Journal of Experimental Psychology:
Animal Behavior Processes
2002, Vol. 28, No. 4, 331–346

0097-7403/02/$5.00 DOI: 10.1037//0097-7403.28.4.331

Mechanisms of Predictive and Diagnostic Causal Induction

Pedro L. Cobos, Francisco J. López, Antonio Caño, and Julián Almaraz
Universidad de Málaga

David R. Shanks
University College London

In predictive causal inference, people reason from causes to effects, whereas in diagnostic inference, they reason from effects to causes. Independently of the causal structure of the events, the temporal structure of the information provided to a reasoner may vary (e.g., multiple events followed by a single event vs. a single event followed by multiple events). The authors report 5 experiments in which causal structure and temporal information were varied independently. Inferences were influenced by temporal structure but not by causal structure. The results are relevant to the evaluation of 2 current accounts of causal induction, the Rescorla–Wagner (R. A. Rescorla & A. R. Wagner, 1972) and causal model theories (M. R. Waldmann & K. J. Holyoak, 1992).

Knowledge about covariations between events is one of the most basic forms of inferential knowledge. It confers on its possessor a very powerful tool to reduce uncertainty about the occurrence of events. For example, an organism may predict the presence of future events from knowing whether a particular present event has occurred. Such inferential knowledge appears to be within the scope of numerous species, as the study of animal conditioning has revealed (Dickinson, 1980). Often, inferential learning takes place within a causal context. In such contexts, the relationships between the events are of a causal nature. In these cases, two different kinds of inference may be distinguished in terms of the temporal order in which the events are known. Thus, in *predictive* causal inference, people are first informed about the occurrence of some potential causes and then make inferences about their potential effects, whereas in *diagnostic* causal inference, they are first informed about the occurrence of some effects and then make inferences about their potential causes.

Different explanations have been provided about how this inferential knowledge is induced. According to one explanation, the processes involved are of an associative nature and are directly related to conditioning mechanisms in animals (Shanks & López, 1996). Thus, inferential learning is grounded on the acquisition of knowledge that captures the statistical or covariational structure of the environment. It is important to note that the acquisition of inferential knowledge may proceed independently from any general and abstract causal knowledge people may have (e.g., that causes precede effects). Associative models conceive the process of knowledge acquisition as a bottom-up process exclusively guided by data concerning pairings between events without the intervention of any general abstract knowledge.

On the other hand, an alternative view on how individuals acquire inferential knowledge in causal contexts is represented by the causal model theory (CMT; Waldmann & Holyoak, 1992). According to this account, this acquisition cannot proceed independently of general and abstract causal knowledge. Hence, top-down influences are present throughout the process of such knowledge acquisition.

The main objective of the present article is therefore an evaluation of the potential influence of general and abstract causal knowledge on the acquisition and use of inferential knowledge to test the theories' predictions. As in previous studies (e.g., Shanks & López, 1996; Waldmann & Holyoak, 1992), *cue competition* will serve as the basic tool for making this evaluation. Cue competition has received different explanations by CMT and the associative account. Each theory proposes different boundary conditions for the occurrence of cue competition and, interestingly, the influence of causal knowledge on inferential learning can be evaluated by looking at these boundary conditions.

Let us take the design of Shanks and López's (1996) Experiment 3 as an illustration of a cue competition effect. Let us further suppose that participants are allergists trying to predict the type of allergic reaction (outcome) a series of patients will develop after having eaten particular foods (cues). The left side of Table 1 shows the different relationships arranged between the various cues and outcomes. In a *noncontingent* condition, participants see that Foods A and B predict the Type 1 allergic reaction (AB $\rightarrow$ Outcome 1, O_1, trials) and also Food B on its own (B $\rightarrow O_1$ trials), whereas patients eating Food C do not develop any allergic reaction (C $\rightarrow$ no O trials). In a *contingent* condition, participants see that Foods D and E predict the Type 2 allergic reaction (DE $\rightarrow O_2$ trials), whereas Food E does not predict any reaction (E $\rightarrow$ no O trials), and Food F also predicts the Type 2 reaction (F $\rightarrow O_2$ trials). As can be noted, the number of Food A–Type 1 pairings

Pedro L. Cobos, Francisco J. López, Antonio Caño, and Julián Almaraz, Departamento de Psicología Básica, Universidad de Málaga, Málaga, Spain; David R. Shanks, Department of Psychology, University College London, London, England.

The research described here was supported by research grants from the United Kingdom Biotechnology and Biological Sciences Research Council and by Dirección General de Investigación Grant BSO2000-1216 and Junta de Andalucía Grant HUM0105 from Spain.

Correspondence concerning this article should be addressed to Pedro L. Cobos, Departamento de Psicología Básica, Facultad de Psicología, Campus de Teatinos, Universidad de Málaga, Málaga 29071, Spain. E-mail: p_cobos@uma.es

Table 1
Design, Sample Size, Mean Ratings, and Standard Errors for the Target Relationships Across the Different Experimental Groups in Experiments 1a–3

	Experiment 1a				Experiment 1b				Experiment 2				Experiment 3							
	CE (N = 30)		EC (N = 28)		CE (N = 18)		EC (N = 18)		CE (N = 29)		EC (N = 28)		CE–McSo (N = 55)		EC–McSo (N = 56)		CE–ScMo (N = 37)		EC–ScMo (N = 37)	
Contingency condition	M	SE	M	SE	M	SE	M	SE	M	SE	M	SE	M	SE	M	SE	M	SE	M	SE
Noncontingent[a]																				
AB → O_1	44.83	6.50	53.39	7.62	45.28	9.26	55.83	9.18	38.97	7.18	46.07	7.17	41.46	4.20	46.07	4.32	78.24	3.98	73.38	5.05
B → O_1																				
C → no O																				
Contingent[b]																				
DE → O_2	76.17	4.72	71.79	5.14	86.94	5.00	81.67	5.95	71.90	4.61	72.86	5.07	67.64	4.23	64.11	4.15	80.95	3.96	84.46	3.89
E → no O																				
F → O_2																				

Note. A, B, C, D, E, and F refer to cues (see text for further details). CE = cause–effect; EC = effect–cause; McSo = multiple cues–single outcome; ScMo = single cue–multiple outcomes; O = outcome; no O = no outcome.
[a] Target Cue A. [b] Target Cue D.

(noncontingent condition) and Food D–Type 2 pairings (contingent condition) are the same. Despite this, however, people perceive a stronger predictive relationship between Food D and Type 2 reaction than between Food A and Type 1 reaction. This has been interpreted as reflecting competition between cues. Note that in the contingent condition, Food D co-occurred with another food (E) that does not predict the Type 2 reaction on its own, whereas in the noncontingent condition, Food A co-occurred with Food B, which itself predicts the Type 1 reaction. Thus, Food D may be regarded as a relevant predictor of the Type 2 reaction, and Food A may be regarded as a redundant predictor of the Type 1 reaction in the sense that it does not convey any relevant information. In other words, Cue D is a better relative predictor of O_2 than Cue A is of O_1.

How does each theory account for this contingency effect in causal contexts? As described by Waldmann and Holyoak (1992), basically CMT makes three assumptions concerning the process of induction and inference. According to the first assumption, the specific causal role (cause or effect) played by the cues and the outcomes influences the nature of the knowledge acquired in the learning task. Such knowledge consists of the acquisition of a directed link connecting a cause and an effect. Thus, the direction of the links acquired depends on the causal role played by cues and outcomes and not on the temporal order in which such events are known. The acquisition of these directed cause–effect links may be thought of as the acquisition of the causal structure of the task. As a consequence, the knowledge acquired in a given predictive or diagnostic situation may be identical, provided that the causal structure underlying the situations is the same. The specific cause–effect direction of the causal links acquired simply reflects the a priori knowledge people have concerning the temporal precedence of causes over effects.

According to the second assumption, such directed causal links are induced by means of a contrast between conditional probabilities (Cheng & Holyoak, 1995; Cheng & Novick, 1990, 1992). Specifically, the contrast is between the probability (P) of the effect (E) given the target cause (C), P(E/C), and the probability of the effect given the absence of the target cause, P(E/–C), everything else being held constant (i.e., keeping constant the status of alternative causal factors).[1]

Finally, according to the third assumption, the directed causal links that are learned may be flexibly used for making two structurally different kinds of inferences: *predictive inferences*, going from observed causes to possible effects, and *diagnostic inferences*, going from observed effects to possible causes. The study of these structural differences has been an issue of philosophical debate (e.g., Pearl, 1988; Reichenbach, 1956). The implications for inference may be summarized as follows. A predictive inference of an effect (outcome) from a given cause (cue) only involves an evaluation of the causal link connecting the cause and the effect. However, the inference of a cause (outcome) from a given effect (cue) cannot be made on the basis of any single causal link,

[1] More recently, however, Cheng has proposed her power PC theory (Cheng, 1997) as a new way to understand the acquisition of causal links still consistent with the sort of theorizing underlying CMT. It is important to note that this modification does not affect the present analysis.

because the diagnostic value of an effect depends not only on the causal link from the cause to this particular effect but also on the diagnostic value of the effect for other possible causes. Therefore, diagnostic inferences involve a search for alternative possible causes of the effect in question followed by a comparison of the plausibility of those competing causes.

Let us illustrate these differences with the following example. Imagine that a physician is trying to estimate the chances of a particular patient developing a certain symptom (or effect; e.g., a high temperature) from her or his knowledge that the patient suffers from a particular disease (or cause; e.g., influenza). All that is required to make this predictive inference is knowledge about the causal strength of influenza to produce a high temperature in this particular patient, and the physician should be quite confident that the patient will develop a high temperature if the causal strength between this particular cause (the disease) and effect (the symptom) is known to be high. Let us now imagine this physician trying instead to estimate the chances of a particular patient suffering from a certain disease (or cause; e.g., influenza) from her or his knowledge that the patient has a particular symptom (or effect; e.g., a high temperature). This diagnostic inference should be based not only on the causal strength of influenza to produce a high temperature but also on all the alternative causes that might have produced the symptom. In this case, a high temperature should be viewed as a weak diagnostic sign for influenza, as it may be attributable to any of several other causes.

