

1 **Interactions between the elements of an outcome in human**
2 **associative learning**

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Abstract

When a cue is established as a reliable predictor of an outcome (A-O1), this cue will typically block learning between an additional cue and the same outcome if both cues are subsequently trained together (AB-O1). Three experiments sought to explore whether this effect extends to outcomes in humans using the food allergist paradigm. In all three experiments an outcome facilitation effect was observed as opposed to outcome blocking. That is, prior learning about an element of an outcome compound (A – O1) appeared to facilitate learning about a novel outcome when (A – O2) these outcomes were presented together (A – O1 O2) relative to a control stimulus that first received C – O3 trials prior to C – O1 O2 trials. In Experiment 2, however, participants were also presented with an additional set of control trials, which were presented during Stage 2 only and reliably predicted the outcome compounds. At test participants displayed more learning about these additional control trials relative to the blocked outcomes, thus displaying an outcome blocking effect alongside an outcome facilitation effect. In Experiment 3 a one-trial outcome blocking procedure was employed in order to distinguish theoretical accounts of these findings. This procedure revealed an outcome facilitation effect but not an outcome blocking effect. These results can be understood in terms of an account derived from Wagner's (1981) model. The implications of these findings are discussed.

Keywords: Outcome interactions; outcome blocking; outcome facilitation; outcome associability; associative learning.

1 When two or more cues are presented in compound this can impact upon the
2 way in which these cues are learned about relative to when they are presented alone
3 (e.g., Kamin, 1968; 1969; Wagner, Logan, Haberlandt, & Price, 1968; Pavlov, 1927).
4 This is perhaps best demonstrated by the blocking effect first reported by Kamin
5 (1968, 1969). In Kamin's experiments pretraining with an element (A+) of a
6 compound stimulus (AX+) and an unconditioned stimulus (US), resulted in an
7 attenuated conditioned response to the stimulus which received no prior training (X).
8 This effect has subsequently been reported in a diverse range of species including rats
9 (e.g., Bonardi, 1991; Dopson, Pearce, Haselgrove, 2009; Jones & Haselgrove, 2013,
10 Mackintosh, 1975), mice (Sanderson, Jones & Austen, 2016), snails (Prados et al.,
11 2013), honeybees (Cheng & Spetch, 2001; Guerrieri, Lachit, Gerber & Giurfa, 2005)
12 and humans (Crookes & Moran, 2003; Dickinson, Shanks & Eveden, 1984; Le Pelley,
13 Oakeshott, McLaren, 2005), and a variety of theoretical accounts have been provided
14 in order to account for its ubiquitous nature. According to the performance-deficit
15 account an association is formed between the US and the additional element of the
16 compound (X), but the expression of this association (X – US) is prevented during a
17 testing phase (e.g., Acrediano, Escobar & Miller, 2004; Denniston, Savastano &
18 Miller, 2001; Miller & Matzel, 1988). According to the learning-deficit account (e.g.,
19 Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972), blocking occurs
20 due to an attenuation in learning about the association between the additional element
21 of the compound (X) and the US, as a result of the pretraining with the other stimulus
22 element (A - US).

23 The latter account of blocking typically appeals to the idea that there is some
24 form of competition between the elements of the compound as they enter into an
25 association with the US. For instance, according to the Rescorla and Wagner (1972)

1 model, learning will not take place between the additional element (e.g. X) of the
2 compound and the US, following training with A+ trials, as the effectiveness of the
3 US is reduced. The Mackintosh (1975), and Pearce and Hall (1980) models state that
4 blocking occurs, for rather different reasons, because the associability of X diminishes
5 after the first compound conditioning trial with AX. Although the literature is replete
6 with demonstrations of blocking between cues, relatively little research has explored
7 whether this effect extends to outcomes. That is, whether prior learning about a cue
8 and one element of an outcome (e.g., shock) will block learning about the association
9 between the cue and an additional element (e.g., a startling noise) if it were added to
10 the original outcome to form an outcome compound. This lack of investigation could
11 be partly attributable to the biases present in the associative learning models noted
12 above. These models afford cues and outcomes differential statuses, with mechanisms
13 provided to explain how cues interact with one another, whilst remaining silent as to
14 whether outcomes are granted the same status.

15 Nevertheless, there is evidence to suggest that outcomes may produce
16 interaction effects comparable to those observed in cues. For instance, in his 1980
17 book, *Pavlovian Second-Order Conditioning*, Rescorla reported a series of
18 experiments that explored whether interactions can occur between the elements of a
19 compound US. In these experiments a second-order autoshaping procedure was
20 employed with pigeons where the presentation of visual cues was followed by
21 second-order USs. In an experimental group, pigeons were presented with
22 presentations of A – O1 and A – O1&O2. A control group of pigeons were also
23 presented with A – O1&O2 trials; however, in this group subjects were not exposed to
24 the A – O1 pairings. The question of interest concerned the extent to which each
25 group learned the A – O2 association. The results revealed that subjects in the control

1 group, who had no exposure to A-O1, demonstrated greater learning about the
2 association between A – O2 than those in the experimental group, therefore
3 demonstrating an ‘outcome blocking’ effect. Rescorla suggested that these results
4 could be explained by assuming that the previously autoshaped O1 in the
5 experimental group acquired substantial biological significance and hence distracted
6 the pigeons from attending to O2, when these were presented in compound. This
7 interpretation suggests that rather than the associative properties of the previously
8 exposed outcome blocking learning about the novel outcome, the animals simply
9 failed to notice it, thus providing an alternative non-associative account of the
10 findings. Nevertheless, it remains possible that the A-O1 association prevented
11 learning about the A-O2 association in a manner that is conceptually comparable to
12 the way in which blocking occurs in cues.

13 Miller and Matute (1998) further investigated this phenomenon in a sensory-
14 preconditioning experiment with rats. In their experiment rats in Group Outcome
15 Blocking were first presented with a click train which served as a signal (A) for a
16 white noise (O1). In Stage 2, rats were then presented with the cue (A) and a
17 compound outcome (O1 O2) featuring the white noise and a tone. In a subsequent
18 stage O2 was reliably paired with a footshock. At test, subjects were presented with
19 the cue A. The question of interest was whether the A – O1 pairings in Stage 1
20 blocked learning about the A – O2 association, thus resulting in the presentation of
21 the cue at test failing to elicit the anticipation of O2, and hence the expectation of a
22 footshock. This was precisely the effect observed: subjects displayed less fear towards
23 cue A relative to a control group which had not received exposure to the cue in Stage
24 1. This result is therefore comparable to the outcome blocking effect reported by
25 Rescorla (1980), however, Miller and Matute’s (1998) results are less easily explained

1 by the “distraction” account that can be developed to explain Rescorla’s (1980)
2 results as O1 did not come to acquire biological significance until after the outcome
3 compound training stage.

4 Similar studies have also been conducted in humans however, these studies
5 have produced heterogenous findings. For example, Cobos, Lopez, Caño, Almaraz
6 and Shanks (2002) employed a causal-judgement task in which participants were
7 required to take on the role of workers in a chemical plant which leaked dangerous
8 substances. The substances resulted in different indicator lights being triggered and
9 participants were required to learn the association between the substances and the
10 indicator lights. In Experiment 3 of their study participants’ ratings were comparable
11 for an outcome which was exclusively predicted by a cue, and an outcome presented
12 in compound with another outcome which was reliably predicted on a series of
13 separate elemental trials. These results thus demonstrate the absence of an outcome
14 blocking effect, and are therefore inconsistent with the findings of Miller and Matute
15 (1998) and Rescorla (1980); comparable findings have also been reported by Price
16 and Yates (1995).

17 Alternatively, however, a study which may also have some relevance, by
18 Flach, Osman, Dickinson and Heyes (2006), found a different set of results.
19 Experiment 1 of Flach et al. sought to observe the response-priming effect first
20 observed by Elsner and Hommel (2001). In their procedure, a training stage was first
21 presented to participants where participants were asked to make a response by
22 pressing one of two keys. Upon selecting a key, a coloured background was briefly
23 presented. In a subsequent Test Stage, participants were presented with the
24 backgrounds and were asked to make a specified response as quickly as possible. For
25 Group Congruent the response was the same as during the training stage, for Group

1 Incongruent the response was the opposite to the training stage. The results revealed
2 that whilst participants made a comparable number of errors in each group, response
3 times were shorter in Group Congruent than Group Incongruent, replicating the
4 response-priming effect reported by Elsner and Hommel (2001). In Experiment 2 of
5 their study, participants received the same Stage 1 training as in Experiment 1,
6 however, in Stage 2 an auditory stimulus was presented alongside the visual stimulus
7 upon responding, thus forming what could be conceived of as an outcome compound.
8 In a Pretrained Condition the response for the visual stimulus in Stage 2 was the same
9 as in Stage 1. In a Control Condition, however, the visual stimulus and the auditory
10 stimuli were both novel in Stage 2. At a test stage the question of interest was how
11 quickly participants were able to provide the required response to the auditory
12 stimulus. As such, these results could be taken to provide evidence of an interaction
13 effect comparable to the outcome blocking effect reported in animals (e.g., Miller and
14 Matute, 1998; Rescorla, 1980). In Experiment 3 of Flach et al., however, the auditory
15 stimuli were presented in the initial training stage prior to being presented in
16 compound with visual stimuli in Stage 2. Under these circumstances, the opposite
17 effect was observed. That is, pretraining with an element of an outcome compound
18 enabled better learning about the added visual stimulus, therefore producing what can
19 be referred to as an outcome-facilitation effect. Importantly, this study differs from
20 the others in that the interaction effects observed in Flach et al.'s Experiments 2 and 3
21 were more comparable to overshadowing and potentiation rather than blocking.
22 Moreover, the experimental paradigm employed (i.e., response-effect priming) differs
23 to that of standard conditioning procedures. Nevertheless, given that the temporal
24 order of the compound stimuli during training is comparable to the aforementioned

1 studies (arguably serving as outcomes) it is appropriate to take these findings into
2 consideration.

3 Taken together the results of the above studies suggest that interaction effects
4 are not unique to cues (also see, Arcediano, Matute, Escobar & Miller, 2005; Matute,
5 Arcediano & Miller, 1996; Miller & Escobar, 2002). These results therefore question
6 the assumptions of key associative learning models, which go to great lengths to
7 explain how cues may interact to modulate associative learning, but which are,
8 largely, silent about comparable situations in outcomes. Indeed, Miller and Matute
9 (1998) note that demonstrations of outcome blocking appear to challenge the
10 differential treatments of cues and outcomes within these models. As can be seen
11 from the above studies, however, outcome blocking is not the only effect to be
12 observed, with facilitation effects also being observed (e.g., Flach et al. 2006) and
13 null effects also being reported (e.g., Cobos et al. 2002; Price & Yates, 1995), the
14 latter of which are consistent with the predictions of most associative learning models.
15 Given the heterogenous results of the above studies and the discrepancy between
16 theory and data, it is important to further explore if, and how, the elements of an
17 outcome may interact during learning. Aside from the theoretical conundrum that the
18 presence of this effect may pose to theories of associative learning, it would seem
19 prudent to explore the parameters of interaction effects in outcomes given the relative
20 scarcity of research on outcome interaction, and the prevalence and importance of this
21 effect - in cues - to associative learning. As such, the following experiments sought to
22 explore the generality of outcome interaction effects in human participants using a
23 food-allergist paradigm. If previous learning about an element of a compound
24 outcome can block learning between a cue and an additional outcome in a manner
25 consistent with Miller and Matute (1998) and Rescorla (1980), it would be expected

1 that a ‘blocked’ outcome would be learned about less well than a control outcome
2 which is surprising. If, however, the elements of an outcome do not compete for
3 associative strength as a result of their associative history – in the same manner as
4 cues – it would be expected that the outcomes will be learned about to the same
5 extent, as is suggested by some studies (Cobos et al. 2002; Price & Yates, 1995) and
6 models of associative learning.

8 **Experiment 1**

9
10 Experiment 1 employed a causal-judgement version of the food allergist task
11 (e.g., Le Pelley & McLaren, 2003; Van-Hamme & Wasserman, 1994), in which
12 participants were asked to imagine themselves as a food allergist tasked with
13 identifying which foods caused certain allergic reactions. Participants were informed
14 about the foods which a fictitious patient (‘Mr. X’) had consumed and were then
15 presented with information about the reaction the patient received. The foods (A – D)
16 served as cues, whilst the reactions (O1-O4) served as outcomes. The design of
17 Experiment 1 can be seen in Table 1.

