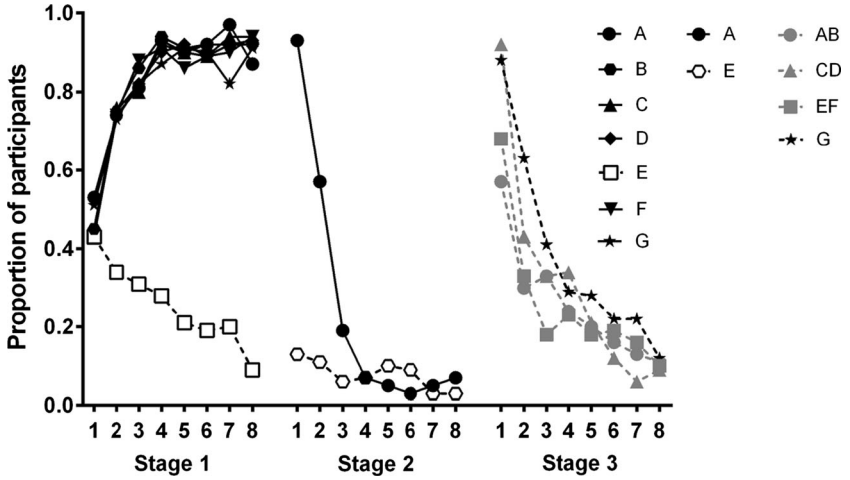

Fig. 3 Proportions of participants who responded "allergy" to each cue or compound across individual trials in Stages 1–3 of Experiment 2. During Stage 3, B was extinguished in compound with the already extinguished cue A, D was extinguished in compound with the nonextinguished cue C, F was extinguished in compound with the nominally neutral control cue E, G was extinguished alone, and K was a nonextinguished control cue

D, and F in causing an allergic reaction: 49 participants (50.5%) chose B as being more causal than D; 48 participants (49.5%) chose B as being more causal than F; and 54 participants (55.7%) chose F as being more causal than D. Binomial tests revealed that no cue was chosen significantly more frequently than its alternative, largest $z = 1.12$.

Individual cue ratings Figure 4 shows the final test ratings for all of the cues in Experiment 1. The test ratings of individual cues were compared using a set of planned nonorthogonal contrasts with a Bonferroni adjustment to control for the five contrasts tested. The first contrast tested whether extinction was maintained on testing through comparison of the average ratings of the cues subjected to extinction for the first time in Stage 3—B, D, F, and G—with a cue not subjected to extinction, K (i.e., B, D, F, and G vs. K). The nonextinguished cue, K, was rated as being significantly more allergenic than the average of the extinguished cues, $F(1, 96) = 42.17$, $p < .001$, CI = 0.75–1.21. The second contrast tested whether the cues only extinguished in compound were protected from the effects of extinction relative to cues that had been extinguished in isolation. That is, whether the average ratings of B, D, and F were higher than the ratings of the cues extinguished in isolation, G and A (i.e., B, D, and F vs. G and A). On average, cues B, D, and F were rated as being more allergenic than cues A and G, which had been extinguished in isolation, $F(1, 96) = 17.80$, $p < .001$, CI = 0.30–0.82.

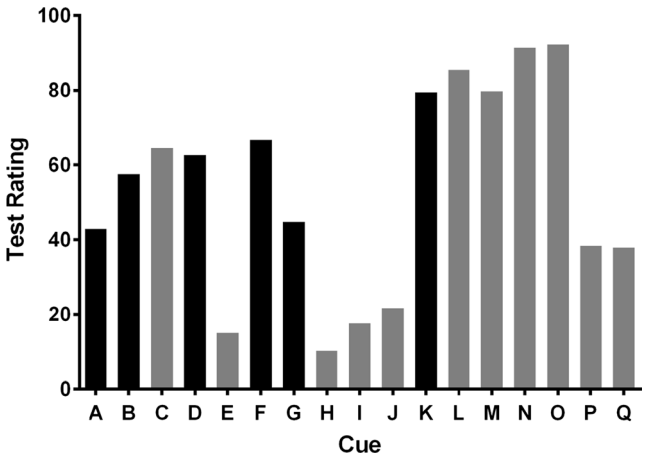

Fig. 4 Mean causal ratings of each cue in Experiment 2. Target cues are in black: B had been extinguished in compound with the already extinguished cue A, D had been extinguished in compound with the nonextinguished cue C, F had been extinguished in compound with the nominally neutral control cue E, G had been extinguished alone, and K had not been extinguished

