Interactions between the elements of an outcome in human associative learning

Martyn Quigley[1]

Mark Haselgrove[2]

[1] Swansea University

[2] The University of Nottingham

Correspondence Address: Martyn Quigley

Department of Psychology, Vivian Tower,

College of Human & Health Sciences,

Swansea University, Swansea, SA2 8PP, United Kingdom.

E-mail: m.c.quigley@swansea.ac.uk

Short title: Interactions between the elements of an outcome

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.

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

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

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

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

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

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

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

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

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

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

conditioning procedures. Nevertheless, given that the temporal order of the compound stimuli during training is comparable to the aforementioned studies (arguably serving as outcomes) it is appropriate to take these findings into consideration.

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

consistent with Miller and Matute (1998) and Rescorla (1980), it would be expected that a 'blocked' outcome would be learned about less well than a control outcome which is surprising. If, however, the elements of an outcome do not compete for associative strength as a result of their associative history – in the same manner as cues – it would be expected that the outcomes will be learned about to the same extent, as is suggested by some studies (Cobos et al. 2002; Price & Yates, 1995) and models of associative learning.

Experiment 1

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

Insert Table 1 here

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

Method

Participants

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

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

Materials

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

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

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

Insert Figure 1 here

Procedure

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

"Thank you for participating in this experiment.

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

Stage 1

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

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

Stage 2

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

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

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

Test

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

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, η_P^2 = .58. There was no significant effect of outcome, F (1, 30) = .67, MSE = .02, p = .42, η_P^2 = .02, and no interaction between outcome and trial, F (23, 690) = .58, MSE = .02, p = .95, η_P^2 = .02. The ANOVA therefore confirmed the impressions of the graph, revealing that participants learned about both

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

Stage 2

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

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

between these two factors, F (7, 210) = 14.55¸ MSE = .08, p = <.001, η_P^2 = .33. To explore the source of the interaction, simple main effects analyses were conducted. These analyses revealed significant differences between the outcomes on Tria1 1, F (1, 30) = 55.18, MSE =.02, p <. 001, η_P^2 = .65, and Trial 2, F (1, 30), = 5.09, MSE = .04, p <. 05, η_P^2 = .15, all other trials p >.05 (smallest p = .37). Overall, these results demonstrate that participants initially produced more correct responses for the blocking trials than the control trials, however, by Trial 3 onward participants responses were comparable.

Test Stage

The bottom left panel of Figure 2 illustrates participants' outcome specific ratings for the Blocked cues (A/B) and Control cues (C/D) at test. To calculate an outcome specific measure of participants' ratings for cues A – D and O2 and O4, participants' ratings for the novel outcome in Stage 2 which was not paired with the cue of interest, was subtracted from the ratings provided for the novel outcome which the cue was paired with in Stage 2 (see: Le Pelley, Beesley & Griffiths, 2014; Le Pelley & McLaren, 2003). For instance, participants' ratings of A – O4 (the novel outcome in Stage 2 which A was not paired with) were subtracted from participants' ratings of A – O2 (the novel outcome in Stage 2 which A was paired with). As can be seen participants ratings were higher for A/B – O2/O4 than C/D – O2/O4.[1] A paired 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).

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

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

Experiment 2

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

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

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

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

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

Insert Table 2 here

Method

Participants

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

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

Materials

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

Procedure

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

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

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

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

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

Stage 1

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

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

Stage 2

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

Test

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

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

Results and Discussion

Insert Figure 3 here

Stage 1

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

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

Stage 2

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

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

main effect of the factor of task type, F (1, 62) = .54, MSE = .19, p = .46, η_P^2 = .00, and all other interactions were non-significant, largest F (2, 124) = 1.15, MSE = .10, p = .32, η_P^2 = .02. Simple main effects identified the cause of the Cue-Outcome pairing × Trial interaction. There was an effect of outcome on Trials 1 to 3, largest F (2, 61) = 48.14, p = <. 001, η_P^2 = .61. All other trials were non-significant, smallest p = .08. Bonferroni corrected (adjusted p =.005) t-tests revealed that for Trial 1 the mean proportion of correct responses for A-O1O2/B-O3O4 was higher than C-O1O2/D-O3O4, t (63) = 9.68, p = < .001, and E-O1O2/F-O3O4, t (63) = 3.69, p = < .001. The mean proportion of correct responses for E-O1O2/F-O3O4 was also higher than C-O1O2/DO3O4, t (63) = 6.69, p = < .001. The t-test comparisons on Trials 2 and 3 were non-significant with the Bonferroni correction (smallest p = .01).

Insert Figure 4 here

Test Stage

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

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

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

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

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

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

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

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

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

Experiment 3

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

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

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

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

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

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

Method

Participants.

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

Materials & Procedure.