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20 Insert Table 1 here
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1 In Stage 1, four cues (A – D) were presented, each of which reliably predicted
2 one of two outcomes (O1 or O3) and participants were tasked with learning which
3 outcomes were paired with which cues. Prior to Stage 2, participants were informed
4 that Mr. X had started to display some additional reactions. Once Stage 2 commenced
5 the same four cues were presented. On blocking trials, cues A and B continued to
6 predict the same outcomes as in Stage 1 (O1 and O3 respectively) and an additional
7 novel outcome (O2 and O4 respectively), to serve as a blocked outcome. For the
8 control trials, participants were presented with cues C and D, which now predicted
9 outcomes which these cues had not previously been paired with in Stage 1 (i.e. O1 and
10 O3 respectively), and a novel outcome (i.e. O2 and O4), therefore rendering all
11 outcomes surprising. During a subsequent test stage, participants were required to
12 provide ratings concerning how predictive the cues (A - D) were of each of the
13 outcomes. If the Stage-1 training with A – O1 / B - O3 blocked learning about A - O2 /
14 B - O4, it would be expected that participants would provide lower test ratings
15 concerning the relationship between A - O2 / B- O4 relative to ratings for C - O2 / D -
16 O4.

17 **Method**

18 **Participants**

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22 Thirty-two participants (24 females; 8 males) were recruited from the
23 University of Nottingham's School of Psychology. Participants ranged from 18 – 30

1 years of age ($M = 21.7$; $SEM = 0.61$). All participants had normal or corrected-to-
2 normal vision. Participants received course credit for their participation or a £3
3 inconvenience allowance. The study received ethical approval from the University of
4 Nottingham's Psychology ethics committee.

6 **Materials**

7 All stimuli were presented (and responses recorded) in the graphical
8 experimental software package PsychoPy2, v1.83.01 (see Peirce, 2007; Peirce 2009),
9 running on Windows 10 on a standard desktop computer (screen size: 27cm × 46cm; h
10 × w). The foods Mr. X had eaten served as the cue stimuli. The reactions Mr. X
11 experienced served as the outcomes. Each of the foods and reactions were assigned to
12 the letters and outcomes in Table 1 using a Latin-Square counterbalancing technique
13 with each food serving as cues A – D, and each reaction serving as outcomes O1 – O4,
14 across the course of the experiment. There were four pictures of foods: broccoli,
15 cabbage, onion and potato (see Figure 1). Each food picture (9mm × 8mm; h × w) was
16 presented in the centre of the screen against a grey background. The reactions Mr. X
17 experienced served as the outcome stimuli. There were four different types of reactions
18 Mr. X experienced: diarrhoea, fever, skin rash and vomiting.

19 In Stage 1, participants were only presented with two of these reactions (e.g.
20 vomiting and diarrhoea). In Stage 2, participants were presented with all four of the
21 reactions, with each of the novel reactions from Stage 2 being presented alongside the
22 Stage 1 reactions to form a compound outcome stimulus (e.g. vomiting was presented
23 alongside fever to form the compound stimulus 'VOMITING AND FEVER', whilst

1 diarrhoea was paired with skin rash to form the compound stimulus ‘DIARRHOEA
2 AND SKIN RASH’). Each of these reactions was presented in capitalised white Arial
3 text (font size: 32) at the bottom of the screen. The reactions were positioned beneath a
4 white scale (length: 35cm). Participants could select their choice of reaction by
5 positioning a cursor on the scale above the reaction they wished to select (see Figure 1
6 for example). Participants could only select the ends of the scale and thus had to make a
7 binary choice. The order of the reactions beneath the scale was counterbalanced
8 between participants. At test, each of the cues was presented individually (positioned
9 on the left of the screen) and participants were asked to rate how predictive each cue
10 was of each of the reactions. Each of the reactions were positioned, alongside the food,
11 on the right of the screen (see Figure 1 for example). Participants made their ratings for
12 each reaction by moving a cursor on a Likert scale which ranged from 0 – 100 [‘0’ =
13 ‘Very unproductive’, ‘100’ = Very predictive”].

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15 -----
16 Insert Figure 1 here
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19 Procedure

20 Participants were tested individually or in small groups of two to three. Once
21 participants had provided informed consent they read the following instructions on a
22 computer monitor before proceeding to Stage 1:

23 *“Thank you for participating in this experiment.”*

1 *In this experiment we would like you to imagine that you are an allergist (i.e.*
2 *someone who tries to discover the cause of allergic reactions). You have just been*
3 *presented with a new patient who suffers from different types of allergic reactions as*
4 *a result of eating certain foods. In an attempt to discover which foods cause the*
5 *different types of allergic reaction in Mr. X, you arrange for him to eat a number of*
6 *different foods on their own, and observe the type of allergic reaction he suffers. On*
7 *the following screens, you will be shown the foods Mr. X has eaten, and you will be*
8 *asked to predict what type of allergic reaction he will suffer as result of eating each*
9 *meal. Each allergic reaction will be presented at the bottom of the screen. Make your*
10 *prediction by selecting one of the allergic reactions below each of the foods. You will*
11 *then be provided with feedback about what reaction Mr. X experienced. You will have*
12 *to guess at first, but with the aid of the feedback your predictions should soon start to*
13 *become more accurate. The participant with the highest overall accuracy will receive*
14 *a £10 cash prize. Please press the SPACE BAR to begin.”*

16 **Stage 1**

17 In Stage 1 participants were exposed to 6 blocks of 16 trials (96 trials in total).
18 Trial order was block randomised. There was no break between blocks. In each block
19 participants received 4 pairings of each cue – outcome pairing (A – O1, B – O3, C – O3
20 and D – O1). On each trial, participants were presented with the following text at the
21 top of the screen: *“After eating this food, what allergic reaction did Mr. X*
22 *experience?”*. This text was presented above the food which was being presented. To

1 proceed to the next trial, participants needed to make a response by selecting a reaction
2 beneath the food. Once they had made a response, by selecting a reaction out of the
3 binary choices available to them, they were presented with feedback informing them
4 about the reaction Mr. X had experienced. On this screen participants were presented
5 with the following text: “*Mr X. experienced: [reaction name here]*”. The reaction Mr.
6 X experienced was presented in the centre of the screen. Participants could take as
7 much time as they needed when selecting which reaction they thought Mr. X had
8 experienced. The feedback screen was presented for 1.5 seconds, before the next trial
9 commenced automatically. Once all 96 trials were complete, Stage 2 began.

11 **Stage 2**

13 Prior to commencing Stage 2, participants were presented with the following
14 text: “*Mr X has started to display additional reactions when eating these foods. The
15 reactions will be listed at the bottom of the screen as before. Please press the SPACE
16 BAR to continue.*”

17 In Stage 2, participants were exposed to 2 blocks of 16 trials (in line with Stage
18 1, trials were block randomised) with each trial type being presented a total of 4 times
19 per block. As in Stage 1 participants were presented with the text: “*After eating this
20 food, what allergic reaction did Mr. X experience?*” which was positioned above the
21 food being presented. However, this time there were additional reactions presented
22 alongside the Stage 1 reactions (e.g. ‘VOMITING’ from Stage 1, would now be

1 presented with 'FEVER' in Stage 2 to form 'VOMITING AND FEVER', see Figure 1).
2 For half of the participants the Stage 2 reaction was presented on the right of the
3 reaction from Stage 1 (e.g. O1 O2); for the other half the Stage 2 reaction was
4 positioned on the left of Stage 1 reaction (e.g. O2 O1). Once participants completed all
5 32 trials of Stage 2, they proceeded to the test stage.

7 **Test**

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9 Prior to commencing the test stage, participants were presented with the
10 following text: "*You will now be asked to rate how predictive each food was of each of*
11 *the reactions. Please press the SPACE BAR to continue*". Each of the foods were then
12 presented at test, one per screen (see Figure 1). On each screen participants were
13 presented with the text: "*Please rate how predictive this food was of each of the*
14 *reactions. Make your rating and then click on the grey box beneath the scale to confirm*
15 *your rating [0 = very unpredictable; 100 = very predictive]*". Participants were then
16 required to make a rating for each of the reactions using the Likert scales positioned to
17 the right of the reactions. Once they had rated each food's ability to predict each
18 reaction, they were informed that the experiment was complete.

19

Results and Discussion

Insert Figure 2 here

Stage 1

The top left panel of Figure 2 shows the mean proportion of correct responses for each cue – outcome pairing (A-O1, B-O3, C-O3 and D-O1) across the 24 trials of Stage 1. Individual data were averaged across participants' responses for each trial for the blocking stimuli A and B, and for the control stimuli C and D. As can be seen in Figure 2, learning progressed rapidly for both the A-O1/B-O3 trials and the C-O3/D-O1 trials, with both cue-outcome associations being learned about at a comparable rate. From Trial 4 onward participants were approaching asymptote for both A-O1/B-O3 and C-O3/D-O1 with participants producing a mean correct response rate in excess of 90%.

A 2×24 repeated measures analysis of variance (ANOVA) was performed on these data with the factors of Stimulus (A-O1/B-O3 vs C-O3/D-O1) and Trial (1 – 24). The ANOVA revealed a significant main effect for the factor Trial, $F(23, 690) = 40.73$, $MSE = .02$, $p < .001$, $\eta_p^2 = .58$. There was no significant effect of outcome, $F(1, 30) = .67$, $MSE = .02$, $p = .42$, $\eta_p^2 = .02$, and no interaction between outcome and trial, $F(23, 690) = .58$, $MSE = .02$, $p = .95$, $\eta_p^2 = .02$. The ANOVA therefore confirmed the impressions of the graph, revealing that participants learned about both

1 A-O1/BO3 and C-O3/D-O1 at comparable rates, with most learning occurring on the
2 first two trials before reaching asymptote.

3 4 **Stage 2**

5 The top right panel of Figure 2 shows the mean proportion of correct
6 responses for each cue – outcome compound (A-O1O2, B-O3O4, C-O1O2 and D-
7 O3O4) across the 8 trials of Stage 2. These data were again averaged across each trial
8 type (Blocking: A-O1O2/B-O3O4; Control: C-O1O2/D-O3O4). As can be seen in
9 Figure 2, participants initially demonstrated a higher proportion of correct responses
10 for the blocking trials than the control trials, suggesting they selected the outcome
11 compound featuring the outcome which the cue was paired with during Stage 1
12 (yielding correct responses for the blocking trials, and incorrect responses for the
13 control trials). From Trial 3 onward, however, both trials produced a comparable
14 percentage of correct responses. Furthermore, participants appeared to reach
15 asymptote from Trial 6 onward, with a mean percentage of correct responses in
16 excess of 90%.

17 A 2×8 repeated measures ANOVA was performed on these data with the
18 factors of Outcome type (A-O1O2/B-O3O4 vs C-O1O2/D-O3O4) and Trial (1 – 8). In
19 light of sphericity violations for the factor ‘Trials’ $\chi^2(27) = 124.58, p < .001$ and
20 the interaction between the factors, $\chi^2(27) = 89.89, p < .001$, Greenhouse-Geisser
21 values are reported. The ANOVA revealed a main effect for the factor of Outcome
22 type, $F(1, 30) = 19.36, MSE = .07, p < .001, \eta_p^2 = .39$, a main effect for the factor
23 trial, $F(7, 210) = 51.27, MSE = .05, p < .001, \eta_p^2 = .63$, and a significant interaction

1 between these two factors, $F(7, 210) = 14.55$, $MSE = .08$, $p < .001$, $\eta_p^2 = .33$. To
2 explore the source of the interaction, simple main effects analyses were conducted.
3 These analyses revealed significant differences between the outcomes on Trial 1, F
4 $(1, 30) = 55.18$, $MSE = .02$, $p < .001$, $\eta_p^2 = .65$, and Trial 2, $F(1, 30) = 5.09$, $MSE =$
5 $.04$, $p < .05$, $\eta_p^2 = .15$, all other trials $p > .05$ (smallest $p = .37$). Overall, these results
6 demonstrate that participants initially produced more correct responses for the
7 blocking trials than the control trials, however, by Trial 3 onward participants
8 responses were comparable.