The third contrast tested whether the amount of protection conferred by a cue depended on its associative value: that is, whether the average ratings of B and F—extinguished in compound with the already extinguished A and the safe E, respectively—were higher than the ratings of D, extinguished in compound with the allergenic C (i.e., B and F vs. D). No significant differences between these cues were observed, $F < 1$. The fourth contrast tested the amount of protection afforded by an extinguished cue, A, relative to a nominally safe cue, E, by comparing the ratings of the Stage 3 associates of these cues, B and F, respectively (i.e., B vs. F). The cue extinguished in compound with a safe partner, F, was not rated as being more allergenic than the cue extinguished in compound with the already extinguished cue B, $F(1, 96) = 5.35$. Finally, the fifth contrast assessed whether cue A, which was extinguished in isolation in Stage 2 and then in compound in Stage 3, underwent more extinction than did cue G, which was only extinguished in isolation in Stage 3. Cue as A was not rated as being less allergenic than cue G, $F < 1$.

Discussion

Overall, participants rated cues subjected to extinction as being less allergenic than cues not subjected to extinction. Moreover, they rated a cue extinguished in isolation as being less allergenic than cues extinguished in compound, implying that cues extinguished in compound are protected from extinction. However, we found no support for the prediction that the associative value of a cue determines the degree to which it protects its partner from extinction. The results from studies with animal subjects suggest that, relative to a cue extinguished in compound with a safe partner, a cue extinguished in compound with an already extinguished partner would have undergone less extinction, whereas a cue extinguished in compound with a nonextinguished partner would have undergone greater extinction. However, the cue extinguished in compound with a nonextinguished partner was not, in fact, rated as being less allergenic than cues extinguished in compound with either a safe or an extinguished partner, and the cue extinguished in compound with an already extinguished partner was not rated as being more allergenic than the cue extinguished in compound with a safe partner. The protective effect of extinguishing cues in compound was further evidenced by the fact that, among the two cues that had been extinguished alone, A and G, additional extinction of A in compound (with B) did not deepen its extinction relative to G. These data are consistent with O. Griffiths and Westbrook's (2012) investigation of the influence of prediction error on the extinction of human causal leaning, in that they too had failed to observe any influence of a partner cue's associative status on a target cue extinguished in compound.

Experiment 3

In the previous experiment, no evidence was found that a cue extinguished in compound with an excitatory partner underwent greater extinction than did a cue extinguished in compound with either a safe cue or an already extinguished cue. In the present experiment, we assessed the effects on an already extinguished target when it received additional extinction in compound. The question of interest was whether the associative value of the partner across compound extinction would regulate the causal effectiveness of the already extinguished target, as it does in animal studies (Leung et al., 2012; Rescorla, 2006). The design is shown in Table 3. Participants first learned that Mr. X suffered an allergic reaction after eating some foods—notably, A+, B+, C+, D+, E+, and G+—but not others, notably, F–. Subsequently, Mr. X ate five of these foods, A–, C–, D–, E–, and G–, without suffering an allergic reaction. This extinction training was followed by additional extinction of A, C, E, and G: A was extinguished in compound with the dangerous B (AB–); C in compound with the already extinguished D (CD–); E in compound with the safe F (EF–); and G continued to be extinguished in isolation. On the basis of findings reported in animal studies, we anticipated that A would undergo the most extinction, due to the presence of the excitatory B; that C would undergo moderate extinction, due to the presence of the previously excitatory D; and, if anything, that E would undergo as much extinction as G, extinguished alone. Thus, the final ratings, from lowest to highest in terms of causal effectiveness, should be $A < C < G \leq E$.

Method

Participants A group of 109 second-year psychology students participated in partial fulfillment of course requirements. The mean age was 21 years, and 69 were female.

Procedure The procedure was similar to that used in the previous experiment, but differed in two respects. The filler cues were altered to balance the changes made to the treatments of the critical and comparison cues (see Table 3), and the items presented in the forced choice consisted in A versus C; A versus E; and C versus E. As in Experiment 1, these choices were shown in a random order, and the left/right screen position of each response key was randomly determined for each trial. In all other respects, the procedure was that described previously.