Having described the main aspects of CMT, we now turn to the explanation it provides of the contingency effect considered previously. A key aspect is the probabilistic contrast mentioned in the second assumption. In the noncontingent condition, the causal link between Cause A (Food A) and Effect 1 (Type 1 allergic reaction) will depend on the contrast between the probability of Effect 1 given Cause A and that of Effect 1 given the absence of Cause A, everything else held constant (e.g., provided the presence of Cause B). Only $AB \rightarrow O_1$ and $B \rightarrow O_1$ trials are relevant to this contrast. Given that Food B on its own always produces the Type 1 reaction, we cannot conclude anything regarding the causal status of Food A, as it always co-occurred with Food B. In the contingent condition, however, the causal link between Cause D and Effect 2 will depend on the contrast between the probabilities of Effect 2 given the presence and absence of Cause D, everything else held constant (e.g., provided the presence of Cause E). Here, only $DE \rightarrow O_2$ and $E \rightarrow$ no O trials are relevant. As Food E on its own never produces the Type 2 allergic reaction, we can be confident that Food D is a cause of this allergy. If we assume that predictive inferences are mediated by the contrasts described, ratings of the Cause D–Effect 2 relationship should be higher than ratings of the Cause A–Effect 1 relationship. Consequently, the contingency effect may be viewed as a form of competition between causes during the induction process. If a target cause always appears with another competing cause, its causal status to produce a certain effect would remain uncertain if the competing cause on its own always produces the effect, even when the target cause and the effect are reliably paired.

To illustrate the implications of the structural differences between predictive and diagnostic inferences, let us now see what the predictions would be if cues and outcomes were interpreted as effects and causes, respectively, and if diagnostic ratings (from observed effects to possible causes) were required. Now, the causal interpretation of trial types for the noncontingent and contingent condition would be as follows: $E_AE_B \leftarrow C_1$, $E_B \leftarrow C_1$, and $E_C \leftarrow$ no cause and $E_DE_E \leftarrow C_2$, $E_E \leftarrow$ no cause, and $E_F \leftarrow C_2$, respectively. If the probabilistic contrast formula is applied, it can be readily shown that Cause 1 produces Effect A to the same extent that Cause 2 produces Effect D. However, we are interested in the diagnostic value of Effects A and D regarding their respective causes. To evaluate this diagnostic value, not only the causal links just mentioned need to be considered but also the general capacity of the effects to diagnose their respective target causes. As Effects A and D have each only been produced by one potential cause, their respective diagnostic values will not be compromised by any other causes, which might explain their origin. Thus, in both conditions, the effects equally and maximally diagnose the presence of their corresponding causes, as these causes are the only possible explanations for their occurrences, and hence no contingency effect should be expected. In summary, CMT predicts a cue competition effect under causal but not diagnostic conditions.

A totally different account of how inferential knowledge is induced and used for inference is provided by associative models. The Rescorla and Wagner model (RW; Rescorla & Wagner, 1972) is a very well-known and prototypical representative of this class of models. We will center our discussion on RW because of its simplicity and explanatory power in the context of human contingency learning (see Allan, 1993, and Shanks, 1995, for reviews) and because it captures all of the main aspects of an associative account. From this perspective, inferential learning, even within causal contexts, is viewed as a bottom-up or data-driven process. The description that follows tries to parallel the analysis made regarding CMT and is also organized around three assumptions. This description helps to contrast the differences between the two accounts.

According to the first assumption, in situations where individuals have first-hand experience with how cues and outcomes are related across a series of trials, the knowledge developed is conceived as a set of associative links from the mental representations of the cues to the mental representations of the outcomes. It is important to note that the direction of the links is only determined by the temporal order in which the events are presented and is independent of the causal role of cues and outcomes, as there is no associative basis for distinguishing causes from effects (though see Van Hamme, Kao, & Wasserman, 1993, for a different interpretation). These links are of a variable magnitude or *associative strength* that represents the extent to which cues and outcomes covary.

The second assumption concerns the learning rule controlling the updating of associative strengths between the cues and outcomes. This rule operates on a trial-by-trial basis according to an error-correcting procedure that is equivalent to the delta rule frequently used in connectionist models (Gluck & Bower, 1988; McClelland & Rumelhart, 1985; Sutton & Barto, 1981). This delta rule has been derived from the least-mean-squared-error method to minimize the average error across trials. Specifically, changes in the associative strength between a cue and an outcome are proportional to the difference between the extent to which the outcome is expected to occur, on the basis of the cue or cues present on a given trial, and what actually happens (see, e.g., Shanks,

1995, for further details and for a recent review of the properties of this rule).

The third assumption refers to the way in which inferences are produced in predictive and diagnostic learning situations. As RW does not conceive any role for abstract and general causal knowledge people may possess during the acquisition of inferential knowledge, predictive and diagnostic judgments are produced on exactly the same basis. Remember that the associative mechanism is opaque to the causal role of cues and outcomes, as stated in the first assumption.

The RW model was originally conceived precisely to explain the contingency effect and other related effects found in animal conditioning. We will not go into the specific details of how the learning rule accounts for the effect (see López, Cobos, Caño, & Shanks, 1998, or Shanks, 1995, for such specification). Suffice it to say that the nature of the learning algorithm forces competition between cues that occur together (as was the case in Shanks & López's, 1996, design) to gain associative strength when paired with a common outcome. Consequently, those cues that are redundant in predicting the outcome (e.g., Cue A in the noncontingent condition) tend to lose associative strength, whereas those cues that are informationally valid (e.g., Cue D in the contingent condition) tend to gain associative strength. Notice that to the extent that the contingency effect is due to a process of cue competition, it should be expected in both predictive and diagnostic situations. That is, no matter what the causal role (cause or effect) such cues play, competition and thus the contingency effect should occur.

To summarize, CMT assumes that the processes involved in the acquisition of inferential knowledge are sensitive to general and abstract causal knowledge, specifically to the causal role played by the events over which the relationship is defined. This sensitivity has two consequences. First, it determines the structure of the causal knowledge acquired during the task. Cause–effect links are learned, regardless of whether the events are presented within a predictive (cause–effect, CE) or a diagnostic (effect–cause, EC) format. Second, predictive judgments only involve an evaluation of the causal link connecting the cause and the effect, whereas diagnostic judgments involve a search for the alternative possible causes of the effect in question, followed by a comparison of the plausibility of those competing causes. This double influence has clear implications for the occurrence of contingency effects. Specifically, in terms of Shanks and López's (1996) design, the contingency effect should only be obtained in a CE situation but not in an EC situation.[2] On the other hand, RW assumes that the contingency effect arises when judgments concern cues whose relative validity has been manipulated, regardless of the causal role such cues play. According to the associative account, only this manipulation will activate the process of cue competition underlying the contingency effect. Hence, in terms of Shanks and López's design, the contingency effect should be obtained in both a CE situation and in an EC situation.

There has been previous research concerning the evaluation of both theories' predictions, but this research has yielded contradictory results (e.g., Matute, Arcediano, & Miller, 1996; Price & Yates, 1995; Shanks & López, 1996; Van Hamme et al., 1993; Waldmann, 2000, 2001; Waldmann & Holyoak, 1992). Despite their somewhat inconclusive results, however, this research has yielded a set of agreed conditions concerning how this evaluation should be made, and the present experimental series fulfils all of them (see Shanks & López, 1996, and Waldmann & Holyoak, 1997, for discussion of these conditions). We will briefly consider some of these previous studies in the General Discussion in light of the findings obtained here.

Overview of the Experiments

In Experiments 1a and 1b, we improved on Shanks and López's (1996) methodology so that all these conditions were simultaneously met. First, this involved using a causal scenario that presented no ambiguity regarding the causal role assigned to cues and outcomes. Second, the wording used for eliciting the ratings was clearly directed and explicitly stated their inferential nature. Hence, in the CE and EC conditions, judgments of the predictive/diagnostic value of the target cause/effect were specified. Third, causal order was manipulated in all the experimental series, and also the probabilistic contrasts yielded identical values in the contingent and noncontingent conditions of the EC condition.

In Experiment 2, we further explored the possible influence of causal knowledge on predictiveness ratings through changing the wording of the predictiveness question used in the EC condition. Though Waldmann and Holyoak's (1997) requirements favored the use of predictiveness questions, the use of the verb *predict* (from Latin, *to say before hand*) in the wording of such judgments may not be the most appropriate way to activate causal knowledge in the EC direction, as this conveys a clear temporal direction from past to future events. As the question goes from effects to causes in the EC condition and causes obviously precede effects, there is a certain contradiction between the meaning of *predict* and the temporal order of the events involved in the actual question, thus potentially making the use of causal knowledge more complex.

In Experiment 3, the impact of causal knowledge on inference was evaluated in a different way. In some of the previous attempts to specifically evaluate such influence (e.g., Shanks & López, 1996; Waldmann, 2000; Waldmann & Holyoak, 1992), the causal structure underlying the relationships arranged between cues and outcomes differed in the CE and EC situations, but the cue–outcome structure remained identical. That is, if we consider the learning task as comprised of trials where cues precede outcomes, no change of the structure of the task from this cue–outcome perspective occurred in the CE and EC conditions. However, this

[2] It may be argued, as one of the reviewers of a previous version of this article did, that a causal model for the contingent condition in the EC or diagnostic situation may involve more than a single cause (e.g., Cause 2 interacting with some unknown factor), as this cause has disjunctive effects, that is, either produces Effects D and E or Effect F by itself, but no other configuration of effects is ever present. Still, though, there is no basis for CMT to expect a higher diagnostic value for Effect D than for Effect A, and hence the contingency effect is still predicted only to occur in the CE situation. In this case, according to CMT, the information provided during the task is not sufficient to properly evaluate the magnitude of the Target $E_D \leftarrow C_2$ causal link, and hence there is no reason to expect that this uncertainty should lead participants to give a higher diagnostic value for Effect D than for Effect A (which, in fact, is maximal). If anything, this uncertainty should lower the diagnostic value of Effect D. All in all, the contingency effect should only occur in the CE or predictive case, according to CMT, even if the contingent condition of the EC case is not compatible with a common cause interpretation.

cue–outcome structure can be manipulated by reversing the temporal order in which cues and outcomes occur, that is, interchanging cues and outcomes. In Experiment 3, we manipulated the cue–outcome structure without altering the causal structure underlying the task. This represents an opportunity to further contrast the predictions of the associative and CMT accounts. According to the associative account, an alteration of the cue–outcome structure of the task should influence the contingency effect, whereas according to CMT, such alteration, if it does not further alter its causal structure, should not influence the contingency effect.

In Experiment 4, we extended the evaluation of the influence of causal order to a situation in which the underlying causal structure is simpler than in Shanks and López's (1996) contingency design. Specifically, we used an overshadowing design that contrasted participants' inferences in two target conditions (overshadowing: $AB \rightarrow O_1$ vs. control: $C \rightarrow O_2$), and again the causal role of cues and outcomes was manipulated. As in previous experiments, CMT again predicts that only predictive or CE inferences, but not diagnostic or EC inferences, should be sensitive to the contingency manipulation (overshadowing effect), whereas RW predicts perfectly symmetrical results for CE and EC inferences regarding this contingency manipulation.