10 **Test Stage**

12 The bottom left panel of Figure 2 illustrates participants' outcome specific
13 ratings for the Blocked cues (A/B) and Control cues (C/D) at test. To calculate an
14 outcome specific measure of participants' ratings for cues A – D and O2 and O4,
15 participants' ratings for the novel outcome in Stage 2 which was not paired with the
16 cue of interest, was subtracted from the ratings provided for the novel outcome which
17 the cue was paired with in Stage 2 (see: Le Pelley, Beesley & Griffiths, 2014; Le
18 Pelley & McLaren, 2003). For instance, participants' ratings of A – O4 (the novel
19 outcome in Stage 2 which A was not paired with) were subtracted from participants'
20 ratings of A – O2 (the novel outcome in Stage 2 which A was paired with). As can be
21 seen participants ratings were higher for A/B – O2/O4 than C/D – O2/O4.¹ A paired
22 samples t-test of participants ratings for A/B – O2/O4 and C/D – O2/O4 revealed a

¹ Participants' raw ratings followed the same pattern as participants' outcome specific ratings (see Appendix).

1 significant difference between these ratings, $t(31) = 2.06$, $p = <.05$, $d = .36$. These
2 results reveal that pairing a cue with a novel outcome in compound with a well-
3 predicted outcome, facilitates learning about the association between the cue and the
4 novel outcome.

5 In summary, Experiment 1 demonstrated that when participants experienced
6 Stage 1 training featuring A–O1, B–O3, C–O3 and D–O1, followed by Stage 2 training
7 with A–O1O2, B–O3O4, C–O1O2 and D–O3O4, participants displayed greater
8 learning about A–O2 and B–O4, than to C–O2 and D–O4. These results are
9 inconsistent with those reported by Miller and Matute (1998) and Rescorla (1980),
10 which both report an outcome blocking effect, and those reported by Cobos et al.
11 (2002) and Price and Yates (1995) which reported null effects. In contrast, the results
12 from the current experiment demonstrate a facilitation effect comparable to that
13 reported by Flach et al. (2006).

14 15 16 17 **Experiment 2** 18

19 Experiment 1 demonstrated an outcome facilitation effect. That is, presenting a
20 novel outcome element alongside a well-predicted outcome resulted in better learning
21 about the added outcome, relative to a control where both outcome elements were
22 surprising. One possible explanation of these results may appeal to the type of control
23 employed within Experiment 1. A variety of controls have also been employed within
24 cue-blocking studies which has resulted in different effects being produced (see Blaser,
25 Couvillion & Bitterman, 2006; Guez & Miller, 2008). One of the most common control
26 types which has reliably demonstrated blocking is that reported by Kamin (1968, 1969).
27 In Kamin's studies the control trials simply involved omitting any Stage 1 training and

1 hence exposure to the cues and the outcome only in Stage 2. This control trial differs
2 from that employed in Experiment 1, as the control cues were exposed prior to Stage 2.
3 Given the relative novelty of the outcome facilitation effect, Experiment 2 sought to
4 examine whether this effect was reproduced with the control trial employed in
5 Experiment 1, and assess whether an outcome interaction effect could be observed
6 when an additional control trial was employed. More specifically, the additional control
7 used in this experiment was comparable to that employed by Kamin - but applied to an
8 outcome blocking procedure (henceforth referred to as the Kamin control). As such,
9 two additional cues were provided in a second stage of training which reliably
10 predicted the outcome compounds.

11 In addition, in Experiment 2, the nature of the task also varied between
12 participants. Half of the participants completed the same version of the task as in
13 Experiment 1 (i.e., a causal judgement task), where foods served as cues and reactions
14 served as outcomes. For the other half of the participants, however, the order of the
15 events was reversed. That is, the reactions served as cues and the foods served as
16 outcomes. As such, participants in this group were required to make a diagnostic
17 judgement, where they made a decision about the food Mr. X consumed on the basis of
18 the reaction he received. This was done to assess whether the effect was sensitive to the
19 type of judgement participants were required to make. Cobos et al. (2002) tested
20 whether the type of judgement participants made (i.e., causal or diagnostic) impacted
21 upon the cue-interaction effects observed in their experiments. In their study the type of
22 task participants completed had little influence on the effect observed. However,
23 similar studies have reported conflicting results, for example, a study by Waldmann
24 (2000) found the type of judgement task completed did indeed influence whether or not
25 blocking would be demonstrated amongst cues (also see Van Hamme, Kao &

1 Wasserman, 1993). In Waldmann’s experiments participants demonstrated blocking
2 amongst cues when a causal judgment task was employed, however, when participants
3 completed a diagnostic judgment task a blocking effect was not observed. As such, this
4 experiment also sought to assess if the type of judgement participants were required to
5 make influenced the interaction effect observed in Experiment 1.

6 The full design of Experiment 2 can be seen in Table 2. Given the introduction
7 of the Kamin control trials, this resulted in two additional cues being included in the
8 experiment, represented by stimuli E and F. Additionally, as both causal and diagnostic
9 versions of the task were employed, the letters in Table 2 represented foods for the
10 causal judgement version of the task, with the outcomes as reactions, and in the
11 diagnostic judgement version of the task, the letters represented the reactions, whilst the
12 outcomes represented the foods.

13
14 -----
15 Insert Table 2 here
16 -----

17
18 **Method**

19
20 **Participants**

21
22 Sixty-four participants (57 females; 7 males) were recruited from the
23 University of Nottingham’s School of Psychology. Participants ranged from 18 – 48
24 years of age (M = 23.84; SEM = 0.76). Participants were allocated randomly (on the
25 condition that there were a comparable number of participants in each group) to

1 complete either the causal judgment task (n = 32) or the diagnostic judgement task (n
2 = 32). Participants received course credit or received a £3 inconvenience allowance.

3 **Materials**

4
5 The six food stimuli were: broccoli, cabbage, mushroom, pepper, potato and
6 tomato. The six reaction stimuli were: diarrhoea, fever, no appetite, skin rash,
7 stomach cramps and vomiting. The causal-judgement version of the task was identical
8 to that employed in Experiment 1 with the inclusion of the additional foods, thus,
9 unless otherwise stated, the following refers exclusively to the diagnostic judgement
10 task. During the training stage for this task, the allergic reactions (serving as the cues)
11 that Mr. X experienced were presented in centre of the screen in capitalized white
12 Arial font (text size: 32), and pictures of the foods (serving as outcomes) were
13 presented above a white scale (length: 45cm), which was positioned beneath the
14 reaction. During the test stage, each of the reactions were presented individually on
15 the left of the screen, on the right pictures of each of the foods were presented.
16 Participants would then be required to rate each reaction's ability to diagnostically
17 predict which of the foods had been consumed. The Likert scale displayed to
18 participants when making their rating was identical to that used in Experiment 1.

19 **Procedure**

20
21 As in Experiment 1 both groups experienced 6 blocks of 16 trials in Stage 1. In
22 Stage 2, however, due to the additional Kamin control trials, participants were
23 presented with 2 blocks of 24 trials in Stage 2, before proceeding to test. There were
24 four of each trial type in each block [$6 \times 4 = 24$]. The procedure for Experiment 2 was
25 identical to Experiment 1 for those completing the causal judgment version of the task,

1 with the exception of the added Kamin control trials. As such, the following refers
2 exclusively to the diagnostic judgement task (unless otherwise stated).

3 For those completing the diagnostic judgement task, participants were presented
4 with the following instructions:

5 *“In this experiment we would like you to imagine that you are an allergist (i.e.*
6 *someone who investigates reactions to foods). You have just been presented with a*
7 *new patient “Mr. X”, who suffers from different types of reactions as a result of*
8 *eating certain foods. In an attempt to discover the relationship between the reactions*
9 *Mr. X experiences and the different types of foods he has eaten, you observe the type*
10 *of reactions he has experienced and try and workout which foods he had eaten.*

11 *On the following screens, you will be shown the REACTIONS Mr. X has*
12 *experienced, and you will be asked to predict which food he had eaten. Each FOOD*
13 *will be presented at the bottom of the screen. Make your prediction by selecting one*
14 *of the FOODS below each of the reactions. You will then be provided with feedback*
15 *about what FOOD Mr. X had eaten. You will have to guess at first, but with the aid of*
16 *the feedback your predictions should soon start to become more accurate. Please*
17 *press the SPACE BAR to begin”*

18 **Stage 1**

19

20 Once Stage 1 commenced, participants were presented with the following text at
21 the top of the screen: “Mr. X experienced the below reaction, which food did he eat?”
22 This text was presented above the reaction which was being presented. To proceed to
23 the next trial participants needed to make a response by selecting a food beneath the
24 reaction (the order of the foods was counterbalanced, i.e. each food occurred on either

1 side of the scale). Once they had made a response, they were then presented with
2 feedback informing them about the food Mr. X had eaten.

3 **Stage 2**

4
5 Prior to commencing Stage 2, participants were presented with the following
6 text: *“Mr X has started to eat additional foods. The foods he has eaten will be*
7 *presented at the bottom of the screen as before. Please press the SPACE BAR to*
8 *continue.”* On each trial participants were presented with the text: *“Mr. X experienced*
9 *the reaction (below), which foods did he eat?”* which was positioned above the reaction
10 which was being presented. However, this time there were additional foods which were
11 presented alongside the Stage 1 foods (e.g. ‘Potato’ from Stage 1, would now be
12 presented with ‘Pepper’ in Stage 2). The order in which the Stage 2 foods were
13 presented alongside the Stage 1 foods was counterbalanced across experimental
14 conditions (i.e., the Stage 2 foods were either presented first in the compound or
15 second, e.g., Potato and Pepper or Pepper and Potato).

16 **Test**

17
18 Prior to commencing the test stage, participants were presented with the
19 following text: *“You will now be asked to rate how predictive each reaction was of*
20 *each of the foods. Please press the SPACE BAR to continue”*. Each of the reactions
21 were then presented at test, one per screen. On each screen participants were presented
22 with the text: *“Please rate how predictive the reaction (below) was of each of the foods*
23 *[0 = very unpredictable; 100 = very predictive]. Make your rating and then press the*
24 *spacebar to proceed”*. Participants were then required to make a rating for each of the

1 foods using the Likert scales positioned to the right of the foods. Once they had rated
2 each reaction's ability to predict each food, they were informed that the experiment was
3 complete.

6 **Results and Discussion**

8 -----
9 Insert Figure 3 here
10 -----

12 **Stage 1**

14 Figure 3 show the mean proportion of correct responses for each cue-outcome
15 pairing (A-O1, B-O3, C-O3 and D-O1) across Stage 1 training for Group Causal (top
16 left panel) and Group Diagnostic (top right panel). As can be seen, regardless of the
17 task completed, participants rapidly learned the cue-outcome associations, reaching
18 asymptote from Trial 5 onward. A three-way mixed model ANOVA was performed
19 with a between-subject factor of task type (Causal vs Diagnostic), and within-subjects
20 factors of Stimulus (A-O1/B-O3 vs C-O3/D-O1) and Trial (1 – 24). The ANOVA
21 revealed a main effect of trial, $F(23, 1426) = 84.98, MSE = .05, p < .001, \eta_p^2 = .58,$
22 demonstrating that participants learned the associations as training progressed. There
23 was no effect of task type, $F(1, 62) = .93, MSE = .06, p = .34, \eta_p^2 = .02,$ and no effect

1 of outcome, $F(1, 62) = .03$, $MSE = .02$, $p = .86$, $\eta_p^2 = .00$. All interactions were non-
2 significant, largest $F(23, 1426) = 1.72$, $MSE = .06$, $p = .13$, $\eta_p^2 = .03$.

4 **Stage 2**

5 Figure 3 demonstrates the mean percentage of correct responses for each cue –
6 outcome compound (Blocking: A-O1O2/B-O3O4, Control: C-O1O2/D-O3O4, Kamin
7 control: E-O1O2/F-O3O4) during Stage 2 training for both the causal (bottom left
8 panel) and diagnostic groups (bottom right panel). As can be seen, participant's
9 performance followed a similar trend regardless of whether participants were
10 completing the causal or diagnostic task. When Stage 2 commenced, participants
11 primarily selected the outcome compound which contained one of the outcomes that
12 the respective cue was previously paired with. Therefore, participants demonstrated a
13 high proportion of correct responses for A and B trials, and an initially low (below
14 chance) proportion of correct trials for C and D. For cues E and F participants mean
15 percentage of correct responses were initially at chance level, but by the end of
16 training all stimuli were responded to equivalently.