Results

Exclusion criteria Of the total sample of 109 participants, 12 were excluded from the analysis using the same criteria that had been applied in Experiment 2. Specifically, nine participants did not satisfy the extinction criterion for either AB, CD, EF, or G in Stage 3; one participant had an average rating greater than 50 for cues that had never been allergenic (F, H, I, and J); and two participants had an average rating less than 50 for cues that had been consistently allergenic (L, M, N, and O). This yielded a final sample of 97 participants.

Training data Figure 5 (left) shows that participants learned that Mr. X suffered an allergic reaction after eating the critical foods A+, B+, C+, D+, E+, and G+, but not after eating other foods, notably F–. They then learned that Mr. X could now eat foods A–, C–, D–, E–, and G– without suffering that reaction (center). Participants appeared to exhibit different expectancies when exposed to the four critical cues (AB–, CD–, EF–, and G–) presented in Stage 3 (right), such that more participants responded "allergy" to the compound of the extinguished A with the nonextinguished B than to any other trial type.

Forced choice data For each of the three forced choice test questions (A vs. C; A vs. E; C vs. E), a binomial test was used

Table 3 Design of Experiment 3

Stage 1	Stage 2	Stage 3	Test	Comment
A+	A–	AB–	A vs. E	A: extinguished with excitatory partner
B+				B: excitatory partner for A
C+	C–	CD–	A vs. C	C: extinguished with already extinguished partner
D+	D–			D: already extinguished partner for C
E+	E–	EF–	E vs. C	E: extinguished with safe partner
F–				F: safe partner for E
Controls				
G+	G–	G–		G: extinguished alone
H–	H–	H–		H: safe cue
I–	I–	IJ–		I: compound safe cue
J–	J–			J: compound safe cue (2)
K+				K: trained but not extinguished
Fillers				
L+	L+	LM+		
M+	M+			
N+	N+	N+		
O+	O+	O+		
P–		PQ+		
Q–				

The letters A–Q denote cues (foods). The "+" symbol denotes the presence of the allergy outcome, the "–" represents a trial in which feedback was provided about the absence of the outcome, and the "?" symbol indicates a trial on which no feedback was provided.

to compare the proportions of participants who selected either of the two cues as being more allergenic. These comparisons failed to reveal any differences in judgments of the effectiveness of A, C, and E in causing an allergic reaction: 54 participants (55.7%) chose A as being less causal than C; 40 participants (41.2%) chose C as being less causal than E; and 47 participants (48.5%) chose cue A as being less causal than E. Binomial tests revealed that no cue was chosen significantly more frequently than its alternative, largest $z = 1.73$.

Individual cue ratings The individual test ratings for all cues are summarized in Fig. 6. The test ratings of individual cues were compared using planned orthogonal contrasts. The first contrast tested whether extinction was maintained on testing

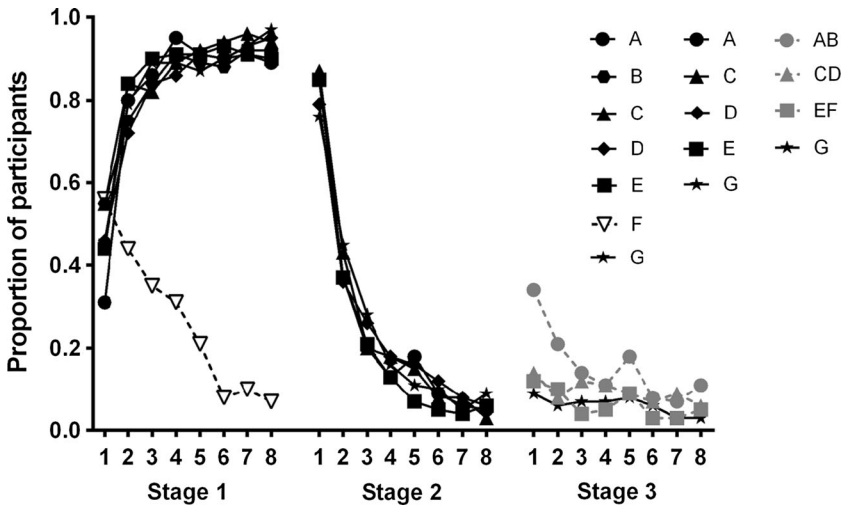

Fig. 5 Proportions of participants who responded "allergy" to each cue or compound across individual trials in Stages 1–3 of Experiment 3. During Stage 3, A was subjected to additional extinction in compound with the nonextinguished cue B, C was subjected to additional extinction in compound with the already extinguished D, E was subjected to additional extinction in compound with the nominally neutral control cue F, G was subjected to additional extinction alone, and K was a nonextinguished control cue

