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Cue interaction and judgments of causality: Contributions of causal and associative processes

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In four experiments, the predictions made by causal model theory and the Rescorla–Wagner model were tested by using a cue interaction paradigm that measures the relative response to a given event based on the influence or salience of an alternative event. Experiments 1 and 2 uncorrelated two variables that have typically been confounded in the literature (causal order and the number of cues and outcomes) and demonstrated that overall contingency judgments are influenced by the causal structure of the events. Experiment 3 showed that trial-by-trial prediction responses, a second measure of causal assessment, were not influenced by the causal structure of the described events. Experiment 4 revealed that participants became less sensitive to the influence of the causal structure in both their ratings and their predictions as trials progressed. Thus, two experiments provided evidence for high-level (causal reasoning) processes, and two experiments provided evidence for low-level (associative) processes. We argue that both factors influence causal assessment, depending on what is being asked about the events and participants' experience with those events.

In the past decade, the debate between causal model and associative learning theorists has centered on whether or not human inferences are sensitive to the causal structure of contingent events (see Waldmann, 2000, for a review). Whereas causal models code events in terms of *causes* and *effects*, associative models disregard the causal description of the events, instead coding them solely in terms of their temporal order, in which antecedent events are referred to as *cues* and subsequent events as *outcomes*. The disagreement has concerned the nature of the processes involved in making causal inferences. According to causal model theory, expectations of causal structure guide learning about the relevant causal events in a top-down fashion. In contrast, an associative account maintains that causal learning is modeled by the bottom-up acquisition of associative weights guided by simple event pairings. In this article, the extent to which and the circumstances in which these two factors influence causal assessments are examined, and the conditions under which they operate are described.

As researchers started applying the principles of associative learning theories to humans (e.g., Shanks & Dickinson, 1987), Waldmann and Holyoak (1992) argued that humans are capable of more sophisticated forms of causal learning than simply reacting to contingencies in their environment. They argued that people conceptualize the asymmetry of causal relationships. Causes influence effects, but effects do not influence causes. "In addition to using perceived or imagined causes to predict future events, people can use perceived or imagined effects as cues to diagnose their unseen causes" (Waldmann & Holyoak, 1997, p. 125). Our knowledge of causal asymmetry provides us with the capacity to ignore the order in which events are presented, thereby transforming them into causal model representations that reflect their asymmetry (Waldmann, 2000). The Rescorla–Wagner model (which embodies the essential and salient characteristics of associative models) neglects the causal status among events by simply encoding their temporal order. Events that occur first are encoded as cues, and subsequent events are encoded as outcomes. It follows from causal model theory that causes interact and effects do not. That is, we judge one *cause* in light of another, but judge two *effects* independently. According to the Rescorla–Wagner model, *cues* compete, and *outcomes* do not. The term *cue interaction* refers broadly to the relative assessment of two events, without reference to the mechanism of interaction.

Causal model and associative theories have often been pitted against one another in the context of cue interaction paradigms, such as blocking (e.g., Waldmann, 2000; Waldmann & Holyoak, 1992), relative cue validity (e.g., Matute, Arcediano, & Miller, 1996; Van Hamme, Kao, & Wasserman, 1993), and overshadowing (e.g., Waldmann, 2001). Of interest in each of these paradigms is the extent to which participants regard one cue in light of another or consider each cue independently. In the present series of experiments, the one-phase simultaneous blocking task (Baker, Mercier, Vallée-Tourangeau, Frank, & Pan, 1993) was used to provide a novel test of causal model theory by means of the conditional ΔP ac-

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count (Spellman, 1996a, 1996b). According to causal model theory, when two causes produce one effect, one should consider each cause as conditional upon the other, because causes interact. When one cause produces two effects, one should consider each effect independently of the other, because effects do not interact. The one-phase simultaneous blocking design provides a strong test of the model's predictions because it enables participants to conditionalize on two differentially predictive causes, either one upon the other. When two causes produce one effect, a conditional ΔP account applied to causal model theory predicts that participants should rate the influence of each cause in accordance with *conditional* ΔP. When one cause produces two effects, participants should rate the influence of the cause on each effect in accordance with *unconditional* ΔP.

In a task involving two cues and a single outcome, one of four cue combinations is possible on a given trial: Both cues may be present (*AB*), one cue may be present and the other absent (*A*~*B* or ~*AB*), or both cues may be absent (~*A*~*B*). For each cue combination, the outcome either occurs (*O*) or does not occur (~*O*), resulting in eight possible cue–outcome combinations, as is illustrated in Figure 1. Thus, each cue can be expressed in terms of its respective unconditional ΔP value, defined as

$$\Delta P_A = P(O \mid A) - P(O \mid \sim A) = \frac{a+c}{a+b+c+d} - \frac{e+g}{e+f+g+h} \quad (1)$$

and

$$\Delta P_B = P(O \mid B) - P(O \mid \sim B) = \frac{a+e}{a+b+e+f} - \frac{c+g}{c+d+g+h}, \quad (2)$$

where each equation corresponds to the difference between the proportion of times the outcome occurs given the cue and the proportion of times the outcome occurs not given the cue (Allan, 1980). Alternatively, Cues *A* and *B* can be expressed in terms of their respective *conditional* ΔP values, defined as

$$\Delta P_{A|B} = P(O \mid AB) - P(O \mid \sim AB) = \frac{a}{a+b} - \frac{e}{e+f}, \quad (3)$$

$$\Delta P_{A|\sim B} = P(O \mid A \sim B) - P(O \mid \sim A \sim B) = \frac{c}{c+d} - \frac{g}{g+h}, \quad (4)$$

$$\Delta P_{B|A} = P(O \mid BA) - P(O \mid \sim BA) = \frac{a}{a+b} - \frac{c}{c+d}, \quad (5)$$

and

$$\Delta P_{B|\sim A} = P(O \mid B \sim A) - P(O \mid \sim B \sim A) = \frac{e}{e+f} - \frac{g}{g+h}. \quad (6)$$

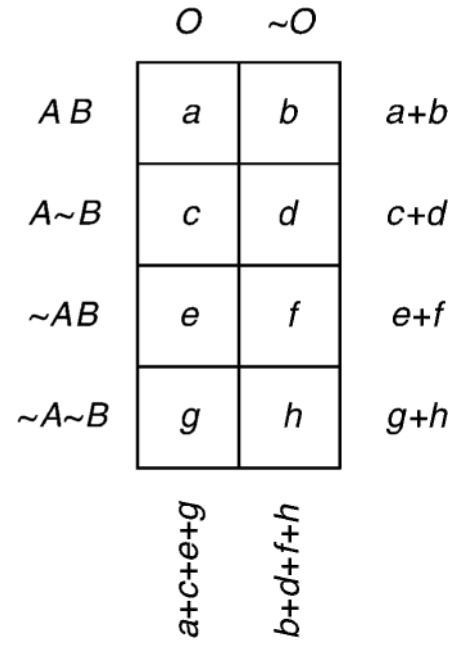

Figure 1. Summary 4 × 2 contingency matrix illustrating each of the possible cause–effect combinations for two cues. Each cell represents the frequency of each event type.

The conditional ΔP values in Equations 3–6 allow one to assess the influence of each cue in both the presence and the absence of the other cue. For example, to assess the influence of Cue *A*, Equation 3 describes only the cases in which Cue *B* is present by taking the difference between the proportion of times the outcome occurs given *A* and the proportion of times the outcome occurs not given *A*. Moreover, Equation 4 describes only the cases in which Cue *B* is absent, by taking the difference between the proportion of times the outcome occurs given *A* and the proportion of times the outcome occurs not given *A*.

Therefore, when two causes produce one effect, a conditional ΔP account applied to causal model theory predicts that, because each cause should be assessed in light of the other, participants should rate the influence of each cause in accordance with conditional ΔP (Equations 3–6). When one cause produces two effects, because each effect should be assessed independently, participants should rate the influence of the cause on each effect in accordance with unconditional ΔP (Equations 1 and 2). Under these circumstances, with only one cause and two effects, one must rotate the 4 × 2 contingency matrix shown in Figure 1 to form a 2 × 4 matrix in which the two rows represent the presence and the absence of the cause and the columns represent the four combinations of the two effects. By doing so, it is impossible to calculate the conditional contingencies for *A* and *B* defined in Equations 3–6.

Experiments designed to test causal model theory have typically compared two causal scenarios in which two (or more) causes precede a single effect or in which

two (or more) effects precede a single cause, thereby confounding causal order (CE vs. EC) and the number of causes and effects (2–1 vs. 1–2; see, e.g., Matute et al., 1996; Van Hamme & Wasserman, 1993; Waldmann, 2000; Waldmann & Holyoak, 1992). As is illustrated in Figure 2, four cause–effect scenarios are possible by crossing the two variables: Two cues can be followed by one outcome and can be described as two causes producing an effect (2C–1E) or as two effects resulting from a cause (2E–1C), and one cue can be followed by two outcomes and can be described as a cause producing two effects (1C–2E) or as an effect resulting from two causes (1E–2C). According to causal model theory, participants should be sensitive to the interaction between causal order and the number of the causes and effects, which is defined as the *structure* of the causal relationship (Waldmann, 2000, 2001; Waldmann & Holyoak, 1992, 1997). The model predicts that pairs of causes will interact in the 2C–1E and 1E–2C scenarios (i.e., the negative diagonal of Figure 2) and predicts that pairs of effects will not interact in the 2E–1C and 1C–2E scenarios (i.e., the positive diagonal of Figure 2). In contrast, according to the Rescorla–Wagner model, participants should be sensitive only to the number of the cues and outcomes in which *cues* interact regardless of their causal order. The model therefore predicts that pairs of cues will interact in the 2C–1E and 2E–1C scenarios (i.e., the left column of Figure 2) and predicts that pairs of outcomes will not interact in the 1C–2E and 1E–2C scenarios (i.e., the right column of Figure 2).

To summarize, a conditional ΔP account applied to causal model theory predicts that judgments of a pair of differentially predictive *causes* should elicit a cue interaction effect, whereas judgments of a pair of differentially diagnostic *effects* should not. In contrast, the Rescorla–Wagner model predicts that judgments of a pair of differentially contingent *cues* should elicit a cue interaction effect, whereas judgments of a pair of differentially contingent *outcomes* should not.