17 A three-way mixed model ANOVA was performed on these data with a
18 between subjects factor of Task type (causal vs diagnostic), and within-subjects
19 factors of Cue-outcome pairing (A-O1O2/B-O3O4 vs C-O1O2/D-O3O4 vs E-
20 O1O2/F-O3O4) and Trial (1 – 8). The ANOVA revealed a main effect for the cue-
21 outcome pairings, $F(2, 124) = 21.26$, $MSE = .09$, $p < .001$, $\eta_p^2 = .26$, and Trial, F
22 $(7, 434) = 96.42$, $MSE = .07$, $p < .001$, $\eta_p^2 = .61$, and a significant interaction between
23 these factors, $F(14, 868) = 12.87$, $MSE = .08$, $p < .001$, $\eta_p^2 = .17$. There was no

1 main effect of the factor of task type, $F(1, 62) = .54$, $MSE = .19$, $p = .46$, $\eta_p^2 = .00$,
2 and all other interactions were non-significant, largest $F(2, 124) = 1.15$, $MSE = .10$, p
3 $= .32$, $\eta_p^2 = .02$. Simple main effects identified the cause of the Cue-Outcome pairing
4 \times Trial interaction. There was an effect of outcome on Trials 1 to 3, largest $F(2, 61) =$
5 48.14 , $p < .001$, $\eta_p^2 = .61$. All other trials were non-significant, smallest $p = .08$.
6 Bonferroni corrected (adjusted $p = .005$) t-tests revealed that for Trial 1 the mean
7 proportion of correct responses for A-O1O2/B-O3O4 was higher than C-O1O2/D-
8 O3O4, $t(63) = 9.68$, $p < .001$, and E-O1O2/F-O3O4, $t(63) = 3.69$, $p < .001$. The
9 mean proportion of correct responses for E-O1O2/F-O3O4 was also higher than C-
10 O1O2/D03O4, $t(63) = 6.69$, $p < .001$. The t-test comparisons on Trials 2 and 3
11 were non-significant with the Bonferroni correction (smallest $p = .01$).

12
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14 Insert Figure 4 here

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17

18 Test Stage

19 Figure 4 shows participants' ratings at test of cues A – F for outcomes O1 –
20 O4 by task type; the dependent variable was the same as Experiment 1 (mean
21 difference rating at test). In keeping with Experiment 1, participants rated the A/B
22 stimuli higher than the stimuli C/D stimuli, however, the E/F stimuli were rated
23 higher than both A/B and C/D, this pattern of results was consistent across the causal

1 and diagnostic learning groups. A 2×3 mixed model ANOVA with a between
2 subjects factor of Group (Causal vs Diagnosis) and a within-subjects factor of cue-
3 outcome pairing (A/B – O2/O4, C/D – O2/O4, E/F – O2/O4), revealed a significant
4 main effect of cue-outcome pairing, $F(2, 122) = 16.08$, $MSE = 1163.43$, $p < .001$,
5 $\eta_p^2 = .20$. There was no main effect of task type, $F(1, 61) = 1.95$, $MSE = 3266.47$, p
6 $= .17$, $\eta_p^2 = .03$. The interaction between these two variables was also non-significant,
7 $F(2, 122) = 1.20$, $MSE = 1163.43$, $p = .30$, $\eta_p^2 = .02$. Bonferroni adjusted paired
8 samples t-tests were performed to explore the main effect of cue-outcome pairings
9 (adjusted $p = .017$). These t-tests revealed that ratings for A/B-O2/O4 were lower than
10 ratings for E/F-O2/O4, $t(62) = 3.67$, $p < .001$, but were higher than ratings for C/D-
11 O2/O4, $t(62) = 2.55$, $p < .017$. Ratings for C/D-O2/O4 were also lower than E/F-
12 O2/O4, $t(62) = 5.06$, $p < .001$. In summary, Experiment 2 replicated the outcome-
13 facilitation effect when employing the control trial type from Experiment 1, however,
14 when the Kamin control was employed, an outcome-blocking effect was observed.
15 This blocking effect is consistent with the findings of some previous studies,
16 particularly Miller and Matute (1998) who also included a Kamin-type control.

17 As was noted in the introduction, many theories of associative learning treat
18 cues and outcomes rather differently – permitting cues to interact (usually in a
19 competitive manner) with one another for associative strength, whilst outcomes are
20 treated in a more binary manner – they are either present or absent (e.g. Mackintosh,
21 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972). However, a theory of
22 associative learning that includes a more balanced treatment of cues and outcomes has
23 been provided by Wagner (1981). According to Wagner’s model, stimuli, be they

1 cues or outcomes, can occupy one of three different states: a primary state of
2 activation (A1) which has a relatively limited capacity, a secondary state of activation
3 (A2), which has a rather larger capacity, or an inactive state (I). Before being
4 presented, a stimulus is in the inactive state where it is unable to attract attention and
5 produce a response. Following the first presentation of a stimulus, however, the
6 stimulus is promoted from inactivity into an A1 state, where it can produce its
7 strongest response, before it decays into the A2 activity state, where the stimulus
8 produces a weaker response and is considered to be at the periphery of an organism's
9 focus. As previously noted, the model states that a stimulus can enter into its A2 state
10 indirectly from inactivity by decaying from A1, however stimuli can also enter their
11 A2 state directly from the inactive state by the presentation of another stimulus that it
12 is associated with. The co-activational states of stimuli is also key to the type of
13 learning which takes place between them. When two stimuli are both in their A1 state,
14 an excitatory association is assumed to form between them. If, however, one stimulus
15 is an A1 state, and the other stimulus is in its A2 state, then an inhibitory association
16 is assumed to form from the stimulus in its A1 state to the stimulus in its A2 state.

17 Wagner's SOP model can explain both the outcome-facilitation effect and
18 outcome-blocking effect observed in the current experiment. The facilitation effect is
19 most naturally dealt with by the model and can be accounted for by assuming that
20 Stage 1 training on a blocking trial (e.g., A – O1) will result in the Stage 1 outcome
21 being activated directly into the A2 state during Stage 2 by A. This therefore allows
22 the novel outcome in Stage 2 (e.g., O2) to fully occupy the, limited capacity, A1 state,
23 permitting greater learning to take place between the cue and the novel outcome (e.g.,

1 A – O2) both of which will be in A1 states. In essence, participants can fully attend to
2 the novel outcome being presented without devoting any central attentional resources
3 to the outcome which was presented in Stage 1. However, on control trials, during
4 Stage 2, the novel outcome is presented alongside an outcome which the control cue
5 was not previously paired with during Stage 1 (e.g., C – O1 O2), therefore the cue
6 *and* both outcomes enter their A1 state and compete for the limited capacity available
7 in this state. Relative to blocking trials, therefore, learning about the cue and the novel
8 outcome will be attenuated on the control trials, which is the result that was observed
9 in Experiment 1 and Experiment 2.

10 Other things being equal, it would seem that Wagner's (1981) model would
11 struggle to explain why learning was successful between the Kamin-control stimuli
12 (e.g. E) and, for example, O2 relative to the blocking stimuli (e.g. A). Like the control
13 trials, the Kamin-control trials establish a situation in which the cue is paired, for the
14 first time in stage 2, with two outcomes. Thus, like during the control trials, we might
15 anticipate the outcome elements to compete for the limited capacity of A1 on the
16 Kamin-control trials, thus restricting learning. However, other things are not equal,
17 the Kamin-control trial cues, E and F, are novel at the outset of stage 2, and therefore
18 can be expected to be learned about more successfully than either the blocking (A and
19 B) or control (C and D) trials, both of which will less-successfully support learning
20 during stage 2 as they are, at least in part, associatively activated into A2 by
21 contextual cues (i.e. they are latently inhibited).

22 Notably, however, the SOP account of the Kamin control trials is parameter
23 specific, with parameters such as the salience of novel stimuli ($p1$), and the extent to

1 which the stimuli are associatively activated by the context, crucial in determining the
2 predictions of the model. For instance, if the salience of the Kamin control stimuli is
3 insufficiently strengthened by their novelty, or the trials presented in Stage 1 are not
4 associatively activated into their A2 state by the context, it is indeed possible that a
5 facilitation effect would be anticipated as opposed to a blocking effect. In any case, as
6 long as the effect of the novelty of the cues is greater than the competition between
7 the elements of the outcome, then Wagner's theory can provide a full account of the
8 results of Experiment 1 and Experiment 2.

9 10 **Experiment 3** 11

12 Experiment 2 reproduced the outcome facilitation effect observed in
13 Experiment 1, and extended the generality of the effect from causal learning to
14 diagnostic learning. Furthermore, Experiment 2 revealed, again using both causal and
15 diagnostic learning scenarios, that an outcome blocking effect can be obtained when a
16 different control type is employed.

17 As noted in our discussion to Experiment 2, Wagner's (1981) SOP model can
18 explain these findings. However, an additional account can also be provided by the
19 model proposed by Mackintosh (1975). According to this model, cues which are
20 reliable predictors of an outcome come to capture more attention and are learned
21 about more rapidly than cues which are poorer predictors of an outcome. According
22 to this model, the introduction of Stage 2 in Experiment 1 will create a difference in
23 the predictive validity of the blocking and control trials, which may have facilitated

1 learning to the blocking cues. This follows because, by the end of Stage 1, both sets of
2 stimuli (A/B and C/D) will have acquired comparable, high, attention and
3 associability as they are equally predictive of their respective outcomes. In Stage 2 the
4 surprising (i.e. non-predicted) addition of the novel outcome following all of these
5 cues can be expected to reduce attention to them. However, the addition of the novel
6 outcome will be additionally surprising in the case of the control trials (e.g. C) as the
7 expected outcome (O2) is replaced with a different outcome (O1). The overall
8 prediction error encountered on the control trials will therefore be more substantial
9 than that encountered on the blocking trials, which have only one source of surprise.
10 Consequently, learning about the blocking cues should be more successful than about
11 the control cues. Furthermore, given the novelty of the Kamin-control trial cues, E
12 and F, at the outset of stage 2, and the fact that these stimuli serve as good predictors
13 of their respective outcomes, it may be expected that participants will learn more
14 successfully about E and F than any of the remaining cues. Thus, as long as it is
15 assumed that the combined novelty and predictive ability of E and F is sufficient to
16 generate better learning about the outcomes on these trials than the blocking trials it is
17 possible to account for the findings according to the Mackintosh model.

18 Although both Wagner's (1981) and Mackintosh's (1975) models can provide
19 accounts for the findings of Experiments 1 and 2 they do make different predictions
20 as to when the various effects observed at test should begin to emerge during training
21 in Stage 2. The Mackintosh account predicts that the effects observed in Experiment 2
22 are dependent upon the prediction error from the first trial in Stage 2 influencing the
23 associability of cues on the next trial. For instance, upon first presentation of the

1 stimuli in Stage 2, the model predicts that the associability of the cues will be
2 comparable. However, following the first trial, the associability of the stimuli will be
3 altered based on the information provided on the first trial. As such, the associability
4 of the control C/D stimuli will decrease, relative to A/B, but only from trial 2
5 onwards. Consequently, the outcome facilitation effect observed in Experiments 1 and
6 2 should not be evident if only one training trial is given in stage 2. The account
7 derived from Wagner's (1981) model does not make the same prediction. According
8 to this analysis one compound trial in Stage 2 should be sufficient to obtain an
9 outcome facilitation effect, as the competition for limited capacity in the A1 state,
10 should restrict learning about the novel outcome on the very first control trial in Stage
11 2.