Experiment 1a

Experiments 1a and 1b may be viewed as an extension of Shanks and López's (1996) work. The causal role of cues and outcomes (CE and EC) was manipulated, as described previously, and hence two different causal structures were defined. However, the cue–outcome structure was kept constant, as shown in Table 1. If causal knowledge exerts the influence CMT proposes, a contingency effect should only be observed in the CE direction. On the other hand, if inferential knowledge is acquired independently of the general causal knowledge people have, as the associative account assumes, the manipulation of the causal role of the events should not influence the contingency effect.

The task scenario ensured that the causal roles of cues and outcomes were not confounded. Participants had to detect relationships between different substances that leaked from a chemical plant and the different indicator lights that came on in the control room of the plant as a consequence of the leakage of such substances. According to the causal scenario, the substances were unequivocally the causes of the activation of the different indicator lights. In the CE condition, participants could observe across trials the substances that had leaked, and they had to guess which indicator light would come on. After this training phase, they had to make explicit ratings about the predictive value of the Target Substances A and D regarding their corresponding Indicator Lights 1 and 2 (see Table 1). In the EC condition, participants could observe on each trial which indicator light had come on, and they had to decide which substance they thought was the cause. Again, after the learning phase, participants had to rate the diagnostic value of the target Indicator Lights A and D for their corresponding Substances 1 and 2.

Method

Participants and apparatus. A total of 73 psychology undergraduates from Málaga University in Spain volunteered to take part in Experiment 1a for course credits. The task was performed on IBM-PC compatible computers. Judgments were required through a paper-and-pencil questionnaire.

Procedure. After reading the instructions on the computer screen and getting familiarized through a pretraining task with the different parts of the experimental task, the participants began the learning phase. Participants acted as workers in a chemical plant whose faulty functioning produced leakages of different substances. These leaks produced in turn the activation of different indicator lights in the control room of the plant (see the Appendix for the instructions used). Participants had to learn the different relationships arranged between different chemical substances and indicator lights. This learning phase consisted of 240 trials. Table 1 shows half of all the programmed trial types; the other half was identical in terms of the relationships programmed except for the use of different cues and outcomes. Thus, two sets of contingent and noncontingent relationships were programmed, including a total of 12 trial types. The sequence of trials was randomized for each participant, with the single restriction of being presented in blocks of 12 trials that included every trial type, though such blocks were not marked to participants.

On each trial, participants could see the relevant cue or cues, and they had to guess the correct outcome. Once they had made their choice, feedback was provided (participants could read at the bottom of the screen what the right choice was for that particular trial) so that they could learn the programmed relationships. Incorrect choices were accompanied by a beep. In addition, from Trial 24 onwards, each trial included explicit visual information about the percentage of correct responses across the last 24 trials. A yellow rectangle in the upper right-hand corner of the screen indicated the percentage of correct responses. A learning criterion was included so that the training phase stopped whenever 240 trials had occurred or the participant had correctly responded in two complete blocks of trials, whichever occurred first.

In the CE group, participants received information about the substances (causes), and they had to predict the correct indicator light (effect). On each trial, participants read the following: "In example [#] the following substances have leaked . . . ." Then, they read the actual substances that were represented by fictitious names and their initials (for ease of responding). Fictitious names were written in a less salient color than their corresponding initials. For half of the participants, the roles of Cues A to F (shown in Table 1) were assigned to the following specific fictitious substances, respectively: betacina, fenolina, xeronina, polixina, dioxina, glicolina. For the other half, the target substances betacina and polixina (A and D, respectively) were swapped. Orthogonally to this counterbalancing, for half of the participants, the roles of Outcomes O_1 and O_2 (shown in Table 1) were assigned to different indicator lights, specifically Indicator 1 and Indicator 2, respectively. For the other half, these target indicators were swapped. In addition, an identical second set of contingent and noncontingent relationships was simultaneously programmed. In this case, the roles of Cues A to F were assigned to the fictitious names melacina, acetina, licaina, clorixina, neocina, and oxatina, respectively, and the roles of Outcomes O_1 and O_2 were assigned to indicator lights Indicator 3 and Indicator 4, respectively. An identical counterbalancing procedure was also adopted within this second set of relationships. Consequently, a set of five choices was listed in every trial, including four different indicator lights and the absence of any light: 1 = Indicator 1, 2 = Indicator 2, 3 = Indicator 3, 4 = Indicator 4, N = no indicator light is on. Participants typed in the corresponding numbers (or N) as their choices on every trial using the computer keyboard.

In the EC group, cues represented information concerning indicator lights (effects), and the outcomes were substances (causes), which may have caused the lights to come on. Participants first read information concerning the indicator lights (now represented by different numbers), and then the five possible outcomes were listed, now represented by fictitious names (in a less salient color) and their initials. These outcomes were the leak of four different substances and the absence of any leak. Participants

typed in these initials (or N for no substance has leaked) as their choices on every trial using the computer keyboard. For half of the participants, the indicator lights (Cues A to F) were numbered from 1 to 6, respectively. For the other half, Indicators 1 and 4 (A and D, respectively) were swapped. Orthogonally to this, for half of the participants, the roles of Outcomes O_1 and O_2 were assigned to different substances, specifically betacina and dioxina. For the other half, these substances were swapped. As before, an identical second set of contingent and noncontingent relationships was programmed, and the same counterbalancing procedure was adopted. In this case, the indicator lights (Cues A to F) were numbered from 7 to 12, respectively, and the roles of Outcomes O_1 and O_2 were assigned to substances fenolina and licaína.

Once the training phase had finished, participants had to answer the questionnaire, which required predictiveness ratings of the target cues regarding the different outcomes: "To what extent does substance/indicator 'X' predict the illumination/leak of the different indicators/substances?"[3] Although participants made inferential judgments on each target cue and the different outcomes, only those judgments concerning the Target A $\rightarrow$ O_1 and D $\rightarrow$ O_2 relationships were analyzed. As two sets of cues and outcomes were included and a counterbalancing procedure was adopted, participants' judgments were collapsed into a single measure, one per contingency condition.

Participants gave their ratings by marking with an *X* on a 0 to 100 scale divided in units of 10. Given the differences between the learning (a computerized task) and the rating phase (paper-and-pencil task), the questionnaire included some control questions regarding the predictive–diagnostic value (in the CE and EC groups, respectively) of certain nontarget cues. The objective was to know whether participants had adequately transferred the knowledge acquired during the training stage to the judgment phase. Specifically, two ratings were required: (a) the predictive–diagnostic relationship between Cue C and the different outcomes (this cue was always paired with the absence of any outcome, and hence a correct answer would mean a 0 rating for the predictive–diagnostic value of the cue with all different outcomes) and (b) the predictive–diagnostic relationship between Cue F and the different outcomes (this cue was only paired with Outcome O_2 during training). For half of the participants, Questions (a) and (b) began and ended the questionnaire, respectively, whereas for the other half, this order was reversed.

Results and Discussion

In the analysis, only the data from participants who satisfied the learning criterion (100% correct) and showed good knowledge transfer on the control questions of the questionnaire were included. Specifically, only participants who rated the value of Cue F above or equal to 80 with its target Outcome O_2 and that of Cue C lower than or equal to 20 with all different outcomes were included in the analysis. Thus, ratings of 58 participants were included in the analysis. Note that this selection process is justified on the basis that the predictions derived from the theories being contrasted assume asymptotic (or near asymptotic) performance.

It is important to note that the number of participants who were selected from each of the two target causal order conditions was practically identical. Specifically, the original sample included 37 and 36 participants in the CE and EC groups, respectively, whereas the selected sample included 30 and 28 in the CE and EC groups, respectively.

All of the statistical analyses reported in this series of experiments adopted a confidence level of 95% ($\alpha = .05$). The mean number of trials to reach the learning criterion was similar in both the CE and the EC causal order, 142.00 and 140.57, respectively ($F < 1$). Table 1 shows the mean ratings of Cues A and D with regards to Outcomes 1 and 2, respectively.

A 2 (contingency: contingent vs. noncontingent) $\times$ 2 (causal order: CE vs. EC) mixed analysis of variance (ANOVA) was performed on participants' mean ratings. The main effect of contingency was significant, $F(1, 56) = 17.48$, $p < .01$, $MSE = 1{,}024.55$. Neither the main effect of causal order nor the Contingency $\times$ Causal Order interaction was significant, $F(1, 56) = 0.11$, $p = .74$, $MSE = 1{,}117.80$, and $F(1, 56) = 1.18$, $p = .28$, $MSE = 1{,}024.55$, respectively. According to these results, participants' knowledge about the causal roles played by the different events did not exert the influence on the acquisition and use of inferential knowledge proposed by CMT. The nonsignificant interaction shows that the contingency effect in the CE and EC condition did not statistically differ, contrary to what CMT predicts.

Despite this nonsignificant interaction, we conducted a simple effect comparison to see whether the crucial CMT prediction concerning the null effect of the contingency factor within the EC group occurred. This analysis revealed a reliable contingency effect within this group, $F(1, 56) = 4.62$, $p = .03$, $MSE = 1{,}024.55$, contradicting this specific CMT prediction.

On the other hand, the absence of a significant interaction together with the contingency main effect are consistent with the RW model's predictions. Nevertheless, although the Contingency $\times$ Causal Order interaction was not statistically reliable, in absolute terms the magnitude of the contingency effect tended to be greater in the CE than in the EC group (mean values of 31.34 and 18.40, respectively). Thus, the possibility of an influence of causal order cannot be completely rejected. Before drawing conclusions from these results, however, one issue needs to be explored. There was an asymmetry with the experimental procedure used in the groups defined by the causal order manipulation, and this is analyzed in Experiment 1b.

Experiment 1b

In the CE group of Experiment 1a, cues (substances) were represented by letters, whereas outcomes (indicators) were represented by numbers. In the EC group, on the other hand, cues (indicators) were represented by numbers, and outcomes (substances) were represented by different letters. This labeling factor might be responsible for the difference in absolute magnitude between the contingency effects obtained in the CE and EC groups. Alternatively, this labeling factor might have attenuated a true Contingency $\times$ Causal Order interaction. Thus, the possible influence of this factor is controlled in Experiment 1b. For half of the participants in each group, substances were represented by letters and indicators were represented by numbers, and for the other half, substances were represented by numbers and indicators were represented by letters.