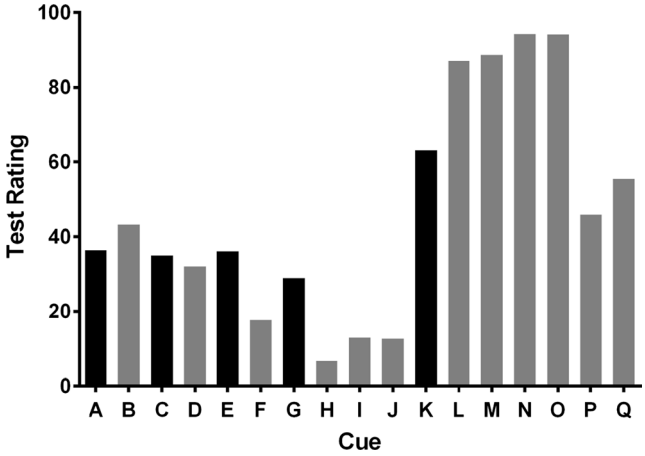

Fig. 6 Mean causal ratings of each cue in Experiment 3. Target cues are in black: A had been subjected to additional extinction in compound with the nonextinguished cue B, C had been subjected to additional extinction in compound with the already extinguished D, E had been subjected to additional extinction in compound with the nominally neutral control cue F, G had been subjected to additional extinction alone, and K had not been extinguished

through comparison of the average ratings of the cues subjected to extinction—A, C, E, and G—with a cue not subjected to extinction, K (i.e., A, C, E, and G vs. K). The nonextinguished cue K was rated as being significantly more allergenic than the average of the extinguished cues, $F(1, 96) = 76.95$, $p < .001$. The second contrast tested whether the effects of extinction were deepened for cues extinguished in compound: that is, whether the average ratings of A, C, and E were lower than ratings of the cue extinguished in isolation, G (i.e., A, C, and E vs. G). On the basis of results from animal studies, the cues (A, C, E) that were subjected to additional extinction in compound (with B, D, and F, respectively) should have been rated as less allergenic than the cue (G) given further extinction in isolation. The opposite was observed: The cues given further extinction in compound (A, C, E) were rated as being significantly more allergenic than the cue (G) extinguished in isolation, $F(1, 96) = 15.10$, $p < .001$.

The third contrast tested whether the amount of deepening varied with the associative value of the partner cues: that is, whether ratings of A, extinguished in compound with the allergenic B, were lower than the average ratings of C and E, extinguished in compound with the already extinguished D and the safe F, respectively (i.e., A vs. C and E). The average ratings of cues C and E did not significantly differ from the ratings of cue A, $F = 1.73$. Finally, the fourth contrast tested the amount of deepening that occurred when a target cue, C, was extinguished in compound with an already extinguished cue, D, versus a target cue, E, extinguished in compound with a safe cue, F (i.e., C vs. E). No significant differences were observed in the ratings given to these two cues, $F < 1$.

Finally, post-hoc comparisons revealed that the failure to detect differences among A, C, and E was not due to the ineffectiveness of their B, D, and F associates in the extinguished compounds: B, extinguished in compound with the already extinguished A, was rated as being significantly more allergenic than D, extinguished in compound with C, $t(96) = 3.11$, $p < .01$, which, in turn, was rated as being significantly more allergenic than the safe F, extinguished in compound with E, $t(96) = 4.68$, $p < .001$.