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

Results and Discussion

Insert Figure 5 here

Stage 1

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

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

Stage 2

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

A two-way mixed model ANOVA was performed on these data with a between-subjects factor of task type (causal vs diagnostic), and within-subjects factor of cue-outcome pairing (A-O1O2/B-O3O4 vs C-O1O2/D-O3O4 vs E-O1O2/F-O3O4). The ANOVA revealed a main effect for the cue-outcome pairings, F (2, 124) = 41.63, MSE = .12, p = < .001, η_P^2 = .40. There was no main effect of the factor of task ty pe F (1, 62) = .61, MSE = .08, p = .44, η_P^2 = .01, and the interaction between these factors was non-significant, F (2, 124) = 2.28, MSE = .12, p = .11, η_P^2 = .04. To identify the source of the main effect for the cue-outcome pairings, Bonferroni adjusted paired samples t-tests were performed (adjusted p = .017). These t-tests revealed that the mean proportion of correct responses for A-O1O2/B-O3O4 was higher than C-O1O2/D-O3O4, t (63) = 8.81, p = < .001, but not E-O1O2/F-O3O4, t (63) = 1.82, p = .07. The mean proportion of correct responses for E-O1O2/F-O3O4 was also higher than C-O1O2/DO3O4, t (63) = 7.60, p = < .001. These results demonstrate that at the outset of Stage 2 participants displayed a higher proportion of correct responses to the blocking and Kamin control trials than to the control trials.

Test Stage

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

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

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

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

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

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

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

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

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

General Discussion

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

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

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

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

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

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

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

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

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

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

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

An alternative, and interesting, explanation for the outcome facilitation effect observed in Experiment 3 can be developed by appealing to the influence of within-compound associations. Note that in Experiment 3 the stimuli which are paired together most frequently in Stage 2 are the outcome compounds (i.e., O1 and O2, and O3 andO4, each of which is paired together 3 times). Given that only one trial is presented in Stage 2 for each of the cue-outcome pairings, it is conceivable that insufficient learning took place about the relationship between the cues and the added outcomes to directly determine participants' ratings at test. In light of this, it is possible that Stage 1 learning is mediating performance at test. For example, when stimulus C was presented at test, it may provoke participants to think of the outcome which it was paired with in Stage 1 (O3), which in turn, associatively activates the outcome which it was paired with most frequently during Stage 2 (O4). This analysis would explain the mildly negative ratings given to C and D at test, as participants are selecting the outcomes that

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

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

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

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

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

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

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

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

References

Arcediano, F., Escobar, M., & Miller, R. R. (2004). Is stimulus competition an acquisition deficit or a performance deficit? *Psychonomic bulletin & review, 11(6),* 1105-1110. https://doi.org/10.3758/BF03196744

Arcediano, F., Matute, H., Escobar, M., & Miller, R. R. (2005). Competition between antecedent and between subsequent stimuli in causal judgments. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 31*(2), 228. http://dx.doi.org/10.1037/0278-7393.31.2.228

Batsell Jr, W. R., & Batson, J. D. (1999). Augmentation of taste conditioning by a preconditioned odor. *Journal of Experimental Psychology: Animal Behavior Processes, 25(*3), 374. http://dx.doi.org/10.1037/0097-7403.25.3.374

Blaser, R. E., Couvillon, P. A., & Bitterman, M. E. (2006). Blocking and pseudoblocking: New control experiments with honeybees. *The Quarterly journal of experimental psychology, 59*(1), 68-76. http://dx.doi.org/10.1080/17470210500242938

Bonardi, C. (1991). Blocking of occasion setting in feature-positive discriminations. *The Quarterly Journal of Experimental Psychology, 43(*4), 431-448. http://dx.doi.org/10.1080/14640749108401278

Cheng, K., & Spetch, M. L. (2001). Blocking in landmark-based search in honeybees. *Animal Learning & Behavior, 29*(1), 1-9. http://dx.doi.org/10.1037/a0012905

Cobos, P. L., López, F. J., Cano, A., Almaraz, J., & Shanks, D. R. (2002). Mechanisms of predictive and diagnostic causal induction. *Journal of Experimental Psychology: Animal Behavior Processes, 28*(4), 331. http://dx.doi.org/10.1037/0097-7403.28.4.331

Crookes, A. E., & Moran, P. M. (2003). An investigation into age and gender differences in human Kamin blocking, using a computerized task. *Developmental Neuropsychology, 24*(1), 461-477. http://dx.doi.org/10.1207/S15326942DN2401_03

Denniston, J. C., Savastano, H. I., & Miller, R. R. (2001). The extended comparator hypothesis: Learning by contiguity, responding by relative strength. In R. R. Mowrer & S. B. Klein (Eds.), *Handbook of contemporary learning theories* (pp. 65-117). Mahwah, NJ: Erlbaum.

Dickinson, A., Nicholas, D. J., & Mackintosh, N. J. (1983). A re-examination of one-trial blocking in conditioned suppression. *The Quarterly Journal of Experimental Psychology, 35*(1), 67-79. http://dx.doi.org/10.1080/14640748308400914

Dickinson, A., Shanks, D., & Evenden, J. (1984). Judgement of act-outcome contingency: The role of selective attribution. *The Quarterly Journal of Experimental Psychology, 36*(1), 29-50. http://dx.doi.org/10.1080/14640748408401502

Don, H. J., & Livesey, E. J. (2018). Is the blocking effect sensitive to causal model? It depends how you ask. In T.T. Rogers, M. Rau, X. Zhu, & C. W. Kalish (Eds.), *Proceedings of the 40th Annual Conference of the Cognitive Science Society* (pp. 1630- 1635). Austin, TX: Cognitive Science Society.

