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Cue Competition in Causality Judgments: The Role of Nonpresentation of Compound Stimulus Elements

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College students rated the causal efficacy of Elements X, A, and B of food compounds AX and BX in producing the allergic reaction of a hypothetical patient. The results of a 16-day allergy test were presented to subjects in a serial, trial-by-trial manner. The response format used was a running estimate, in which subjects were asked to rate *all* of the three foods after *each* of the 16 trials. Ratings of distinctive Elements A and B diverged and ratings of common Element X decreased as the difference in the correlation of AX and BX with the occurrence and nonoccurrence of the allergic reaction increased. These human causal judgments closely correspond with stimulus selection effects observed in the conditioned responses of animals in associative learning studies. The experiment also directly demonstrated the fact that significant changes in the causal ratings of a stimulus occur on trials in which the cue is *not* presented. Associative theories such as that of Rescorla and Wagner (1972) predict changes in associative strength only for those stimulus elements that *are* presented on a particular trial. A modification of the Rescorla–Wagner model is described that correctly predicts immediate changes in the associative strengths of *all* relevant cues on each trial—whether presented or not. © 1994 Academic Press, Inc.

Many investigators have suggested close correspondences between the mechanisms of instrumental and classical conditioning, on the one hand, and those of human causality judgments and diagnostic reasoning, on the other (Algom & Bizman, 1983; Alloy & Tabachnik, 1984; Chapman, 1991; Gluck & Bower, 1988; Lovibond, 1988; Shanks & Dickinson, 1987; Wasserman, 1990a, b). A conditioned stimulus, discriminative stimulus, or response may be seen as the predictor of an outcome (reinforcement) in these diverse learning and judgment paradigms. Shanks and Dickinson (1987) proposed an associative account of human causality judgments and sensitivity to interevent contingencies that is consistent with recent theories of animal conditioning. Empirical work has shown that the acquisition

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functions (learning curves) for human judgments of causality parallel those observed in animal conditioning experiments under various contingencies of reinforcement (Gluck & Bower, 1988; Shanks, 1987). Such incremental learning functions are expected by conditioning theories like that of Rescorla and Wagner (1972), which postulate an increment in associative strength to a stimulus each time it is paired with an outcome.

Earlier theories of conditioning generally assumed that the joint occurrence (temporal contiguity) of events is sufficient for associative learning (e.g., Hull, 1943; Spence, 1956). Later research, however, demonstrated that temporal contiguity is insufficient (Kamin, 1969; Rescorla, 1968; Wagner, 1969); a contingency between the events is also necessary for associative learning. In classical conditioning, the probability of occurrence of the unconditioned stimulus (US) must be higher when the conditioned stimulus (CS) is present than when the CS is absent in order for conditioning to occur. The CS must also reliably and nonredundantly predict the occurrence of the US. Conditioning of a stimulus may be blocked (Kamin, 1969) if the information it provides about the occurrence of an outcome is more reliably conveyed by another stimulus.

The Rescorla–Wagner model and other more recent theories of conditioning (e.g., Mackintosh, 1975; Pearce & Hall, 1980) were developed to provide an explanation of the selective nature of conditioning disclosed by such phenomena as blocking, overshadowing, and conditioned inhibition. Rescorla and Wagner (1972) argued that a reinforcer will sustain only a limited amount of associative strength; therefore, simultaneously presented cues compete for association with an outcome. Their formulation proposed that the increment in associative strength that accrues to a stimulus on a given trial is proportional to the degree to which the outcome is *un*predicted by the combined associative strengths of all stimuli present on that trial. On trials in which the stimulus (i) is followed by the outcome, the increase in associative strength (V_i) between Stimulus i and the outcome is described by

$$\delta V_i = \alpha_i \beta_1 (L - \Sigma V_k), \tag{1}$$

where α_i and β_1 are learning rate parameters associated with the CS and US, respectively, L is the maximum level of associative strength that the US will support, and ΣV_k is the sum of the associative strengths of all of the CS elements present on the trial. If a stimulus is presented on a trial but is not followed by the reinforcing outcome, then the association between that stimulus and the outcome is decreased according to

$$\delta V_i = \alpha_i \beta_2 (0 - \Sigma V_k), \tag{2}$$

where 0 is the level of associative strength supported by the nonpresentation of the US, and β_2 is the learning rate parameter for nonreinforced trials.