[3] Note that predictiveness ratings were required even in the diagnostic situation. Because of this fact, in the remainder of the article the use of the term *predictiveness ratings* is intended to evoke the exact wording that prompted participants' judgments. However, such judgments are conceptually referred to as diagnostic judgments because they were made in a diagnostic situation (Waldmann & Holyoak, 1997).

Again, as in Experiment 1a, the contingency effect should not differ in the CE and EC groups according to RW, whereas for CMT, the contingency effect should be sensitive to the causal order of the task. The experiment potentially allows us, then, to replicate the results found in the previous experiment.

Method

Participants and apparatus. A total of 78 further psychology undergraduates from Málaga University volunteered to participate in this experiment for course credits. Again, the task was presented using IBM-PC compatible computers, and the ratings were taken using a pencil-and-paper questionnaire.

Procedure. All procedural details were the same as described for Experiment 1a. The only difference concerns the way in which the labels for cues and outcomes were assigned. Fictitious substances were indexed by their initials or by a number. Thus, for half of the participants in each group, substances were represented by letters and indicators were represented by numbers, and for the other half, substances were represented by numbers and indicators were represented by letters.

Results and Discussion

The same criteria as in Experiment 1a were adopted for participant inclusion in the analysis. These criteria allowed us to select the ratings of those participants who had correctly learned the programmed relationships and had correctly transferred their knowledge to the judgment stage. On this basis, the ratings of 36 participants were included in the analysis. The number of participants who were selected from each of the two target causal order conditions was practically identical. Specifically, the original sample included 40 and 38 participants in the CE and EC groups, respectively, whereas the selected sample included 18 participants in both the CE and EC groups.

The mean number of trials to reach the learning criterion was similar in the CE and the EC causal order conditions, 184.17 and 172.83, respectively ($F < 1$). Table 1 shows the mean ratings for the Target Cues A and D regarding O_1 and O_2, respectively. A 2 (contingency: contingent vs. noncontingent) $\times$ 2 (causal order: CE vs. EC) mixed ANOVA was performed on these ratings. The main effect of contingency was significant, $F(1, 34) = 30.87$, $p < .01$, $MSE = 664.15$. Neither the main effect of causal order nor the Contingency $\times$ Causal Order interaction was significant, $F(1, 34) = 0.09$, $p = .76$, $MSE = 1{,}409.17$, and $F(1, 34) = 1.70$, $p = .20$, $MSE = 664.15$, respectively. As in Experiment 1a, the contingency effect was not statistically different in the CE and EC groups. Thus, the results contradicted CMT's predictions. We again conducted a simple effect comparison to see whether the crucial null effect of the contingency factor within the EC group occurred. This analysis revealed a reliable contingency effect within this group, $F(1, 34) = 9.04$, $p < .01$, $MSE = 664.15$, again contradicting this specific CMT prediction. Nevertheless, as was the case in Experiment 1a, the magnitude of the contingency effect in absolute terms tended to be greater in the CE group than in the EC group (mean values of 41.66 and 25.84, respectively).

To conclude, the results from Experiments 1a and 1b revealed a contingency effect and the absence of any Contingency $\times$ Causal Order interaction. These results are clearly inconsistent with CMT's predictions. Despite the interaction not being significant, the contingency effect in the CE group tended to be of a greater magnitude in absolute terms than in the EC group. Hence, although the results clearly favor the associative account over the CMT account, there is nevertheless a hint of an influence of abstract causal knowledge on the acquisition and use of inferential knowledge.

Experiment 2

In this experiment, we further explored the contrast between CMT and RW's predictions concerning the influence of causal knowledge. It may be the case that the wording of the predictiveness rating instructions in the EC group in Experiments 1a and 1b precluded, or at least made more difficult, the use of abstract causal knowledge. Note that the word *predict* involves a clear temporal direction from past to future events (i.e., if X is said to predict Y, X occurs before Y). Despite this fact, in the EC group, the inference question went from effects to causes (i.e., "To what extent does indicator 'X' predict the leak of the different substances?"), and according to the causal scenario used, the illumination of the indicator lights does not occur before the leak of the substances. Hence, there was an element of contradiction between the temporal direction conveyed by the word *predict* and the actual temporal order of the events involved in the inference (see Matute et al., 1996, for a similar argument). This inconsistent use of the word *predict* was also present in Waldmann and Holyoak (1992), and as described in the introduction, its use was suggested in Waldmann and Holyoak (1997). However, this may have hindered the use of causal knowledge for some of the participants.

In Experiment 2, we used the design of Experiments 1a and 1b but adopted a new wording for the question in the EC group. Rather than using the word *predict*, the word *indicate* was adopted. The word *indicate* is free from the connotations mentioned previously in the sense that it lacks any temporal meaning that could contradict participants' knowledge about causal precedence.

Method

Participants and apparatus. A total of 83 psychology undergraduate students from Málaga University took part in the experiment for course credits. The materials and apparatus were the same as in previous experiments.

Procedure. Procedural details were as described before, except in the following aspects. In the CE group, substances were always represented by color names and their initials. Those color names were presented in a less salient color and between brackets. Indicator lights were always represented by numbers. In the EC group, indicators were always represented by color names and their initials, and substances were always represented by numbers. Again, color names appeared in a less salient color and between brackets. In doing so, we ensured that cues and outcomes received exactly the same labels in the CE and EC conditions and, at the same time, the use of pseudowords as substance labels was avoided, as they could make the learning task harder. Specifically, the roles of Cues A to F (shown in Table 1) were assigned to the following specific colors and corresponding initials, respectively: B[4] (white), F (fuchsia), E (emerald), N (orange), V (violet), O (ochre), and outcomes were numbered from 1 to 2 (Outcomes O_1 and O_2, respectively, shown in Table 1). For the second set of contingent and noncontingent relationships that was programmed, the roles of Cues A to F were assigned to the following specific colors and initials, respectively:

[4] Initials refer to Spanish color names.

M (brown), R (red), G (gray), T (turquoise), C (pale blue), and A (blue), and outcomes were numbered from 3 to 4 (Outcomes O_1 and O_2, respectively).

Once the training stage had finished, participants made inferential judgments. In the CE group, participants answered the question "To what extent does substance 'X' predict the illumination of the different indicators?," whereas in the EC group, participants had to answer the question "To what extent does indicator 'X' indicate the leak of the different substances?"

Results and Discussion

The criteria for participant selection were the same as in Experiments 1a and 1b. A total of 57 participants reached the learning criterion and showed good transfer of knowledge in the test stage. The number of participants rejected from each of the two target causal order conditions was identical. Specifically, the original sample included 42 and 41 participants in the CE and EC groups, respectively, whereas the selected sample included 29 and 28 in these groups, respectively.

The mean number of trials to reach the learning criterion was similar in the CE and EC causal orders, 128.28 and 138.00, respectively ($F < 1$). Table 1 shows the mean ratings for the Target A–O_1 and D–O_2 relationships. The main effect of contingency was again significant, $F(1, 55) = 27.42$, $p < .01$, $MSE = 926.24$. Neither the main effect of causal order nor the Contingency × Causal Order interaction was significant, $F(1, 55) = 0.38$, $p = .53$, $MSE = 1{,}209.74$, and $F(1, 55) = 0.29$, $p = .59$, $MSE = 926.24$, respectively. As in previous experiments, the contingency effect was not statistically different in the CE and EC groups and was independent of the causal roles played by the cues and outcomes, contrary to CMT's predictions (mean values of 32.93 and 26.79 for the CE and EC groups, respectively). And again, the crucial contingency effect was significant in the EC group, $F(1, 55) = 10.84$, $p < .01$, $MSE = 926.24$, at variance with CMT. The hypothesis that the word *predict* in the rating instructions in Experiments 1a and 1b precluded the use of causal knowledge within the EC group did not find empirical support because, if anything, the difference between the magnitude of the contingency effect in the CE and EC groups was even smaller in this experiment. Thus, from a descriptive point of view, the results of this experiment are more consistent with the associative account and more problematic for CMT than the results of the previous experiments.

Experiment 3

In Experiments 1a, 1b, and 2, the causal structure underlying the task was explicitly manipulated, whereas its cue–outcome structure was kept constant across the causal order conditions. According to CMT, this manipulation should affect inferential judgments. In Experiment 3, we extended our previous results concerning the evaluation of the influence of causal knowledge to a situation in which the cue–outcome structure of the task was manipulated but the causal structure was held constant. As will be shown, CMT and RW make different predictions concerning this manipulation.

The manipulation of the cue–outcome structure is orthogonal to the previous causal order manipulation. Table 2 illustrates the design. This new experimental manipulation included a temporal reversal of the cues and outcomes presented in previous experiments. Thus, cues and outcomes from one of the experimental groups (multiple cues–single outcome group; McSo) turn into the outcomes and cues for the other group (single cue–multiple outcomes group; ScMo). Orthogonally, the causal order was also manipulated: Cues and outcomes could play the role of causes and effects or of effects and causes. Both manipulations allowed us to further evaluate the influence of causal knowledge on the acquisition of inferential knowledge and in turn its influence on judgments. In addition, the design allowed us to replicate the results found in Experiments 1a–2. The McSo conditions exactly replicate those used in the previous experiments.

Table 2
Design and Trial Types in Experiment 3

	McSo (CE–EC)		ScMo (CE–EC)	
Contingency condition	Trial types	Target relationship	Trial types	Target relationship
Noncontingent				
	$AB \rightarrow O_1$ $B \rightarrow O_1$ $C \rightarrow$ no O	$A \rightarrow O_1$	$1 \rightarrow O_{AB}$ $1 \rightarrow O_B$ $0 \rightarrow O_C$	$1 \rightarrow O_A$
Contingent				
	$DE \rightarrow O_2$ $E \rightarrow$ no O $F \rightarrow O_2$	$D \rightarrow O_2$	$2 \rightarrow O_{DE}$ $0 \rightarrow O_E$ $2 \rightarrow O_F$	$2 \rightarrow O_D$

Note. A, B, C, D, E, and F refer to cues; O and no O refer to the outcome and the absence of any outcome, respectively (see text for further details). McSo = multiple cues–single outcome; ScMo = single cue–multiple outcomes; CE = cause–effect; EC = effect–cause.