12 The purpose of Experiment 3, therefore, was to employ an outcome blocking
13 procedure but with only one trial in stage 2 in order to distinguish the accounts
14 provided by Wagner's (1981) and Mackintosh's (1975) theories. One-trial blocking
15 procedures have been used a number of times in the cue-blocking literature to test
16 various attentional accounts of blocking (e.g., Dickinson, Nicholas & Mackintosh,
17 1983; Gillan & Domjan, 1977; Mackintosh 1975b; Willner, 1978). This procedure
18 does not appear to have been used to assess outcome interaction effects, however. The
19 design of Experiment 3 was therefore the same as Experiment 2, with the exception of
20 the number of trials presented in Stage 2 (see Table 2). For completeness, we also
21 retain in Experiment 3 the Kamin-control trials employed in Experiment 2, as well as
22 the comparison between causal and diagnostic learning.
23

1 **Method**

2 **Participants.**

3
4 Sixty-four participants (46 females; 18 males) were recruited from the
5 University of Nottingham’s School of Psychology. Participants ranged from 18 – 50
6 years of age ($M = 22.45$; $SEM = 0.78$). Again, participants were randomly allocated
7 (on the condition that there were a comparable number of participants in each group)
8 to complete either the causal judgment task ($n = 32$) or the diagnostic judgement task
9 ($n = 32$). Participants received course credit or received a £3 inconvenience allowance

10 **Materials & Procedure.**

11
12 The materials and procedure were identical to that reported in Experiment 2,
13 with the exception that in Stage 2 only one trial of each cue-outcome pairing was
14 presented. As such, Stage 2 only consisted of 6 trials.

15
16 **Results and Discussion**

17
18 -----

19 Insert Figure 5 here

20 -----
21

22 **Stage 1**

23
24 Figure 5 shows the mean proportion of correct responses for each cue-
25 outcome pairing (A-O1, B-O3, C-O3 and D-O1) across Stage 1 training for both the

1 causal (top left panel) and diagnostic (top right panel) groups. Again, participants
2 generally displayed the same pattern of results regardless of the task completed (e.g.
3 Causal/Diagnostic), with participants rapidly learning the relationships between the
4 cue-outcome pairings. A three-way mixed model ANOVA was performed with a
5 between-subject factor of task type (Causal vs Diagnostic), and within-subjects
6 factors of Stimulus (A-O1/B-O3 vs C-O3/D-O1) and Trial (1 – 24). The ANOVA
7 revealed a main effect of trial, $F(23, 1426) = 93.26$, $MSE = .01$, $p < .001$, $\eta_p^2 = .60$,
8 demonstrating that participants learned the associations as training progressed. There
9 was no effect of task type $F(1, 62) = .10$, $MSE = .06$, $p = .75$, $\eta_p^2 = .00$, and no effect
10 of outcome $F(1, 62) = .47$, $MSE = .02$, $p = .49$, $\eta_p^2 = .00$. All interactions were non-
11 significant, largest $F(1, 62) = .87$, $MSE = .02$, $p = .35$, $\eta_p^2 = .01$.

12 **Stage 2**

13 Figure 5 shows the mean proportion of correct responses for each cue –
14 outcome compound (A-O1O2/B-O3O4, C-O1O2/D-O3O4, E-O1O2/F-O3O4) during
15 Stage 2 training. For the causal group (bottom left panel of Figure 5), the mean
16 proportion of correct responses for the blocking and the Kamin control stimuli were
17 comparable, whilst the proportion of correct responses to the control stimuli which
18 featured in Stage 1 were lower. For the diagnostic group (bottom right panel of Figure
19 5) participants demonstrated a relatively high proportion of correct responses for the
20 blocking stimuli, an approximately chance level of correct responding was displayed
21 to the Kamin control, and lower than chance levels of responding to the control
22 stimuli.

1 A two-way mixed model ANOVA was performed on these data with a
2 between-subjects factor of task type (causal vs diagnostic), and within-subjects factor
3 of cue-outcome pairing (A-O1O2/B-O3O4 vs C-O1O2/D-O3O4 vs E-O1O2/F-
4 O3O4). The ANOVA revealed a main effect for the cue-outcome pairings, $F(2, 124)$
5 $= 41.63$, $MSE = .12$, $p < .001$, $\eta_p^2 = .40$. There was no main effect of the factor of
6 task type $F(1, 62) = .61$, $MSE = .08$, $p = .44$, $\eta_p^2 = .01$, and the interaction between
7 these factors was non-significant, $F(2, 124) = 2.28$, $MSE = .12$, $p = .11$, $\eta_p^2 = .04$. To
8 identify the source of the main effect for the cue-outcome pairings, Bonferroni
9 adjusted paired samples t-tests were performed (adjusted $p = .017$). These t-tests
10 revealed that the mean proportion of correct responses for A-O1O2/B-O3O4 was
11 higher than C-O1O2/D-O3O4, $t(63) = 8.81$, $p < .001$, but not E-O1O2/F-O3O4, t
12 $(63) = 1.82$, $p = .07$. The mean proportion of correct responses for E-O1O2/F-O3O4
13 was also higher than C-O1O2/D-O3O4, $t(63) = 7.60$, $p < .001$. These results
14 demonstrate that at the outset of Stage 2 participants displayed a higher proportion of
15 correct responses to the blocking and Kamin control trials than to the control trials.

17 **Test Stage**

19 Figure 6 shows participants' difference ratings at test of how predictive cues A
20 – F were for outcomes O1 – O4, separated by the diagnostic and causal task type. The
21 dependent variable was the same as Experiments 1 and 2 (mean difference rating at
22 test). As can be seen, in the causal group, participants rated the A/B stimuli higher than
23 the C/D stimuli, and slightly higher than the E/F stimuli. Notably, C/D ratings were
24 negative suggesting participants were rating these cues as predictive of the novel

1 outcome that was presented alongside the outcome these stimuli had previously been
2 paired with. In the diagnostic group, ratings for C/D were also lower than A/B and E/F,
3 with E/F receiving the highest ratings. A one sample t-test was performed comparing
4 the collapsed mean ratings (across task type) of C/D – O2/O4 to 0, to assess whether
5 the ratings were significantly negative. The t-test revealed that participants ratings were
6 not significantly below 0, $t(63) = 1.61, p = .11$.

7 A 2×3 mixed model ANOVA with a between subjects factor of task type
8 (Causal vs Diagnostic) and a within-subjects factor of cue-outcome pairing (A/B –
9 O2/O4, C/D – O2/O4, E/F – O2/O4), revealed a significant main effect of cue-outcome
10 pairing, $F(2, 124) = 10.39, MSE = 2198.64, p < .001, \eta_p^2 = .14$. There was no main
11 effect of task type, $F(1, 62) = .04, MSE = 1485.43, p = .83, \eta_p^2 = .01$. The interaction
12 between these two variables was significant, however, $F(2, 124) = 3.29, MSE =$
13 $2198.64, p < .05, \eta_p^2 = .05$. Simple main effects were performed to reveal the source
14 of the interaction. There was an effect of task type for stimuli A/B with participants in
15 the Causal group providing higher ratings than the Diagnostic group, $F(1, 62) = 5.35,$
16 $MSE = 1439.03, p < .05, \eta_p^2 = .08$, but no effect for the C/D or E/F stimuli, largest F
17 $(1, 62) = 3.16, MSE = 2136.36, p = .08, \eta_p^2 = .05$. To further explore the main effect of
18 cue-outcome pairings, Bonferroni adjusted paired samples t-tests were performed
19 (adjusted $p = .017$). These t-tests revealed that ratings for A/B-O2/O4 were higher than
20 ratings for C/D-O2/O4, $t(63) = 3.93, p < .001$, but not E/F-O2/O4, $t(63) = .65, p =$
21 $.51$. Ratings for C/D-O2/O4 were also lower than E/F-O2/O4, $t(63) = -3.57, p <$
22 $.001$.

23 Experiment 3 revealed that when employing a one-trial procedure in stage 2, an
24 outcome facilitation effect was present. This result is inconsistent with the account of

1 the Experiments 1 and 2 that can be derived from Mackintosh's (1975) model.
2 According to the Mackintosh model the outcome facilitation effect observed in
3 Experiments 1 and 2 is dependent upon the prediction error generated on learning trial
4 N changing the associability of the cues on learning trial N+1. If trial N+1 is absent
5 then it follows that the outcome facilitation effect should be similarly absent. In
6 contrast the results of the current experiment can still be reconciled with the account of
7 learning provided by Wagner (1981).

8 The absence of an outcome blocking effect, however, suggests that the account
9 of Experiment 2 derived from Wagner's (1981) model is incomplete. According to the
10 account outlined previously, the outcome blocking effect observed in Experiment 2
11 would be expected after a single trial of Stage 2 training. By having only one trial in
12 Stage 2 means that stimuli E/F are highly novel – more so than in Experiment 2 –
13 therefore any explanation based on novelty would anticipate that the ratings for stimuli
14 E/F would be higher than stimuli C/D. Thus, on this basis the outcome blocking effect
15 would be expected to be enhanced as opposed to abolished. Given that only one trial is
16 presented in Stage 2, it is possible that blocking is difficult to detect as participants'
17 ratings are understandably low (due to the lack of trials). However, the ratings for A/B
18 and E/F are higher than C/D, and thus it is not the case that a floor effect is obscuring
19 any differences. It is also worth noting though, that some cue-blocking studies which
20 have employed one-trial blocking procedures have also reported an absence of blocking
21 when only trial is employed (see Mackintosh, 1975b; Mackintosh, Dickinson & Cotton,
22 1980).

23 It is also worth noting the absence of a difference between participants'
24 proportion of correct responses for the blocking trials and the Kamin control trials in
25 Stage 2 for Group Causal. Assuming participants used the information gained from

1 Stage 1 it would be anticipated that the proportion of correct responses for the blocking
2 trials would be higher at the outset of Stage 2 than the Kamin control trials, yet this was
3 not the case. The the source of this inconsistency remains to be determined.

4 The analysis of the test trial data for stimuli A/B, revealed that participants
5 provided higher ratings in the causal task than the diagnostic task. This could be
6 accounted for by assuming that participants are more likely to consider other potential
7 causes when completing the diagnostic task (e.g., Waldmann & Holyoak, 1992;
8 Waldmann, 2000) and therefore provided lower ratings in the diagnostic task. As to
9 why it is only observed with blocking stimuli, one possible explanation could be that
10 cognitive, or reasoned, judgements play less of a role with the C/D and E/F stimuli as
11 uncertainty or novelty (respectively) are more salient factors for these trials in both
12 versions of the tasks. One potential way of gaining further insights into the role of
13 uncertainty in influencing these ratings would be to collect confidence ratings alongside
14 predictiveness ratings (e.g., Jones, Zaksaitė & Mitchell, 2019; Livesey, Greenaway,
15 Schubert & Thorwart, 2019).

17 **General Discussion**

18
19 Three experiments explored how training with an outcome (e.g., A – O1) would
20 influence learning about a novel outcome (e.g., O2), when these two outcomes were
21 subsequently presented in compound (e.g., A – O1 O2). In Experiment 1 participants
22 displayed a bias toward learning about the novel element of an outcome, when
23 presented in compound with an outcome which a cue had previously predicted, relative
24 to a control where both elements of the outcome compound were surprising (e.g. C –
25 O3 followed by C – O1 O2). As such, previous training with an element of an outcome
26 appeared to facilitate learning about the novel outcome, as opposed to block learning

1 about the novel outcome. Experiment 2 reproduced this finding in a causal judgment
2 task where participants were first presented with foods (i.e., cues) which predicted
3 certain reactions (i.e., outcomes), and in a diagnostic judgment task where reactions
4 were presented first, and participants were required to determine which foods caused
5 these reactions. Experiment 2 included an additional control trial, that omitted any
6 training with the cue in Stage 1 (e.g. E- O1 O2 during only stage 2), a control trial that
7 is the outcome blocking equivalent to that employed by Kamin (1968) in his studies of
8 cue blocking. When learning about the Blocking cue was compared to the Kamin
9 control cue, an outcome blocking effect was observed. Experiment 3 replicated the
10 design of Experiment 2, but only included a single trial with the outcome compounds in
11 Stage 2. The purpose of this experiment was to distinguish between two theoretical
12 accounts of the results reported in Experiment 2 – derived from the Mackintosh (1975)
13 model and Wagner’s (1981) SOP model. Although both accounts can accommodate the
14 results from Experiments 1 and 2, they differ as to when these effects should manifest.
15 According to the Mackintosh model any effect should only be present after two training
16 trials or more According to the SOP model one trial should be sufficient. In Experiment
17 3 an outcome facilitation effect was observed, following one trial of training in Stage 2,
18 but not an outcome blocking effect. The maintenance of the outcome facilitation effect
19 is more consistent with the analysis that can be derived from Wagner’s theory than
20 Mackintosh’s. However, the abolition of the outcome blocking effect is somewhat
21 problematic for both of these theories.