Dopson, J. C., Pearce, J. M., & Haselgrove, M. (2009). Failure of retrospective revaluation to influence blocking. *Journal of Experimental Psychology: Animal Behavior Processes, 35*(4), 473. http://dx.doi.org/10.1037/a0014907

Durlach, P. J., & Rescorla, R. A. (1980). Potentiation rather than overshadowing in flavor-aversion learning: an analysis in terms of within-compound associations. *Journal of Experimental Psychology: Animal Behavior Processes, 6*(2), 175. http://dx.doi.org/10.1037/0097-7403.6.2.175

Elsner, B., & Hommel, B. (2001). Effect anticipation and action control. *Journal of experimental psychology: human perception and performance, 27(*1), 229. http://dx.doi.org/10.1037/0096-1523.27.1.229

Flach, R., Osman, M., Dickinson, A., & Heyes, C. (2006). The interaction between response effects during the acquisition of response priming. *Acta Psychologica, 122*(1), 11-26. http://dx.doi.org/10.1016/j.actpsy.2005.09.001

Gillan, D. J., & Domjan, M. (1977). Taste-aversion conditioning with expected versus unexpected drug treatment. *Journal of Experimental Psychology: Animal Behavior Processes, 3*(4), 297. http://dx.doi.org/10.1037/0097-7403.3.4.297

Guerrieri, F., Lachnit, H., Gerber, B., & Giurfa, M. (2005). Olfactory blocking and odorant similarity in the honeybee. *Learning & Memory, 12*(2), 86-95. http://dx.doi.org/10.1101/lm.79305

Guez, D., & Miller, R. R. (2008). Blocking and pseudoblocking: The reply of Rattus norvegicus to Apis mellifera. *The Quarterly Journal of Experimental Psychology, 61*(8), 1186-1198. http://dx.doi.org/10.1080/17470210701480238

Jones, P. M., & Haselgrove, M. (2013). Overshadowing and associability change: Examining the contribution of differential stimulus exposure. *Learning & Behavior, 41*(1), 107–117. http://dx.doi.org/10.3758/s13420-012-0089-z

Jones, P. M., Zaksaite, T., & Mitchell, C. J. (2019). Uncertainty and blocking in human causal learning. *Journal of Experimental Psychology: Animal Learning and Cognition, 45*(1), 111. https://doi.org/10.1037/xan0000185

Kamin, L.J. (1968). “Attention-like” processes in classical conditioning. In M.R. Jones (Ed.). *Miami Symposium on the Prediction of Behavior: Aversive Stimulation*. Coral Gables, Florida (pp. 9-31). Florida: University of Miami Press.

Kamin, L.J. (1969). Predictability, surprise, attention, and conditioning. In B. A. Campbell & R. M. Church (Eds.), *Punishment and Aversive Behavior* (pp. 279-296). New York: Appleton-Century-Crofts.

Le Pelley, M. E., Beesley, T., & Griffiths, O. (2014). Relative salience versus relative validity: Cue salience influences blocking in human associative learning. *Journal of Experimental Psychology: Animal Learning and Cognition, 40*(1), 116. http://dx.doi.org/10.1037/xan0000006

Le Pelley, M. E., & McLaren, I. P. L. (2003). Learned associability and associative change in human causal learning. *The Quarterly Journal of Experimental Psychology, 56B*(1), 68–79. http://dx.doi.org/10.1080/02724990244000179

Le Pelley, M. E., Oakeshott, S. M., & McLaren, I. P. L. (2005). Blocking and unblocking in human causal learning. *Journal of Experimental Psychology: Animal Behavior Processes, 31*(1), 56. http://dx.doi.org/10.1037/0097-7403.31.1.56

Livesey, E. J., Greenaway, J. K., Schubert, S., & Thorwart, A. (2019). Testing the deductive inferential account of blocking in causal learning. *Memory & cognition, 47*(6), 1120-1132. https://doi.org/10.3758/s13421-019-00920-w

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review, 82*(4), 276–298. http://dx.doi.org/10.1037/h0076778

Mackintosh, N. J. (1975b). Blocking of conditioned suppression: Role of the first compound trial. *Journal of Experimental Psychology: Animal Behavior Processes, 1*, 335-45.

Mackintosh, N. J., Dickinson, A., & Cotton, M. M. (1980). Surprise and blocking: Effects of the number of compound trials. *Animal Learning & Behavior, 8(3),* 387-391. http://dx.doi.org/10.3758/BF03199622

Matute, H., Arcediano, F., & Miller, R. R. (1996). Test question modulates cue competition between causes and between effects. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 22*(1), 182. http://dx.doi.org/10.1037/0278-7393.22.1.182

McLaren, I. P. L., McAndrew, A., Angerer, K., McLaren, R., Forrest, C., Bowditch, W., Monsell, S., & Verbruggen, F. (2019). Mackintosh lecture: Association and cognition: Two processes, one system. *The Quarterly Journal of*

Experimental Psychology, *72*(2), 98-117. https://doi.org/10.1177/1747021818766287

Miller, R. R., & Escobar, M. (2002). Associative interference between cues and between outcomes presented together and presented apart: An integration. Behavioural Processes, 57(2-3), 163-185. https://doi.org/10.1016/S0376-6357(02)00012-8

Miller, R. R., & Matute, H. (1998). Competition between outcomes. *Psychological Science, 9*(2), 146-149. http://dx.doi.org/10.1111/1467-9280.00028

Miller, R. R., & Matzel, L. D. (1988). The comparator hypothesis: A response rule for the expression of associations. In Bower, G. H. (Ed.), The *Psychology of learning and motivation* (Vol. 22, pp. 51-92). San Diego, CA: Academic Pres Academic Press.