Many workers have recently proposed that selectional processes in human causality judgments can also be described by associative models like that of Rescorla and Wagner (Chapman, 1991; Chapman & Robbins, 1990; Gluck & Bower, 1988; Shanks, 1986, 1989; Shanks & Dickinson, 1987; Van Hamme, Kao, & Wasserman, 1993; Wasserman, 1990a, 1990b). These workers have attempted to evaluate this proposal by seeking empirical convergence between the results of animal associative learning studies and investigations of complex human judgment. They have obtained evidence that suggests the presence of cue competition due to blocking, overshadowing, or conditioned inhibition in human contingency judgment and causal or diagnostic inference (Algom & Bizman, 1983; Chapman, 1991; Chapman & Robbins, 1990; Dickinson, Shanks, & Evenden, 1984; Gluck & Bower, 1988; Shanks, 1986, 1989).

Wasserman (1990a) provided one very clear example of stimulus competition in human causality judgments in an experimental procedure modeled on work originally done by Wagner, Logan, Haberlandt, and Price (1968) and later extended by Wasserman (1974) involving animal Pavlovian and operant conditioning. That original work by Wagner *et al.* (1968) plus Kamin's (1969) arguably better-known "blocking" effect were the key reasons for Rescorla and Wagner to devise their extremely influential theory of learning.

In the work by Wagner *et al.* (1968) and Wasserman (1974), the amount of conditioned responding to one element (X) of a compound stimulus depended on the *differential* predictiveness of two other stimuli (A or B) with which X was concurrently presented, even though the correlation of Stimulus X with the outcome remained the *same* in all experimental conditions. The human subjects in Wasserman's (1990a) study were given the task of diagnosing the source of a hypothetical patient's allergic reaction. College students judged the causal efficacy of common (X) and distinctive (A or B) elements (food items) of the compound stimuli AX and BX. As the difference in the correlations of AX and BX with the occurrence and nonoccurrence of the allergic reaction increased from .00 to 1.00, ratings of the common X element decreased, even though the correlation of X with the allergic reaction remained at .50 in all experimental conditions. These human causal judgments thus exhibited selective attributional effects parametrically parallel to those seen in the conditioned responses of the animals in the associative learning studies of Wagner *et al.* (1968) and Wasserman (1974).

The Wasserman (1990a) study involved two methods of information presentation: (a) A 2 × 2 summary table indicating the number of times an allergic reaction did or did not occur following consumption of Food 2 + Food 1 (AX) or Food 3 + Food 1 (BX) and (b) a serial listing of the individual trials displayed in rows representing the consumption of Food 1, Food 2, or Food 3 and the occurrence or nonoccurrence of an

allergic reaction for each meal of the allergy test series. Each of these procedures involved the simultaneous presentation of all contingency information. Van Hamme and Wasserman (1993) extended the Wasserman (1990a) study to compare the effects of serial and simultaneous presentation of information. Similar stimulus selection effects were obtained regardless of whether the information was described to subjects in a summarized manner or experienced trial-by-trial. Subjects in each of these conditions made a single final rating of each cue after receipt of all of the contingency information.

In the present experiment, a serial presentation format was used, and the response format was a *running* estimate, in which subjects were asked to rate the causal efficacy of each cue after each trial. The purpose of this procedural modification was to assess the adequacy of the Rescorla–Wagner model of associative learning in accounting for subjects' utilization of all contingency information. The Rescorla–Wagner model predicts changes in the associative strengths of only those stimulus elements that are presented on a particular trial. Work on contingency judgments has shown, however, that subjects' ratings reflect the use of information about both the *occurrence* and the *nonoccurrence* of potential causal factors (Arkes & Harkness, 1983; Levin, Wasserman, & Kao, 1993; Wasserman, Dorner, & Kao, 1990). The present experiment was designed to allow the measurement of changes in the causal ratings of each cue after each trial, *whether or not* the cue was presented on that trial. A modified associative model will be outlined that, unlike the original Rescorla–Wagner account, directly and immediately utilizes all of the pertinent contingency information for each cue.