22 Given the heterogeneous nature of the previous literature, these results are both
23 consistent and inconsistent with previous studies which have explored outcome
24 blocking. Notably, the outcome facilitation effect reported in these experiments is
25 inconsistent with previous animal studies such as Rescorla (1980) and Miller and

1 Matute (1998), which demonstrated an outcome blocking effect (and also Price and
2 Yates, 1995, who demonstrated no effect). Yet they are consistent with Experiment 3 of
3 Flach et al. (2006). These authors demonstrated a comparable effect within a response
4 priming task, when the pretrained-outcome element was an auditory stimulus and the
5 added outcome was a visual stimulus. However, a number of procedural differences
6 exist between the aforementioned studies and the experiments reported in the paper,
7 particularly the animal studies. Firstly, the animal studies cited above employed
8 indirect conditioning procedures with animals which could potentially influence results.
9 Indeed, Rescorla (1980) noted that as a result of employing a second-order conditioning
10 autoshaping procedure with biologically significant outcomes, it could be that the
11 pigeons in this experiment were simply distracted by the ‘blocking’ outcome element,
12 as opposed to a form of associative competition occurring between the elements of the
13 outcome; although this issue is neatly circumvented by Miller and Matute (1998) by
14 employing a sensory pre-conditioning paradigm (also see Miller & Escobar, 2002, for
15 discussion). Furthermore, differences also exist between the aforementioned studies
16 and the experiments reported here, in terms of species, the experimental task
17 completed, or the type of control employed.

18 Perhaps most importantly, however, the experiments reported in this paper
19 highlight how different controls can result in different effects being produced, as the
20 introduction of the Kamin control in Experiment 2 resulted in an outcome blocking
21 effect. Two possible theoretical accounts have been considered for why these different
22 types of effects are observed, depending on the control employed. According to the
23 Mackintosh (1975) model the outcome facilitation effect can be explained as a
24 consequence of a difference in the predictive validity of the blocking and control
25 stimuli included in Experiment 1, whilst the outcome blocking effect occurred due to

1 the absence of this difference and the novelty of the cues employed in the Kamin
2 control. According to SOP, Stage 1 learning (e.g., A – O1) ensures that the pretrained
3 outcome is in the A2 state during stage 2, thus allowing the added outcome on the
4 blocking trials (e.g., A – O1 O2) to fully occupy its A1. On the control trials, however,
5 both outcomes are surprising and thus compete to enter into the limited capacity A1,
6 therefore producing an outcome facilitation effect. To account for the outcome blocking
7 effect observed with the addition of the Kamin control, the effect of novelty can once
8 again be appealed to as the cues already presented in Stage 1 in the control and
9 blocking conditions, but not the Kamin control condition, will be activated into their
10 A2 state by the context.

11 As noted previously, however, the SOP account of the outcome blocking effect
12 is parameter specific. If, for instance, the novelty of the Kamin control stimuli does not
13 warrant them fully occupying their A1 state to a greater extent than the alternative
14 control trials (C and D), and thus competition between the elements of the compound
15 occur in their A1 state, this would result in each outcome element being learned about
16 less than the novel outcome element presented on a blocking trial in Stage 2 (which can
17 fully occupy its A1 state). If, however, the novelty of the Kamin control stimuli renders
18 the salience of these stimuli to be high, thus being at the focal point of attention, and
19 the blocking and control trials from Stage 1 are (partly) associatively activated into
20 their A2 state by the context, then an outcome blocking effect would be anticipated.
21 This is most likely to be the case in the experiments reported here, according to the
22 model, as the stimuli presented in Stage 1 will be latently inhibited due to previous
23 exposure with these stimuli.

24 The Mackintosh account predicts that the effects observed in Experiments 1 and
25 2 are dependent upon the prediction error from the first trial in Stage 2 influencing the

1 associability of cues on the next trial. As such, according to the Mackintosh model no
2 differences should be present after only one trial of Stage 2. The SOP account does not
3 make the same prediction. According to this analysis, one compound trial in Stage 2
4 should be sufficient to obtain the effect observed. Whilst the presence of a difference
5 between the blocking and the control conditions in Experiment 2 seem to support the
6 SOP prediction, the absence of outcome blocking in Experiment 3 does suggest
7 however, that additional training trials are needed to obtain the effect. There is
8 therefore, at least, a conceptual consistency between the effects of a single compound
9 training trial in cue blocking, and outcome blocking. It remains to be determined
10 whether this result is fully consistent with the analysis of the current results that can be
11 provided by Mackintosh (1975).

12 The contrasting results produced with the two different control types employed
13 in Experiments 2 and 3 raises questions over which control is deemed to be the most
14 suitable. Arguably, the Kamin control would appear to be the most appropriate. On
15 these trials a comparable number of outcomes are presented to the blocking trials (i.e.,
16 each cue is paired with two outcomes). On the alternative control trials (C/D), however,
17 an additional outcome is presented in stage 1, thus creating a discrepancy with the
18 blocking trials. Furthermore, due to the fact the control trials featuring C/D are paired
19 with different outcomes across the two stages of training, these stimuli will also
20 undergo reversal training which is likely to disrupt learning on these trials. As such, the
21 Kamin control trials would appear to overcome some of the potential issues
22 encountered when employing the alternative control trials. If the Kamin control trials
23 are considered the most appropriate control, the results of the above experiments
24 suggest that blocking can indeed be observed in outcomes in a similar manner to cues
25 when Stage 2 training is provided of an appropriate duration.

1 However, the Kamin control trials also have their shortcomings due to the lack
2 of exposure to the cues during Stage 1. For instance, on a blocking trial participants'
3 have already had exposure to the blocking cues (A/B) presented in Stage 2, yet on a
4 Kamin control trial, the cues (E/F) are novel and thus the respective cues potentially
5 differ in their salience. In light of this discrepancy, it could thus be argued that the
6 blocking effect observed with the Kamin control was driven by a difference in the
7 saliences of the cues as opposed to an outcome interaction effect. The alternative
8 control trial, however, mitigates this issue as participants have had comparable
9 exposure to the cues (C/D) on these trials in Stage 1 training as the blocking cues
10 (A/B). Yet, when employing this control an outcome facilitation effect is observed as
11 opposed to an outcome blocking effect. Thus, it is clear that the type of control selected
12 is crucial. Yet, determining which type of control is appropriate is a difficult task.

13 An alternative, and interesting, explanation for the outcome facilitation effect
14 observed in Experiment 3 can be developed by appealing to the influence of within-
15 compound associations. Note that in Experiment 3 the stimuli which are paired together
16 most frequently in Stage 2 are the outcome compounds (i.e., O1 and O2, and O3
17 and O4, each of which is paired together 3 times). Given that only one trial is presented
18 in Stage 2 for each of the cue-outcome pairings, it is conceivable that insufficient
19 learning took place about the relationship between the cues and the added outcomes to
20 directly determine participants' ratings at test. In light of this, it is possible that Stage 1
21 learning is mediating performance at test. For example, when stimulus C was presented
22 at test, it may provoke participants to think of the outcome which it was paired with in
23 Stage 1 (O3), which in turn, associatively activates the outcome which it was paired
24 with most frequently during Stage 2 (O4). This analysis would explain the mildly
25 negative ratings given to C and D at test, as participants are selecting the outcomes that

1 these cues were not paired with in Stage 2 to a greater extent than the outcomes that
2 these cues were paired with. If we apply the same analysis to the blocking cues then
3 presentation of cue A at test would associatively activate the outcome it was most
4 frequently paired with in Stage 1 (O1) which itself has a strong within compound
5 association with only O2 – which is the correct outcome. As such, a within-compound
6 association account predicts a positive rating to cues A and B at test, which is what was
7 observed. The ratings for the E/F stimuli are difficult to accommodate according to this
8 account though. Given that these stimuli are not presented in Stage 1, there is no
9 opportunity for these stimuli to form associations with a Stage 1 outcome which could
10 associatively activate an outcome from Stage 2. As such, the positive ratings given to
11 these stimuli at test imply that at least some excitatory learning between the cues and
12 the outcomes takes place during Stage 2.

13 Across the course of, for example, Experiment 1 the control stimuli are also
14 associated with a greater number of outcomes than the blocking stimuli. For example,
15 cue A is paired with O1 and O2, whilst cue C is paired with O1, O2 and O3. It is
16 therefore possible that this disparity in the number of associates could result in reduced
17 learning about the outcomes associated with the C/D control stimuli, relative to the
18 blocking stimuli. For instance, Anderson (1974) observed that when a stimulus accrues
19 a greater number of associates, participants display slower response times when tasked
20 with identifying an associate of this stimulus, thus displaying what Anderson referred
21 to as the “fan effect”. It is possible this effect could account for the outcome facilitation
22 results provided here. However, this would not account for the outcome blocking effect
23 which was observed when the blocking cues are compared with the Kamin control
24 cues. These control cues have a comparable number of associates to the blocking
25 stimuli.

1 An additional point to consider is the potential role of proactive interference,
2 particularly in relation to the C and D control trials. Given that these controls are paired
3 with different outcomes across the two stages of training, it is possible that proactive
4 interference (produced by Stage 1 training), results in participants failing to recall the
5 associations between C and D and the outcomes they are paired with in Stage 2 at the
6 test stage. The fact that participants' learning had reached asymptote at the end of Stage
7 2 for these trials in Experiments 1 and 2 would suggest that this may be less of a
8 problem for these experiments. However, in Experiment 3, participants are presented
9 with a single trial with each cue-outcome pairing in Stage 2, and as such it is possible
10 that their learning about the cue-outcome pairings in Stage 1 interferes with their
11 ratings at test.

12 Interestingly, despite the contradictory nature of the effects produced in these
13 experiments when employing different controls, both types of effects challenge
14 associative learning models which assume that outcomes do not interact and compete
15 with one another in a comparable manner to cues. Moreover, the observation of both
16 facilitation and blocking within outcomes could indeed highlight a further similarity
17 between the behaviour of cues and outcomes when presented in compound. Although
18 competition effects are often observed when cues are presented in compound (e.g.,
19 blocking, overshadowing), facilitation effects have also been observed under certain
20 circumstances such as augmentation (e.g., Batsell & Baston, 1999) and potentiation
21 (Durlach & Rescorla, 1980). Indeed, Miller and Escobar (2002) have noted how
22 interaction effects can occur in both cues and outcomes and have proposed possible
23 mechanisms (i.e., a comparator mechanism and a priming mechanism) which could
24 account for interaction effects (competitive and facilitative) in both cues and outcomes.
25 Urcelay (2017) also notes that cue-interaction effects occur on a spectrum ranging from

1 competition, to no effect, to facilitation, despite the fact that focus typically tends to be
2 placed on competition effects.

3 Finally, it is also worth considering the impact on these results of the type of
4 judgement task completed. According to associative learning theories the type of
5 judgement task completed should have little effect (e.g. Mackintosh, 1975; Pearce &
6 Hall, 1980; Rescorla & Wagner, 1972). The key factor which will influence results is
7 the temporal order compound stimuli are presented in. Cues refer to the stimuli which
8 are presented prior to an outcome; outcomes are the stimuli which are presented after a
9 cue. According to causality models developed to account for human reasoning, the type
10 of task completed should have an effect. For instance, causal model theory (Waldmann
11 & Holyoak, 1992) assumes that participants differentiate between causes and effects
12 regardless of which order they are presented in. Moreover, they make different
13 judgements based on whether a cause or effect is presented first. The model assumes
14 that when participants are presented with a diagnostic judgement task where they are
15 presented with an outcome first and then presented with the causes, they are more
16 likely to consider alternative causes which could have accounted for this outcome. The
17 results of these experiments provide only slight support to this model. There was a
18 difference in Experiment 3 with participants in the Causal group providing higher
19 ratings to the blocking stimuli than the Diagnostic group. However, collectively the
20 results demonstrate little impact of the type of task completed. That is, whether or not
21 an interaction effect was observed was not influenced by the type of judgement task
22 completed. This could potentially be accounted for by the multi-outcome design
23 employed in these experiments (which differ to standard cue-blocking studies) and the
24 nature of the test question. For instance, Don and Livesey (2018) found that the test
25 question presented to participants was crucial in determining whether task type would

1 influence the presence of cue-blocking. Thus, it is possible that the test question in the
2 current experiments was insensitive to detect any differences between the two types of
3 judgement tasks.