Pavlov, I. P. (1927). *Conditioned reflexes* (G. V. Anrep, Trans.). London: Oxford University Press.

Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, *87*(6), 532–52. http://dx.doi.org/10.1037/0033-295X.87.6.532

Perruchet, P. (1985). A pitfall for the expectancy theory of human eyelid conditioning. *The Pavlovian journal of biological science: official journal of the Pavlovian, 20*(4), 163-170. http://dx.doi.org/10.1007/bf03003653

Prados, J., Alvarez, B., Acebes, F., Loy, I., Sansa, J., & Moreno-Fernández, M. M. (2013). Blocking in rats, humans and snails using a within-subjects design. *Behavioural processes, 100*, 23-31. http://dx.doi.org/10.1016/j.beproc.2013.07.014

Price, P. C., & Yates, J. F. (1995). Associative and rule-based accounts of cue interaction in contingency judgment. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *21*(6), 1639. http://dx.doi.org/10.1037/0278-7393.21.6.1639

Rescorla, R. A. (1980). *Pavlovian second-order conditioning: Studies in associative learning*. Hillsdale, NJ: Erlbaum.

Rescorla, R. A. & Wagner, A. R. (1972). A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In: Black, A. H., & Prokasy, W. F. (Eds.), *Classical conditioning II: Current research and theory* (pp. 64-99). New York: Appleton-Century-Crofts.

Sanderson, D. J., Jones, W. S., & Austen, J. M. (2016). The effect of the amount of blocking cue training on blocking of appetitive conditioning in mice. *Behavioural processes, 122,* 36-42.

http://dx.doi.org/10.1016/j.beproc.2015.11.007

Urcelay, G. P. (2017). Competition and facilitation in compound conditioning. *Journal of Experimental Psychology: Animal Learning and Cognition, 43*(4), 303. http://dx.doi.org/10.1037/xan0000149

Van Hamme, L. J., & Wasserman, E. A. (1994). Cue competition in causality judgments: The role of nonpresentation of compound stimulus elements. *Learning and motivation, 25*(2), 127-151. http://dx.doi.org/10.1006/lmot.1994.1008

Van Hamme, L. J., Kao, S. F., & Wasserman, E. A. (1993). Judging interevent relations: From cause to effect and from effect to cause. *Memory & Cognition, 21*(6), 802-808.

Wagner, A. R., Logan, F. A., Haberlandt, K., & Price, T. (1968). Stimulus selection in animal discrimination learning. *Journal of experimental psychology, 76*(2p1),

Wagner, A.R. 1981. SOP: a model of automatic memory processing in animal behavior. In: Spear, N.E., Miller, R.R. (Eds.), *Information Processing in Animals: Memory Mechanisms*. Erlbaum, Hillsdale, NJ, pp. 5–47.

Waldmann, M. R. (2000). Competition among causes but not effects in predictive and diagnostic learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 26*(1), 53. http://dx.doi.org/10.1037/0278-7393.26.1.53

Waldmann, M. R., & Holyoak, K. J. (1992). Predictive and diagnostic learning within causal models: asymmetries in cue competition. *Journal of Experimental Psychology: General, 121*(2), 222. http://dx.doi.org/10.1037//0096-3445.121.2.222

Willner, J. A. (1978). Blocking of a taste aversion by prior pairings of exteroceptive stimuli with illness. *Learning & Motivation, 9*, 125-140. https://doi.org/10.1016/0023-9690(78)90016-4

Footnote to title page.

Martyn Quigley, Department of Psychology, Swansea University. Mark Haselgrove, School of Psychology, University of Nottingham.

This work was supported by the Economic and Social Research Council [grant number ES/I021108/1] and contributed to Martyn Quigley's doctorate degree by funding a studentship.

Correspondence concerning this article should be addressed to Martyn Quigley, Department of Psychology, Vivian Tower, College of Human & Health Sciences, Swansea University, Swansea, SA2 8PP, United Kingdom. E-mail: m.c.quigley@swansea.ac.uk

Tables

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.

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 causal task, A-E were reactions and O1-O4 were foods.