Associative accounts of causality judgment have been suggested as alternatives to statistical approaches, such as Delta P (Wasserman, Elek, Chatlosh, & Baker, 1993), Bayes' Theorem (Fales & Wasserman, 1992), regression (Schustack & Sternberg, 1981), and ANOVA (Cheng & Novick, 1990, 1992; Forsterling, 1989; Kelley, 1967). There are, however, some significant correspondences among the different types of model. Several authors have noted that both the Rescorla–Wagner associative learning rule and a multiple linear regression solution operate to select cue weights that minimize the error or discrepancy between expected and actual outcomes (Chapman, 1991; Gluck & Bower, 1988; Shanks, 1991). As the structural properties of the analysis of variance are formally equivalent to those of multiple regression, the solutions of associative learning models will approach those of linear regression analysis as well as those of other statistical models.

Chapman (1991) has argued, however, that the two types of model make different predictions about the effects of trial order on the relative strengths of competing causal cues. In associative models, the strength of each cue is updated after each trial on which it is presented; therefore,

trial order will affect the resulting cue weights. Statistical models, in contrast, assume an analysis based on summarized data, which presumably would not be affected by the order in which the information is presented.

Both associative and statistical models can account for the selective nature of causal judgments (the so-called "discounting" of alternatives). The associative account can also explain the sequential integration of information and the effects of the temporal order of events. However, the Rescorla–Wagner model, in its current form, does *not* directly and immediately utilize all of the covariation information represented in the four cells of the contingency table for each of the competing cues.

One normative statistical rule for the use of contingency information in the assessment of covariation is referred to as Delta P. Delta P is equal to the probability of an outcome given the occurrence of an event minus the probability of an outcome given the nonoccurrence of that event [P(O|E) − P(O|No E)]. In terms of a standard 2 × 2 contingency table, Delta P = a/(a + b) − c/(c + d), where the cell entries represent the following event combinations: Cell a = Event–Outcome, Cell b = Event–No Outcome, Cell c = No Event–Outcome, and Cell d = No Event–No Outcome. This expression utilizes the information in all four cells of the table and represents the true contingency between the events. Normative use of the Delta P rule also requires that all of the information in the contingency table be given *equal* weight; but, subjects' utilization of cell information has generally been found to reflect the following *biased* weighting of the four types of information: Cell a > Cell b <=> Cell c > Cell d (Crocker, 1982; Kao & Wasserman, 1993; Levin *et al.*, 1993; Wasserman *et al.*, 1990).

It can be seen that the Rescorla–Wagner model directly utilizes only Cell a [Eq. (1)] and Cell b [Eq. (2)] information for each of the relevant possible causes. Equation 1 is applied when both the cue and the outcome (the CS and the US) are present on a trial; Eq. 2 is applied when the cue is present and the outcome does not occur (CS only trials). These situations are represented, respectively, by Cells a and b of a contingency table. When multiple cues are intercorrelated (co-occur), they compete in terms of their sufficiency as predictors. The model does not directly or completely utilize Cell c or Cell d information, because cues *not* present on a trial are assumed *not* immediately to change in associative strength.

The Rescorla–Wagner model utilizes Cell c and Cell d information in an *indirect, nonimmediate* way because of the assumption that there is a limited amount of associative strength available between a US and *all* of the cues presented on a trial. On reinforced (US present) trials in which a particular cue (N) is *not* presented (Cell c for Cue N), any cue (P) that *is* presented on those trials acquires a portion of this limited amount of associative strength; this portion of the associative strength is then unavailable on subsequent trials on which P is presented, including later

compound-cue (NP) trials, in which this strength would not be available to N. A "Cell c" trial, therefore, results in the loss of *availability* of associative strength rather than an immediate decrease in *existing* strength. The sequence of trial types (P – NP) generates the *forward* blocking effect described by Kamin (1969), which is one of the stimulus selection effects the Rescorla–Wagner model was designed to explain. However, the Rescorla–Wagner model cannot explain the occurrence of *backward* blocking, in which the reverse sequence of trial types (NP – P) results in a reduction in the strength of Stimulus N after the entire sequence compared to its strength after the NP phase only (Chapman, 1991; Shanks, 1985). According to the Rescorla–Wagner model, the strength of N should not change in the second phase because it is not presented in that phase.