What are the predictions made by CMT and RW concerning this new, more complex design? Let us first consider the predictions derived from RW. As described in the introduction, two (or more) cues compete to gain associative strength with a particular outcome whenever such cues co-occur prior to the outcome. Thus, cue competition is responsible for the contingency effect described previously. However, if a cue is consistently followed by more than one outcome, as is the case in the ScMo groups of this design, it may form independent associations with each outcome. Thus, the predictive value of each cue regarding a particular outcome is not compromised at all by the fact that the cue may also predict different outcomes. In this particular case, the associative strength of each cue will equal asymptotically the difference between the probability of the target outcome given the cue in question and the probability of the outcome given the absence of the cue, everything else being held constant (as Chapman & Robbins, 1990, demonstrated). Because these differences are identical in both contingency conditions, no contingency or cue competition effect should be expected. Again, these predictions are not altered by the causal role that cues and outcomes play. Consequently, no contingency effect should be observed in the ScMo groups regardless of the causal roles of cues and outcomes.

According to CMT, on the other hand, causal knowledge should influence inferential judgments in the ScMo groups, that is, judgments should be sensitive to the causal role that cues and outcomes play. In the CE group, the predictions the theories make are identical regarding the contingency effect. In this case, the temporal order of the events (cue → outcome) coincides with the

primed causal direction (cause → effect). As there is only one potential cause of the different effects in both contingency conditions, the predictive value of the target cues will, according to CMT, be given by the difference between P(E/C) and P(E/–C). This difference is equal to 0.5 in both the contingent and noncontingent conditions, P(E/C) = 0.5, and P(E/–C) = 0 (see Table 2).

On the other hand, in the EC group (note that its causal structure is as in the CE−McSo group), diagnostic rather than predictive judgments are required. However, there is an important structural difference compared with the EC−McSo condition previously described, though in both cases judgments are diagnostic. In this case, there are several causes potentially producing the same effect (see Table 2). According to CMT, the diagnostic value of an effect regarding a cause depends not only on the predictive value of the cause for the target effect but also depends on the plausibility of other alternative causes for the effect. Thus, in the noncontingent condition, Effect 1 (E_1) may have been produced by Causes A (C_A) and B (C_B), or alternatively by Cause B (C_B) on its own. In the contingent condition, E_2 may have been produced by C_D and C_E or alternatively by C_F. In terms of CMT, then, the diagnostic values of E_1 and E_2 with regards to C_A and C_D, respectively, will depend on two factors: (a) the causal values of C_A and C_D regarding E_1 and E_2, respectively, and (b) the plausibility of the alternatives C_B and C_F, respectively. As C_B and C_F are equally valid predictors of their corresponding E_1 and E_2 and moreover are equally frequent, both may be regarded as equally plausible alternative causes. Thus, in this particular situation, the diagnostic values of E_1 and E_2 with regards to C_A and C_D will crucially depend on the first factor mentioned. As described for previous experiments, whereas C_D clearly produces E_2, C_A has an unclear status as a cause of E_1. Therefore, judgments about the diagnostic value of E_1 for C_A should be lower than judgments about the diagnostic value of E_2 for C_D. Note that in its actual level of specification, CMT makes only this qualitative prediction. As presently formulated, it does not detail how diagnostic inferences are actually made, and hence no predictions concerning the actual magnitude of inferential judgments may be derived. In other words, all that may be said is that in the EC direction, judgments should reflect a process of competition between causes (or outcomes). Consequently, the contingency effect should only be observed in the EC but not in the CE causal order within ScMo conditions, whereas within McSo conditions, the contingency effect should only be found in the CE but not in the EC causal order, as previously described. See Table 3 for a summary of the predictions the two theories make.

Table 3
Predictions Made by CMT and RW, Magnitude of the Contingency Effect, and Standard Errors for the Different Groups of Experiment 3

Cue–outcome structure	CMT	RW	*M* judgment	*SE*
McSo				
CE	+	+	26.18	5.54
EC	−	+	18.04	5.51
ScMo				
CE	−	−	2.70	2.91
EC	+	−	11.08	3.95

Note. CMT = causal model theory (Waldmann & Holyoak, 1992); RW = Rescorla and Wagner (1972) model; McSo = multiple cues–single outcome; CE = cause–effect; EC = effect–cause; ScMo = single cue–multiple outcomes; + = contingency effect expected; − = no contingency effect expected.

Method

Participants and apparatus. A total of 240 psychology undergraduate students from Málaga University took part in Experiment 3 for course credits. The materials and apparatus were the same as in Experiments 1a–2.

Procedure. As in previous experiments, participants had to learn relationships between the substances that leaked from a chemical plant and the indicator lights that came on in the control room of the plant.

Procedural details were the same as described in Experiment 2, except in the following aspects. In the ScMo groups, the procedure was arranged to be as similar as possible to that adopted for McSo groups. As shown in Table 2, cues were now numbered from 1 to 2, whereas the role of Outcomes A to F was assigned to the different color names used in Experiment 2. As in previous experiments, Table 2 shows half of the total number of relationships that were actually programmed during the task. Thus, a total of 12 response options (one per trial type) were programmed: compound responses (e.g., A and B, Outcome O_{AB}) and single responses (e.g., B, Outcome O_B). Nevertheless, to keep the same number of response options as in McSo groups, participants from these new groups only saw 5 possible response options on every trial (see Figure 1 for details concerning the actual display). There are two important reasons for this. First, including 12 response options would have made the task harder to learn than in McSo groups where the correct response was selected out of a set of only 5 response options. Second, including the whole set of response options would have made the task of a nondeterministic nature, at variance with the situation in the McSo groups. Note that each type of cue was paired with at least 2 different outcomes, and consequently at least 2 different response options would have been correct. For example, Cue 1 may have been paired with Outcome O_{AB} and Outcome O_B, thus both response options would have been correct. However, as shown in Table 2, each of these 2 outcomes is the correct response just in half of the trials, and thus the task would have become nondeterministic. This, in turn, would have made the task harder and precluded participants from reaching the learning criterion adopted in previous experiments. To avoid this situation, a subset of 5 response options was selected for each trial so that only one of the possible correct outcomes was included. For example, in a trial type including Cue 1, either O_{AB} or O_B was included, but never both of them simultaneously. The accompanying 4 response options were randomly selected from the remaining set of options on a trial-by-trial basis. For example, in a Cue 1 trial type, the list of responses may include Outcomes O_{AB}, O_F, O_C, O_E, or O_{DE}. Note that if O_{AB} is the outcome selected as the correct response, O_B cannot also be included in the list of response options. However, both O_{AB} and O_B could be included within the same subset of response options in those trial types not including Cue 1. The relative position within the list of the 5 response options and the order of actual outcomes within compound responses (e.g., Compound A and B or Compound B and A) were randomized for every trial.

Participants typed in the initial(s) of the color(s), in any order, representing what they thought was the correct response on every trial. It should be mentioned that this procedural modification does not affect at all any of the predictions derived from the theories that are being contrasted.

In addition, in the ScMo groups, all of the response options listed explicitly stated that they were the only outcomes present on that trial. The objective was to solve a possible ambiguity in the interpretation of the information presented. Participants could reason that those outcomes not listed (and hence about which no information was provided regarding their presence or absence) were also present. Such ambiguity would have

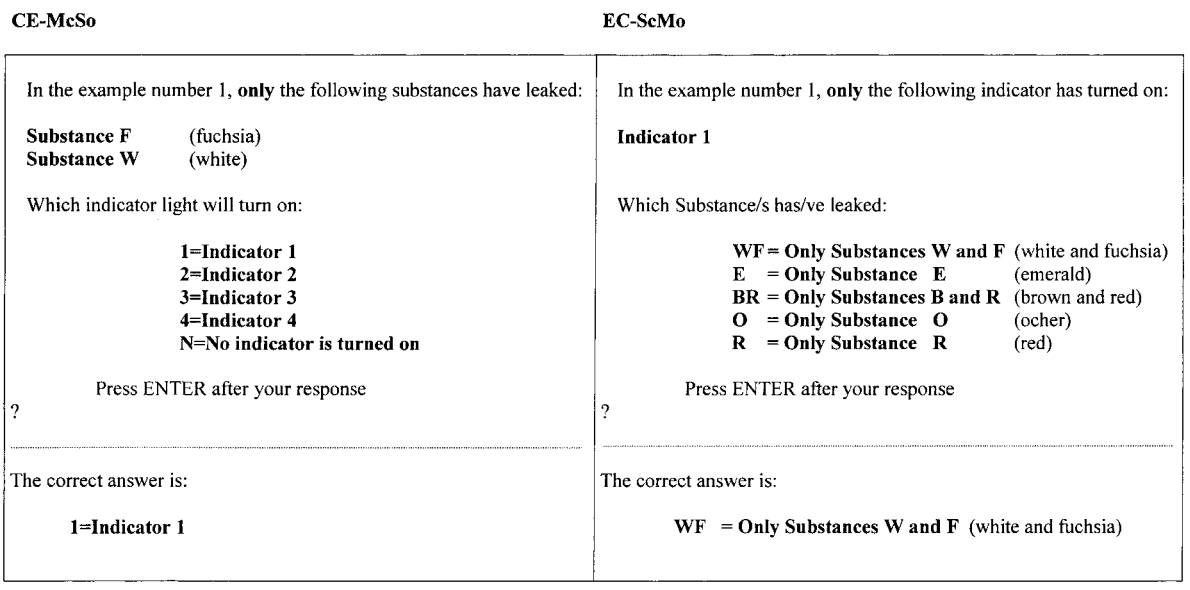

Figure 1. A representation of the displays that participants saw in McSo (multiple cues–single outcome) and ScMo (single cue–multiple outcomes) groups of Experiment 3 is shown. Specifically, the displays for the CE (cause–effect)−McSo and the EC (effect–cause)−ScMo groups are shown. The displays for the remaining groups (EC−McSo and CE−ScMo) were equivalent. In the McSo group, an AB $\rightarrow O_1$ trial type is shown (Cue A = W [white], Cue B = F [fuchsia], and O_1 [Outcome 1] = 1), whereas in the ScMo group, a 1 $\rightarrow O_{AB}$ (Outcome AB) trial type is displayed. Once participants had typed their response, they received corrective feedback concerning the correct response for that particular trial. This information is represented under the dotted line (this line did not actually appear and has been included to represent the fact that the corrective feedback was not available at the time participants made their response). An English version of the information provided has been used.

affected the computation of the conditional probabilities on which the predictive value of causes is defined, as assumed by CMT. Furthermore, such ambiguity would have also affected the computation of the predictive value of cues for RW. Correspondingly, in McSo groups, the cues presented were said to be the only cues present on a particular trial. The wording of the inferential ratings required in the EC and CE groups was the same as in Experiments 1a and 1b (i.e., predictiveness ratings), as the alternative wording used in Experiment 2 did not alter the use of causal knowledge.

