Journal of Experimental Psychology:
Learning, Memory, and Cognition
2005, Vol. 31, No. 2, 228–237

0278-7393/05/$12.00 DOI: 10.1037/0278-7393.31.2.228

Competition Between Antecedent and Between Subsequent Stimuli in Causal Judgments

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In the analysis of stimulus competition in causal judgment, 4 variables have been frequently confounded with respect to the conditions necessary for stimuli to compete: causal status of the competing stimuli (causes vs. effects), temporal order of the competing stimuli (antecedent vs. subsequent) relative to the noncompeting stimulus, directionality of training (predictive vs. diagnostic), and directionality of testing (predictive vs. diagnostic). In a factorial study using an overshadowing preparation, the authors isolated the role of each of these variables and their interactions. The results indicate that competition may be obtained in all conditions. Although some of the results are compatible with various theories of learning, the observation of stimulus competition in all conditions calls for a less restrictive reformulation of current learning theories that allows similar processing of antecedent and subsequent events, as well as of causes and effects.

Stimulus competition is defined as the phenomenon in which responding to a target stimulus (*X*), on the basis of its signaling some event, is weakened as a consequence of *X*'s being trained in the presence of another stimulus (*A*) that better signals the same event (e.g., Kamin, 1968; Pavlov, 1927; Wagner, Logan, Haberlandt, & Price, 1968; Wasserman, 1974). Overshadowing (Pavlov, 1927) is one example of stimulus competition. In overshadowing, presentations of two stimuli in compound, *A* and *X* (e.g., a tone and a light), signaling an impending event (e.g., food) results in weaker conditioned responding to *X* compared with a condition in which *X* is presented alone as a signal for food. That is, although *X* is presented signaling the food an equal number of times in both conditions, participants seem to attribute greater signal value to *X* for the impending food when *X* is presented alone than when it is presented in compound with *A*.

The phenomenon of stimulus competition, which was originally observed in animal conditioning, has also been demonstrated in human causal judgment experiments (e.g., Baker, Mercier, Vallée-Tourangeau, Frank, & Pan, 1993; Dickinson, Shanks, & Evenden, 1984; Matute, Arcediano, & Miller, 1996; Shanks, 1985; Van Hamme, Kao, & Wasserman, 1993; Waldmann & Holyoak, 1992; Wasserman, 1990). These experiments with human participants were similar to the examples mentioned above, but here, instead of lights, tones, and food, participants were exposed to a series of different causes (e.g., fictitious medicines) signaling the occurrence of an effect (e.g., an allergic reaction), and then, at test, they were asked to indicate the degree to which they thought each one of the potential causes was a cause of the effect. Stimulus competition in causal judgment is of theoretical importance because it speaks to the conditions necessary for causal judgment (and learning in general) to occur. Moreover, stimulus competition is widely used nowadays as a tool with which to discriminate between the two most prevalent frameworks that seek to explain causal judgments in humans.

On the one hand, causal judgment has been frequently explained in the framework of the modern associative learning theories (e.g., Cobos, López, Caño, Almaraz, & Shanks, 2002; Shanks & López, 1996). In this framework, the same principles that are applied to animal conditioning are applied to causal judgment. In the majority of associative theories (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972), it is assumed that the establishment of an association (i.e., learning) between two events (e.g., a cause and an effect) requires that the two events occur temporally proximate to each other, and that the event presented first in the event sequence (the antecedent event or cue) predicts nonredundantly the occurrence of the event presented second in the event sequence (the subsequent event or outcome). Thus, in this framework, stimulus competition occurs unidirectionally between antecedent events predicting the same subsequent event, regardless of whether these events are causes or effects (either of which could be

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Support for this research was provided by Departamento de Educación, Universidades e Investigación of the Basque Government Grant PI-2000-12, and by an Auburn University Competitive Research Grant award. We thank Robert Cole, James Denniston, Mirko Gerolin, Lisa Gunther, Philippe Oberling, Nuria Ortega, Oskar Pineño, Gonzalo Urcelay, and Sonia Vegas for comments on a draft of the article, and Abraham Arias, Kenya Castellanos, Jamie Francis, Leanne Scalli, and Tyson Platt for their help in conducting the experiments.

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the antecedent event in the information sequence provided to the participant). In this account, causal judgments proceed independent of any general and abstract causal knowledge that people may have. That is, the causal relationship between the events is not relevant to stimulus competition, but their temporal order of presentation is critical.

On the other hand, human causal judgment has been considered by some researchers as a special type of learning with principles different from those of associative learning. In the causal model theory (Waldmann & Holyoak, 1992), it is assumed that people are sensitive to general and abstract causal knowledge (i.e., people are sensitive to how causal relationships between events are defined), and that people use this knowledge to attribute causal relationships between events. According to this account, the acquisition of causal knowledge cannot proceed independent of the knowledge of the underlying causal structure of the events. Thus, according to Waldmann and Holyoak, people are aware that multiple potential causes of an effect can compete as causes of that effect, independent of their temporal order of presentation (i.e., their being antecedent or subsequent events is irrelevant) and that multiple potential effects of a cause cannot compete as consequences of the cause regardless of whether they are presented first or second (e.g., Waldmann, 2000; Waldmann & Holyoak, 1992).

Experiments analyzing stimulus competition within causal judgment situations have become the benchmark with which to support or reject these two different families of accounts of causal judgments. Most contemporary theories of associative learning (e.g., Rescorla & Wagner, 1972) assume that associations are unidirectional, from antecedent events (cues) to subsequent events (outcomes). This assumption has led to their postulating learning mechanisms that predict stimulus competition only between antecedent events, regardless of whether they are causes or effects. Thus, stimulus competition is expected in animal conditioning between multiple conditioned stimuli that signal an impending unconditioned stimulus but not between multiple unconditioned stimuli that follow a conditioned stimulus (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972); in associative neural networks, competition is expected to occur between inputs but not between outputs (e.g., Shanks & López, 1996); and in causal judgment situations, competition is expected to occur between antecedent stimuli (causes or effects) but not between subsequent stimuli (e.g., Cobos et al., 2002). In contrast, as mentioned above, in the causal model theory (Waldmann & Holyoak, 1992), it is postulated that competition occurs between causes (antecedent or subsequent events) but not between effects.