Figures

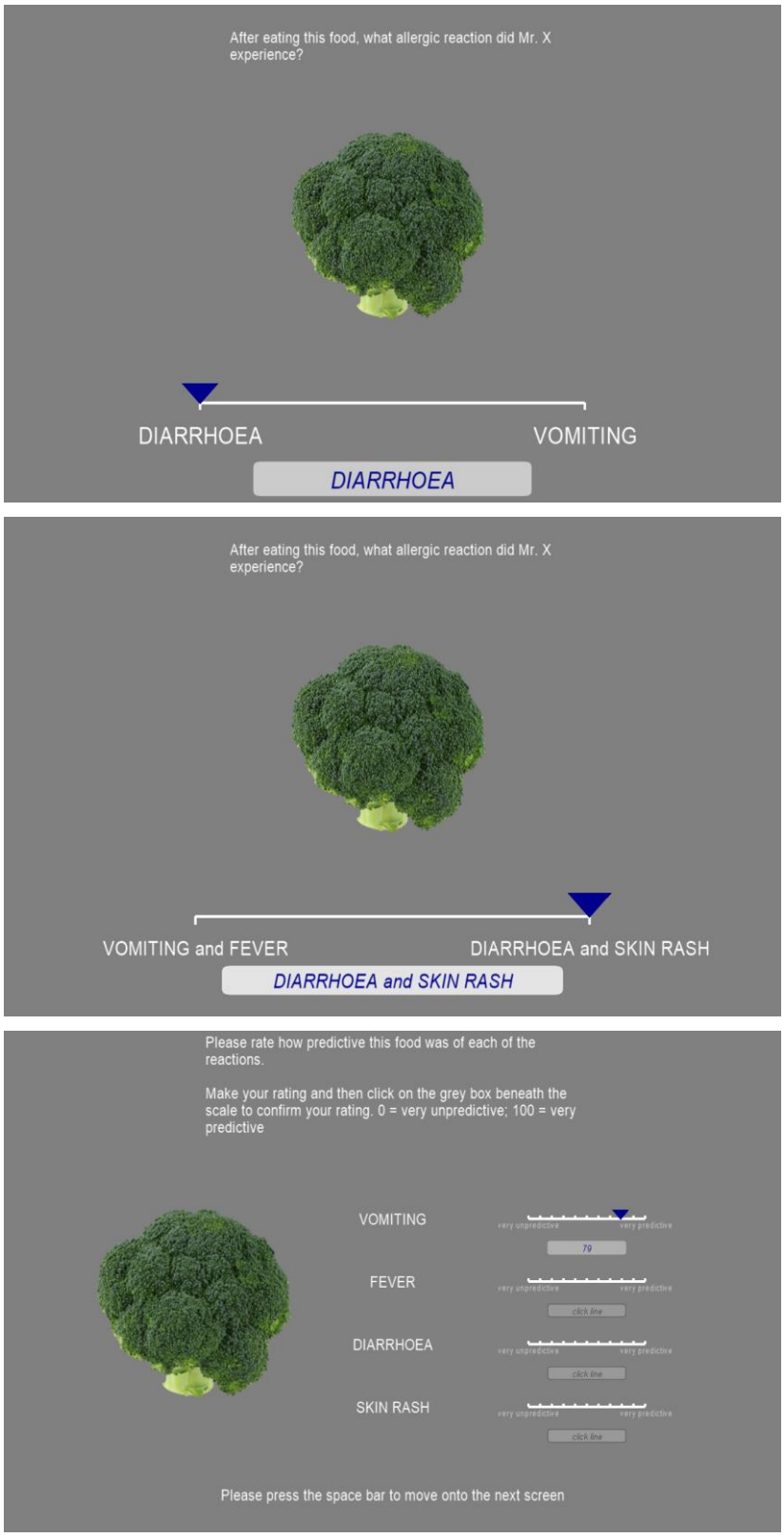

Figure 1. An example screen participants would be presented with during Stage 1 (top), Stage 2 (middle) and at test (bottom).