EXPERIMENT 1

Experiment 1 was designed to test the predictions made by causal model theory and the Rescorla–Wagner model by independently manipulating causal order and the number of cues and outcomes. Thus, four causal scenarios were presented to participants, using the one-phase simultaneous blocking task described above. Two cues were described either as causes of an effect (2C–1E) or as effects of a cause (2E–1C), or one cue was described either as a cause of two effects (1C–2E) or as an effect of two causes (1E–2C), as is shown in Figure 2. In each of the four scenarios, the two events that were presented simultaneously were either differentially predictive or diagnostic of the single event. Event *A* had a moderately positive unconditional ΔP of .5 and was paired with *B*, which had an unconditional ΔP of 0 or 1. Causal model theory predicts that participants will demonstrate a cue interaction effect in the 2C–1E and 1E–2C scenarios (and not in the other two), and the Rescorla–Wagner model predicts that participants will demonstrate a cue interaction effect in the 2C–1E and 2E–1C scenarios (and not in the other two).

Method

Participants and Design

Forty-eight undergraduate students at McMaster University participated for course credit. The experiment was designed to test how ratings of a moderately positive contingency varied in the presence of a zero or a perfect contingency as a function of causal order and

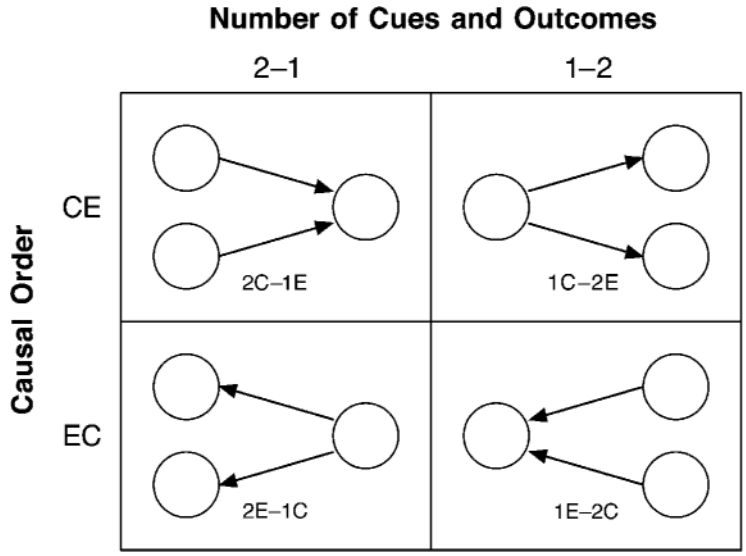

Figure 2. Four possible causal scenarios generated by crossing causal order (CE vs. EC) with the number of cues and outcomes (2–1 vs. 1–2).

the number of cues and outcomes. A four-factor mixed design was used, with causal order as a between factor with two levels (CE and EC) and the number of cues and outcomes as a within factor with two levels (2–1 and 1–2). Thus, half of the participants were assigned to the CE group and were presented with the 2C–1E and 1C–2E scenarios, and half were assigned to the EC group and received the 2E–1C and 1E–2C scenarios. Within each group, the order in which the scenarios were presented was counterbalanced. A third within factor was the contingency of Event B ($\Delta P_B = 0$ and $\Delta P_B = 1$), in which the order of presentation was also counterbalanced. The fourth factor was a within factor representing the number of trials prior to the participants' ratings (32 and 48). Table 1 illustrates the trial frequencies obtained by combining an unconditional contingency for A ($\Delta P_A = .5$) with one of two unconditional contingencies for B: a zero contingency ($\Delta P_B = 0$) or a perfect contingency ($\Delta P_B = 1$). We use the notation introduced by Baker et al. (1993) to represent the unconditional contingencies of the two events, $\Delta P_A/\Delta P_B$. The designation for the two examples in Table 1 are .5/0 and .5/1, in which the value on the left of the solidus represents ΔP_A and the value on the right represents ΔP_B.

Procedure and Materials

The design and procedure for Experiment 1 were adapted from Mehta (2000). The participants received instructions on a computer screen, where they were informed about four strains of bacteria that have been discovered in the mammalian digestive system. In the 2C–1E and 1E–2C scenarios, they were told that scientists were testing whether a pair of chemicals affected the strain's survival, whereas in the 2E–1C and 1C–2E scenarios, the scientists were testing whether the bacteria affected the production of a pair of chemicals.

Up to 4 participants at a time performed the experiment on Power Macintosh computers located in separate rooms. The entire experiment was programmed in MetaCard 2.3.1. In the instructions, the four causal scenarios were identified as separate "experiments" designed to test the influence of the chemicals on the bacterial strain or vice versa. Within each scenario, 48 trials were presented in random order, according to the frequencies presented in Table 1. The addition or production of a chemical was indicated by a computer-rendered movie of a colored three-dimensional chemical spinning along its axis, and actual footage of moving bacteria was displayed when the bacterial strain survived or was added. Faded, unmoving grayscale images of the same chemicals and bacteria were displayed to indicate their absence on a given trial. The names of the chemicals and bacteria were displayed only when the events occurred. Each of the movies and images was randomly assigned a fictitious name from a set of eight chemicals and four bacteria. Chemical A was always presented on the left-hand side of the display, and Chemical B was always presented on the right. The observer initiated a condition by clicking the Begin button on the computer screen and initiated each subsequent trial by clicking the Next Trial button.

Table 1
Frequency of Events in Experiment 1

	$\Delta P_A/\Delta P_B$	
Trial Type	.5/0	.5/1
ABO	9	18
A~BO	9	0
~ABO	3	6
~A~BO	3	0
AB~O	3	0
A~B~O	3	6
~AB~O	9	0
~A~B~O	9	18
Total trials	48	48
ΔP_A	.5	.5
$\Delta P_{A\|B}$	.5	0
$\Delta P_{A\|\sim B}$	.5	0
ΔP_B	0	1
$\Delta P_{B\|A}$	0	1
$\Delta P_{B\|\sim A}$	0	1

Note—The unconditional ΔP values were calculated using Equations 1 and 2. The conditional ΔP values were calculated using Equations 3–6. A and B are cues, O represents outcome. For further explanation, see text.

The materials for the four causal scenarios are described as follows.

2C–1E. The participants were instructed that each of the two chemicals would either be added to the bacterial strain or not, resulting in the survival or death of the bacterial strain. They were then presented with a series of trials in which one, both, or neither chemical was added, followed by the survival or death of the bacterial strain.

1C–2E. The participants were instructed that the bacterial strain would either be added to a human digestive environment or not, resulting in the production of each of a pair of chemicals or not. They were then presented with a series of trials in which the bacterial strain was either added or not, followed by the production of one, both, or neither chemical.

2E–1C. The participants were instructed that the bacterial strain would either be added to a human digestive environment or not, resulting in the production of each of a pair of chemicals or not. They were then presented with a series of trials in which one, both, or neither chemical was produced, followed by the addition of the bacterial strain or not.

1E–2C. The participants were instructed that each of the two chemicals would either be added to the bacterial strain or not, resulting in the survival or death of the bacterial strain. They were then presented with a series of trials in which the bacterial strain survived or not, followed by the addition of one, both, or neither chemical.

After passively viewing a series of 32 trials, the participants in the 2C–1E and the 1E–2C scenarios were asked to rate how strongly each chemical affected the survival of the bacteria, and those in the 2E–1C and 1C–2E scenarios were asked to rate how strongly the bacteria affected the production of each chemical. Ratings were made on a scale ranging from −100 to 100 by using a mouse to move a horizontal scroll bar from −100 at the leftmost position to 100 at the rightmost position, anchored at 0 at the center. After they had rated A, they were prompted to rate B, followed by another 16 trials in which they would repeat the rating process. After observing two "experiments" in which ΔP_B was either 0 or 1, a second set of instructions was presented, nearly identical to the first, differing only in the number of cues and outcomes, as has been described above. Again, ΔP_B was either 0 or 1 for the latter two "experiments," comprising a total of four conditions.

Results and Discussion

Mean ratings of Event A after 48 trials are illustrated in Figure 3A (error bars represent standard errors of the means). Ratings for each of the four scenarios are plotted as a function of the two ΔP_B values. According to causal model theory, when two causes produce a single effect (2C–1E and 1E–2C), ratings of A, which was always moderately positive, should remain moderately positive in the presence of a zero contingency and should be much less positive in the presence of a perfect contingency (tracking the conditional ΔP values in Table 1). When two effects result from a single cause (2E–1C and 1C–2E), A should be rated as moderately positive in the presence of both a zero and a perfect contingency (tracking the unconditional ΔP values in Table 1). According

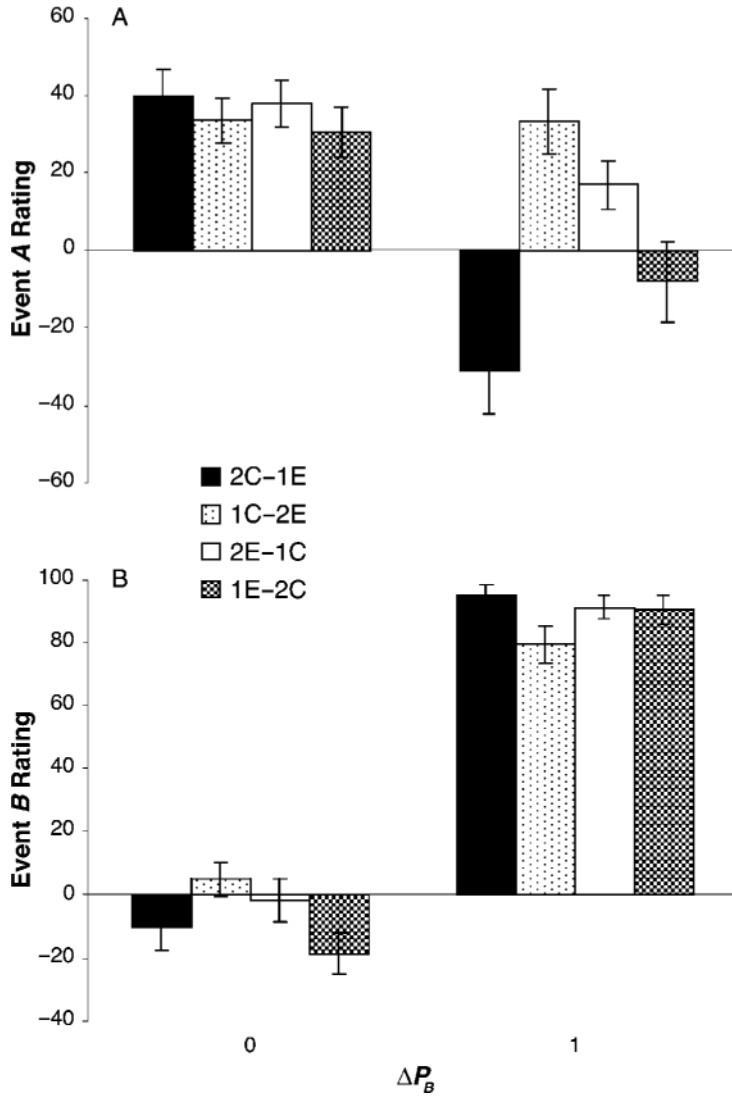

Figure 3. Mean ratings in Experiment 1 after 48 trials of (A) Event *A* and (B) Event *B*. For each event, the ratings are shown as a function of ΔP_B separately for each of the four scenarios. Error bars represent standard errors of the means.

to the Rescorla–Wagner model, cue interaction should be present only in the 2C–1E and 2E–1C scenarios. The pattern of results presented in Figure 3A is consistent with causal model theory. Only in the 2C–1E and 1E–2C scenarios are ratings of the moderately positive contingency noticeably lower in the presence of a perfect contingency ($\Delta P_B = 1$) than in the presence of a zero contingency ($\Delta P_B = 0$). Although *noticeably lower* here refers to a sizeable negative rating of *A*, what is relevant is that the *trend* in the participants' ratings of *A* demonstrate conditionalization (see also Spellman, 1996a).