As can be seen in Figure 1, empirical data often seem to provide support for (but sometimes refute) the claims from each family of accounts of causal judgment. The associative accounts are supported by findings of stimulus competition between antecedent stimuli (Panels A and D) regardless of whether they are causes (with predictive training, i.e., causes followed by effects; e.g., Cobos et al., 2002; Shanks & López, 1996; Waldmann, 2000, 2001; Waldmann & Holyoak, 1992) or effects (with diagnostic training, i.e., effects followed by causes; e.g., Cobos et al., 2002; Price & Yates, 1993; Shanks, 1991; Shanks & López, 1996). However, this evidence has been challenged by failed demonstrations of competition between antecedent effects (e.g., Van Hamme et al., 1993; Waldmann, 2000, 2001; Waldmann & Holyoak, 1992) and by evidence of stimulus competition between subsequent effects (e.g., Esmoris-Arranz, Miller, & Matute, 1997; Matute et al., 1996; Miller & Matute, 1998; but see Cobos et al., 2002).

In contrast with the associative account, the causal model theory has been supported by the findings of stimulus competition between causes (e.g., Cobos et al., 2002; Shanks & López, 1996; Waldmann, 2000, 2001; Waldmann & Holyoak, 1992) and the failed demonstrations of competition between antecedent effects (e.g., Van Hamme et al., 1993; Waldmann, 2000, 2001; Waldmann & Holyoak, 1992). However, this last observation has been challenged by repeated demonstrations of competition between effects (e.g., Cobos et al., 2002; Matute et al., 1996; Price & Yates, 1993; Shanks, 1991; Shanks & López, 1996) and by the failed demonstrations of competition between causes with diagnostic training (i.e., when the effect is presented first; e.g., Cobos et al., 2002; Price & Yates, 1995). Note that the causal model theory predicts competition between causes even when they are presented as subsequent events because, for this account, the causal structure is the relevant factor, and the temporal order of the stimuli is irrelevant. However, competition between subsequent causes is not predicted by the associative accounts because these models assume that subsequent stimuli cannot compete.

Interpreting the results from studies of stimulus competition in causal judgments has been complicated because every reported experimental finding seems to be susceptible to more than one interpretation. For example, the most straightforward and best supported evidence of stimulus competition, competition between causes with predictive training (e.g., lower ratings to Cause X when *X* was presented together with Cause A predicting a subsequent effect) can be interpreted as evidence for (a) competition between causes, (b) competition between antecedent stimuli, and (c) competition with predictive training. However, to conclude that causes always compete (as is predicted in the causal model theory), one must demonstrate competition between causes with diagnostic training, not just with predictive training. To conclude that antecedent stimuli always compete (as is predicted in associative models), one must demonstrate competition between causes presented as antecedents during predictive training and competition between effects presented as antecedents during diagnostic training. Finally, to conclude that competition occurs exclusively when training is predictive, one must demonstrate competition when causes are followed by effects during training and not when effects precede causes.

The situation is further complicated by test variables. The questions used to assess stimulus competition can also be predictive (from causes to effects) or diagnostic (from effects to causes). In the associative account, it is assumed that competition can occur only unidirectionally between antecedent stimuli signaling a subsequent stimuli, and, probably because of this assumption, its supporters have tested only competition between causes with predictive training and predictive test questions and competition between effects with diagnostic training and diagnostic test questions (e.g., Cobos et al., 2002; Shanks, 1991; Shanks & López, 1996). That is, it has been assumed that training and testing must have the same forward directionality to observe competition (i.e., from antecedent events to subsequent events, regardless of their causal status). In contrast, in the causal model theory, it is assumed that competition between causes can occur and competition between effects will never be observed (e.g., Waldmann, 2000; Waldmann & Holyoak, 1992, 1997).