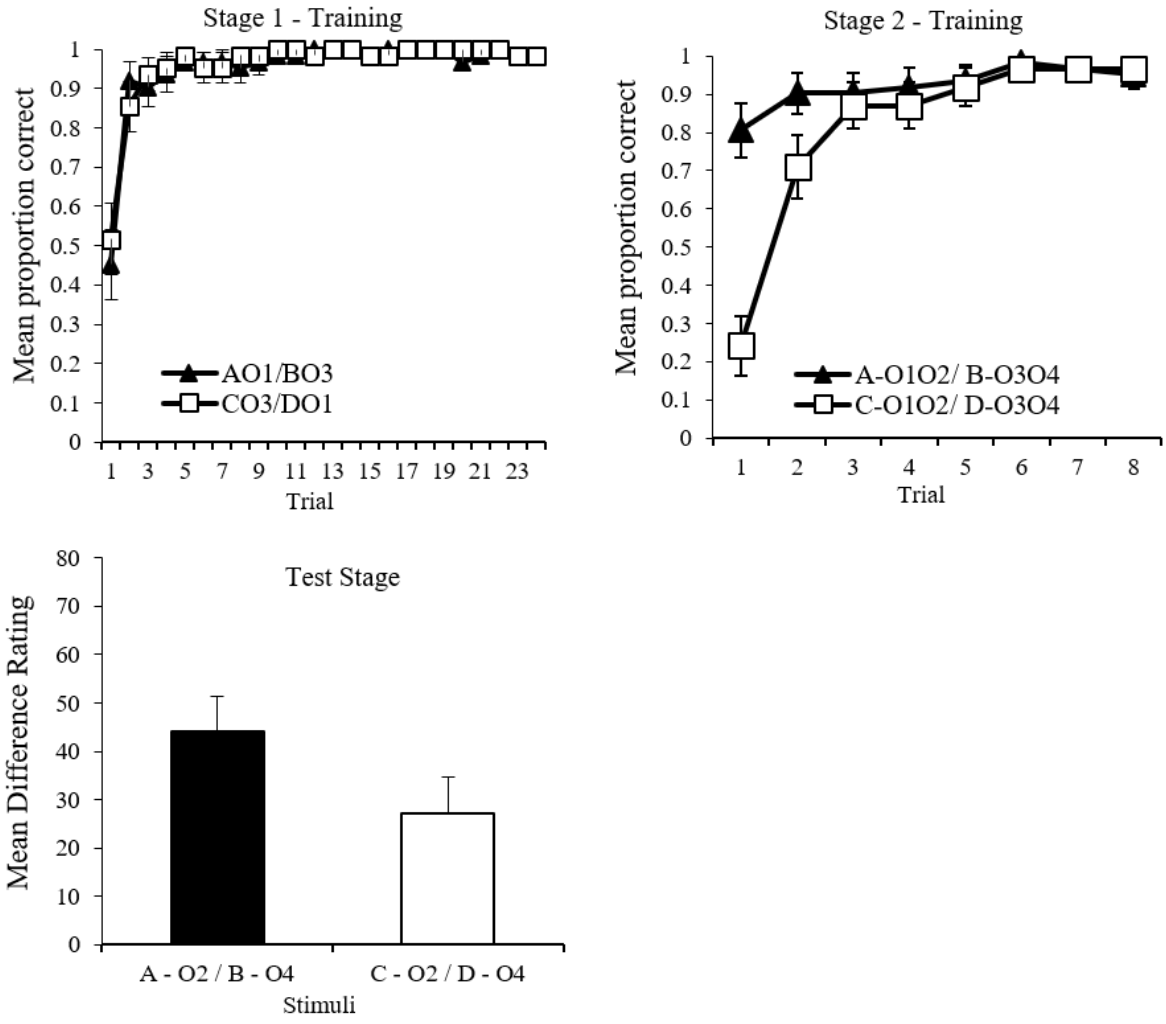

Figure 2. Mean proportion of correct responses for A – D: O1 and O3 during Stage 1 training (top left), for A – D: O1O2 and O3O4 during Stage 2 (top right) and the mean of participants' difference ratings for A/B – O2/O4 and C/D - O2/O4 at test. Error bars represent SEM.

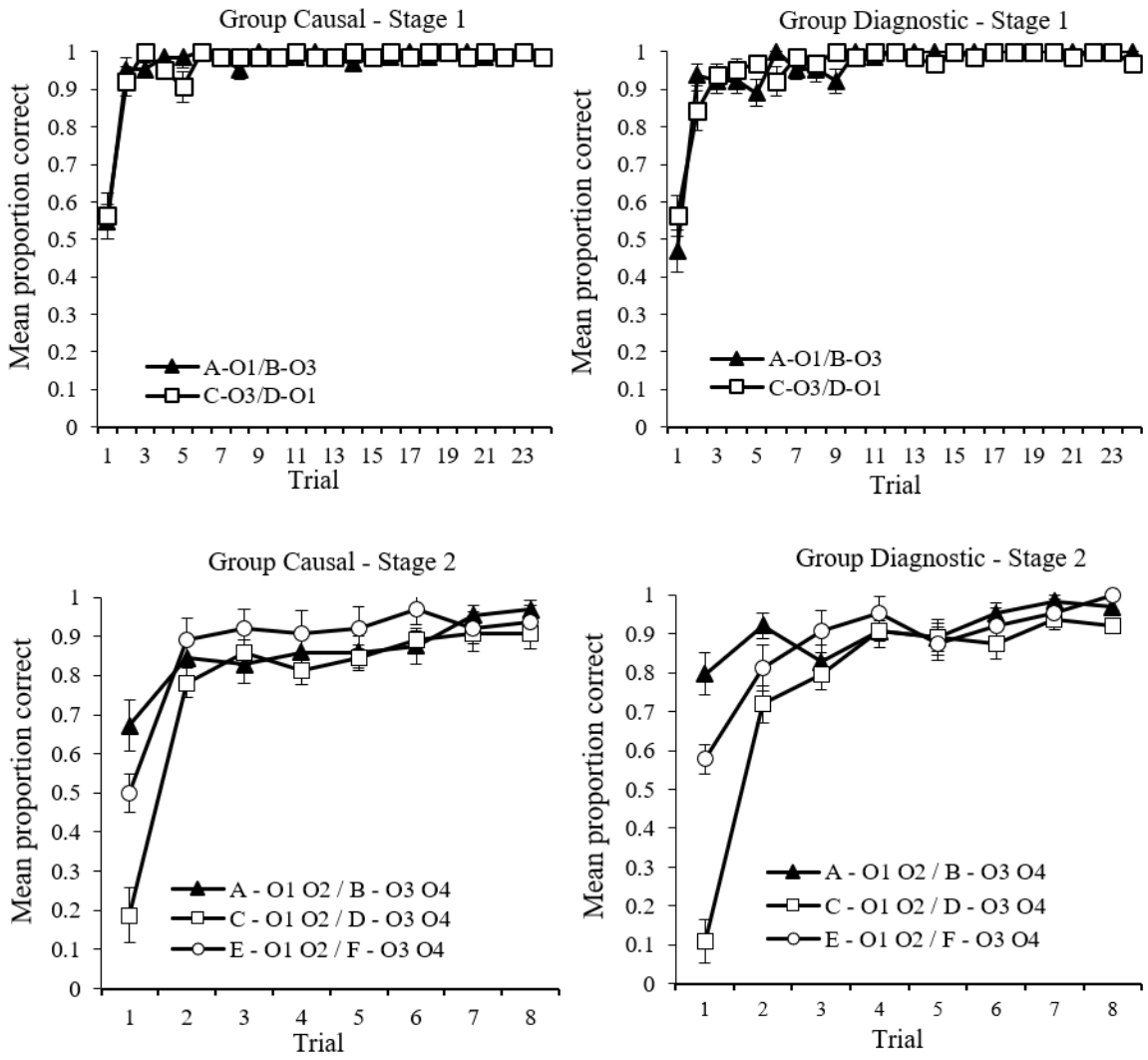

Figure 3. Mean proportion of correct responses for each trial type for the Causal (top left panel) and Diagnostic (top right panel) groups during Stage 1, and for each trial type during Stage 2 for the Causal (bottom left panel) and Diagnostic groups (bottom right panel) for Experiment 2. Error bars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