A four-way mixed analysis of variance (ANOVA; effects were assessed for significance at the $\alpha = .05$ level), with ratings of *A* as the dependent variable, revealed significant main effects of contingency for Event *B* [$\Delta P_B = 0$ vs. $\Delta P_B = 1$; $F(1,46) = 40.12$, $MS_e = 3{,}112.23$] and the number of cues and outcomes [2–1 vs. 1–2; $F(1,46) = 4.55$, $MS_e = 1{,}388.45$]. The trial main effect (rating after 32 vs. 48 trials) was not significant [$F(1,46) = 0.28$, $MS_e = 609.23$], nor did it interact with any of the other factors. The main effect of causal order (CE vs. EC), although not significant [$F(1,46) = 0.04$, $MS_e = 2{,}573.04$], did interact with the number of cues and outcomes and the contingency for Event *B* [$F(1,46) = 17.78$, $MS_e = 2{,}635.78$]. This significant three-way interaction was further examined using the Tukey test. When $\Delta P_B = 0$, the ratings were not significantly different among the four scenarios. Moreover, these ratings did not differ from the ratings in the two scenarios in which one cause produced two effects (1C–2E and 2E–1C) when $\Delta P_B = 1$.

In contrast, ratings in the two scenarios in which two causes produced one effect (2C–1E and 1E–2C) when $\Delta P_B = 1$ were significantly lower than the other ratings and did not differ from each other.

Mean ratings of Event *B* are shown in Figure 3B. Table 1 indicates that for both .5/0 and .5/1, the conditional probabilities are the same as the unconditional probabilities. Therefore, ratings of *B* should be the same for the four scenarios and should be lower for .5/0 than for .5/1. It is clear from Figure 3B that the ratings for *B* are consistent with causal model theory. With ratings of Event *B* as the dependent variable, a four-way ANOVA revealed that the only main effect that was significant was ΔP_B [$F(1,46) = 628.13$, $MS_e = 1{,}394.64$]. None of the interactions involving ΔP_B were significant, confirming that ratings for a constant ΔP_B did not differ across causal order or number of cues and outcomes. The only other significant outcome was the interaction between causal order and trial [$F(1,46) = 4.14$, $MS_e = 474.94$]. The Tukey test revealed that this interaction reflected higher ratings for the CE order than for the EC order after 32 trials, but not after 48 trials.

In summary, Experiment 1 resulted in a significant interaction between causal order and the number of cues and outcomes. When two causes resulted in one effect (2C–1E and 1E–2C), the participants rated the moderately contingent Cause *A* as less predictive when it was paired with a perfect predictor ($\Delta P_B = 1$) than when it was paired with a nonpredictor ($\Delta P_B = 0$). When one cause resulted in two effects (2E–1C and 1C–2E), the participants rated the moderately contingent Effect *A* as equally diagnostic, both when the effect it had been paired with was perfectly diagnostic ($\Delta P_B = 1$) and when it was nondiagnostic ($\Delta P_B = 0$). These results indicate that cue interaction occurs when two causes produce one effect, regardless of whether the causes are presented before or after the effect, thus providing clear support for causal model theory. The participants' overall ratings seem to be sensitive to the causal structure of contingent events.

Both causal model theory and the Rescorla–Wagner model predict a cue interaction effect when two causes precede a single effect (2C–1E) and no cue interaction when one cause precedes two effects (1C–2E). However, only causal model theory predicts the pattern of results obtained in Experiment 1, in which a cue interaction effect occurs when one effect precedes two causes (1E–2C) and no cue interaction occurs when two effects precede one cause (2E–1C). Note, however, that ratings of *A* in the presence of a perfect predictor are lower in the 2E–1C scenario than in the 1C–2E scenario. Similarly, ratings of *A* in the presence of a perfect predictor are lower in the 2C–1E scenario than in the 1E–2C scenario. According to causal model theory, when two effects precede a single cause (2E–1C), there should be no difference between ratings of *A* when *B* is perfectly predictive or nonpredictive, and these ratings should not differ from those in the 1C–2E scenario. In contrast, when one effect precedes two causes (1E–2C), there *should* be a difference between ratings of *A* when *B* is perfectly predictive or nonpredictive, and these ratings should not differ from those in the 2C–1E scenario. The data indicate, however, that when the effects come first, the influence of the causal model seems to lessen, or perhaps, the influence of an associative mechanism may increase. We will revisit this point in the Discussion section of Experiment 2.

EXPERIMENT 2

The data provided in Experiment 1 indicate that participants' overall ratings are sensitive to the causal structure of events. Following the suggestion that causal order and the number of cues and outcomes had been confounded in previous investigations of cue interaction, four causal scenarios were tested in Experiment 1, in which a moderately positive contingency ($\Delta P_A = .5$) was paired with either a zero contingency ($\Delta P_B = 0$) or a perfect contingency ($\Delta P_B = 1$). Experiment 2 was designed to replicate the results from Experiment 1 and to generalize from the extreme contingencies used to less extreme values, by including three intermediate ΔP_B values (.25, .5, .75). The ΔP_B values chosen for the three intermediate contingency pairs were selected to best contrast the predictions made by the Rescorla–Wagner model and causal model theory through participants' ratings of *A* and were not chosen for their intrinsic value. To clarify, several different frequencies can be selected to fill the eight cells of the 4×2 matrix, each resulting in various combinations of unconditional and conditional ΔP values. The frequencies for Experiment 2 (shown in Table 2) were selected to produce a descending pattern of conditional ΔP_A values while maintaining identical unconditional ΔP_A values. As well, they were selected so that the unconditional and the conditional ΔP_B values would be as closely matched as possible. The ΔP_B values were, therefore, selected only for their influence on the conditional ΔP_A values. The frequencies were also selected so that the respective conditional contingencies for *A* and *B* would be identical, where $\Delta P_{A|B} = \Delta P_{A|\sim B}$ and $\Delta P_{B|A} = \Delta P_{B|\sim A}$, resulting in the symmetry observed in the two columns of the 4×2 contingency matrix for each of the five conditions (see Spellman, 1996b, Property 4).

In addition, Experiment 2 was designed to independently test each of the four causal scenarios. In Experiment 1, half of the participants were presented with both the 2C–1E and the 1C–2E scenarios, and the other half were presented with the 2E–1C and the 1E–2C scenarios. In Experiment 2, however, each group was presented with only one causal scenario (2C–1E, 2E–1C, 1C–2E, or 1E–2E).

Method

Participants and Design

Sixty undergraduate students at McMaster University participated for course credit. The experiment was designed as a replica-

Table 2
Frequency of Events in Experiments 2 and 3

	$\Delta P_A/\Delta P_B$				
Trial Type	.5/0	.5/.25	.5/.5	.5/.75	.5/1
ABO	6	8	10	11	12
A~BO	6	4	2	1	0
~ABO	2	2	2	3	4
~A~BO	2	2	2	1	0
AB~O	2	2	2	1	0
A~B~O	2	2	2	3	4
~AB~O	6	4	2	1	0
~A~B~O	6	8	10	11	12
Total trials	32	32	32	32	32
ΔP_A	.5	.5	.5	.5	.5
$\Delta P_{A\|B}$	.5	.47	.33	.17	0
$\Delta P_{A\|\sim B}$	.5	.47	.33	.17	0
ΔP_B	0	.25	.5	.75	1
$\Delta P_{B\|A}$	0	.13	.33	.67	1
$\Delta P_{B\|\sim A}$	0	.13	.33	.67	1

Note—Unconditional ΔP values were calculated using Equations 1 and 2. Conditional ΔP values were calculated using Equations 3–6. For further explanation, see text.

tion of Experiment 1, using five contingency pairs rather than two, casual scenario as a between factor, and a total of 32, rather than 48, trials with a single overall rating. The 60 participants were randomly assigned to one of the four causal scenarios (i.e., 2C–1E, 1C–2E, 2E–1C, or 1E–2C). Within each group, the presentation order of the five ΔP_B values was randomized. Table 2 illustrates the trial frequencies obtained by combining $\Delta P_A = .5$, with each of the five ΔP_B values.

Procedure and Materials

The procedure and materials in Experiment 2 were very similar to those in Experiment 1. The difference was in the total number of trials and the number of ΔP_B values. The participants were presented with 32 trials before rating Events *A* and *B*, where they would repeat the process after observing each of the five "experiments." Two more fictitious chemicals and one more bacterial strain were added among those to be presented.

Results and Discussion

Mean ratings of Event *A* are illustrated in Figure 4A. Ratings for each of the four causal scenarios are plotted as a function of the five ΔP_B values. According to causal model theory, ratings of *A* in the 2C–1E and 1E–2C scenarios should track the pattern of conditional ΔP_A values presented in Table 2. The conditional ΔP_A values decrease as ΔP_B increases, and therefore, ratings of *A* should also decrease. Causal model theory also predicts that the ratings of *A* in the 1C–2E and 2E–1C scenarios should track the pattern of unconditional ΔP_A values presented in Table 2. The unconditional ΔP_A values are constant, and therefore, the ratings of *A* should not change across the five ΔP_B values. The Rescorla–Wagner model makes similar predictions, but for different scenarios: Ratings of *A* should be a decreasing function of ΔP_B for the 2C–1E and 2E–1C scenarios and should be independent of ΔP_B for the 1C–2E and 1E–2C scenarios. As we noted above, the predictions for both models are ordinal. Thus, we are examining not only the presence or absence of cue interaction, but also the ordinal level of cue interaction among the four causal scenarios.