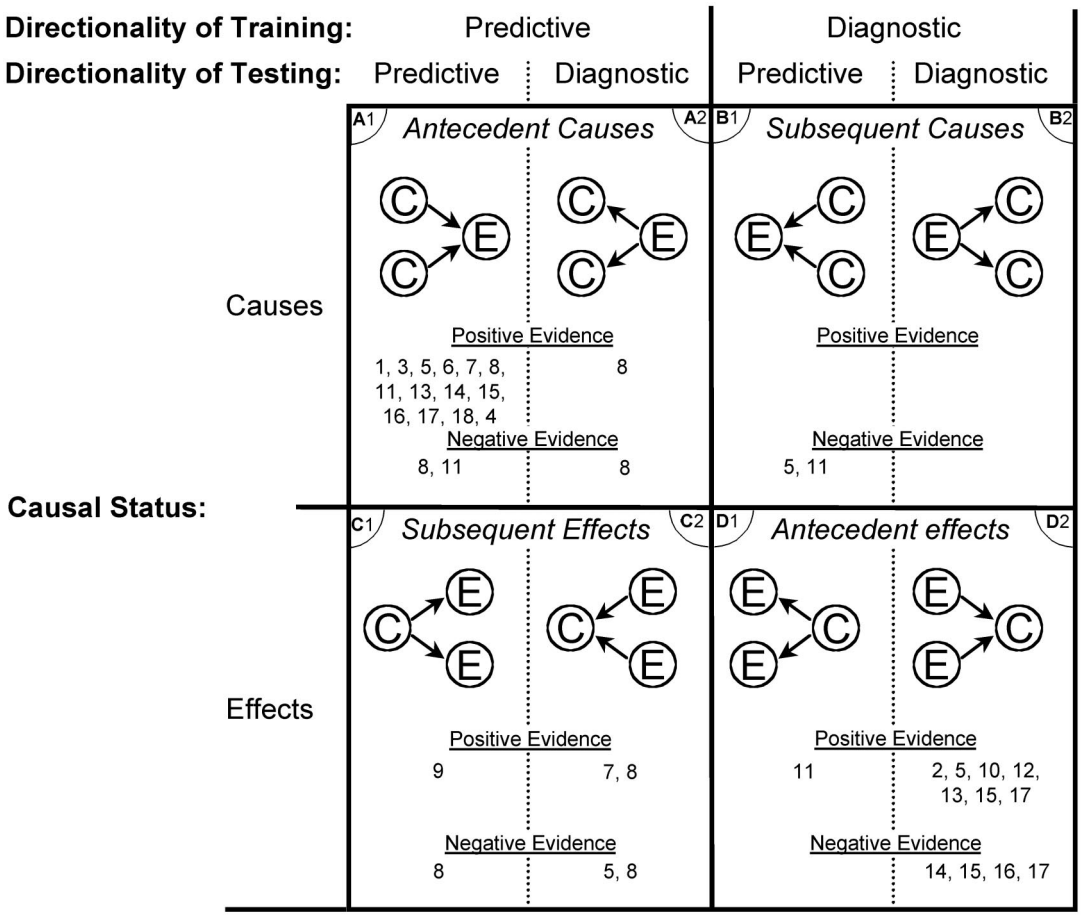

Figure 1. The eight panels show the critical variables examined in this research. The variables that may be responsible for competition in causality judgments are (a) causal status of the competing stimuli: causes (C) versus effects (E); (b) directionality of training: predictive (cause-to-effect) versus diagnostic (effect-to-cause); (c) directionality of the test question: predictive (cause-to-effect) versus diagnostic (effect-to-cause); and (d) temporal order of presentation of the competing stimuli: antecedent versus subsequent. For each form of competition, the positive and negative evidence is listed in the corresponding panel. 1 = Baker et al., 1993; 2 = Chapman, 1991; 3 = Chapman & Robbins, 1990; 4 = Cobos et al., 2000; 5 = Cobos et al., 2002; 6 = Dickinson et al., 1984; 7 = Esmoris-Arranz et al., 1997; 8 = Matute et al., 1996; 9 = Miller & Matute, 1998; 10 = Price & Yates, 1993; 11 = Price & Yates, 1995; 12 = Shanks, 1991; 13 = Shanks & López, 1996; 14 = Van Hamme et al., 1993; 15 = Waldmann & Holyoak, 1992; 16 = Waldmann, 2000; 17 = Waldmann, 2001; 18 = Wasserman, 1990.

In summary, four variables can be identified that may have potential impact on stimulus competition in human causal judgment (see Figure 1):

1. Causal status. Whether the competing stimuli are causes (Panels A1, A2, B1, and B2 of Figure 1) or effects (Panels C1, C2, D1, and D2), regardless of their temporal order of presentation in training or testing.

2. Temporal order of the stimuli. Whether competition occurs between antecedent stimuli (Panels A1, A2, D1, and D2) or between subsequent stimuli (Panels B1, B2, C1, and C2), independent of their being causes or effects.

3. Directionality of training. Whether training is predictive (i.e., causes are presented first; Panels A1, A2, C1, and C2) or diagnostic (i.e., effects are presented first; Panels B1, B2, D1, and D2), independent of the competing stimuli being causes or effects.

4. Directionality of testing. Whether the test question used to assess competition is predictive (i.e., asking for effects of causes; A1, B1, C1, and D1) or diagnostic (i.e., asking for causes of effects; A2, B2, C2, and D2).

For instance, competition between causes does not necessarily require predictive training and testing (Panel A1) but can also be studied with any other combination of predictive or diagnostic training and testing (Panels A2, B1, and B2). Likewise, studying competition between effects does not necessarily require diagnostic training and testing (Panel D2) but can also be accomplished

with any other combination of predictive and diagnostic training and testing (Panels D1, C1, and C2). More generally, any condition in which there are multiple stimuli linked to one stimulus is potentially susceptible to competition among the multiple stimuli regardless of whether they are causes or effects, antecedent or subsequent stimuli, whether they are trained in the predictive or diagnostic direction, and whether they are tested in the predictive or diagnostic direction. Each of these four potentially critical variables in stimulus competition should be studied, without their being confounded with any of the other three variables, and the potential interactions between them also requires examination in order to determine the roles they play in producing competition and their relative weights in doing so.

In the following experiment, we manipulated the role of the aforementioned four variables within an overshadowing procedure. Training information was presented to the participants in a trial-by-trial format. While so doing, we systematically varied the causal status of the competing stimuli (causes vs. effects), the directionality of training (predictive vs. diagnostic), the directionality of testing (predictive vs. diagnostic test questions), and the temporal order of presentation of the competing stimuli (antecedent vs. subsequent) during training, all in a single integrative study.

Overview of the Experiment

We used an overshadowing procedure (e.g., Pavlov, 1927) in order to analyze some of the different variables that play a role in stimulus competition in causality judgment (see Figure 1). As previously mentioned, in an overshadowing procedure, the target stimulus is trained in compound with another stimulus as a signal for an impending event, which results in lower ratings of the target stimulus than would be obtained in a control condition in which it was paired alone with the impending event.

We manipulated whether causes or effects were the potentially competing stimuli (i.e., multiple causes of one effect or multiple effects of one cause) and whether causes or effects were presented first (antecedent) or second (subsequent) in the event sequence on each training trial (see Figure 1 for an explanation of the variables and Table 1 for the experimental design). Thus, we controlled for the influence on stimulus competition of the causal status of the competing stimuli (causes vs. effects), the directionality of training (predictive vs. diagnostic), and the temporal order of presentation of the competing stimuli (antecedent vs. subsequent). In addition, we controlled for the influence of the directionality of the test question, asking participants to answer predictive and diagnostic test questions at the end of training.

The experiment was completely within-subjects (i.e., every participant was exposed to all the conditions and was tested with all the test questions). There was a single cover story for all the conditions. Participants had to learn the relationships among eight causes and eight effects. The causes were the ingestion of various foods by fictitious patients, and the effects were adverse reactions to those foods. The role of every food as a cause and the role of every adverse reaction as an effect were counterbalanced between participants to control for any potential impact of their familiarity or prior experience. During training, participants had to guess, after the presentation of the first stimulus or stimuli on each trial, what stimulus or stimuli would follow. Thus, we were able to record the progress of learning of these relationships during training and assess whether participants were able to learn these relationships despite the complexity of the task. This allowed us to determine whether a low rating to a given stimulus during testing was the result of a deficit in acquiring the information.