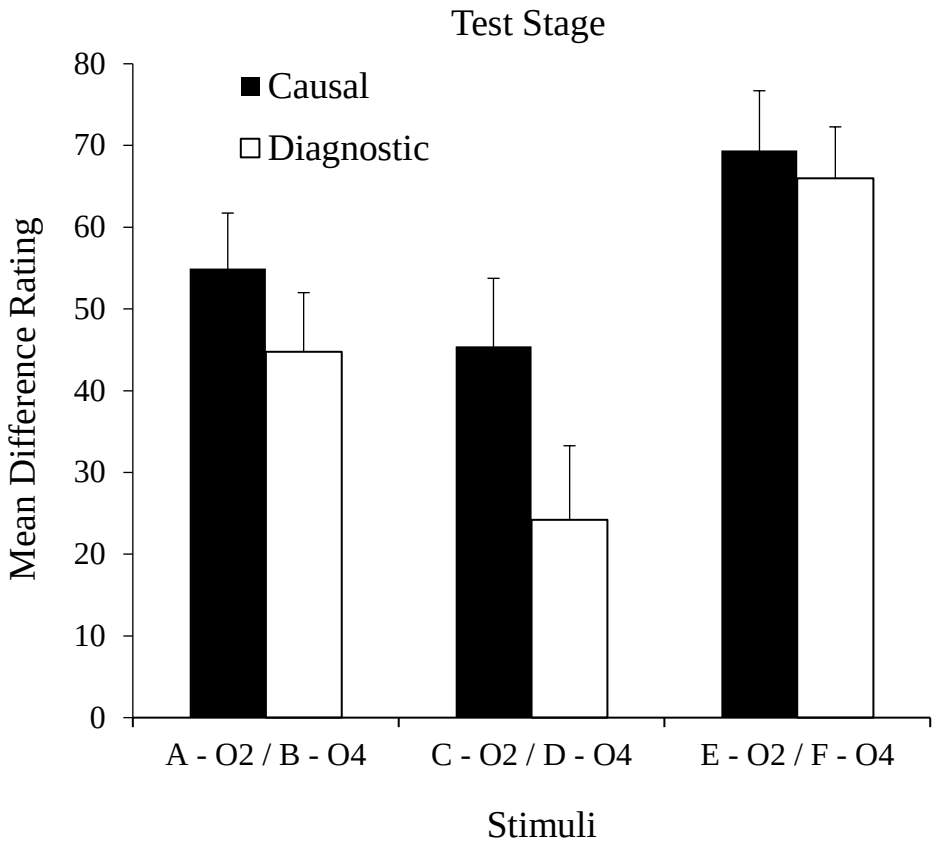

Figure 4. Mean of participants' difference ratings for the outcomes at test, for both causal and diagnostic judgment tasks. Error bars represent SEM.