The ratings of *A* appear to support the predictions made by causal model theory. In the 2C–1E and 1E–2C scenarios, ratings of *A* decline as ΔP_B increases, tracking the pattern of conditional ΔP_A values presented in Table 2. In the 2E–1C and 1C–2E scenarios, ratings of *A* remain relatively constant regardless of the contingency for Event *B*, tracking the pattern of unconditional ΔP_A values presented in Table 2.

With four causal scenarios (2C–1E, 1C–2E, 2E–1C, and 1E–2C) as a between factor and five ΔP_B values (0, .25, .5, .75, and 1) as a within factor, a mixed ANOVA was conducted on the ratings of *A*. As was expected, the analysis revealed main effects of causal scenario [$F(3,56) = 14.83$, $MS_e = 2{,}098.34$] and ΔP_B [$F(4,224) = 13.32$, $MS_e = 1{,}348.32$], as well as a significant interaction between them [$F(12,224) = 3.98$, $MS_e = 1{,}348.32$]. The Tukey test was used to examine this significant interaction in order to see whether the results replicated those found in Experiment 1. The ratings of *A* for the two ΔP_B values used in Experiment 1 ($\Delta P_B = 0$ and $\Delta P_B = 1$) were compared, and the ratings were not significantly different among the four causal scenarios when $\Delta P_B = 0$. Also, these ratings did not differ from the ratings when $\Delta P_B = 1$ if one cause produced two effects (1C–2E and 2E–1C). In contrast, when two causes produced a single effect (2C–1E and 1E–2C) and $\Delta P_B = 1$, the ratings were significantly lower than the other ratings and did not differ from each other. Thus, the ratings of *A* in Experiment 2 provide a replication of the Experiment 1 results. Cue interaction occurs when two causes result in one effect, regardless of whether the causes precede or follow the effect, and cue interaction does not occur when a single cause results in two effects, regardless of their causal order.

According to causal model theory, ratings of *A* should decrease as ΔP_B increases when two causes produce one effect (2C–1E and 1E–2C) and should remain constant when one cause produces two effects (1C–2E and 2E–1C). A linear trend analysis was conducted on the *A* ratings, separately for each scenario, across the five ΔP_B values.[1] As is predicted by causal model theory, the linear trend was significant for the 2C–1E [$F(1,56) = 40.10$] and the 1E–2C [$F(1,56) = 29.60$] scenarios and was not significant for the 1C–2E [$F(1,56) = 0.09$] and 2E–1C [$F(1,56) = 0.74$] scenarios ($MS_e = 1{,}751.69$ for each comparison).

Mean ratings of Event *B* are illustrated in Figure 4B. The ratings of *B* clearly increase with ΔP_B. Table 2 indicates that for .5/.25, .5/.5, and .5/.75, the conditional values of ΔP_B are less than the unconditional values. Thus, according to causal model theory, ratings of *B* when two causes produce one effect (2C–1E and 1E–2C) should be less than when one cause produces two effects (1C–2E and 2E–1C). Although the data tend in that direction, the statistical analysis indicated that the scenario effect was not significant. With ratings of *B* as the dependent measure, a mixed ANOVA revealed only a significant main

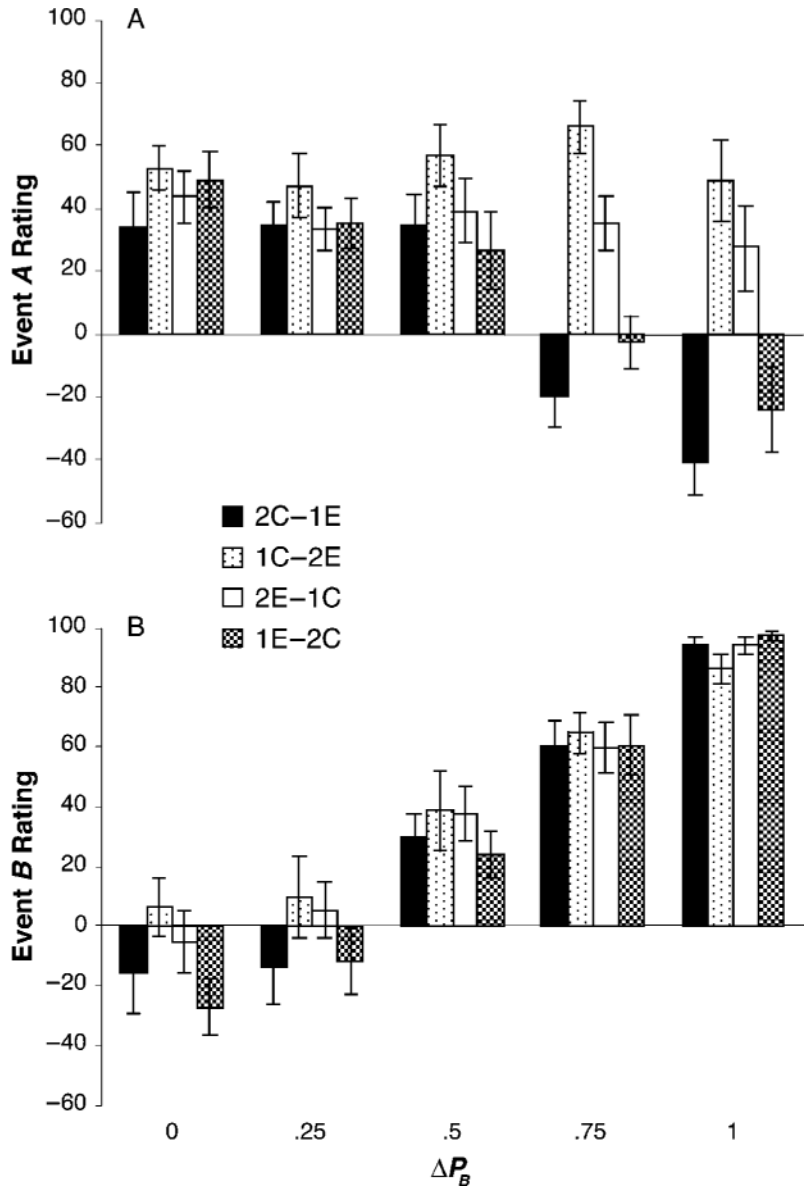

Figure 4. Mean ratings in Experiment 2 after 32 trials of (A) Event *A* and (B) Event *B*. For each event, the ratings are shown as a function of ΔP_B (0, .25, .5, .75, or 1) separately for each of the four scenarios. Error bars represent standard errors of the means.

effect of ΔP_B [$F(4,224) = 107.84, MS_e = 1{,}047.12$]. The main effect of causal scenario was not significant [$F(3,56) = 1.16, MS_e = 2{,}296.1$], nor was the interaction between causal scenario and ΔP_B [$F(12,224) = 0.89$, $MS_e = 1{,}047.13$]. To evaluate whether the absence of a significant scenario effect was attributable to the cases in which the conditional and the unconditional values of ΔP_B were the same (.5/0 and .5/1), an ANOVA was conducted on the three other pairings (.5/.25, .5/.5, and .5/.75). Again, only the main effect of ΔP_B was significant [$F(2,112) = 50.51, MS_e = 1{,}223.04$].

In summary, the ratings of *A* in Experiment 2 provide a direct replication of the ratings in Experiment 1 and generalize the results to less extreme ΔP_B values. When two causes produced one effect, the participants rated the moderately positive cause as less predictive when it was paired with a strong predictor than when it was paired with a weak predictor. When a single cause produced two effects, the participants rated the moderately positive effect as equally diagnostic, regardless of the diagnosticity of the effect that it was paired with. This interaction between causal order and the number of cues and outcomes is consistent with the predictions of causal model theory. Although not statistically significant, the ratings of *B* were also consistent with causal model theory. It must be emphasized that the *B* ratings do not pro-

vide a strong assessment of the models, because the ΔP_B values were selected only for their influence on the conditional ΔP_A values.

As in Experiment 1, the causal model effect was not as strong when the effect(s) preceded the cause(s). In Experiment 2, we see that ratings of *A* were consistently lower in the 2E–1C scenario than in the 1C–2E scenario. Similarly, ratings of *A* tended to be lower in the 2C–1E scenario than in the 1E–2C scenario. Again, although the differences are not significant, when the effect(s) precede the cause(s), participants' ratings seem to be influenced less by the causal description of the events and more by their associative strength. Although the data from Experiments 1 and 2 provide conclusive evidence that participants' judgments are driven primarily by the structure of the causal relationship, we will demonstrate the significant role of associative processes in the following two experiments.

EXPERIMENT 3

A conditional ΔP account applied to causal model theory allows one to generate dichotomous predictions in which cue interaction should occur or not (as has been done in previous investigations), but in addition, it allows for ordinal predictions where the relative effectiveness of each event determines the degree to which they interact. The data from Experiments 1 and 2 provide solid evidence for the influence of causal expectation on human inference. In Experiment 3, we demonstrate that these high-level processes may not occur independently of basic low-level (associative) processes, by exploring a different measure of causal assessment.

In Experiments 1 and 2, the participants passively viewed a series of trials before providing an overall rating of the relationship between the events. Our methodology differs from that reported by others (e.g., Cobos, López, Caño, Almaraz, & Shanks, 2002; Price & Yates, 1995; Shanks & López, 1996; Waldmann & Holyoak, 1992), who required participants to predict the outcome of each trial and provided corrective feedback on their predictions. For example, on each trial, the participants in Experiment 1 of Waldmann and Holyoak (1992) would see descriptions of people on a computer screen and were to use those descriptions to predict whether they thought a person had the described disease (by pressing a *Yes* key) or did not have the disease (by pressing a *No* key). After indicating their response, they received *correct* or *incorrect* as feedback. If participants are presented with four types of event combinations (*AB*, *A*~*B*, ~*AB*, ~*A*~*B*) and are asked to predict the outcome of each trial (*Yes* or *No*), a 4 × 2 matrix, such as the one presented in Figure 1, can be constructed in which the columns represent the two prediction responses (*Yes* or *No*), rather than the actual outcomes. These predictions can then be used as an indirect measure of their conditional ΔP estimates (López, Shanks, Almaraz, & Fernandez, 1998; Tangen & Allan, 2003).

We have shown in Experiments 1 and 2 that participants demonstrate a sensitivity to the structure of causal relationships that is consistent with the predictions made by causal model theory. To further investigate the participants' sensitivity to causal structure, we required participants in Experiment 3 to predict the outcome of each trial, in addition to providing an overall rating of the relationship between the events. Thus, we obtained both a measure of causal assessment derived from prediction responses and explicit overall judgments between the events, to determine whether the two measures were congruent as we varied the structure of the causal relationship.