Method

Participants and Apparatus

Twenty-eight undergraduate students from the State University of New York at Binghamton and 22 undergraduate students from Auburn Univer-

Table 1
Design Summary

	Training variables				Test variables	
Condition	Training	Causal status	Temporal order	Directionality	Directionality	
Competing cause → effect (control)	C1 → E1		Antecedent	Predictive	C1 → E1? (pred)	E1 → C1? (diag)
Competing causes → effect (experimental)	C2 C3 → E2	Multiple causes	Antecedent	Predictive	C2 → E2? (pred) C3 → E2? (pred)	E2 → C2? (diag) E2 → C3? (diag)
Effect → competing cause (control)	E3 → C4		Subsequent	Diagnostic	C4 → E3? (pred)	E3 → C4? (diag)
Effect → competing causes (experimental)	E4 → C5 C6	Multiple causes	Subsequent	Diagnostic	C5 → E4? (pred) C6 → E4? (pred)	E4 → C5 (diag) E4 → C6 (diag)
Cause → competing effect (control)	*C1 → E1*		Subsequent	Predictive	*C1 → E1? (pred)*	*E1 → C1? (diag)*
Cause → competing effects (experimental)	C7 → E5 E6	Multiple effects	Subsequent	Predictive	C7 → E5? (pred) C7 → E6? (pred)	E5 → C7? (diag) E6 → C7? (diag)
Competing effect → cause (control)	*E3 → C4*		Antecedent	Diagnostic	*C4 → E3? (pred)*	*E3 → C4? (diag)*
Competing effects → cause (experimental)	E7 E8 → C8	Multiple effects	Antecedent	Diagnostic	C8 → E7? (pred) C8 → E8? (pred)	E7 → C8? (diag) E8 → C8? (diag)

Note. C1–C8 are eight different causes; E1–E8 are eight different effects. Temporal order refers to whether the potentially competing stimuli were presented in the antecedent (cue) or subsequent (outcome) position of the trial pairing. Predictive or pred = predictive directionality (from cause to effect), and diagnostic or diag = diagnostic directionality (from effect to cause). Conditions listed twice (for clarity) are in italics the second time they are listed.

sity participated in the study. All Binghamton students participated in the study as partial fulfillment of a course requirement. All Auburn University students received extra credit within a course in return for their participation. The experiment was conducted in small rooms containing one to four personal computers.

Design

The five variables manipulated in the experiment were the following.

Experimental condition (experimental vs. control). There were four experimental conditions and two control conditions. For the experimental (overshadowing) conditions, a compound of two causes or two effects was paired with its corresponding effect or cause. For the control conditions, one cause or one effect was paired with its corresponding effect or cause.

Causal status (multiple causes vs. multiple effects). The potentially competing stimuli were causes for half of the conditions and effects for the remaining half.

Temporal order of presentation of the competing stimuli (antecedent vs. subsequent). The potentially competing stimuli were antecedent (i.e., presented first in the event sequence; either causes or effects) for one half of the conditions and subsequent (i.e., presented second in the event sequence; either causes or effects) for the remaining half.

Directionality of training (predictive vs. diagnostic). Half of the conditions received predictive training; that is, causes were presented before effects during training. The other half received diagnostic training; that is, effects were presented before causes during training. It is important to note that directionality of training was not independent of the temporal order of presentation of the competing stimuli and causal status but, rather, represented the interaction of these two variables. For example, if causes are antecedent stimuli, the directionality of training is, by definition, predictive (although, as shown in Figure 1, either competition between causes or between effects can be studied in such a predictive training condition); if causes are subsequent stimuli, the directionality of training is diagnostic (although competition between causes and between effects can also be studied in this condition). In statistical analysis, any two of these variables could serve as independent variables, with the third variable emerging as the interaction of the first two variables.

Directionality of test (predictive vs. diagnostic). All participants received predictive (from cause to effect) and diagnostic (from effect to cause) test questions for each cause and each effect. The words *predictive* and *diagnostic* were not used on the test questions to facilitate the parallelism between the two types of test questions.

In Table 1, we summarize the design. We manipulated five variables in six within-subjects conditions (four experimental and two control conditions). Note that, in Table 1, eight (not six) conditions were depicted for the sake of clarity with respect to the pairwise comparisons. The C1 → E1 (i.e., Cause 1 predicting Effect 1) condition served as the control condition for competition between causes in the C2 C3 → E2 condition (i.e., Cause 2 and Cause 3 predicting Effect 2). In addition, the C1 → E1 condition served as the control condition for competition between effects in the C7 → E5 E6 condition (i.e., Cause 7 predicting Effect 5 and Effect 6). Likewise, the E3 → C4 condition served as the control condition for competition between effects in the E7 E8 → C8 condition (i.e., Effect 7 and Effect 8 diagnosing Cause 8) and competition between causes in the E4 → C5 C6 condition (i.e., Effect 4 diagnosing Cause 5 and Cause 6).

Procedure

In order to manipulate whether the competing stimuli were causes or effects and whether training was predictive (cause-to-effect direction) or diagnostic (effect-to-cause direction), we introduced a cover story in which the causes were foods that fictitious patients ate and in which the effects were adverse reactions to these foods. Participants were informed that they would first see for each patient either a record of what food(s) the patient ate or what adverse reaction(s) the patient developed. When food(s) were presented first, participants were asked to guess which adverse reaction(s) that patient had developed and were then told what adverse reaction(s) there were; when adverse reaction(s) were presented first, participants were asked to guess which food(s) that patient had eaten and were then told what food(s) had been eaten.

Training and testing was implemented with personal computers. Participants entered their responses by pointing with the mouse cursor and pressing either of the mouse buttons. There were 48 training trials, each of which represented a different fictitious patient. These training trials were organized in eight blocks (undifferentiated for the participant) with one trial of each of the six conditions per block. The order of the conditions within each block for each participant was pseudorandomized and counterbalanced among participants. On each trial, participants were shown two charts, one of which contained causes (i.e., the foods that the fictitious patient ate) and the other of which contained effects (i.e., the adverse reactions that the patient had developed to those foods). Each chart depicted one or two causes or effects, depending on the condition. The foods that served as causes were *chicken*, *cheese*, *mushrooms*, *bananas*, *fish*, *onions*, *rice*, and *carrots*; the adverse reactions that served as effects were *nausea*, *fever*, *rash*, *itching*, *headache*, *constipation*, *insomnia*, and *cramps*. The role of the foods as causes and the adverse reactions as effects were counterbalanced between participants to control for any effect of familiarity. That is, any food could be any of the eight causes, and any adverse reaction could be any of the eight effects.