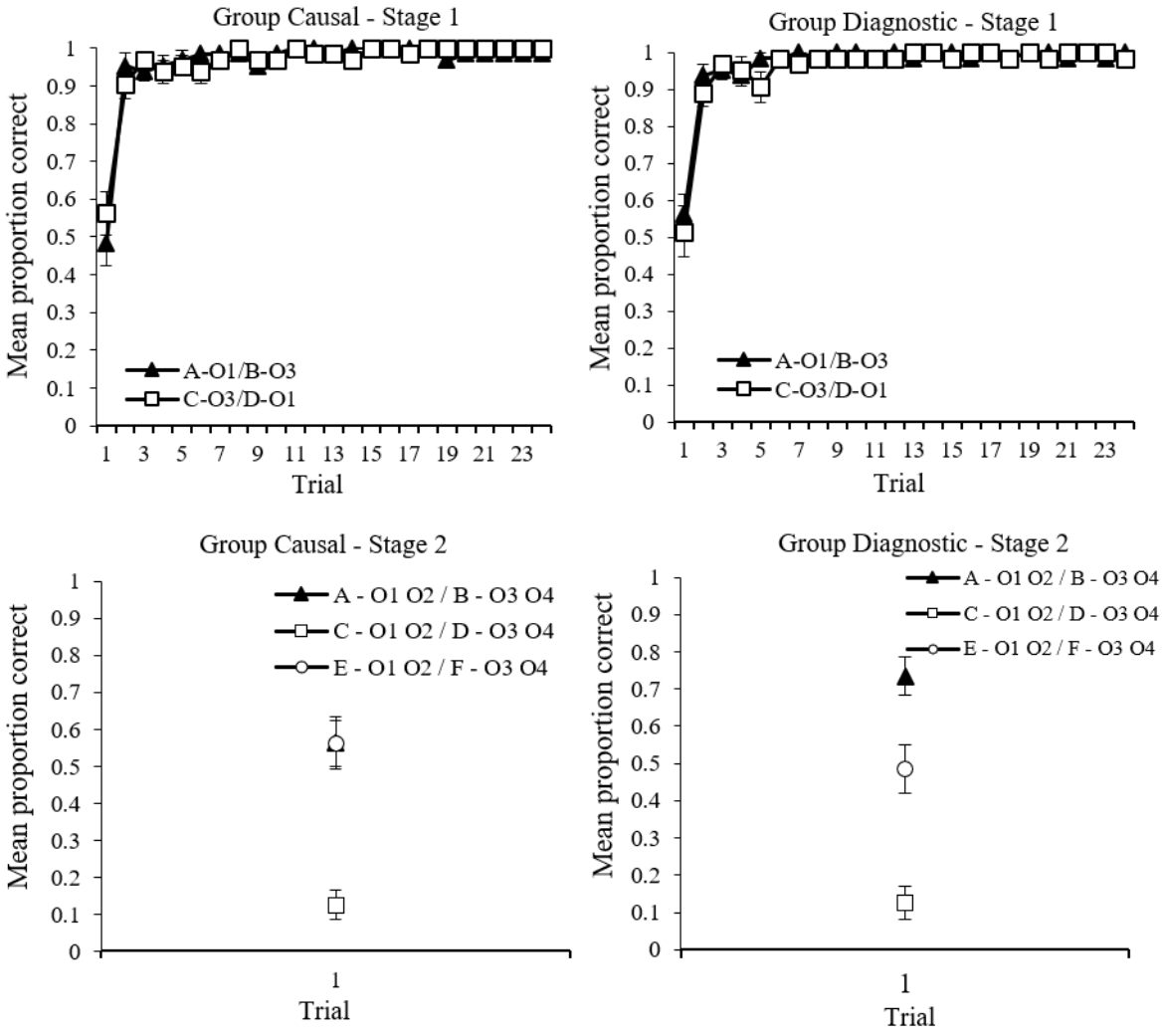

Figure 5. Mean proportion of correct responses for each trial type for the Causal (top left panel) and Diagnostic (top right panel) groups during Stage 1, and for each trial type during Stage 2 for the Causal (bottom left panel) and Diagnostic groups (bottom right panel) for Experiment 3. Error bars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

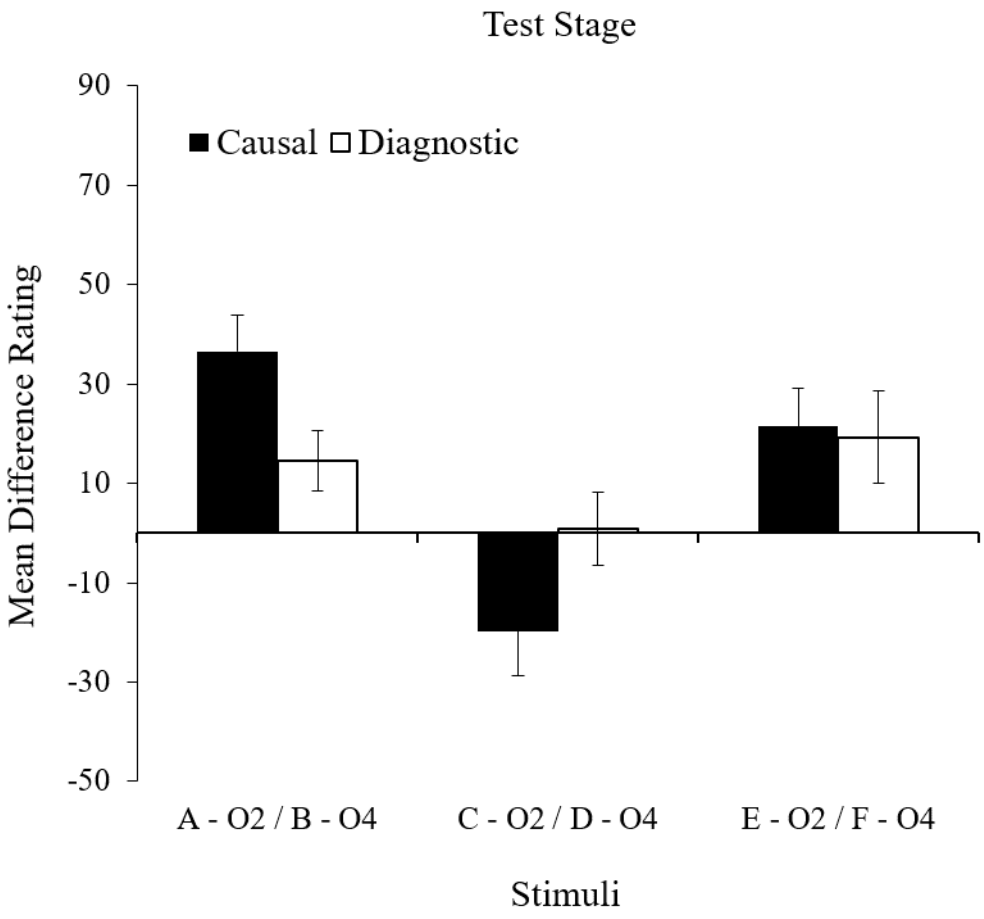

Figure 6. Mean of participants' difference ratings for the outcomes at test, for both causal and diagnostic judgment tasks. Error bars represent SEM.

Appendix

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

		Group Causal		Group Diagnostic	
Experiment	**Cue Type**	**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.