The Rescorla–Wagner model can, however, be modified to *directly* and *immediately* utilize and appropriately weight all of the types of covariation information by the inclusion of two additional equations to reflect Cell c and Cell d information (see Fig. 1). In this revised model, the strengths of *all* relevant cues are updated on *all* trials.[1]

For any particular cue, there are four types of trial, representing the four possible types of contingency table information (Cells a, b, c, and d). Revision of the Rescorla–Wagner model to enable appropriate and immediate updating of all of this information requires a modification of the manner in which the learning rate parameters are applied. In the original version of the model—applied to all causal cues presented on a trial—the β_1 parameter is usually assumed to be larger than the β_2 parameter. (Cell a information is weighted more heavily than Cell b information). The β_2 parameter retains a nonzero value even when the outcome event (the US in classical conditioning) is not presented. But, when the causal cue (the CS in classical conditioning) is not presented, the α parameter is always reduced to *zero*, resulting in no change in the associative strength of the cue on that trial. (Cell c and Cell d information each carry a weight of zero.) A modified model in which the α parameter *also* retains a *nonzero* (negative) value when the relevant cue does not occur might more accurately reflect the manner in which conditioning or causal judgments occur. The combined $\alpha\beta$ values for the resulting four equations are then equivalent to cell information weightings (see Fig. 1).

This modification of the Rescorla–Wagner model allows CS and US nonoccurrence to be treated in a parallel manner. The Rescorla–Wagner model currently does not treat these two types of event consistently. The

[1] A cue would become relevant after acquiring some level of positive or negative associative strength. For animal subjects, it would be necessary to present the stimulus at least once in the experimental context, either followed by reinforcement or presented together with a cue that had previously been reinforced. For human subjects, the relevance of a cue as a potential cause could be established with verbal instructions.

Contingency Table

	Outcome Present	Outcome Absent
Cue Present	Cell a	Cell b
Cue Absent	Cell c	Cell d

Original Rescorla-Wagner Model

Cue Present-Outcome Present (Cell a): $\delta V_i = \alpha_1\beta_1(L - \Sigma V_k)$

Cue Present-Outcome Absent (Cell b): $\delta V_i = \alpha_1\beta_2(0 - \Sigma V_k)$

Modified Rescorla-Wagner Model

Cue Present-Outcome Present (Cell a): $\delta V_i = \alpha_1\beta_1(L - \Sigma V_k)$

Cue Present-Outcome Absent (Cell b): $\delta V_i = \alpha_1\beta_2(0 - \Sigma V_k)$

Cue Absent-Outcome Present (Cell c): $\delta V_i = \alpha_2\beta_1(L - \Sigma V_k)$

Cue Absent-Outcome Absent (Cell d): $\delta V_i = \alpha_2\beta_2(0 - \Sigma V_k)$

α_1 = learning rate parameter for cue i present
α_2 = learning rate parameter for cue i absent
β_1 = learning rate parameter for outcome present
β_2 = learning rate parameter for outcome absent

FIG. 1. The 2 × 2 contingency table, the original Rescorla–Wagner model, and the modified Rescorla–Wagner model.

modified model makes the assumption that missing input cues—as well as missing output cues—are actively encoded as *absent.* Markman (1989) has previously proposed this modified application of the Rescorla–Wagner equations in a connectionist model. Markman's model encodes the presence of a feature with an activation level of 1 and the absence of a feature with an activation level of −1. (In most connectionist models, a nonpresented cue is assigned an activation level of 0.) His model, therefore, assumed that the *absolute values* of the weights attached to cue presence and cue absence information are equal ($|\alpha_1| = |\alpha_2|$ in the modified Rescorla–Wagner model shown in Fig. 1). And, because this connectionist model was used to simulate a categorization problem—predicting *which* of two outcomes will occur given a particular set of input cues—rather than predicting the presence or absence of a single outcome, the weights

attached to each of the two outcomes were also equal ($\beta_1 = \beta_2$). All four types of contingency table information were thus weighted equally in the application of Markman's model. The four different types of information represented by the four possible combinations of presence and absence of the cue and outcome could, however, be appropriately *weighted* according to the type of contingency table information that they represent by choosing values for the α and β parameters such that: $|\alpha_1\beta_1| > |\alpha_1\beta_2| <=> |\alpha_2\beta_1| > |\alpha_2\beta_2|$ (Cell a > Cell b <=> Cell c > Cell d). This strategy would be preferable to that used by Markman because it would more accurately reflect the manner in which subjects have been found to use these four types of information.