In the predictive training conditions, the first screen depicted a chart of causes showing the names of one or two foods that the patient had eaten. Below the chart with causes, participants could see all eight potential adverse reactions that could follow that cause(s), and they were asked to select the adverse reaction(s) that they thought that patient had developed. There were no restrictions on the number of selections they could make. That is, they could select none of the choices or all of them. Once participants had made their selections, and after clicking with the mouse cursor on the "accept" button on the screen, they received feedback about the actual adverse reactions that the current patient had developed. The diagnostic training conditions were similar to the predictive training conditions, but the order of the causes and effects was reversed. In the diagnostic conditions, participants had to select from the choices what food(s) they guessed were the causes of the adverse reaction(s) the current patient had developed.

After training, participants had to answer one predictive and one diagnostic test question for each cause and each effect. One half of the participants received first the eight predictive test questions and then the eight diagnostic test questions. For the other half, this order was reversed. The predictive test question read *To what degree do you think that having eaten* name-of-food *is indicative that each of the following adverse reactions will occur?* Below the question, participants were presented with the eight potential adverse reactions, and they had to select using the mouse their rating for each of the effects. Participants rated each effect by selecting a value on a response bar presented above the name of each effect. The bar was anchored at 0 and 100; ratings could be increased or decreased in intervals of one. The diagnostic test question read *To what degree do you think that developing* name-of-adverse-reaction *is indicative of having eaten each of the following foods*? As in the predictive test questions, participants had to select with the mouse their rating for each food in a 0–100 scale. The order of questioning concerning each cause and each effect was counterbalanced among participants. The order of the specific effects and causes to be rated in each predictive and test question was not counterbalanced, but their role in the experimental design was. That is, for example, participants always rated *nausea* first when asked a predictive question, but nausea could be any of the eight potential effects counterbalanced between participants.

Results

Stimulus competition was observed regardless of whether the potential competing stimuli were causes or effects, whether the directionality of training was predictive or diagnostic, whether the directionality of testing was predictive or diagnostic, and whether the competing stimuli were presented in the antecedent or the subsequent temporal order. Thus, the only variable that influenced the participants' judgments in an important way seemed to be whether the test stimuli had been in the experimental (overshadowing) or control condition. Figure 2 depicts participants' ratings for each target causal relationship.

Figure 3A depicts participants' responding during training for each one of the conditions. As can be observed, participants were sensitive to the causal relationships between the stimuli during training. At the end of training, participants discriminated between the correct and incorrect choices. Figure 3B depicts the predictive and diagnostic ratings during testing for each cause and each effect. As during training, participants discriminated which causes were followed by which effects and vice versa. Thus, it seems that participants successfully discriminated the stimuli during training, and that stimulus competition at test did not reflect a deficiency of acquisition but a deficit due to overshadowing per se.

For statistical comparisons, when two causes or two effects were presented in compound, we used the average of their predictive–diagnostic ratings as the dependent variable for their respective comparisons. Thus, for example, when comparing competition between causes in predictive training (i.e., C1→E1 vs. C2 C3→E2), we averaged the ratings of Cause 2 and Cause 3 predicting Effect 2, and we compared the mean with the ratings of Cause 1 predicting Effect 1.

A 2 (experimental condition: experimental vs. control) × 2 (causal status: competing causes vs. competing effects) × 2 (directionality of training: predictive vs. diagnostic) × 2 (directionality of test: predictive vs. diagnostic) analysis of variance (ANOVA) performed on the ratings for the target stimulus in each condition yielded a main effect of experimental condition (experimental vs. control), $F(1, 49) = 43.05$, $MSE = 3{,}104$, $p < .0001$, and no other main effect or interaction (all Fs < 1.14, all ps $> .29$). Although there were no interactions, we broke down all the relevant comparisons to see the impact of the variables on each one of the cells of Figure 1. Planned comparisons (see Table 2 for specific statistical values) showed that stimulus competition (lower ratings for the experimental conditions than the control conditions) had occurred regardless of the causal status of the competing stimuli, direction of training, and direction of testing. Thus, the results were indicative of stimulus competition in all possible conditions.

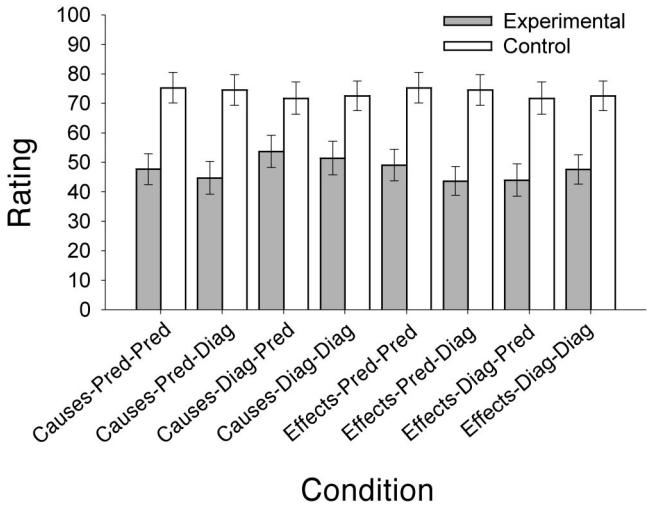

Figure 2. Mean ratings at test. Filled bars represent the experimental conditions, and open bars represent the control conditions. Brackets represent the standard error of the mean. Labels for the conditions represent, from left to right, causal status (causes vs. effects), directionality of training: predictive (Pred) versus diagnostic (Diag), and directionality of testing: predictive (Pred) versus diagnostic (Diag).