Results and Discussion

As in previous experiments, only the ratings of those participants who achieved 100% correct responses by the end of the training phase were included in the analysis. No further selection criterion was now used, at variance with previous experiments. Note that for the ScMo groups the relationships arranged between cues and outcomes were nondeterministic, whereas for the McSo groups they were deterministic. Hence, the use of these ratings as a selection criterion was not justified, as there was no obvious way to specify a comparable selection criterion for the ScMo and McSo groups. The ratings of 185 participants were included. The number of participants who were selected from each of the four experimental groups was practically identical. Specifically, the original sample included 67, 68, 53, and 52 participants for the CE–McSo, EC–McSo, CE–ScMo, and EC–ScMo groups, respectively, whereas the selected sample included 55, 56, 37, and 37 for the corresponding groups.

The mean number of trials to reach the learning criterion was similar in all four experimental groups, 148.58, 139.93, 147.89, and 153.43 for the CE–McSo, EC–McSo, CE–ScMo, and EC–ScMo groups, respectively (all Fs < 1). Table 1 shows the ratings for the contingent and noncontingent target cues across the experimental groups. To facilitate the interpretation of the results, Table 3 shows the magnitude of the contingency effect (the difference between the ratings given to the contingent and the noncontingent target cues) across these experimental groups. A 2 (causal order: CE vs. EC) × 2 (cue–outcome structure: McSo vs. ScMo) between-subjects ANOVA was performed on the contingency effect data. As can be seen in Table 1, there were important differences between groups in sample size, hence the analysis used normalized mean-square-error values. The only significant effect was for the cue–outcome structure factor, $F(1, 181) = 8.56$, $p < .01$, $MSE = 1{,}196.79$. Neither the causal order factor nor the Cue-Outcome Structure × Causal Order interaction was significant, $F(1, 181) = 0.08$, $p = .77$, $MSE = 1{,}196.79$, and $F(1, 181) = 2.53$, $p = .11$, $MSE = 1{,}196.79$, respectively.

The cue–outcome structure effect is specifically predicted by RW: The contingency effect was statistically greater in the McSo group than in the ScMo group. According to CMT, in the EC–McSo and in the CE–ScMo groups no contingency effect should

be obtained. Regarding the other two groups (EC–ScMo and CE–McSo), though a contingency effect should be found in both of them, the formulation of CMT is not specific enough to say whether the contingency effects should be of similar magnitude. Note that CMT has been proposed as a computational analysis rather than as a description of an algorithm for how predictive and diagnostic inferences are made. Despite this fact, the reason for expecting a contingency effect in the EC–ScMo group is that diagnostic ratings are sensitive to the causal structure of the task, and this causal structure is identical to that in the CE–McSo group. Thus, though the theory does not explicitly predict a contingency effect of similar magnitude in the two groups, such an assumption does not seem unreasonable. Should this be the case, then, the cue–outcome structure effect would be a misprediction of the model. In addition, the absence of a significant Cue-Outcome Structure × Causal Order interaction contradicts the CMT's predictions, whereas the absence is specifically predicted by RW.

As in previous experiments, despite the nonsignificant interaction, we conducted simple effects comparisons to see whether any more detailed patterns in the data were theoretically relevant. Contrary to what CMT predicts, causal order yielded significant differences in neither the McSo groups (replicating the results from Experiments 1a, 1b, and 2) nor in the ScMo groups, $F(1, 181) = 1.5$, $p = .22$, $MSE = 1{,}196.79$, and $F(1, 181) = 1.09$, $p = .29$, $MSE = 1{,}196.79$, respectively. In addition, as both theories predict, the cue–outcome structure of the task yielded significant differences in the CE groups, $F(1, 181) = 10.25$, $p < .01$, $MSE = 1{,}196.79$. On the other hand, in the EC groups, CMT predicts a greater contingency effect in the ScMo group than in the McSo group. Such a difference was not obtained, $F(1, 181) = 0.80$, $p = .37$, $MSE = 1{,}196.79$, and in fact, it was in the opposite direction. All of these simple effects were consistent with the associative RW account, except this last analysis which, although in the direction expected, was not significant.

Finally, we also analyzed the magnitude of the contingency effect in each experimental group. The contingency effect was significant in the CE–McSo and the EC–McSo groups, $F(1, 181) = 31.50$, $p < .01$, $MSE = 598.39$, and $F(1, 181) = 15.22$, $p < .01$, $MSE = 598.39$, respectively. Though the result of the former group is consistent with CMT's predictions, the result of the latter is inconsistent. In addition, the contingency effect was not significant in either the CE–ScMo or EC–ScMo groups, $F(1, 181) = 0.23$, $p = .63$, $MSE = 598.39$, and $F(1, 181) = 3.80$, $MSE = 598.39$, respectively, though in this last group the effect was marginally significant ($p = .05$). Again, though the result of the former group is consistent with CMT's predictions, the result of the latter is inconsistent, because CMT predicts this effect to be significant. On the other hand, this whole set of results is in agreement with RW.

To sum up, the results show that participants' ratings were strongly influenced by the cue–outcome structure of the task. This suggests, as predicted by the associative view, that the acquisition and use of inferential knowledge strongly depends on the temporal order in which events are experienced. Contrary to CMT, we failed to obtain any reliable evidence of causal order influencing participants' ratings. The simple effects analysis served to corroborate this main result. Nonetheless, the results were not in complete agreement with the associative account. Specifically, the cue–outcome structure factor did not yield significant differences within the EC groups, and there was a sizable, though not conventionally significant, contingency effect in the EC–ScMo group.

There is a possibility that participants treat cue or outcome combinations as configurations that are unrelated to their constituent elements. To what extent might this possibility alter our theoretical conclusions? First, we regard the likelihood of such configural processing as minimal, because the events were described symbolically and because the screen positions of the elements of each compound were randomly ordered. Second, in the ScMo case, CMT would correctly predict an absence of cue competition under both predictive and diagnostic conditions. This is because the critical trial types are now $1–O_X$, $1–O_B$, and $0–O_C$ in the noncontingent case and $2–O_Y$, $0–O_E$, and $2–O_F$ in the contingent case, where X and Y are the configurations AB and DE. Any conditional probabilities computed for the Target $1–O_X$ and $2–O_Y$ relationships (whether predictive or diagnostic) will be identical, and hence no contingency effect would be anticipated.

However, configural representations of cue compounds do not provide any benefit to CMT in terms of explaining the competition effect seen in the EC–McSo condition. Configuration would create functional trial types $X–O_1$, $B–O_1$, and C–no O in the noncontingent case and $Y–O_2$, E–no O, and $F–O_2$ in the contingent case. As in the ScMo case, such trial types would not support a contingency effect according to CMT, as the contingency calculations for $X–O_1$ and $Y–O_2$ are identical.

Experiment 4

In our previous experiments, no significant influence of general and abstract causal knowledge has been revealed on participants' inferential judgments. One possible explanation for this result may relate to the complexity of the causal structure of the task in the EC or diagnostic situation. The information provided here may not be viewed as compatible with a common-cause situation (see Footnote 2 for an explanation and the implications regarding CMT predictions). Some participants may have lacked the cognitive resources required (according to CMT) to make diagnostic inferences in a task with this greater degree of causal complexity and may have disregarded the causal scenario of the task.

Although one might question the utility of a theory that is inapplicable to diagnostic situations involving more than a single cause, Experiment 4 served to evaluate the influence of general and abstract causal knowledge using a cue–outcome structure that was compatible with a common-cause model in the diagnostic condition, that is, a scenario that should facilitate the use of the type of causal knowledge that CMT describes.

As in previous experiments, this evaluation was made by examining possible asymmetries between CE and EC situations regarding a cue competition effect. Specifically, we used an overshadowing design that contrasted two conditions (overshadowing: $AB \rightarrow O_1$ trials and control: $C \rightarrow O_2$ trials; see Table 4 for details and Waldmann, 2001, for the use of an equivalent design). Overshadowing occurs if inferential judgments concerning the predictive value of Cues A or B regarding Outcome O_1 are of a lower magnitude than those of Cue C regarding Outcome O_2. A further advantage of this design is that cues and outcomes are paired in a one-to-one fashion, that is, the same cues are always paired with

Table 4
Design, Sample Size, Mean Ratings, and Standard Errors for Target Relationships in the CE and EC Groups of Experiment 4

	CE[a]		EC[a]	
Experimental condition	*M*	*SE*	*M*	*SE*
Overshadowing: AB → O_1[b]	70.83	6.73	69.07	6.66
Control: C → O_2	93.70	3.88	97.04	2.96
Filler: ∅ → ∅				

Note. A, B, and C refer to cues. In ∅ → ∅ trials, no cue was present and no outcome occurred. CE = cause–effect; EC = effect–cause; O = outcome.
[a] $N = 18$. [b] Target Cues A and B.

the same outcomes and vice versa. In addition to simplifying the causal model underlying the two conditions, this one-to-one relationship may be considered as more plausible with respect to the specific causal scenario used (i.e., problems in a chemical plant), which, in turn, should further facilitate participants' use of causal knowledge.

Nevertheless, the main predictions the two theories make are unaltered. Again, CMT predicts that the manipulation of the causal order of the task should produce asymmetries in predictive and diagnostic inferences. Specifically, only predictive, but not diagnostic, inferences should show the overshadowing effect. According to the second assumption of the theory, if cues are interpreted as causes and outcomes are interpreted as effects (CE situation), the probabilistic contrast required to estimate the magnitude of the $C_A \rightarrow E_1$ or $C_B \rightarrow E_1$ causal link from the overshadowing condition cannot be computed. In fact, participants cannot be certain regarding the causal values of A or B, as both causes were always simultaneously present or absent (i.e., the contrast cannot be performed keeping constant the presence or absence of alternative causal factors, as required). Thus, predictive inferences should reflect this uncertainty. Though the theory does not specify the precise magnitude of ratings, it does predict that this magnitude should be lower than the magnitude of the predictive judgments obtained in the control condition. In this case, the trial types presented allow the computation of the probabilistic contrast required to evaluate the magnitude of the $C_C \rightarrow E_2$ causal link: C → O_2 and ∅ → ∅ trials (no cue was present and no outcome occurred). Thus, Cause C reliably produces Effect 2.

Regarding the EC situation (cues are interpreted as effects and outcomes are interpreted as causes), the causal interpretation of overshadowing and control trial types would be $E_AE_B \leftarrow C_1$, $E_C \leftarrow C_2$. According to the second assumption of CMT, the information provided allows participants to conclude that Cause 1 deterministically produces Effects A and B, and Cause 2 deterministically produces Effect C. In both cases, the underlying causal model is simple and compatible with a common-cause situation, and thus Effects A, B, and C equally and maximally diagnose the presence of their corresponding causes, as these causes are the only possible explanations for their occurrences. Therefore, no overshadowing should be expected.