Among the four causal scenarios described earlier (2C–1E, 1C–2E, 2E–1C, and 1E–2C), the results from Experiments 1 and 2 revealed that neither causal order nor the number of cues and outcomes were significant factors independently. Instead, the important variable was the interaction between the two factors—that is, the structure of the causal relationship. Therefore, to avoid the potential confound of the number of predictions the participants were making on each trial, we eliminated the right-hand column of Figure 2 and presented them with only two cues and one outcome (2C–1E and 2E–1C). Each group was shown identical stimuli, but the causal description of the stimuli differed between the two groups. According to causal model theory, judgments should vary depending on whether the events are described as two causes resulting in an effect or as two effects resulting from one cause. In contrast, the Rescorla–Wagner model does not make a distinction between the causal description of the events and codes the two scenarios identically as two cues followed by one outcome. On each trial, a participant was presented with one of four event combinations (*AB*, *A*~*B*, ~*AB*, ~*A*~*B*) and then predicted whether the effect/cause occurred, given the information from the preceding pair of events and from previous trials. Corrective feedback (*correct* or *incorrect*) was provided immediately after their decisions had been made. After 32 trials, they were asked to provide an overall rating of the relationship between the events, as in the previous experiments. The same five contingency pairs were used as those in Experiment 2.

Method

Participants and Design

Thirty undergraduate students at McMaster University took part in this experiment for course credit. The design of Experiment 3 was identical to that in Experiment 2, except that the 1C–2E and 1E–2C causal scenarios were eliminated and the participants were asked to predict the outcome of each trial and were provided feedback on their decisions. The frequency of events in Experiment 3 are shown in Table 2.

Procedure and Materials

The same procedure and materials as those in Experiment 2 were used, with the addition of predictions on each trial. The participants were presented with two cues consisting of the presence or absence of two chemicals (2C or 2E) and were then asked to predict whether they thought the bacterial strain survived/was added or not by click-

ing one of two buttons on the computer screen. Once they had made their selection, they were presented with the outcome (1E or 1C) along with *correct* or *incorrect* as feedback. The prediction responses for each event combination were recorded and used to calculate estimated conditional ΔP values by counting the number of *yes* and *no* responses for each event combination (AB, $A\sim B$, $\sim AB$, or $\sim A\sim B$) after 16, 32, 48, and 64 trials and substituting these frequencies into Equations 3–6.

Results

In this experiment there were two dependent measures, ratings and predictions.

Ratings

Figures 5A and 5B depict the mean ratings for Cues *A* and *B*, respectively. The pattern of results for both cues was similar to that observed in Experiment 2. Ratings of *A*, in the 2C–1E scenario, decline as ΔP_B increases, tracking the pattern of conditional ΔP_A values presented in Table 2. Ratings of *A*, in the 2E–1C scenario, remain relatively constant as ΔP_B increases, tracking the pattern of unconditional ΔP_A values presented in Table 2. With ratings of *A* as the dependent variable, a mixed ANOVA, with causal scenario (2C–1E vs. 2E–1C) as a between factor and ΔP_B (0, .25, .5, .75, and 1) as a within factor, revealed significant main effects for scenario [$F(1,28) = 8.03$, $MS_e = 4{,}048.77$] and ΔP_B [$F(4,112) = 5.21$, $MS_e = 1{,}845.58$], as well as a significant interaction [$F(4,112) = 2.95$, $MS_e = 1{,}845.58$]. As in Experiment 2, the linear trend was significant for the 2C–1E scenario [$F(1,28) = 23.80$], but not for the 2E–1C scenario [$F(1,28) = 0.66$ ($MS_e = 2{,}338.93$ for both comparisons)].

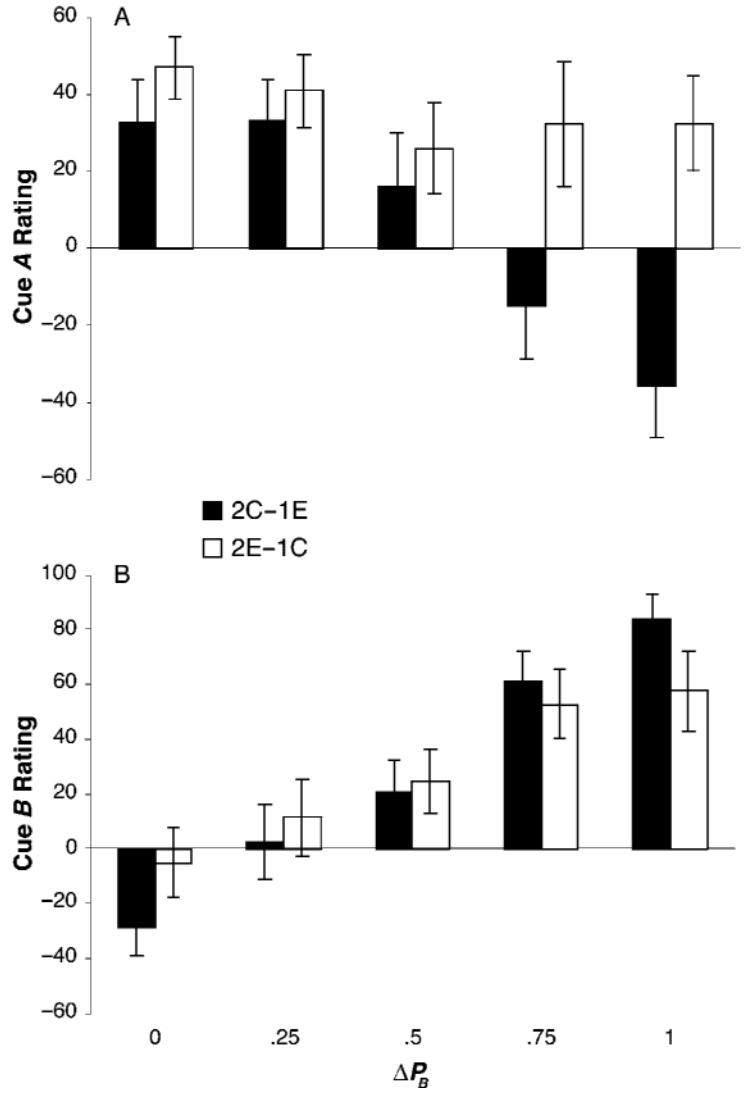

Figure 5. Mean ratings in Experiment 3 after 32 trials of (A) Cue *A* and (B) Cue *B*. For each cue, the ratings are shown as a function of ΔP_B (0, .25, .5, .75, or 1) separately for each of the two scenarios. Error bars represent standard errors of the means.

Figure 5B indicates that the ratings of B increase with ΔP_B and do not appear to depend on causal scenario. With ratings of B as the dependent measure, a mixed ANOVA revealed a significant main effect of ΔP_B [$F(4,112) = 18.59$, $MS_e = 2{,}069.06$]. The main effect of causal scenario was not significant [$F(1,28) = 0.001$, $MS_e = 3{,}001.04$], nor was the interaction between contingency and causal scenario [$F(4,112) = 1.26$, $MS_e = 2{,}069.06$].

Predictions

Figures 6A and 6B plot the estimated ΔP values for Cue A conditional on the presence and absence of B, respectively. It is clear from these two figures that the participants' prediction responses are at variance with their ratings. A comparison of the two figures also indicates that the estimates of $\Delta P_{A|B}$ are different from the estimates of $\Delta P_{A|\sim B}$. A 2 (scenario: 2C–1E or 2E–1C) × 2 (Cue B status: present or absent) × 5 (ΔP_B: 0, .25, .5, .75, or 1) mixed ANOVA on the estimated conditional ΔP values for A confirms these observations. The main effect of causal scenario was not significant [$F(1,28) = 1.25$, $MS_e = 1{,}201.76$], nor did it interact with ΔP_B [$F(4,112) = 1.00$, $MS_e = 1{,}088.09$], Cue B status [$F(1,28) = 2.54$, $MS_e = 1{,}357.18$], or both [$F(4,112) = 0.9$, $MS_e = 603.56$]. The main effect of ΔP_B was significant [$F(4,112) = 3.46$, $MS_e = 1{,}088.09$]. The main effect of Cue B status was also significant [$F(1,28) = 9.91$, $MS_e = 1{,}357.18$], indicating that estimated conditional ΔP for A was lower when B was present (est$\Delta P_{A|B} = .24$) than when it was absent (est$\Delta P_{A|\sim B} = .38$). The interaction between ΔP_B and Cue B status was not significant [$F(4,112) = .54$, $MS_e = 603.56$].

Figures 6C and 6D illustrate the estimated ΔP values for B, conditional on the presence and absence of A, respectively. A 2 (scenario: 2C–1E or 2E–1C) × 2 (Cue A status: present or absent) × 5 (ΔP_B: 0, .25, .5, .75, or 1) mixed ANOVA on the estimated conditional ΔP values for B revealed a main effect of ΔP_B [$F(4,112) = 31.26$, $MS_e = 1{,}137.41$]. The main effect of Cue A status was also significant [$F(1,28) = 10.06$, $MS_e = 1{,}349.84$], indicating that estimated conditional ΔP for B was lower when A was present (est$\Delta P_{B|A} = .31$) than when it was absent (est$\Delta P_{B|\sim A} = .44$). No other effects or interactions reached significance.