The present experiment was designed to begin to assess this potentially important modification of the Rescorla–Wagner model by seeing if the causal ratings of a cue do change when that cue is not presented on a *particular* trial; Markman himself did not test the assumption that subjects actively encode missing cues as absent. Both Chapman (1991) and Shanks (1985)—using backward blocking and backward conditioned inhibition procedures—have demonstrated changes in the associative strength of a stimulus after a *series* of trials on which a stimulus is not presented; these effects were described as unexplainable by associative models because they involve the apparent retrospective processing of a stimulus rather than trial-by-trial increments or decrements in associative strength. The modified Rescorla–Wagner model is an associative model that predicts just such immediate trial-by-trial changes in associative strength for a nonpresented cue. The purpose of the present experiment is to test that prediction by asking subjects to rate all cues on every trial—whether or not a cue was presented on that particular trial. The demonstration of immediate changes in the associative strength of a cue after particular trials on which the cue was not presented would make it unnecessary to posit the retrospective processing of stimuli in order to explain phenomena like backward blocking and backward conditioned inhibition and would suggest that these phenomena can be explained by an associative model.

Subjects were asked to rate the likelihood that three different foods were the cause of an allergic reaction in a hypothetical patient. Three experimental conditions varied the differential correlations (.00, .50, and 1.00) of Food A and Food X (AX) and of Food B and Food X (BX) combinations with the occurrence and nonoccurrence of the allergic reaction. Subjects were asked to rate all three foods after each of the 16 trials, representing the 16 days of a hypothetical allergy test. This procedure is consistent with animal conditioning experiments in which responses are measured on each trial, and it allows the observation of causal acquisition functions. Ratings of each of the three foods were also requested prior to the receipt of any information about the results of the allergy test. These initial ratings allowed us to measure the *change* in

subjects' ratings after receipt of the first piece of contingency information; changes in causal ratings could thus be determined for all 16 trials. The use of human subjects provides the clear advantage of allowing measurement of the judged causal strength of a stimulus on trials when that stimulus is *not* presented.[2] The main purpose for using this rating method was that changes in ratings of all of the individual cues could be observed after each of the four trial types: (a) AX trials in which the allergic reaction occurred (AX+), (b) AX trials in which the allergic reaction did not occur (AX−), (c) BX trials in which the allergic reaction occurred (BX+), and (d) BX trials in which the allergic reaction did not occur (BX−). Trial types AX+, AX−, BX+, and BX− represent Cells a, b, c, and d for Stimulus A, and Cells c, d, a, and b for Stimulus B. The rating scores of most interest involved changes in A on BX trials and changes in B on AX trials, because significant changes in the judged causal strength of a *non*presented cue would provide unique empirical support for the modified Rescorla–Wagner model that—unlike the original account—directly and immediately utilizes Cell c and Cell d information.

Arkes and Harkness (1983, Experiment 7) previously employed such a "running estimate" contingency judgment procedure and demonstrated that subjects used information from all four cells of the contingency table. The Arkes and Harkness study, however, involved only *one* possible cause (cloud seeding) of an effect (rainfall). The present experiment extends the running estimate procedure to assess the impact of all four types of contingency table information in the context of competition among *three* possible causal cues.

METHOD

Subjects

Subjects were 48 University of Iowa undergraduates in an introductory psychology course, who participated in the study in partial fulfillment of a course requirement.