Discussion

On the basis of animal studies, we expected that a cue subjected to additional extinction in compound with an excitatory partner would undergo a greater loss in its allergenic properties than would a cue subjected to additional extinction in isolation. However, we found no evidence that A, extinguished in compound with a nonextinguished allergenic cue, B, underwent more change than did either C or E, which had been extinguished in compound with an already extinguished cue, D, and a safe cue, F, respectively. Instead, the average rating of these target cues was greater than the ratings of a cue extinguished in isolation, suggesting that cues extinguished in compound were protected from the effects of extinction, and that the degree of protection conferred by a partner cue was independent of its training history.

General discussion

In the present study, we used a causal judgment task to examine whether the associative history of a partner cue influences the extinction of a target cue when the two are extinguished in a simultaneous compound. In each of the three experiments, the extinction to a target cue was measured following a manipulation of the associative history of its within-compound partner cue during extinction. The experiments differed with respect to the associative histories of the Stage 3 target cues and their within-compound associates.

Experiment 1 provided some evidence that a food, X—which had signaled that its otherwise allergenic partners C, D, and L were not followed by an allergic reaction—had become a preventative cause of the reaction. Participants were asked to choose between a meal containing X and the allergenic N versus one composed of Y and the allergenic M, where Y was identical to X except that it had been presented in compound with nonallergenic partners. Although the results failed to reach statistical significance, more participants selected NX as being less likely to cause an allergic reaction than MY. Moreover, among participants who selected NX as being less allergenic than MY, we found evidence that X had protected its allergenic partner, A, more than Y had protected its allergenic partner, B, across the extinction of AX and BY

compounds, because A was rated as being more causal than B. The rating test also showed that both A and B were judged as being more causal of allergy than G, extinguished in isolation, showing that both X and Y had protected their partners from extinction.

Contemporary theories of associative learning typically rely on error correction mechanisms to explain Pavlovian acquisition and extinction phenomena (see Le Pelley, 2004). Many of these theories hold that all of the cues present on a trial contribute to the error, whose size determines the amount of associative change and whose sign, positive or negative, determines whether this change is excitatory or inhibitory. According to such theories, the presence of the preventative cause, X, predicted the nonoccurrence of the allergy across the AX extinction trials, thereby reducing the size of the error term that determined how much inhibitory change would accrue to A. The size of this error across the AX trials was less than that across extinction of the BY trials, because X was better able to predict the nonoccurrence of the allergic reaction than was Y, resulting in less associative change (more protection) to A than to B. Moreover, in contrast to the protection afforded to A and B by their X and Y partners, no such protection would be associated with G, which would have accrued all of the associative change across its extinction in isolation. A corollary of such theories is that the presence of X will enhance acquisition to a novel A across pairings of AX and the allergic outcome. This enhancement will occur because the outcome (the allergy) is the opposite of that predicted by X. Therefore, the size of the error term will be large, and the amount of excitatory change to A will be greater across pairings of AX and the allergy than when a novel cue is compounded with one that has already signaled the allergy. Just these results have been reported in judgments of causal relations in people (Aitken, Larkin, & Dickinson, 2000).

Theories that rely on a common error term to explain associative change predict that the amount of change to an allergenic food will be regulated by the associative value of its partner across extinction of the compound. In Experiment 2, we examined whether a target allergenic food undergoes less change when it is extinguished in compound with an already extinguished allergenic food rather than with another equally allergenic food. According to such theories, the target will undergo less change (it will be more protected) when extinguished in compound with the already extinguished food, because the size of the error term is smaller than when the partner is another allergenic food. In fact, such theories predict that, depending on the parameters, an equally allergenic food can enhance extinction to its target partner relative to an allergenic food extinguished in isolation. Neither prediction was confirmed: A target extinguished in compound with an already extinguished food and another extinguished with a second allergenic food were protected equally, both being rated as less causal than an allergenic food extinguished in isolation. The failure to detect a difference between the ratings of targets extinguished in compound with already extinguished or with allergenic foods replicates our previous findings using a similar task with people (O. Griffiths & Westbrook, 2012). The demonstration that these ratings were greater than those shown to an allergenic food extinguished in isolation extends the previous findings, allowing the conclusion of equal protection relative to the food extinguished in isolation.