Discussion

The results from Experiment 3 provide a direct replication of the rating data obtained in Experiments 1 and 2. The participants rated identical contingencies quite differently depending on whether the events had been described as causes or effects. In the 2C–1E scenario, the participants gave lower ratings to the moderately predictive Cause A when it was paired with a highly predictive

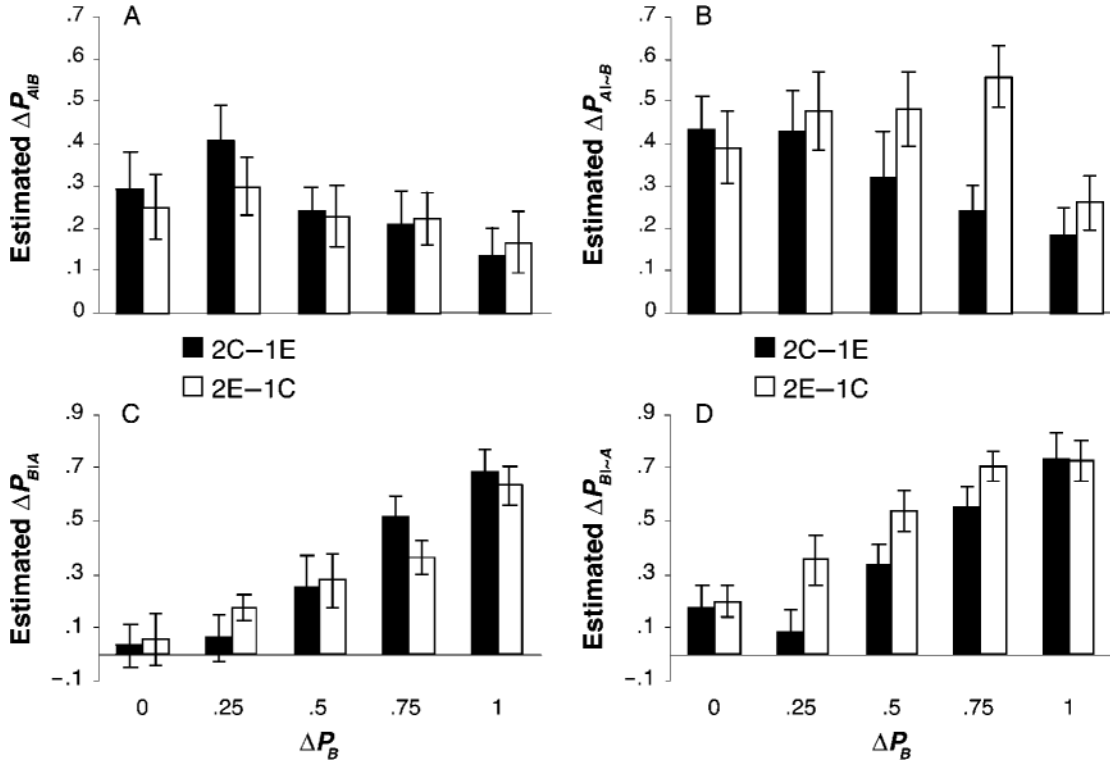

Figure 6. Mean estimated conditional ΔP values in Experiment 3 for Cues A and B as a function of ΔP_B separately for the two causal scenarios: (A) est$\Delta P_{A|B}$, (B) est$\Delta P_{A|\sim B}$, (C) est$\Delta P_{B|A}$, and (D) est$\Delta P_{B|\sim A}$. Error bars represent standard errors of the means.

Cause *B* than when it was paired with a less predictive Cause *B*, indicating that causes interact. In contrast, in the 2E–1C scenario, the ratings of Effect *A* did not depend on the contingency of Effect *B*, indicating that effects do not interact

In contrast to the ratings, a causal scenario effect was not seen with the prediction responses. For both 2C–1E and 2E–1C, the estimated conditional ΔP values for *A* decreased as unconditional ΔP_B increased, indicating that cue interaction occurred in both scenarios. There appears to be a dissociation between the ratings and the prediction responses. Table 2 shows that for each cue, the two conditional ΔP values were always the same—that is, $\Delta P_{A|B} = \Delta P_{A|\sim B}$ and $\Delta P_{B|A} = \Delta P_{B|\sim A}$. This was not the case, however, for the estimates based on the participants' predictions, where $\text{est}\Delta P_{A|B} < \text{est}\Delta P_{A|\sim B}$ and $\text{est}\Delta P_{B|A} < \text{est}\Delta P_{B|\sim A}$. That is, the estimated conditional ΔP value was smaller when the cue conditionalized upon was present than when it was absent. This pattern of results was also found by Tangen and Allan (2003).

In summary, identical stimuli were presented to the participants, which were described either as two causes of an effect (2C–1E) or as two effects of a cause (2E–1C). The participants' overall judgments of these relationships varied systematically depending on their causal labels. In addition to making an overall judgment of the relationship, they were asked to make a prediction as to the whether the outcome would occur or not on each trial. Their prediction responses did not vary according to the causal description of the events.

We have revealed a dissociation between two means of assessing judgments of causality. Trial-by-trial prediction responses require participants to estimate the presence or absence of the outcome. The results suggest that participants manage this task by simply basing their judgments on the current level of associative strength, identifying cues as generic events without any deeper recognition of their causal status. Overall ratings, on the other hand, require participants to not only consider the status of a single outcome, but also take into account the causal relationship among the events presented.

Thus, it seems either that participants can report the current level of associative strength in their predictions by basing their causal assessments on the number of cues and outcomes, rather than on the causal structure of the events, or that their assessments can reflect the causal status of the events by taking into consideration how they are structured. It depends on the nature of the question being asked.

EXPERIMENT 4

The results from Experiment 3 revealed that the participants were sensitive to the causal description of the cues and outcome in rating the overall relationship but that the effect was absent in their trial-by-trial predictions. Experiment 4 was designed to further investigate this dissociation between ratings and prediction responses by increasing the total number of trials in each condition from 32 to 64 and having the participants provide an overall rating after 16, 32, 48, and 64 trials. By increasing the number of ratings, we could compare each measure across trials as a function of causal scenario. Perhaps a greater number of trials would result in a greater sensitivity to the associative processes at work and less sensitivity to the causal description of the events. Increasing the total number of trials resulted in the elimination of the .5/.5 contingency pair in order to maintain a 1-h experimental session.

Method

Participants and Design

Forty undergraduate students at McMaster University took part in this experiment for course credit. The design of Experiment 4 was similar to that in Experiment 3, except that the total number of trials was increased to 64, the participants were asked to rate each cue after 16, 32, 48, and 64 trials, and the .5/.5 contingency pair was eliminated. The event frequencies in Experiment 4 are shown in Table 3.

Procedure and Materials

The procedure and materials for Experiment 4 were similar to those in Experiment 3 apart from the total number of trials presented and the number of ratings provided by the participants. Four contingency pairs were presented to the participants as separate "experiments."

Results

As in Experiment 3, there were two dependent measures, ratings and predictions.

Ratings

Figures 7A and 7B depict the mean ratings after 64 trials for Cues *A* and *B* respectively, and Table 4 depicts the mean and standard error of the ratings for Cue *A* after

Table 3
Frequency of Events in Experiment 4

	$\Delta P_A/\Delta P_B$			
Trial Type	.5/0	.5/.25	.5/.75	.5/1
ABO	12	16	22	24
A~BO	12	8	2	0
~ABO	4	4	6	8
~A~BO	4	4	2	0
AB~O	4	4	2	0
A~B~O	4	4	6	8
~AB~O	12	8	2	0
~A~B~O	12	16	22	24
Total Trials	64	64	64	64
ΔP_A	.5	.5	.5	.5
$\Delta P_{A\|B}$	.5	.47	.17	0
$\Delta P_{A\|\sim B}$	.5	.47	.17	0
ΔP_B	0	.25	.75	1
$\Delta P_{B\|A}$	0	.13	.67	1
$\Delta P_{B\|\sim A}$	0	.13	.67	1

Note—Unconditional ΔP values were calculated using Equations 1 and 2. Conditional ΔP values were calculated using Equations 3–6. For further explanation, see text.

32, 48, and 64 trials. The ratings and estimated ΔP values after 16 trials are not reported, since the participants' prediction responses of the randomly presented events occasionally resulted in 4 × 2 matrices with row frequencies of zero. The pattern of results after 32 trials is similar to that in Experiments 1, 2, and 3. In the 2C–1E scenario, ratings of *A* roughly approximated the conditional ΔP values presented in Table 3, whereas in the 2E–1C scenario the ratings were consistent with the unconditional ΔP values. After 48 and 64 trials, however, a different pattern of results emerged. As is illustrated in Figure 7A, ratings of *A* declined as ΔP_B increased, regardless of the causal scenario. The effect of the causal model seems to have dissipated over trials, and cue interaction occurred for both scenarios. A 2 (scenario: 2C–1E or 2E–1C) × 4 (ΔP_B: 0, .25, .75, or 1) × 3 (trials: 32, 48, or 64) mixed ANOVA on the ratings of *A* revealed only a significant main effect for ΔP_B [$F(3,114) = 21.27$, $MS_e = 3{,}189.24$], which contributed to significant interactions with scenario [$F(3,114) = 3.33$, $MS_e = 3{,}189.24$] and trial [$F(6,228) = 2.60$, $MS_e = 1{,}397.89$], and a three-way interaction with trial and scenario [$F(6,228) = 3.13$, $MS_e = 1{,}397.89$]. A linear trend analysis[2] was conducted on the *A* ratings, separately for each scenario after 32, 48, and 64 trials. For the 2C–1E scenario, the linear trend was significant after 32 trials [$F(1,38) = 13.59$, $MS_e = 2{,}715.31$], 48 trials [$F(1,38) = 19.22$, $MS_e = 2{,}703.87$], and 64 trials [$F(1,38) = 37.95$, $MS_e = 2{,}482.45$]. For the 2E–1C scenario, the linear trend was not significant after 32 trials [$F(1,38) = 2.87$, $MS_e = 2{,}715.31$] but was sig-

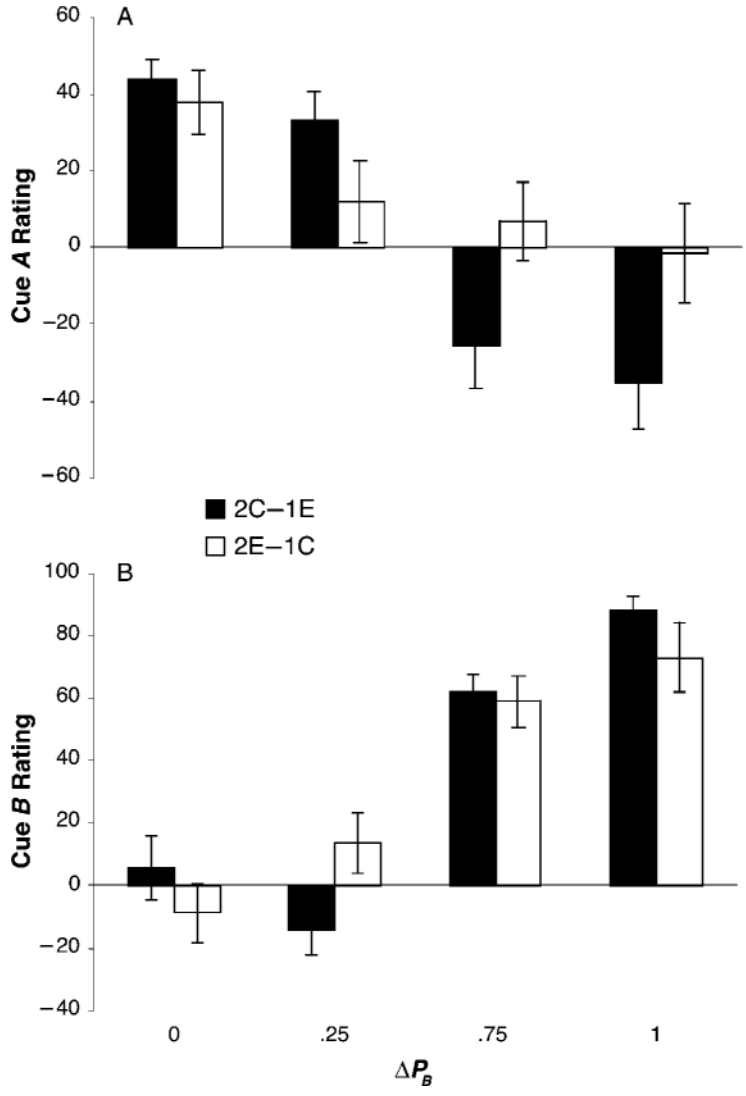

Figure 7. Mean ratings in Experiment 4 after 64 trials of (A) Event *A* and (B) Event *B*. For each event, the ratings are shown as a function of ΔP_B (0, .25, .75, or 1) separately for each of the two conditions. Error bars represent standard errors of the means.