In contrast, according to RW, the magnitude of the overshadowing effect should be equivalent in the CE and the EC situations. As mentioned before, the learning algorithm forces competition between cues that occur together to gain associative strength when paired with a common outcome. Specifically, in the design used, both Cues A and B in the overshadowing condition will asymptotically end up with half of the maximum associative strength available for Outcome O_1 (assuming equal learning rates for the cues), whereas in the control condition, Cue C will end up with all of the associative strength available, as it is the only relevant predictor for Outcome O_2.

Method

Participants and apparatus. A total of 36 psychology undergraduate students from Málaga University took part in Experiment 4 for course credits. The materials and apparatus were the same as in Experiments 1a–3.

Procedure. As in previous experiments, participants had to learn relationships between the substances that leaked from a chemical plant and the indicator lights that came on in the control room of the plant.

Procedural details were the same as those described in Experiment 2, except in the following respects. The learning phase consisted of 210 trials. Table 4 only shows one set of the overshadowing and control relationships programmed plus the filler (∅ → ∅) type: In fact, a total of three sets were included, using different cues and outcomes. Participants could see in the filler trial type that no outcome occurred in the absence of any cue. Thus, a total of seven trial types were presented. The cues were A to C and the outcomes were O_1 and O_2 (see Table 4). As in previous experiments, cues were assigned to different color names and their initials, and outcomes were assigned to numbers. Specifically, in the first set of relationships, the roles of Cues A to C were assigned to the following specific colors and corresponding initials: B (white), F (fuchsia), and E (emerald). In a second counterbalancing condition, the roles of the colors white and emerald (Cues A and C, respectively) were swapped, and in a third condition, the roles of fuchsia and emerald (Cues B and C, respectively) were also swapped. This way, all possible assignments of cues were made for the overshadowing and control conditions. Orthogonally, for half of the participants, the outcomes were numbered from 1 to 2 (Outcomes O_1 and O_2), and for the other half, the roles of these outcomes were swapped. In the case of the second set, the roles of Cues A to C were assigned to the colors C (pale blue), V (violet), and O (ochre), respectively, and the roles of Outcomes O_1 and O_2 were assigned to Numbers 3 and 4, respectively. In the case of the third set, the roles of Cues A to C were assigned to the colors M (brown), R (red), and G (gray), respectively, and the roles of the Outcomes O_1 and O_2 were assigned to Numbers 5 and 6, respectively. The same counterbalancing procedure was also adopted for this second and third set of programmed relationships.

The sequence of trials was randomized for each participant with the single restriction of being presented in blocks of 7 trials that included every trial type, though such blocks were not marked to participants. From Trial 21 onwards, each trial included explicit visual information about the percentage of correct responses across the last 21 trials, as in previous experiments. A learning criterion was included so that the training phase stopped whenever 210 trials had occurred or the participant had correctly responded in three complete blocks of trials, whichever occurred first (in fact, all participants met this second condition first).

Once the training phase had finished, participants were required to make predictiveness ratings of all cues regarding the different outcomes, though again only those judgments concerning the Target A → O_1, B → O_1, and C → O_2 relationships were analyzed. As three set of cues and outcomes were included and a counterbalancing procedure was adopted, participants' judgments were collapsed into a single measure, one each for the overshadowing and control conditions.

Results and Discussion

All participants reached the learning criterion and, consequently, were included in the analysis. As all cues were targets, no control questions referring to nontarget cues were used as a transfer of knowledge selection criterion, at variance with Experiments 1a–2.

The mean number of trials to reach the learning criterion was similar in both the CE and the EC causal order, 72.72 and 64.59, respectively ($F < 1$). Table 4 shows the mean ratings for the target overshadowing and control relationships. The main effect of contingency was significant, $F(1, 34) = 30.92$, $p < .01$, $MSE = 376.09$. Neither the main effect of causal order nor the Contingency × Causal Order interaction was significant, $F(1, 34) = 0.20$, $p = .90$, $MSE = 644.43$, and $F(1, 34) = 0.31$, $p = .58$, $MSE = 376.09$, respectively. As in previous experiments, the contingency effect was not statistically different in the CE and EC groups and was independent of the causal roles of the cues and outcomes (if anything, the effect tended to be greater in the EC group), contrary to CMT's predictions but consistent with the associative account. This result is especially informative, as the potential influence of causal knowledge on inferential judgments was evaluated in a relatively simple causal scenario for which CMT predictions should have been, at least in principle, more easily revealed.

General Discussion

The main goal of this work has been to evaluate the possible influence of causal knowledge on the acquisition and use of inferential knowledge and, in turn, its theoretical implications. This objective has been met by trying to understand the conditions under which participants' inferential judgments are sensitive to the contingency between cues and outcomes. According to CMT, a contingency effect should be observed if it is the contingency of causes that is manipulated but not if it is the contingency of effects. On the other hand, according to RW, a contingency effect should be observed if it is the contingency of cues that is manipulated, beyond the causal interpretation of cues and outcomes.

The results have consistently revealed that inferential ratings are sensitive to contingency, regardless of causal order (Experiments 1–4). This irrelevance of causal order has been shown in several ways. First, causal order was manipulated while keeping constant the cue–outcome structure of the task (Experiments 1a, 1b, and 2). Following this strategy, the results showed that CE and EC inferences were equivalent with respect to the contingency effect. Second, causal order and the cue–outcome structure of the task were orthogonally manipulated (Experiment 3). The results showed that participants' ratings were strongly influenced by the cue–outcome structure. Third, we extended our previous results to the much simpler causal structure embodied in an overshadowing design (Experiment 4), a causal structure that, according to CMT, should have facilitated the use of causal knowledge. Therefore, these results clearly contradicted CMT's assumptions. On the other hand, the general pattern of results conformed to what is predicted by an associative account, according to which cue–outcome structure but not causal order should be important. Nevertheless, there are some aspects that are not consistent with such an account. For example, in Experiment 3, the difference in the magnitude of the contingency effect between the EC–McSo and EC–ScMo groups was not significant, at variance with RW's predictions.

One aspect that we have left aside is the nonsignificant Contingency × Causal Order interaction in Experiments 1a, 1b, and 2 and in the McSo groups in Experiment 3. Bearing in mind the crucial importance of this interaction, specifically predicted by CMT, we conducted a final analysis to provide participants' ratings with a further opportunity to reveal their sensitivity to causal order. In absolute terms, the contingency effect tended to be of greater magnitude in the CE condition than in the EC condition. Given that there were no specially salient procedural differences between the experiments (the consistency of the results speaks in favor of this), we pooled the data from all of those participants who had been selected for previous analyses (Experiments 1a–2 and the McSo groups of Experiment 3) and conducted a new analysis to determine the size of this interaction effect. A 2 (contingency: contingent vs. noncontingent) × 2 (causal order: CE vs. EC) mixed ANOVA was performed on participants' mean ratings. A total sample of 262 participants was included. As in the analysis reported for each of the separate experiments, the main effect of contingency was significant, $F(1, 260) = 102.01$, $p < .01$, $MSE = 868.86$. Neither the main effect of causal order nor the Contingency × Causal Order interaction was significant, $F(1, 260) = 0.38$, $p = .54$, $MSE = 1{,}195.77$, and $F(1, 260) = 3.67$, $MSE = 868.86$, respectively, though this last interaction is now marginally significant ($p = .06$). The size of the nonsignificant interaction effect was small ($f = .12$; Cohen, 1988). And again, a significant contingency effect was found within the EC group, $F(1, 260) = 33.23$, $p < .01$, $MSE = 868.86$. The results from these analyses serve to corroborate the main result obtained in the experimental series, namely, that there is no compelling evidence of an influence of causal knowledge on the acquisition and use of inferential knowledge. The size of such an influence on inference ratings was small and not conventionally significant even with this pooled sample of participants. Furthermore, the specific prediction of CMT concerning the null effect of the contingency factor within the EC group was systematically contradicted.

There has not been extensive previous work on the evaluation of the influence of causal knowledge on the acquisition and use of inferential knowledge, and the results reported thus far are somewhat conflicting. Some of these studies (Cobos, Caño, López, Luque, & Almaraz, 2000; Matute et al., 1996; Price & Yates, 1995, Experiments 3 and 4) have been unable to show the sort of influence of general and abstract causal knowledge envisaged by CMT, consistent with the results reported here. On the other hand, Waldmann (2000, Experiments 3a and 3b) has reported evidence showing an influence of causal knowledge consistent with CMT and contradicting RW predictions. Specifically, he found asymmetries between predictive and diagnostic inferences in situations where the contingency of different target cues was manipulated.

Other studies have found differences between inferences in CE and EC situations, although they provide only partial support for CMT (Van Hamme et al., 1993; Waldmann, 2000, Experiments 1 and 2). However, Van Hamme et al. (1993) also provided only weak evidence against RW, because in their EC condition, the cue–outcome direction of training could be interpreted as inconsistent with the outcome-cue direction of the test question and, in principle, the RW model's predictions are only defined for situations in which the direction of training and testing are identical.

The results of Waldmann's (2000) Experiments 1 and 2, though clearly problematic for the associative account, are not as clearly predicted from CMT as Waldmann argued. In Experiment 1, the results showed that participants' predictive ratings about a blocked and an overshadowed target cue differed, though the relevant conditional contrasts for each target cue could not be computed according to the information provided. Because CMT predicts that all of these ratings should reflect participants' uncertainty regarding the predictive value of the cues, it is hard to see how the obtained difference could be predicted from CMT without further assumptions beyond the theory. In Experiment 2, within a common or single-cause EC situation, the results showed that the diagnostic value of a target effect decreased when the causal power of this single cause to produce the target effect also decreased. Waldmann argued that this effect was predicted by CMT, because diagnostic inferences depend on causal strength in the cause–effect direction. However, this prediction is clearly beyond CMT. The theory assumes that diagnostic inferences also depend on the existence of alternative causes and, because CMT does not specify how these two factors are integrated, it is as compatible with the obtained effect as with the absence of the effect. For example, according to the Bayesian framework for diagnostic reasoning (i.e., a particular way to integrate such factors), if a single cause is the only possible reason for the effect to occur, the diagnostic value of the effect should be maximal regardless of the causal strength in the cause–effect direction (e.g., rain may be taken as a perfect diagnostic signal for clouds, though the causal power of clouds to produce rain is far from perfect).