Such theories make additional predictions with respect to the effects of additional extinction on an already extinguished allergenic food. One prediction is that extinguishing an already extinguished target in compound with another extinguished allergenic food will restore their allergenic properties, and that additional extinction of this compound will deepen extinction to the target relative to an extinguished allergenic food given additional extinction in isolation. A second prediction is that a target allergenic food extinguished first in isolation and then in compound with an allergenic food will produce even more deepening of extinction to the target. These predictions have been confirmed in animal studies (Leung et al., 2012; Rescorla, 2006), but were not confirmed in the present study with people. Participants received extinction of A, C, and E and then additional extinction of these foods in compound with either an allergenic food (AB), another extinguished allergenic food (CD), or a safe food (EF), and were extinguished throughout to G in isolation. Subsequently, participants rated A, C, and E, as being more (not less) allergenic than G, showing that additional extinction of a target in compound with any other partner (currently allergenic, extinguished or safe) protected against extinction, rather than deepening extinction relative to G extinguished in isolation.

The present findings are inconsistent with the use of a common error term to produce change in extinction. It is possible, for example, that during the extinction of a target cue in simultaneous compound with a partner cue, the very presence of the partner may have been sufficient to reduce processing of the target, and therefore, to protect it from extinction (cf. Mackintosh, 1971; Sutherland & Mackintosh, 1971). However, an inhibitory partner conferred more protection to its associated target than did a neutral partner, which challenges this explanation. Alternatively, the theory of associative learning proposed by Pearce (1987, 1994, 2002) also holds that learning about a target cue is regulated by a process of error reduction. However, unlike common error-term theories, in which the associative change produced by the error signal acts on the elements in a compound, Pearce (1987) held that (1)the change accrues to a single unique configural representation constituted by those elements, and (2)the associative strength of the compound influences test responding to the elements through generalization. For example, during the extinction of a target cue,

X, in compound with a neutral cue, N, a node representing the NX compound will accrue inhibition to offset the excitation of X. However, test presentations of X alone will only activate the NX node to half strength (and the X node to full strength), resulting in a loss of inhibition, and therefore, more conditioned responding relative to a control cue, Y, extinguished throughout in isolation (i.e., protection from extinction). As such, the theory anticipates that a target allergenic cue extinguished in compound with a neutral cue will be rated as more allergenic than a control allergenic cue extinguished alone. This result was seen in Experiments 1–3.

Critically, Pearce (1987, 1994, 2002) also allowed the associative history of a partner cue to influence the extinction of a target cue across the extinction of the compound. This is because the partner cue will regulate the amount of inhibition that accrues to the compound representation across extinction, and therefore, the amount of generalized inhibition during test presentations of the target. For example, an inhibitory partner reduces the amount of inhibition that accrues to the configural representation of the inhibitor–target compound. This, in turn, reduces the amount of generalized inhibition to test presentations of the target alone, resulting in more responding to a cue extinguished in the presence of a conditioned inhibitor than to either a control cue extinguished with a neutral partner (above) or to a control cue extinguished in isolation. The theory therefore predicts that a target allergenic cue extinguished in compound with an inhibitory partner (or preventative cause) will be rated as more allergenic than control allergenic cues extinguished in compound with a neutral partner or alone, which was the primary finding in Experiment 1.

For the case of an extinguished partner cue, Pearce (1987, 1994, 2002) held that a completely extinguished cue is functionally equivalent to a neutral cue, and therefore, that a target extinguished in compound with an extinguished partner should evoke as much test responding as a target extinguished in compound with a neutral partner (via the same mechanisms described above), and should evoke more responding than a control cue extinguished in isolation. These results were obtained in Experiment 2 (see also O. Griffiths & Westbrook, 2012).

In contrast, the model's predictions concerning the hypothesized deepening of extinction of cue A, via additional AB– extinction trials in Experiment 3, are more complex. This is because the directional predictions are parameter-dependent. Consider the case in which the target cue (e.g., cue A) is only partially extinguished in isolation (in Stage 2) prior to extinction in compound with the excitor (B) in Stage 3. In this case, test responding will be greater to the target cue than to a control cue extinguished alone; that is, the excitor will protect the target from extinction. This is because the bulk of inhibitory learning in extinction will accrue to the excitor–target compound, and this representation is only activated to half-strength during test presentations of the target alone at test, thereby diminishing its capacity to suppress responding. This result was obtained in Experiment 3 (see also Shanks, Darby, & Charles, 1998, Exp. 1).