4 In summary, the experiments in this paper examined whether prior learning
5 about a cue-outcome association can influence learning about a novel outcome when
6 the two are presented in compound. An assumption common to many associative
7 models of learning is that the associative history of an outcome should have little
8 impact on the learning of a novel outcome when these two outcomes are presented
9 together. The current experiments question this assumption, providing evidence of
10 outcome facilitation and outcome blocking effects dependent on the control employed,
11 and an account of these effects has been derived from Wagner's SOP (1981) model.
12 These experiments provide further insight into the nature of outcome associability and
13 outcome interaction effects and present some factors which should be considered when
14 theories of stimulus interaction are being developed.

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Footnote to title page.

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2

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Tables

2

Table 1. *Design of Experiment 1*

	Stage 1	Stage 2	Test
Blocking	A – O1	A – O1 O2	A: O1- O4
	B – O3	B – O3 O4	B: O1- O4
Control	C – O3	C – O1 O2	C: O1- O4
	D – O1	D – O3 O4	D: O1- O4

Note. A – D refer to foods (i.e. cues), whilst O1 – O4 refer to outcomes. A and B cues represent the blocking stimuli, whilst C and D represent the control stimuli.

3

Table 2. *Design of Experiments 2 and 3*

	Stage 1	Stage 2	Test
Blocking	A – O1	A – O1 O2	A: O1-O4
	B – O3	B – O3 O4	B: O1-O4
Control	C – O3	C – O1 O2	C: O1-O4
	D – O1	D – O3 O4	D: O1-O4
Kamin Control	--	E – O1 O2	E: O1-O4
	--	F – O3 O4	F: O1-O4

Note. A – E refer to cues, whilst O1 – O4 refer to outcomes. A and B represent the blocking trials, C and D represent the control trials, whilst E and F represent the Kamin control trials. A-E were foods for the causal task, with O1-O4 serving as reactions. In the diagnostic task, A-E were reactions and O1-O4 were foods.

Figures

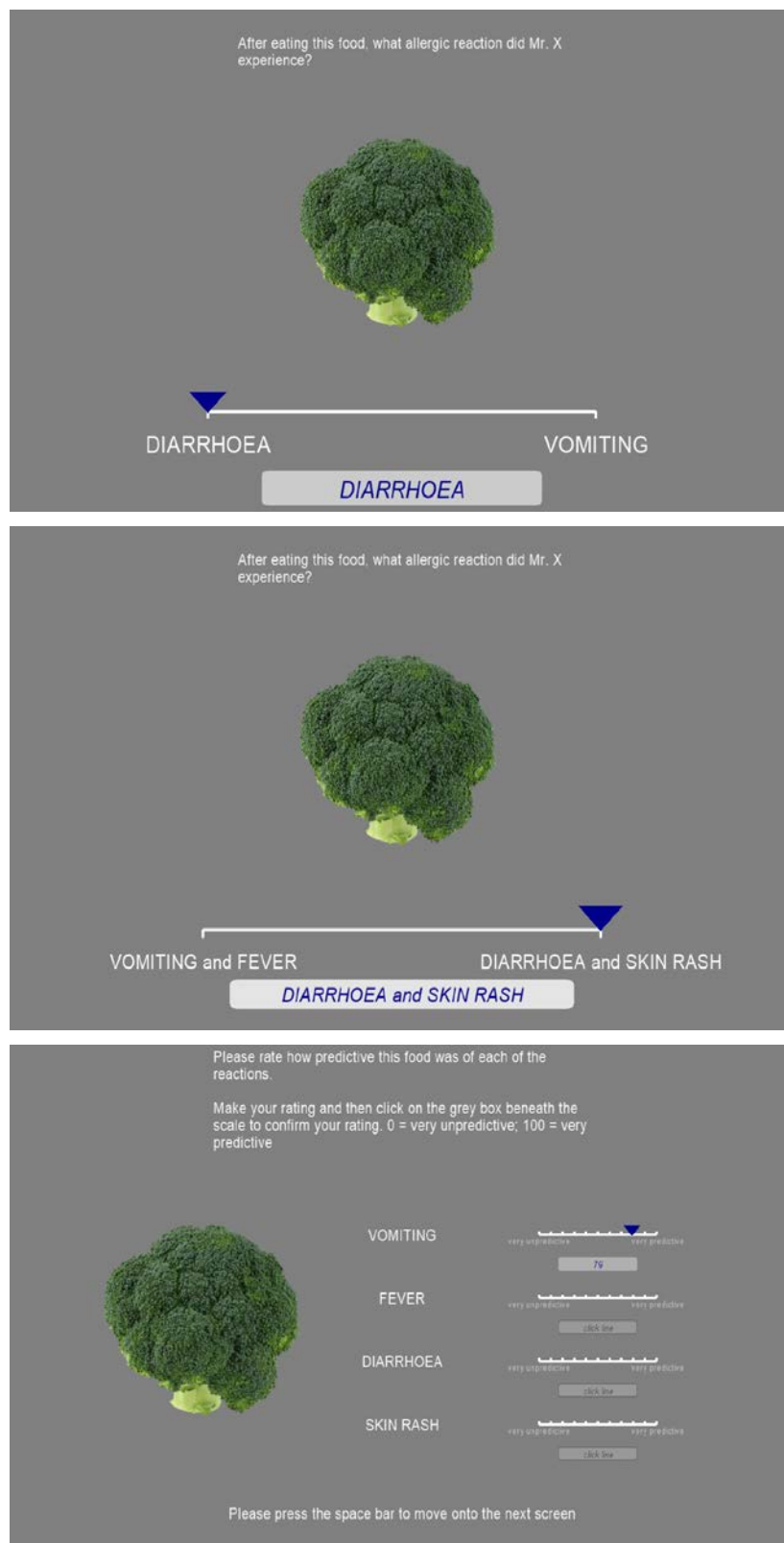


Figure 1. An example screen participants would be presented with during Stage 1 (top), Stage 2 (middle) and at test (bottom).

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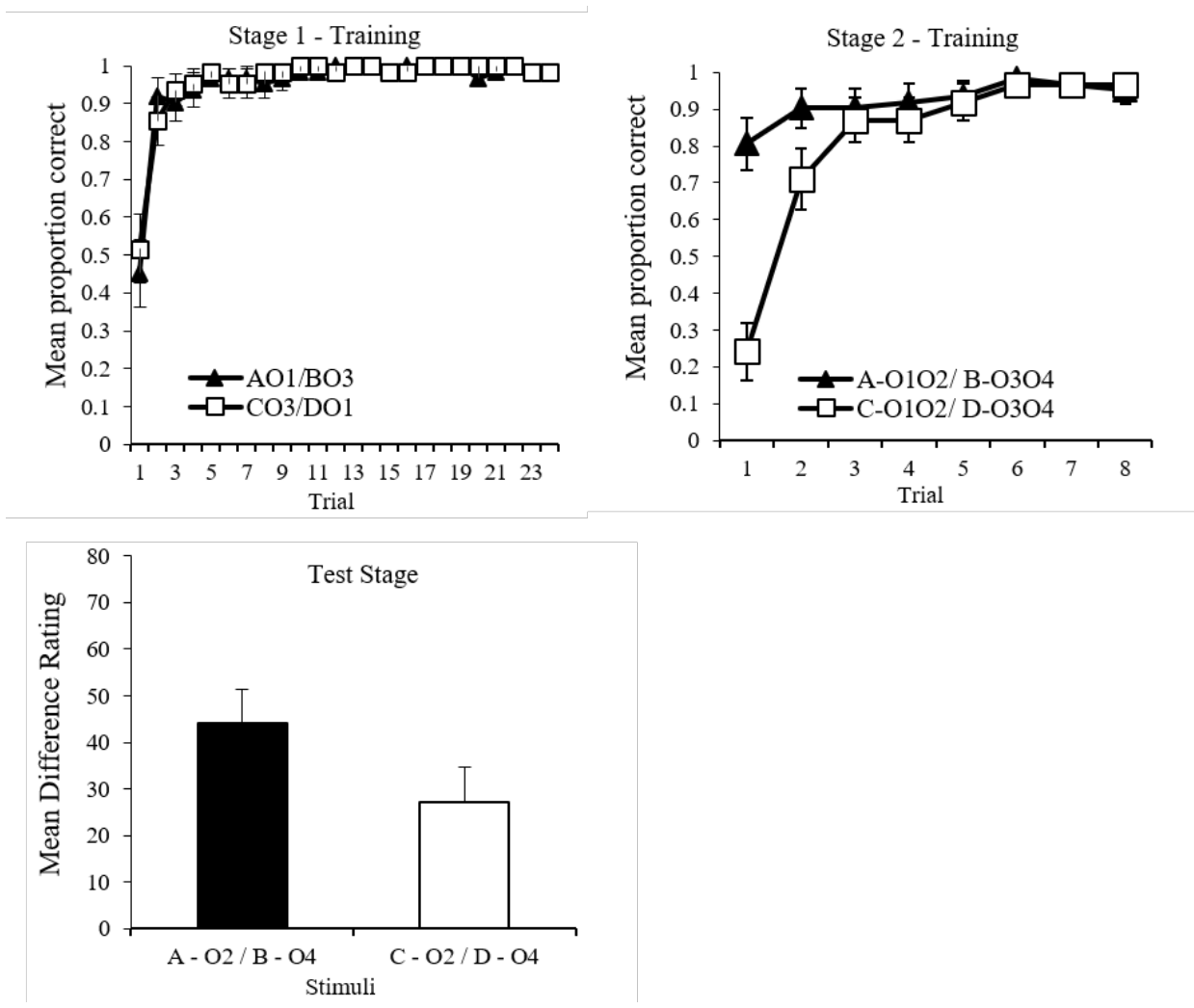


Figure 2. Mean proportion of correct responses for A – D: O1 and O3 during Stage 1 training (top left), for A – D: O1O2 and O3O4 during Stage 2 (top right) and the mean of participants’ difference ratings for A/B – O2/O4 and C/D - O2/O4 at test. Error bars represent SEM.

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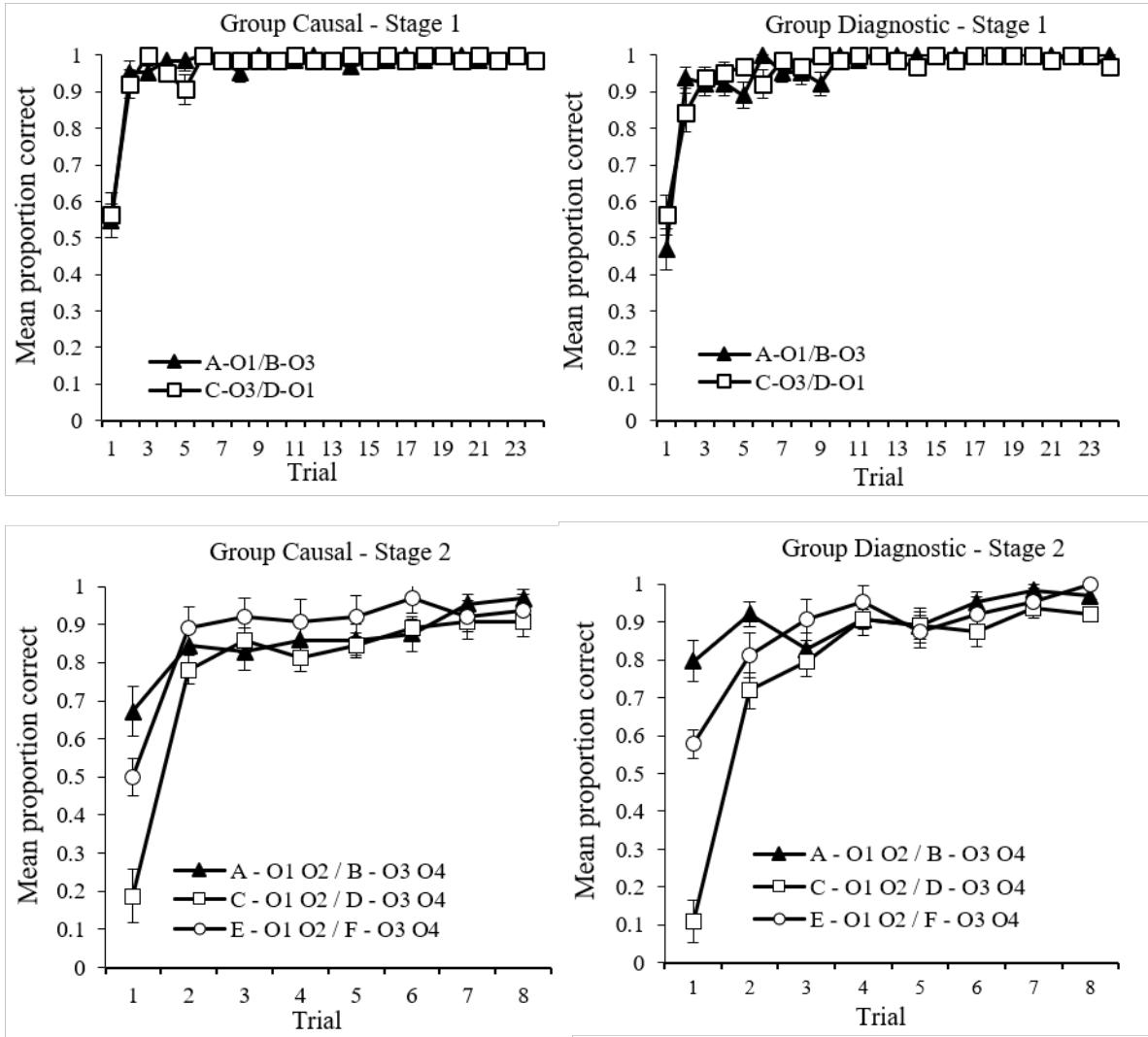


Figure 3. Mean proportion of correct responses for each trial type for the Causal (top left panel) and Diagnostic (top right panel) groups during Stage 1, and for each trial type during Stage 2 for the Causal (bottom left panel) and Diagnostic groups (bottom right panel) for Experiment 2. Error bars represent SEM.