In order to analyze the influence of temporal order (antecedent vs. subsequent, independent of their directionality of training and causal status), we performed an additional 2 (experimental condition: experimental vs. control) × 2 (causal status: competing causes vs. competing effects) × 2 (temporal order: antecedent vs. subsequent) × 2 (directionality of test: predictive vs. diagnostic) ANOVA. Although the main effects and interactions were the same as in the former ANOVA, we needed this additional comparison to analyze the impact of temporal order (antecedent vs. subsequent stimuli) on stimulus competition, because temporal order and directionality of training were nonorthogonal variables. As can be seen in Table 2, stimulus competition was observed with both antecedent and subsequent stimuli regardless of whether they were causes or effects.

Discussion

Our results show that the phenomenon of stimulus competition in human causal judgment tasks does not categorically depend on the causal status of the stimuli (competing causes or competing effects), the temporal order of the competing stimuli (antecedent or subsequent), the directionality of the training procedure (predictive or diagnostic), or the directionality of testing (predictive or diagnostic). That is, stimulus competition in causal judgment can be obtained in every possible manipulation depicted in Figure 1.

In the present research, we systematically manipulated these variables factorially in an integrative study, and its results do not fully support any associative account of causal judgment (e.g., Cobos et al., 2002; Shanks & López, 1996) or any of the models that treat causal learning as a special adaptation (e.g., Waldmann & Holyoak's causal model, 1992, 1997). Our results partially support the assumptions of the associative account because we observed competition between antecedent stimuli, independent of whether they were causes (with predictive testing) or effects (with diagnostic testing). However, our results also showed competition between antecedent causes (i.e., predictive training) with diagnostic testing (from effects to causes) and competition between antecedent effects (i.e., diagnostic training) with predictive testing (from causes to effects). These results seem to refute the assumption that a common directionality during training and testing is necessary to observe stimulus competition; that is, they are contrary to the view that associations are formed only from antecedents to subsequent stimuli (for other data relevant to this conclusion, see Arcediano, Escobar, & Miller, in press). Moreover, our results demonstrated competition between subsequent events in-

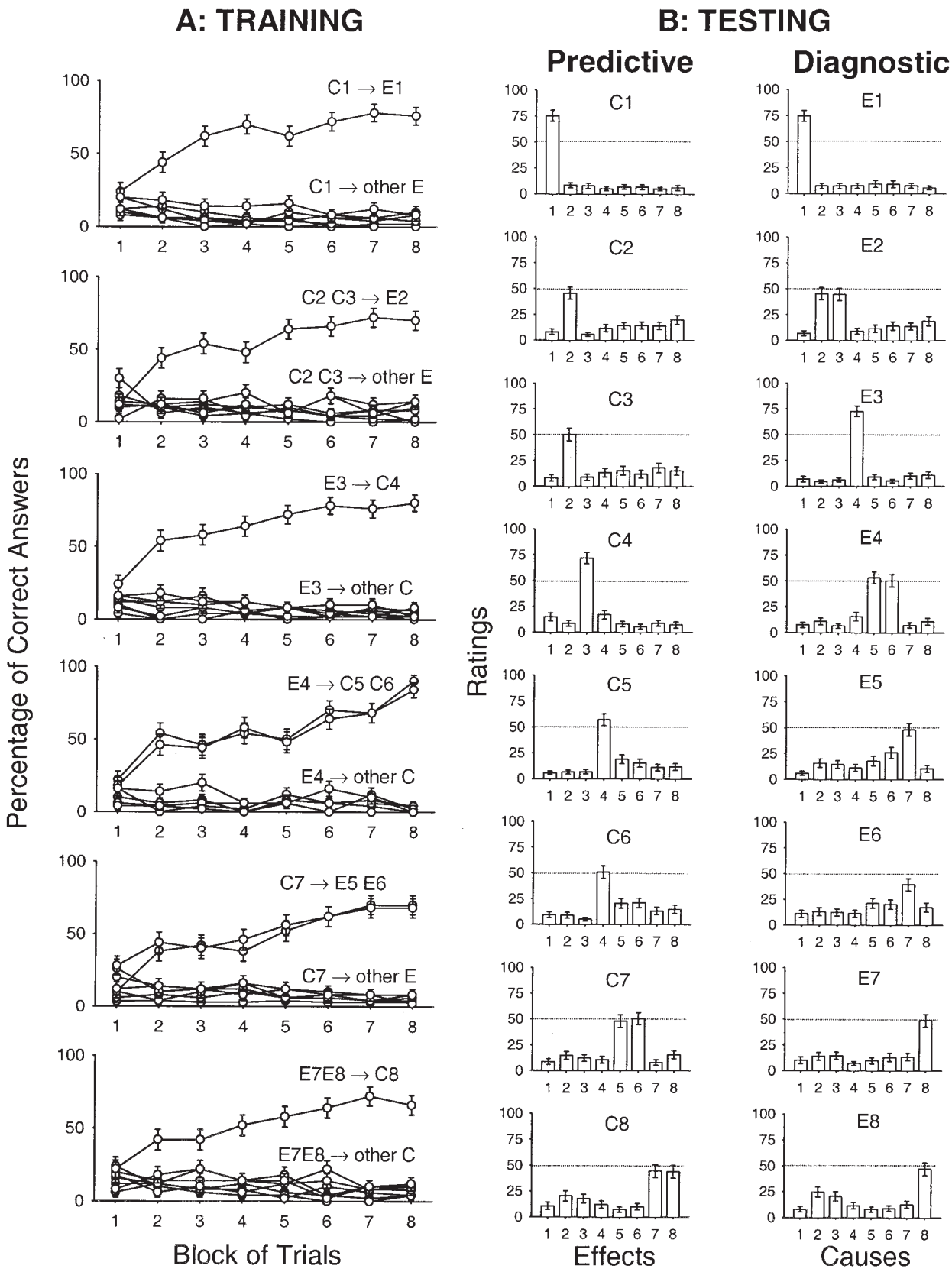

Figure 3. Mean responses during training and testing for all causes (C) and effects (E). A: Acquisition of the relationships between causes and effects. B: The predictive (from causes to effects) and the diagnostic (from effects to causes) ratings at test. Brackets represent the standard error of the mean.