In contrast, if the target cue (e.g., A) is completely extinguished prior to extinction in compound with the excitor (cue B), the model predicts the opposite result (in line with associative models that propose a common error term). Specifically, it predicts that responding to the target cue (A) will be less than that to the elementally extinguished control (cue G); that is, the excitor will deepen the extinction of the target. Initial extinction of the target cue alone returns the strength of the target node to zero, and additional extinction of the excitor–target compound permits accrual of negative associative strength to its representation. Hence, partial activation of the excitor–target representation at test suppresses responding to the target cue (via generalization) below the level of the elementally extinguished control cue.

However, even with favorable parameters, Pearce's (1987, 1994, 2002) theory does not explain all of the results reported in the present study. It incorrectly predicts that a neutral (the safe) partner should (if anything) confer more protection than an extinguished partner, which in turn should confer more protection than an excitatory partner, parameters permitting (neutral≥ extinguished> excitatory). Instead, the neutral, extinguished, and excitatory partners were indistinguishable in the amounts of protection they afforded their respective targets (Exps. 2 and 3). Pearce's model also fails to accommodate results reported in conceptually similar studies that have used the same paradigm. For example, Shanks, Charles, Darby, and Azmi (1998, Exps. 3 and 4) showed that initial learning about target cues presented alone is highly resistant to change when a compound of those cues subsequently signals the opposite outcome (see also the "highlighting" effect: Kruschke, Kappenman, & Hetrick, 2005). This was true when the initial learning was excitatory (A–allergy, B–allergy) and subsequent learning was inhibitory (AB–no allergy), as well as when the initial learning was inhibitory (C–no allergy, D–no allergy) and subsequent learning excitatory (CD–allergy). The latter results were taken to imply that, in the allergist task, participants more rapidly configured simultaneously presented cues than was anticipated by Pearce (1987), and hence, that the learning that accrues to the configural representation of these cues more effectively preserves (or protects) the initial response-evoking tendency of its constituent elements (see also Shanks, Darby, & Charles, 1998).

Nevertheless, the configural theory is perhaps best placed among those in the associative tradition to explain the contrasting results reported here and in previous animal studies. This is specifically because the theory allows an excitatory partner cue to both protect and deepen the extinction of a target cue, depending on the parameters, which may be taken to reflect differences between the procedures used to study the effects of compound extinction in animal and human studies.

For example, extinction in animal studies is evident as a decline in levels of responding across nonreinforced CS exposures; it is usually conducted across multiple sessions on multiple days, until performance reaches an extinction criterion or until extinction is complete. In contrast, the extinction of human causal judgments is usually evidenced by a rapid decline in the numbers of participants who choose the "outcome present" response alternative (e.g., allergy) when faced with a binary choice. Moreover, because this measure changes rapidly, it tends only to be examined across a few brief stages of training. In both cases, the change in responding is taken to infer a decline in US expectancy across extinction; however, only in the former case is the net strength of either a CS–US or a cue–outcome association indexed directly, as in the vigor or intensity of behavioral responses.

Before concluding, it is worth noting that other types of theories—for example, causal models (T.L. Griffiths & Tenenbaum, 2005)—potentially explain various aspects of the present findings. The application of these theories to the present findings is certainly of interest; however, because our study was designed to investigate whether the effects of compound extinction observed in animal studies could be observed in the case of the extinction of causal judgments in humans, we have selectively reviewed the implications of the present findings for models in the associative tradition. It will be for future studies to determine the points of difference between associative and causal models, and to test these points of difference in so far as they apply to judgments of causality in people.

In summary, the present findings have shown that a target cue was more effectively protected from extinction by an inhibitory partner than by a neutral partner (Exp. 1), and that neutral, extinguished, and excitatory partners conferred equal amounts of protection to their target associates, relative to a cue extinguished alone (Exps. 2 and 3). Most of these findings are consistent with the central feature of Pearce's (1987, 1994, 2002) theory that new learning in extinction accrues to a unique configured representation of all of the cues present on a trial. Participants appear to view an allergenic food extinguished in compound as a meal that signals the absence of an allergic reaction, and whose components are largely unaffected by this new learning (see also Shanks, Charles, et al., 1998, and Shanks, Darby, & Charles, 1998).

Author note Authors N.M.H. and O.G. contributed equally to this work.

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