Table 4
Experiment 4 Overall Ratings and Estimated Conditional ΔP Values for Cue *A* After 32, 48, and 64 Trials

No. Trials	Measure	$\Delta P_A/\Delta P_B$ .5/0 *M*	.5/0 *SEM*	.5/.25 *M*	.5/.25 *SEM*	.5/.75 *M*	.5/.75 *SEM*	.5/1 *M*	.5/1 *SEM*
					2C–1E				
32	Rating	37.7	7.6	14.8	8.4	30.7	13.2	−38	11
	est$\Delta P_{A\|B}$	.41	.07	.27	.06	.17	.07	.18	.07
	est$\Delta P_{A\|\sim B}$	.44	.1	.52	.06	.31	.08	.24	.06
48	Rating	29.1	9.6	24	8.6	10.8	10.7	−32.9	12.3
	est$\Delta P_{A\|B}$	.49	.06	.32	.07	.16	.06	.1	.04
	est$\Delta P_{A\|\sim B}$	.47	.09	.51	.07	.26	.05	.11	.04
64	Rating	44	5.1	33.3	7.1	−25.4	11.1	−35.3	11.9
	est$\Delta P_{A\|B}$	.51	.06	.35	.07	.17	.05	.08	.03
	est$\Delta P_{A\|\sim B}$	.51	.07	.47	.06	.23	.04	.08	.02
					2E–1C				
32	Rating	33.6	9.5	26	10.3	5.7	11.7	11.3	14
	est$\Delta P_{A\|B}$	.46	.06	.38	.07	.17	.09	.11	.05
	est$\Delta P_{A\|\sim B}$	.55	.08	.56	.08	.35	.07	.3	.08
48	Rating	32.4	8.8	36.7	9	−12.4	10.1	−3.15	13
	est$\Delta P_{A\|B}$	.47	.06	.45	.06	.14	.07	.09	.04
	est$\Delta P_{A\|\sim B}$	.6	.07	.57	.06	.3	.06	.19	.05
64	Rating	37.7	8.4	12.1	10.7	6.8	10.3	−1.5	12.9
	est$\Delta P_{A\|B}$	.5	.05	.46	.06	.15	.06	.08	.04
	est$\Delta P_{A\|\sim B}$	.58	.07	.59	.06	.31	.05	.17	.04

nificant after 48 trials [$F(1,38) = 11.05, MS_e = 2{,}703.87$] and 64 trials [$F(1,38) = 4.70, MS_e = 2{,}482.45$]. Thus, by 48 trials, cue interaction was seen in both scenarios.

Figure 7B presents the mean and standard error of the ratings for Cue *B* after 64 trials, and Table 5 presents the mean and standard error of the ratings for Cue *B* after 32, 48, and 64 trials. Ratings of *B* seem fairly typical of the results obtained in Experiments 1–3. Mean ratings increased for both scenarios as a function of ΔP_B. A 2 (scenario: 2C–1E or 2E–1C) × 4 (ΔP_B: 0, .25, .75, or 1) × 3 (trials: 32, 48, or 64) mixed ANOVA on the ratings of *B* confirmed this observation. The only signifi-

Table 5
Experiment 4 Overall Ratings and Estimated Conditional ΔP Values for Cue *B* After 32, 48, and 64 Trials

No. Trials	Measure	$\Delta P_A/\Delta P_B$ .5/0 *M*	.5/0 *SEM*	.5/.25 *M*	.5/.25 *SEM*	.5/.75 *M*	.5/.75 *SEM*	.5/1 *M*	.5/1 *SEM*
					2C–1E				
32	Rating	−22.1	7.5	−0.7	8.8	61.5	6.3	84.3	5.7
	est$\Delta P_{B\|A}$	.12	.06	.08	.05	.44	.08	.68	.05
	est$\Delta P_{B\|\sim A}$	.15	.07	.33	.08	.58	.09	.75	.07
48	Rating	−10.5	11.2	9.3	9.5	46.1	10.7	90.7	4
	est$\Delta P_{B\|A}$	.15	.04	.11	.04	.5	.06	.79	.02
	est$\Delta P_{B\|\sim A}$	.12	.06	.3	.09	.6	.07	.8	.06
64	Rating	5.6	10.4	−14.6	7.9	62.1	5.3	87.8	5.2
	est$\Delta P_{B\|A}$	.12	.03	.14	.04	.56	.05	.83	.03
	est$\Delta P_{B\|\sim A}$	.12	.05	.26	.07	.61	.07	.83	.05
					2E–1C				
32	Rating	−5.6	9.2	−3.8	10.2	49.9	11.1	81.1	8.6
	est$\Delta P_{B\|A}$	.03	.06	.1	.06	.46	.08	.61	.07
	est$\Delta P_{B\|\sim A}$	.12	.08	.28	.07	.63	.08	.8	.07
48	Rating	−2.1	8.5	15	8.9	62.8	8	69.4	11.9
	est$\Delta P_{B\|A}$	.01	.05	.1	.06	.54	.07	.73	.04
	est$\Delta P_{B\|\sim A}$	.14	.07	.22	.06	.7	.06	.83	.07
64	Rating	−8.8	9.5	13.6	9.4	58.9	8.1	73	1.9
	est$\Delta P_{B\|A}$	.03	.04	.09	.05	.54	.06	.77	.04
	est$\Delta P_{B\|\sim A}$	.11	.05	.22	.05	.7	.06	.85	.06

cant main effect was for ΔP [$F(3,114) = 77.99$, $MS_e = 2{,}760.85$]. The only other significant effect was a three-way interaction between ΔP_B, trial, and scenario [$F(6,228) = 3.55$, $MS_e = 747.98$], resulting primarily from an exceptionally low mean rating in the 2C–1E scenario, .5/.25 condition, after 64 trials.

Predictions

Figures 8A and 8B plot the estimated ΔP values for Cue *A* conditional on the presence and absence of *B*, respectively, computed after 64 trials. Table 4 also presents the estimated ΔP values for Cue *A* conditional on the presence and absence of *B*, and Table 5 also presents the estimated ΔP values for Cue *B* conditional on the presence and absence of *A*. The data are presented for each of the four contingency pairs after 32, 48, and 64 trials. The estimated ΔP data reported in Tables 4 and 5 correspond to the cumulative values recorded after a given number of trials, in that the 32-trial values are based on the first 32 trials, the 48-trial values are based on the first 48 trials, and the 64-trial values are based on all of the trials.

The mean estimated conditional ΔP values for *A* calculated after 32, 48, and 64 trials closely track the conditional ΔP values presented in Table 3 for both causal scenarios. Also, the estimated ΔP values conditional on the presence of *B* (est$\Delta P_{A|B}$) are lower than the estimated ΔP values conditional on the absence of *B* (est$\Delta P_{A|\sim B}$).

A 2 (scenario: 2C–1E or 2E–1C) × 4 (ΔP_B: 0, .25, .75, or 1) × 3 (trials: 32, 48, or 64) × 2 (Cue *B* status: present or absent) mixed ANOVA on the estimated values for *A* verifies these observations. The ΔP_B main effect was significant [$F(3,114) = 33.71$, $MS_e = 0.21$] and contributed to a significant interaction with trial [$F(6,228) = 9.39$, $MS_e = 0.01$]. The status of the Cue *B* main effect was also significant [$F(1,38) = 19.57$, $MS_e = 0.14$], indicating that the estimated conditional ΔP for *A* was lower when *B* was present (.28) than when it was absent (.38), and the significant Cue *B* status × trial interaction [$F(2,76) = 5.76$, $MS_e = 0.14$] indicates that this difference became less evident across trials. Linear trend analyses were conducted separately for the 2C–1E and the 2E–1C causal scenarios after 32, 48, and 64 trials, both on the estimated ΔP values conditional on the presence and absence of *B*. These analyses revealed a significant linear trend for the prediction responses in both causal scenarios after each of the three trial intervals (32, 48, or 64), regardless of the status of Cue *B*. Cue interaction is evident in the prediction responses regardless of the circumstances.