Table 2
Statistical Comparisons for the Target Stimuli

Conditions	*F*	*MSE*	*p*
Experimental condition	43.05	3104.30	.000001
Causes	38.64	1513.16	.000001
Effects	32.98	2296.62	.000001
Predictive training	31.14	2640.88	.000001
Diagnostic training	25.76	2058.35	.000006
Predictive testing	29.60	2104.91	.000002
Diagnostic testing	40.89	1765.95	.000001
Causes with predictive training	26.18	1578.90	.000005
Causes with predictive training and predictive testing	17.15	1114.99	.000136
Causes with predictive training and diagnostic testing	22.73	979.25	.000017
Causes with diagnostic training	17.02	1129.88	.000143
Causes with diagnostic training and predictive testing	8.70	937.74	.004877
Causes with diagnostic training and diagnostic testing	14.89	751.58	.000333
Effects with predictive training	22.61	1808.79	.000018
Effects with predictive training and predictive testing	14.38	1199.14	.000411
Effects with predictive training and diagnostic testing	20.73	1154.70	.000035
Effects with diagnostic training	21.60	1618.55	.000026
Effects with diagnostic training and predictive testing	16.88	1149.71	.000151
Effects with diagnostic training and diagnostic testing	18.08	865.58	.000095
Antecedent stimuli	33.79	2253.58	.000001
Antecedent causes	26.18	1578.90	.000005
Antecedent effects	21.60	1618.55	.000026
Subsequent stimuli	36.73	1582.09	.000001
Subsequent causes	17.02	1129.88	.000143
Subsequent effects	22.61	1808.79	.000018

Note. Planned comparisons conducted from the 2 (experimental condition: experimental vs. control) × 2 (causal status: competing causes vs. competing effects) × 2 (directionality of training: predictive vs. diagnostic) × 2 (directionality of test: predictive vs. diagnostic) and 2 (experimental condition: experimental vs. control) × 2 (causal status: competing causes vs. competing effects) × 2 (temporal order: antecedent vs. subsequent) × 2 (directionality of test: predictive vs. diagnostic) analyses of variance performed on the ratings of the causes and effects in the experiment, Fs(1, 49) = 43.05, MSE = 3,104, ps < .000001.

dependent of whether they were causes or effects. Competition between subsequent stimuli is not consistent with associative accounts of causal judgment based on learning models in which forward unidirectionality of the associations is assumed (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972). Alternatively, our results partially support the assumptions of Waldmann and Holyoak's (1992, 1997) causal model theory, because we observed competition between causes, independent of whether they were antecedent or subsequent events (i.e., whether they were presented with predictive or diagnostic training, respectively). However, contrary to the postulates of the causal model theory, in which it is assumed that people are sensitive to the causal structure of the events and known that common causes of an effect can compete but common effects of a cause cannot, we also observed competition between effects.

Our results are problematic for the two predominant families of accounts of causal judgment. Two findings would allow us to discriminate between these families of accounts: evidence of competition between antecedent effects, which would support the associative accounts but not the causal model account; and evidence of competition between subsequent causes, which would support the causal model account but not the associative account. With respect to competition between antecedent effects, this evidence has not been conclusive (see Figure 1). For example, there have been reports of competition between antecedent effects (e.g., Chapman, 1991; Cobos et al., 2002; Price & Yates, 1993, 1995; Shanks, 1991; Shanks & López, 1996; Waldmann, 2001, Experiment 1; Waldmann & Holyoak, 1992, Experiment 2). However, there have also been reports of failure to obtain competition between antecedent effects (e.g., Van Hamme et al., 1993; Waldmann & Holyoak, 1992, Experiments 1 and 3; Waldmann, 2000, 2001, Experiment 2). Concerning competition between causes presented as subsequent events, there is little prior evidence of this effect. Indeed, Cobos et al. (2002) and Price and Yates (1995) have reported absence of stimulus competition in this situation. However, the results of the present experiment show evidence of competition between subsequent causes. Finally, our most surprising finding, competition between subsequent effects (i.e., effects presented with predictive training), is in strong contradiction to both families of accounts of causal judgment. However, this observation is consistent with prior evidence of competition in this situation (e.g., Esmoris-Arranz et al., 1997; Matute et al., 1996; Miller & Matute, 1998).

In summary, the role of each of the variables investigated in the present research was not adequately isolated in previous research (i.e., they were not analyzed independent of the values of the other potential variables). The present research demonstrated that none of these variables or their interactions were critical in obtaining stimulus competition in causal judgment. Failure to report competition in some conditions of previous studies does not mean that competition never occurs in these conditions. Instead, it demonstrates that these effects can be elusive. But stimulus competition can be elusive even in the most well documented condition, that is, in the case in which causes are presented as antecedent events during training and testing. However, these null results, although well known to researchers, usually are not submitted for publication. That other studies have failed to obtain competition between, for example, effects presented as antecedents should not be taken as strong evidence in favor of any particular theory, because there are many other studies demonstrating that effects presented as antecedent events can compete. There are now enough data to conclude that stimulus competition is not limited to antecedent stimuli or to situations with common causes competing to predict an effect, as has been suggested by the associative account and the causal model account of causal judgment, respectively. Our results show that competition between stimuli occurred when the target stimulus was trained in the presence of another stimulus, regardless of whether they were causes or effects, of whether they were trained in the predictive or diagnostic direction, of whether they were presented in the antecedent or subsequent temporal order, and of whether they were tested with predictive or diagnostic questions. Although our results could be limited to an overshadowing procedure, we expect that they would generalize to other stimulus competition effects, because prior research has not found overshadowing to have properties that are fundamentally different from blocking or stimulus relative validity.