Materials

Six versions of the experimental sheets were prepared, corresponding with the different combinations of: (a) three levels of differential correlation of the food compounds (AX–BX) with the allergic reaction and (b) two different sets of foods corresponding with the A, B, and X items

[2] Although it is the occurrence or nonoccurrence of the US that changes the associative strength of a CS, this associative strength cannot be assessed with animal subjects without presenting the CS and measuring the response to it. Thus, any trial in which associative strength is measured also necessarily involves a change in the associative relation between stimulus and outcome. The use of appropriately instructed human subjects allowed us to avoid this difficulty.

for each contingency. Each subject received three sheets, corresponding to the three differential contingencies. All sheets began with the following instructions:

> Imagine that you are an allergist who is trying to determine the cause of an allergic reaction shortly after your patient eats dinner. You arrange that the patient eat particular foods at dinner over a series of evenings, and then report to you whether an allergic reaction followed.
>
> The patient will be eating two of the following three foods each day: [The three foods appropriate for the contingency and food conditions were listed here.] Before seeing the test results, please indicate how likely you think it is that each of these foods would cause an allergic reaction by choosing the appropriate number along the following rating scale: [The rating scale was inserted here].
>
> Each slide will be displayed for 15 seconds. During that time, please look at the day's results and then make probability ratings on the appropriate line below for each of the three foods. Use the above rating scale. [The three foods were then listed horizontally with 16 blank lines below each labeled Day 1 through Day 16].

The results were presented on 16 separate slides, displaying information about the occurrence and type of Food 1, Food 2, and Food 3 and whether or not an allergic reaction had occurred on each day of the 16-day test series. Food 1 was eaten every day; either Food 2 or Food 3 was eaten together with Food 1 on a random half of the days. Food 2 + Food 1 combinations were considered AX compounds; Food 3 + Food 1 combinations were considered BX compounds. No compound was listed more than twice in a row.

The order of occurrence of the AX and BX compounds and the occurrence or nonoccurrence of an allergic reaction over the hypothetical test series are listed in Table 1. The experimental conditions—AX–BX of .00, .50, and 1.00—were defined by the difference in the probability of an allergic reaction (R) after the different two-food combinations, AX and BX [$P(R|AX) - P(R|BX)$].

Before seeing any of the hypothetical test results, subjects were asked to make an initial rating of each of the three foods and then to make ratings of each of the foods after viewing each of the 16 slides. Following receipt of the results of each day of the allergy test series, subjects were asked to indicate their diagnosis of the allergic reaction by choosing the appropriate number along a rating scale. The scale ranged from 0 to 8, with the following verbal descriptions at selected points along the scale: (0) *definitely not,* (4) *possibly,* and (8) *definitely* the cause of the allergic reaction. Subjects were not asked for an overall rating at the end of the 16 trials and were not able to change earlier responses.

Procedure

The 48 participants were divided into six groups with 6 to 10 subjects in each; variability in group size was due to the vicissitudes of scheduling.

TABLE 1
Order of Occurrence of AX and BX Compounds and Allergic Reactions for the Three Different AX–BX Correlations

		AX–BX		
		.00	.50	1.00
Trial	Stimulus compound	Allergic reaction?		
1	AX	Yes	Yes	Yes
2	BX	No	No	No
3	BX	Yes	Yes	No
4	AX	No	Yes	Yes
5	AX	No	No	Yes
6	BX	Yes	No	No
7	AX	Yes	Yes	Yes
8	BX	No	No	No
9	BX	Yes	Yes	No
10	AX	No	Yes	Yes
11	BX	No	No	No
12	AX	Yes	Yes	Yes
13	AX	No	No	Yes
14	BX	Yes	No	No
15	AX	Yes	Yes	Yes
16	BX	No	No	No

Each subject in all groups was given three of the experimental sheets and made causal ratings of the three foods in each of the three AX–BX conditions. Different foods were used on each of the three rating sheets. The order of presentation of the AX–BX conditions and the food types was varied across groups, such that all possible combinations of these orders were presented in Groups 1 to 6. Table 2 shows a summary of the experimental procedures and Table 3 shows the foods used in each condition.

RESULTS

Figure 2 shows the final (Day 16) mean causal ratings of Elements X, A, and B, combined for all groups, as a function of the AX–BX correlation. Increases in the AX–BX correlation produced increases in the causal ratings of Element A and corresponding decreases in the ratings of Elements B and X. Three separate within-subjects analyses of variance assessed these effects of increases in the AX–BX correlation on ratings of Elements X, A, and B. The correlation factor was significant in all three, $F(2, 47) = 16.85$, $p < .001$; $F(2, 47) = 38.70$, $p < .001$; and, $F(2, 47) = 58.91$, $p < .001$, respectively, for Elements X, A, and B.