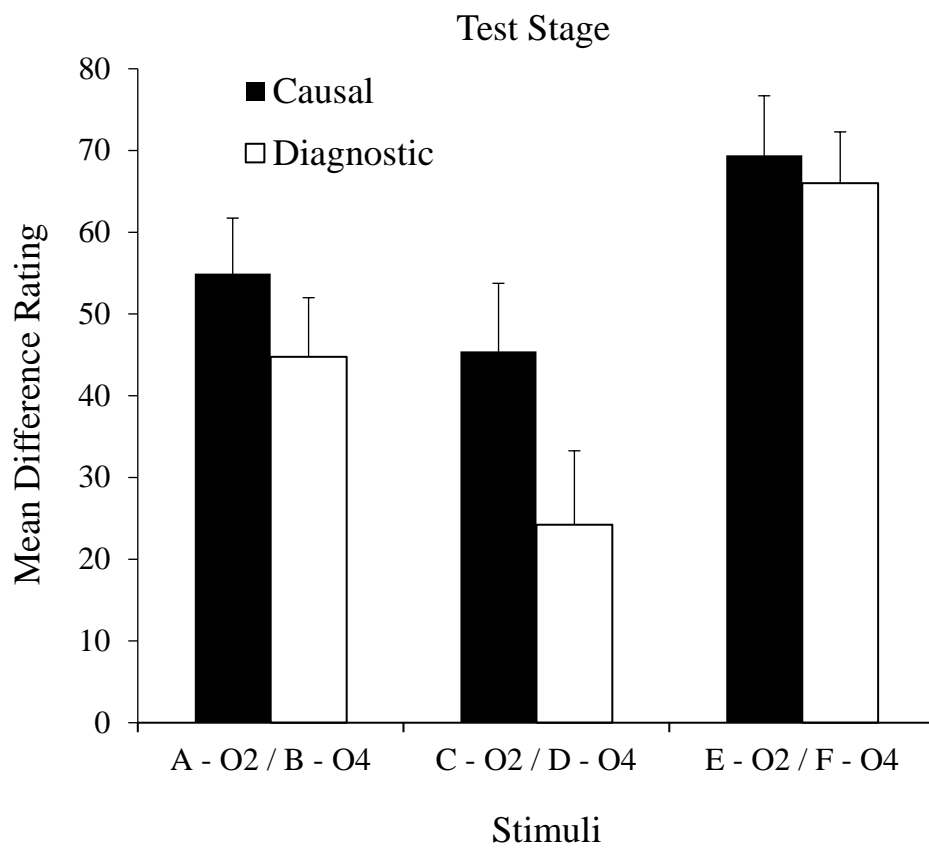


Figure 4. Mean of participants' difference ratings for the outcomes at test, for both causal and diagnostic judgment tasks. Error bars represent SEM.

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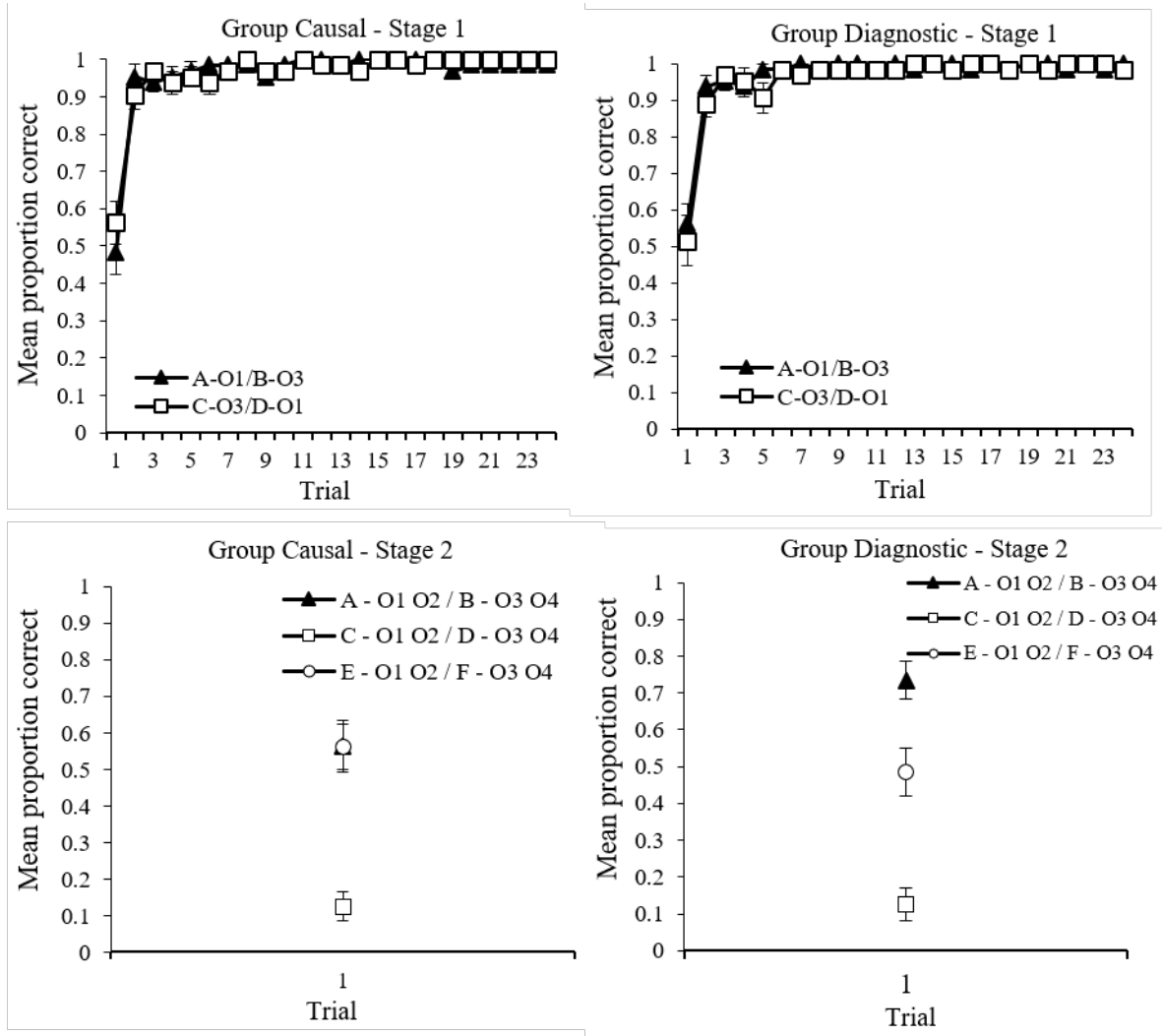


Figure 5. Mean proportion of correct responses for each trial type for the Causal (top left panel) and Diagnostic (top right panel) groups during Stage 1, and for each trial type during Stage 2 for the Causal (bottom left panel) and Diagnostic groups (bottom right panel) for Experiment 3. Error bars represent SEM.

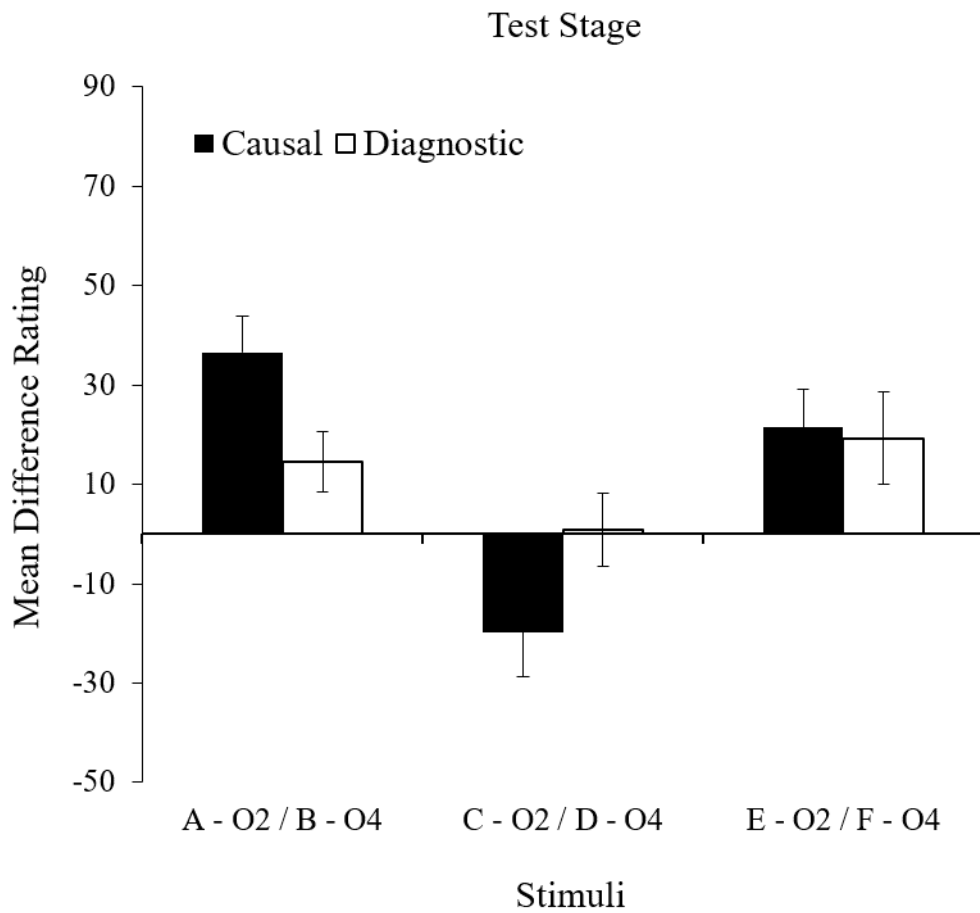


Figure 6. Mean of participants' difference ratings for the outcomes at test, for both causal and diagnostic judgment tasks. Error bars represent SEM.

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Appendix

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Mean rating and standard error of the mean for each cue type

Experiment	Cue Type	Group Causal		Group Diagnostic	
		M	SEM	M	SEM
Experiment 1	A/B	66.46	4.84	-	-
				-	-
	C/D	56.06	5.03	-	-
				-	-
Experiment 2	A/B	75.74	4.08	60.29	4.78
	C/D	69.06	4.64	53.81	5.30
	E/F	84.79	3.17	76.73	4.40
Experiment 3	A/B	63.81	4.35	46.90	4.10
	C/D	36.10	4.46	39.95	4.81
	E/F	59.81	3.89	54.93	5.43

Note. M refers to the mean raw rating provided to each cue type, whilst SEM stands for standard error of the mean for each rating. – signifies that this version of the task was not completed.

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