In our view, during training, events (causes or effects) do not compete to become associated with the paired event (an effect or a cause). Instead, we suggest that all antecedent and subsequent events become associated if they are presented contiguously (e.g., the comparator hypothesis; Denniston, Savastano, & Miller, 2001; Miller & Matzel, 1988). In this view, there is no competition during training; it is at the moment of testing (i.e., responding) when, if there is more than one association to the same event, these associations may compete with one another with respect to being activated. The observation of competition during responding may also depend on the test demands. That is, if the test question fosters competition between stimuli, it will be easier to observe competition between the associated events. However, if the test question does not foster competition (e.g., asking about contiguity, cooccurrences, or frequencies), then stimulus competition may not be observed (e.g., Matute et al., 1996; Price & Yates, 1995; but see also Cobos, Caño, López, Luque, & Almaraz, 2000). In addition, we suggest that the associations between stimuli (and any potential competition at testing) may occur in either temporal direction of the occurring events, either forward (from antecedent to subsequent events) or backward (from subsequent to antecedent events; see, e.g., Arcediano, Escobar, & Miller, 2003, for a demonstration of the use of both forward and backward associations, and Esmoris-Arranz et al., 1997, for stimulus competition between subsequent stimuli with respect to an antecedent one).

The issue that remains is why stimulus competition may be observed under certain conditions and not others (see Figure 1). It is difficult to determine which variables account for such divergent results, because the different studies analyzing stimulus competition have used a broad range of preparations, procedures, cover stories, and test questions. Most of these studies manipulate more than one variable, and frequently these variables are confounded. We believe that the relative difficulty in obtaining certain effects (e.g., competition between subsequent events) should not be viewed as evidence of the nonexistence of that effect. Rather, it should be viewed as indicative of the great response flexibility in causal learning, which may sometimes allow for competition between multiple effects of a cause and sometimes prevent such competition. In our view, the study of causal learning is still only beginning, and it would be quite difficult to develop a formal comprehensive model to account for all the different aspects of causal learning at this time. It is unfortunate that very few studies have tried to isolate the impact of specific variables affecting competition between causes or between effects and that, even when that isolation has been attempted, the results have sometimes been contradictory (see, e.g., the disparate results of Cobos et al., 2000; Matute, Arcediano, & Miller, 1996, regarding the impact of test questions on stimulus competition). Likewise, there are multiple possible interpretations for the same effect. Here we have favored the view that causes and effects are subject to the same rules of associative learning. However, it is possible that causes and effects are processed differently (cf. Waldmann & Holyoak, 1992), and that the instances reporting competition between effects reflect, for example, participants' responding to whether there is a unique association between the target effect and the cause rather than regarding the target as an effect of the cause. In either case, the low ratings of the cause–target effect relationship in our experimental conditions follow the pattern of stimulus competition and suggest that causal inference is sensitive to stimulus competition in situations with multiple outcomes.

To conclude, we suggest there are both training variables and testing variables that collectively could explain the disparity of the reported results in this literature. Thus, some training variables (e.g., cover stories, nature of the stimuli, training procedures, and parameters) and some testing variables (e.g., wording that fosters competition and directionality of the events) seem to promote, on the one hand, different degrees of stimulus competition or its absence, depending on their manipulations, and on the other hand, different degrees in the perceived causality, which could affect the directional use of the information. Further research is needed to analyze the impact on causal learning of the variables at play.

References

Arcediano, F., Escobar, M., & Miller, R. R. (2003). Temporal integration and temporal backward associations in human and nonhuman subjects. *Learning and Behavior, 31,* 242–256.

Arcediano, F., Escobar, M., & Miller, R. R. (in press). Bidirectional associations in humans and rats. *Journal of Experimental Psychology: Animal Behavior Processes.*

Baker, A. G., Mercier, P., Vallée-Tourangeau, F., Frank, R., & Pan, M. (1993). Selective associations and causality judgments: Presence of a strong causal factor may reduce judgments of a weaker one. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 19,* 414–432.

Chapman, G. B. (1991). Trial order affects cue interaction in contingency judgment. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17,* 837–854.

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

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

Cobos, P. L., López, F. J., Caño, A., Almaraz, J., & Shanks, D. R. (2002). Mechanisms of predictive and diagnostic causal induction. *Journal of Experimental Psychology: Animal Behavior Processes, 28,* 331–346.

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

Dickinson, A., Shanks, D., & Evenden, J. (1984). Judgment of act-outcome contingency: The role of selective attribution. *Quarterly Journal of Experimental Psychology: Human Experimental Psychology, 36A,* 29–50.

Esmoris-Arranz, F. J., Miller, R. R., & Matute, H. (1997). Blocking of subsequent and antecedent events. *Journal of Experimental Psychology: Animal Behavior Processes, 23,* 145–156.

Kamin, L. J. (1968). "Attention-like" processes in classical conditioning. In M. R. Jones (Ed.), *Miami symposium on the prediction of behavior: Aversive stimulation* (pp. 9–31). Miami, FL: University of Miami Press.

Mackintosh, N. J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. *Psychological Review, 82,* 276–298.

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

Miller, R. R., & Matute, H. (1998). Competition between outcomes. *Psychological Science, 9,* 146–149.

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

Pavlov, I. P. (1927). *Conditioned reflexes.* Oxford, England: Oxford University Press.

Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: Varia-

tions in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review, 87,* 532–552.

Price, P. C., & Yates, J. F. (1993). Judgmental overshadowing: Further evidence of cue interaction in contingency judgment. *Memory & Cognition, 21,* 561–572.

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

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 research and theory* (pp. 64–99). New York: Appleton-Century-Crofts.

Shanks, D. R. (1985). Forward and backward blocking in human contingency judgment. *Quarterly Journal of Experimental Psychology, 37B,* 1–21.

Shanks, D. R. (1991). Categorization by a connectionist network. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17,* 433–443.

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

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

Wagner, A. R., Logan, F. A., Haberlandt, K., & Price, T. (1968). Stimulus selection and a "modified continuity theory." *Journal of Experimental Psychology, 76,* 171–180.

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 learning: Evidence from an overshadowing paradigm. *Psychonomic Bulletin & 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 Lopez (1996). *Memory & Cognition, 25,* 125–134.

Wasserman, E. A. (1974). Stimulus-reinforcer predictiveness and selective discrimination learning in pigeons. *Journal of Experimental Psychology, 103,* 284–297.

Wasserman, E. A. (1990). Attribution of causality to common and distinctive elements of compound stimuli. *Psychological Science, 1,* 298–302.

Received July 31, 2003
Revision received August 13, 2004
Accepted August 20, 2004 ■