Figures 8C and 8D illustrate the estimated ΔP values for *B* conditional on the presence and absence of *A*, respectively, computed after 64 trials. An identical ANOVA was performed on the prediction response data for *B*, substituting Cue *A* status (present or absent) for Cue *B*

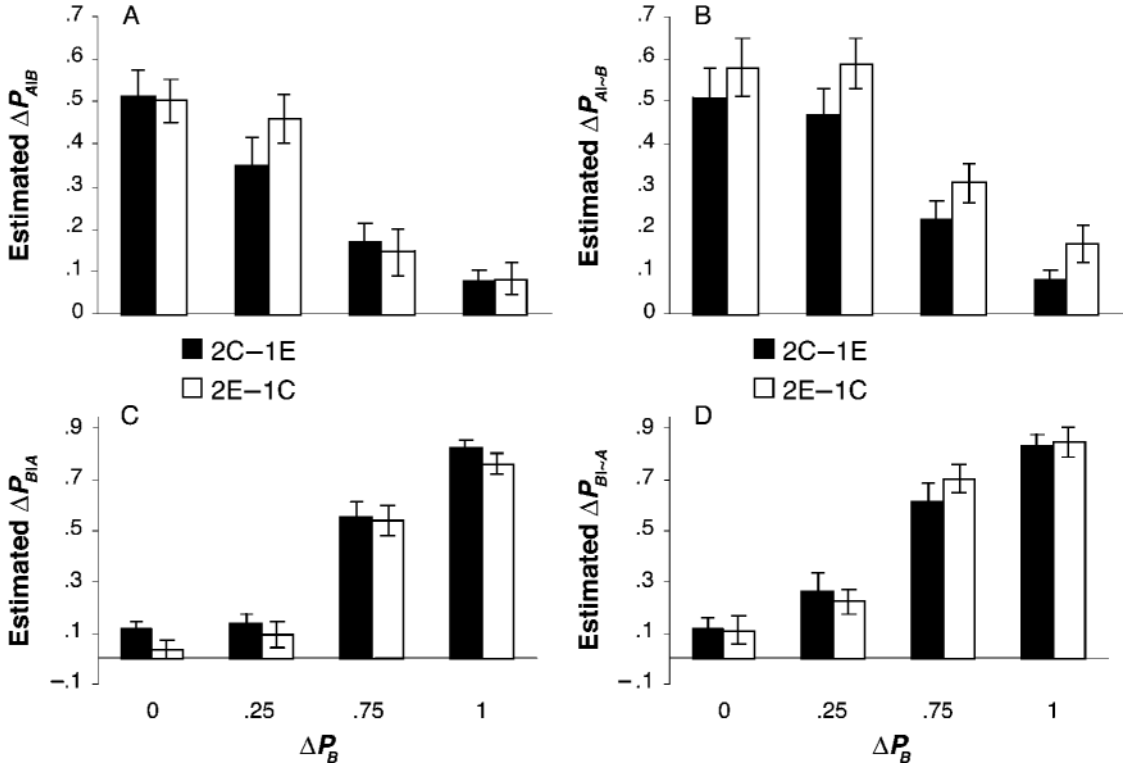

Figure 8. Mean estimated conditional ΔP values in Experiment 4 for Events *A* and *B* as a function of ΔP_B separately for the two causal scenarios after 64 trials: (A) est$\Delta P_{A|B}$, (B) est$\Delta P_{A|\sim B}$, (C) est$\Delta P_{B|A}$, and (D) est$\Delta P_{B|\sim A}$. Error bars represent standard errors of the means.

status. Resembling the data reported in Experiment 3, significant main effects were obtained for ΔP_B [$F(3,114) = 110.28, MS_e = 0.22$] and Cue *A* status [$F(1,38) = 19.57, MS_e = 0.15$]. In addition, the trial factor introduced in Experiment 4 was significant [$F(2,76) = 9.66, MS_e = 0.02$] and led to significant interactions with ΔP_B [$F(6,228) = 6.93, MS_e = 0.01$] and Cue *A* status [$F(2,76) = 5.76, MS_e = 0.02$]. As is indicated by the Cue *A* data, the estimated conditional ΔP values for *B* were lower when *A* was present (.36) than when it was absent (.46), and this difference became less evident across trials.

Discussion

The rating data from Experiment 4 are similar to those obtained in each of the previous experiments and have extended these findings to reveal an interesting scenario × trial interaction. Experiment 4 has shown that cue interaction is evident across the entire span of 64 trials when *A* and *B* are described as two causes of a single effect (2C–1E). When the causal labels are reversed, however, and *A* and *B* are described as two effects resulting from a single cause (2E–1C), we see a very different pattern of results across trials. As in each of the previous experiments, ratings of *A* reveal that cue interaction is not evident in the 2E–1C scenario at 32 trials. After 48 and 64 trials, the cue interaction effect becomes increasingly evident. After 64 trials, ratings of *A* in the 2E–1C scenario are clearly attenuated, as is indicated in Figure 7A. Although the trial × scenario data in Experiment 1 tended in the same direction as those in Experiment 4, the effect was not significant. This trial × scenario interaction may not have been evident in Experiment 1 between 32 and 48 trials because we compared the trend between two trial points (32 and 48), as opposed to three (32, 48, and 64) in Experiment 4. Other data collected in our lab suggest that the trial × scenario interaction is indeed robust (Sadeghi, 2003).

The prediction response values are estimated by separately calculating ΔP conditional on the presence and absence of the other cue. In both Experiments 3 and 4, estimated ΔP conditional on the present cue was significantly lower than estimated ΔP conditional on the absent cue—that is, $\text{est}\Delta P_{A|B} < \text{est}\Delta P_{A|\sim B}$ and $\text{est}\Delta P_{B|A} < \text{est}\Delta P_{B|\sim A}$. Although the conditional ΔP account has not explicitly addressed the relationship between estimated conditional ΔP and actual ΔP, one would expect them to be congruent, as is indicated by the identical conditional ΔP values presented in Table 3. Our data indicating that the estimated values are not congruent with the actual values might raise problems for the conditional ΔP account (see also Tangen & Allan, 2003).

In summary, Experiment 4 provides results similar to those in Experiments 1–3. Overall ratings were influenced by the causal description of the events after 32 trials. Trial-by-trial prediction responses, however, were not influenced by the causal description of the events. In addition, Experiment 4 demonstrates that on later trials, the participants become less sensitive to the difference in description of the two causal scenarios. These data support the argument that causal assessments are not driven solely by associative or causal model processes but, instead, seem be sensitive to both, depending on how and when they are obtained. After repeatedly making trial-by-trial predictions, participants may be disregarding the causal order of the events, which may be reflected in their overall causal ratings. By continually predicting the presence or absence of the outcome, it is likely that participants are treating the events less like causes and effects and more like cues and outcomes. As a consequence, on later trials, their causal assessments are based on the same associative strength as their trial-by-trial predictions.

GENERAL DISCUSSION

Price and Yates (1995) were among the first to suggest that both high- and low-level processes are used in causal assessments (see also Hagmayer & Waldmann, 2000, for a similar two-process position). There has been little work since then to explain the conditions under which these two processes are likely to be operating. Instead, there has been considerable debate between causal model and associative learning theorists as to which of the two theoretical interpretations is correct. The results from our experiments revisit the arguments made by Price and Yates as to the joint contribution of associative and causal factors in judgments of causality.

A similar approach has been taken recently by Collins and Shanks (2002) to account for primacy and recency effects. They described two strategies involved in judgments of causality: the momentary strategy, in which judgments simply reflect the current associative strength of the cue, and the integrative strategy, in which participants do not constrain their judgments on the current perception of the relationship but, instead, integrate information across a number of trials. Although Collins and Shanks were describing judgment strategies in primacy and recency effects, we believe that the same tactics are being used in judgments of causally asymmetric events. Participants are required to estimate the presence or absence of the outcome in their trial-by-trial prediction responses. They likely manage this task by identifying cues as generic events, without any deeper recognition of their causal status, thereby basing their judgments on the current level of associative strength. Overall ratings, on the other hand, require a more global (integrative) strategy, in which participants not only consider the status of a single outcome, but also take into account the causal structure of the events presented.

We have demonstrated that the contribution of causal and associative processes depends on what the participant is being asked about the events and on their experience with those events. Participants recognize that in order to assess the influence of a given cause, they must hold constant (conditionalize on) any alternative causes

(2C–1E). Conversely, they understand that a single cause can independently influence a number of effects (1C–2E). In associative terms, two cues compete to be associated with a single outcome. Conversely, one cue can be associated with a number of outcomes. These results are not surprising to anyone. In fact, both causal model theory and the Rescorla–Wagner model make these predictions. The question, then, is whether the events continue to interact or not when the order of the causal labels are reversed (2E–1C and 1E–2C, respectively). The Rescorla–Wagner model predicts that the events should be treated identically in either instance, and causal model theory predicts that the presence of a cue interaction effect should be reversed along with the causal labels.

Experiments 1 and 2 provide evidence that contingency ratings are influenced by the interaction between causal order (CE vs. EC) and the number of cues and outcomes (2–1 vs. 1–2), indicating that participants are sensitive to the structure of the causal relationship. In Experiment 3, we see that predictions, a second measure of causal assessment, are not so easily swayed by the causal structure of the stimuli. Even though participants assess the same causal relationship in either case, they account for the causal description of the events in one instance (i.e., ratings), but not in the other (i.e., predictions). Finally, in Experiment 4, we see that the relative weighting of causal and associative factors are influenced not only by the means of assessing causal inference (ratings and predictions), but also by the repeated exposure to the events. We cannot argue whether the repeated exposure to trial-by-trial predictions is influencing their causal judgments, or whether it is simply the result of additional trials, since these two factors were not tested independently. Regardless, most experiments that support an associative account use *both* a large number of trials and trial-by-trial predictions, which may explain the discrepant results. The relative contribution of each of these factors remains an open question.

We would expect that if participants were asked to describe how the causal events were interconnected or were required to use the causal model for some particular purpose, they would likely be more sensitive to the structure of the causal relationship than if they were asked to report the probability, covariation, or frequency of the events. Similarly, we might expect participants to consider the causal nature of the events more carefully if several types of causal relationships are presented, rather than repeatedly presenting just one. As is indicated by the results from Experiment 4, participants become less sensitive to the influence of the causal model in both their ratings and predictions as trials progress. One might expect that participants will disregard the causal order of the events if they are presented with a large number of trials. In fact, several experiments supporting an associative interpretation have shown just that. For example, Cobos et al. (2002) required participants to provide a single rating of each event after a learning phase that consisted of as many as 240 trials. Our data from Experiment 4 indicate that any causal model effect would be largely eliminated by then. Although there is no reason to expect the effect of the causal model to diminish over trials, it may be a step forward in understanding the circumstances with which we use them. We suggest that the number of trials presented to the participant is an important factor in determining their sensitivity to the structure of the causal relationship. In fact, many experiments that have provided support for causal model theory have used a smaller number of trials (e.g., Waldmann, 2000, 2001), as compared with those supporting an associative account (e.g., Cobos et al., 2002; Shanks & López, 1996). This finding may help explain much of the contradictory data in the literature.

Over the past decade, associative and causal model theorists have continued to debate whether or not human inferences are guided by causal interpretation. We have described specific circumstances that allow one to find one pattern of results or the other, and we provide evidence for an account in which the two processes operate in conjunction, rather than independently.

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NOTES

1. We are interested in whether there is a significant linear trend among the *A* ratings across the five levels of ΔP_B, tracking the conditional ΔP values for Event *A*. The interval between the levels of the independent variable are unequal (i.e., .5, .47, .33, .17, and 0), whereby the following coefficients were derived: 21, 17, 4, −13, and −29 (see Howell, 1997, for the derivation).

2. With only four levels of ΔP_B in Experiment 4, the following coefficients were derived to test for a linear trend tracking the ordinal pattern of the conditional ΔP values for *A*: 22, 18, −12, and −28.

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