Thus, neither the evidence reviewed nor the experiments reported here should be regarded as implying that individuals either lack general and abstract causal knowledge or that they cannot make use of it under any circumstances. However, this evidence also shows that the circumstances under which a CMT-like influence is observed are quite restricted and, correspondingly, that the circumstances under which causal inferences may be mediated by the operation of an associative learning mechanism are quite broad.

Therefore, a step forward that should guide future research in this field would be to detail the specific circumstances that allow one to find one pattern of results or the other. At present, the procedures used here and in other studies are different in so many ways that little may be concluded regarding the nature of such circumstances. In principle, it seems reasonable to argue that differences in the processing demands of the specific procedures used may be a relevant factor. By way of illustration, the causal scenarios involved in Waldmann's (2000) Experiments 3a and 3b included such a small absolute number of causal events (three different cues and two different outcomes) that his results may be regarded as speaking more of what individuals, under ideal circumstances, would be able to do rather than what individuals actually do in real-world situations. It seems reasonable to argue that very often people have to make predictive or diagnostic decisions in which many relevant causal events need to be considered and numerous causal relationships need to be acquired almost simultaneously. For example, imagine a group of people engaged in lively conversation. Words, gestures, tone of voice, prosody elements, specific expressions in one of the interlocutors, and so forth are all effects caused by certain properties of this interlocutor's mental state, all of which the other interlocutors need to know about for effective conversation. To make things even more complex, some of these causal relationships may well be fairly specific for a particular interlocutor and do not easily generalize to others. Thus, causal learning usually takes place in the context of a complex web of numerous causal relationships that are learned nearly simultaneously. The problem with Waldmann's (2000) result, thus, is that it may not generalize to more complex (i.e., real) situations. And, in fact, this is what the results reported in the present series have shown: In more complex situations (i.e., involving a greater number of causal events in our Experiment 4—nine different cues and six different outcomes), a person's performance is not sensitive to causal order.

No doubt, a complete specification of what these circumstances are would require a clearer specification of the information-processing details of the cognitive processes involved. In this sense, CMT may be regarded as a rational or computational analysis of predictive and diagnostic causal learning rather than a theory of the actual cognitive processes involved in this type of learning. It provides a specification of the objectives or computations an individual has to satisfy to correctly acquire and use inferential knowledge in predictive and diagnostic situations, but it does not describe the details of the cognitive processes that actually carry out such computations. Thus, although CMT provides a step forward in the rational analysis of induction and inference in causal contexts, it only very poorly characterizes the cognitive processes involved in the acquisition and inferential use of knowledge.

On the other hand, the associative account considered here may be regarded as a detailed specification of the processes involved in inferential learning, at least under some circumstances. In relation to what those circumstances are, we may tentatively argue that they are learning tasks that exhibit a certain degree of complexity regarding, for example, the number of events involved or the tasks in which participants are required to make predictions concerning what the correct outcome is on a trial-by-trial basis. Under such circumstances, associative learning mechanisms seem to determine participants' performance during the learning phase and also during the subsequent judgment stage. Thus, the knowledge base developed during the learning task by the associative mechanism consisting of the predictive values of the cues constitutes the main source of knowledge on which inferences are based.

The price the individual has to pay is that of suboptimality, because the operation of the associative mechanism is insensitive to some of the demands made by a rational analysis of inference in causal contexts, for example, sensitivity to the causal order of the task (see López et al., 1998, for a similar analysis regarding other deviations of individuals' performance from a rational analysis of inferential learning or from a rational analysis of some reasoning tasks). Nevertheless, the price does not seem to be very high; as Hume (1777/1993) stated, "nor can an operation of such immense consequence in life, as that of inferring effects from causes, be trusted to the uncertain process of reasoning and argumentation"(p. 71). Moreover, if the inferences carried out by the associative mechanism yield adaptive behavior (e.g., a correct solution of the learning task), then it is not unreasonable to regard such behavior as in some sense rational (Evans & Over, 1996).

In any case, future research should further determine the value of associative mechanisms in our understanding of individuals' performance in inferential learning situations, for example, through an evaluation of whether performance also deviates from the dictums of a rational analysis (e.g., CMT) in other specific ways predicted by these associative mechanisms. Furthermore, future research should also clarify the circumstances under which associative mechanisms are and are not engaged in inferential learning situations.

References

Allan, L. G. (1993). Human contingency judgments: Rule based or associative? *Psychological Bulletin, 114,* 435–448.

Chapman, G. B., & Robbins, S. J. (1990). Cue interaction in human contingency judgment. *Memory and Cognition, 18,* 537–545.

Cheng, P. W. (1997). From covariation to causation: A causal power theory. *Psychological Review, 104,* 367–405.

Cheng, P. W., & Holyoak, K. J. (1995). Complex adaptive systems as intuitive statisticians: Causality, contingency, and prediction. In H. L. Roitblat & J. A. Meyer (Eds.), *Comparative approaches to cognitive science* (pp. 271–302). Cambridge, MA: MIT Press.

Cheng, P. W., & Novick, L. R. (1990). A probabilistic contrast model of causal induction. *Journal of Personality and Social Psychology, 58,* 545–567.

Cheng, P. W., & Novick, L. R. (1992). Covariation in natural causal induction. *Psychological Review, 99,* 365–382.

Cobos, P. L., Caño, A., López, F. J., Luque, J. L., & Almaraz, J. (2000). Does the type of judgment required modulate cue competition? *Quarterly Journal of Experimental Psychology, 53B,* 193–207.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Dickinson, A. (1980). *Contemporary animal learning theory*. Cambridge, England: Cambridge University Press.

Evans, J. St. B. T., & Over, D. E. (1996). *Rationality and reasoning*. Hove, England: Psychology Press.

Gluck, M. A., & Bower, G. H. (1988). From conditioning to category learning: An adaptive network model. *Journal of Experimental Psychology: General, 117,* 227–247.

Hume, D. (1993). *An enquiry concerning human understanding*. Indianapolis, IN: Hacket. (Original work published 1777)

López, F. J., Cobos, P. L., Caño, A., & Shanks, D. R. (1998). The rational analysis of human causal and probability judgment. In M. Oaksford & N. Chater (Eds.), *Rational models of cognition* (pp. 314–352). Oxford, England: Oxford University Press.

Matute, H., Arcediano, F., & Miller, R. (1996). Test question modulates cue competition between causes and between effects. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 22,* 182–196.

McClelland, J. L., & Rumelhart, D. E. (1985). Distributed memory and the representation of general and specific information. *Journal of Experimental Psychology: General, 114,* 159–188.

Pearl, J. (1988). *Probabilistic reasoning in intelligent systems: Networks of plausible inference*. San Mateo, CA: Morgan Kaufmann.

Price, P. C., & Yates, J. F. (1995). Associative and rule-based account of cue interaction in contingency judgment. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 21,* 1639–1655.

Reichenbach, H. (1956). *The direction of time*. Berkeley: University of California Press.

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

Shanks, D. R. (1995). *The psychology of associative learning*. Cambridge, England: Cambridge University Press.

Shanks, D. R., & López, F. J. (1996). Causal order does not affect cue selection in human associative learning. *Memory and Cognition, 24,* 511–522.

Sutton, R. S., & Barto, A. G. (1981). Toward a modern theory of adaptive networks: Expectation and prediction. *Psychological Review, 88,* 135–170.

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

Waldmann, M. R. (2000). Competition among causes but not effects in predictive and diagnostic learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 26,* 53–76.

Waldmann, M. R. (2001). Predictive versus diagnostic causal learning: Evidence from an overshadowing paradigm. *Psychonomic Bulletin and Review, 8,* 600–608.

Waldmann, M. R., & Holyoak, K. J. (1992). Predictive and diagnostic learning within causal models: Asymmetries in cue competition. *Journal of Experimental Psychology: General, 121,* 222–236.

Waldmann, M. R., & Holyoak, K. J. (1997). Determining whether causal order affects cue selection in human contingency learning: Comments on Shanks and López (1996). *Memory and Cognition, 25,* 125–134.

(*Appendix follows*)

Appendix

English Version of the Instructions Used in the Experiments

Instructions for Experiments 1a–4

The following instructions were used for the different experiments, which were conducted in Spanish, both in the CE (cause–effect) and the EC (effect–cause) causal orders. The bracketed text represents the variations for the EC groups.

> Imagine that you work in a chemical plant. Sometimes the leakage of different substances occurs as a result of accidents in the plant. These substances cause the illumination of different indicator lights that warn you about the leaks. [Experiment 4: In fact, the illumination of the lights can only be produced by the leakage of these substances.[a]] However, you have not been told which indicator light comes on [substance has leaked] when different substances leak [indicator lights come on]. It is important for the safety of the plant that you learn the causal relationships between the leakage of substances (causes) [different indicator lights (effects)] and the different indicator lights (effects) [leakage of substances (causes)]. The system is still under test so a substance may leak [an indicator light may be illuminated] without the illumination of any indicator light [any substance leaking].[b]
>
> Your aim is learning which indicator light will come on [substance has leaked] when the different substances leak [indicator lights come on]. For this, you will be shown a series of examples. On each example, you will know which substance or substances have leaked [indicator or indicators have come on] and you should say which indicator will come on [substance has leaked]. If you make a mistake, the computer will make a *beep*. Moreover, after making your response, you will be told what the right answer was. Use this information to learn the relationships between the different substances [indicator lights] and the indicator lights [substances].
>
> Press the space bar to continue reading the instructions . . . .
>
> On the screen you will see your percentage of correct responses. Try to make as many correct responses as possible. The task ends when you reach a certain number of correct responses.

Experiments 1a–b

> To make the task easier, substances [indicators] are numbered from 1 to 12, so you do not need to remember their names. Indicators [substances] are indexed by different letters.[c]

Experiments 2–4

> To ease the task, substances [indicators] are of different colors, though you only need to remember their initials. The indicators [substances] are numbered from 1 to 4 [Experiment 4: from 1 to 6].
>
> Once you have examined all the examples, you will have to evaluate on a questionnaire to what extent each of the different substances [indicator lights] predicts[d] the illumination of the different indicator lights [substances].
>
> Press the space bar to start . . . .

---

[a] This sentence was added in Experiment 4 to make explicit the idea of a closed world causal scenario. [b] This last sentence was not present in Experiment 4, as its design made it unnecessary. [c] In Experiment 1b, this paragraph of the instructions was varied according to the specific way of indexing substances and indicator lights described in the *Procedure* section of this experiment. [d] In Experiment 2 the word *indicates* was used instead.

Received November 16, 2000
Revision received May 2, 2002
Accepted May 2, 2002 ■