TABLE 2
Order of Presentation of Experimental Conditions in Groups 1 to 6

Group	Number of subjects	AX–BX condition order	Food condition order
1	7	.50 .00 1.00	2 1 3
		.50 1.00 .00	5 6 4
2	8	.00 1.00 .50	1 3 2
		.00 .50 1.00	4 5 6
3	6	1.00 .50 .00	3 2 1
		1.00 .00 .50	6 4 5
4	8	.50 1.00 .00	5 6 4
		.50 .00 1.00	2 1 3
5	10	.00 .50 1.00	4 5 6
		.00 1.00 .50	1 3 2
6	9	1.00 .00 .50	6 4 5
		1.00 .50 .00	3 2 1

The increase in ratings of Element A and the decreases in ratings of Elements B and X were, therefore, reliable.

Figures 3, 4, and 5 show the initial and daily mean causal ratings of each of the stimulus elements for AX–BX values of .00, .50, and 1.00, respectively. These figures represent the acquisition functions for the three AX–BX contingency differences and demonstrate the incremental manner in which both accurate and biased judgments of correlation developed. In all three conditions, Element X predicted the allergic reaction 50% of the time. The AX–BX values represented the differential predictiveness of Elements A and B (.00 = .50 − .50; .50 = .75 − .25; 1.00 = 1.00 − .00). Figures 3, 4, and 5 reflect the development of generally accurate correlational ratings in most cases, except for ratings of Element X in the AX–BX = 1.00 condition. In this case, as subjects learned that Foods

TABLE 3
Food Conditions

Food condition	AX–BX	Stimulus element		
		X	A	B
1	.00	Shrimp	Strawberries	Peanuts
2	.50	Yogurt	Bran	Cabbage
3	1.00	Bananas	Chicken	Mustard
4	.00	Wheat	Walnuts	Peaches
5	.50	Corn	Horseradish	Lobster
6	1.00	Blueberries	Cheese	Pork

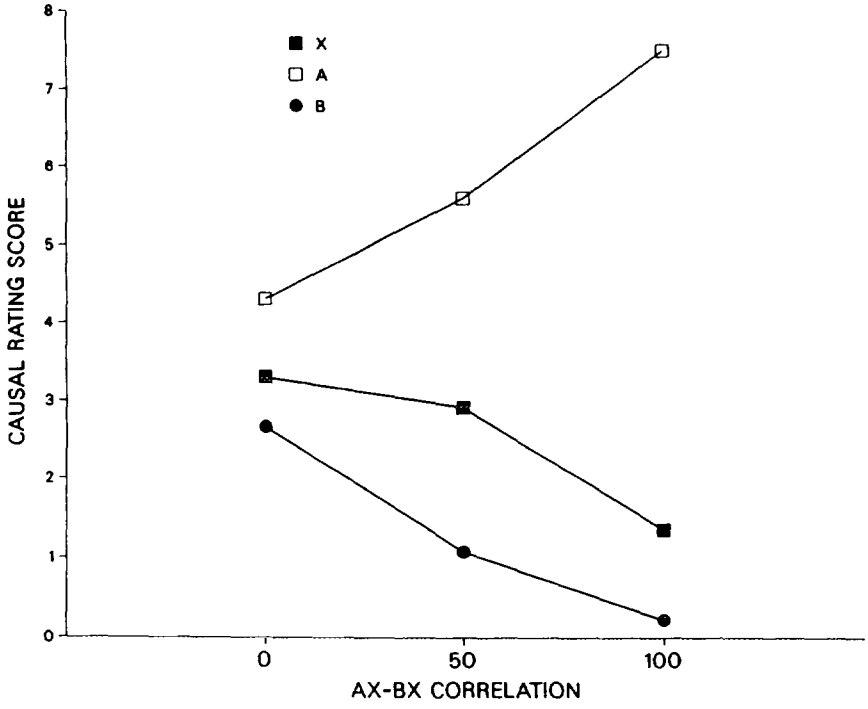

FIG. 2. Final (Day 16) mean causal rating scores of subjects in all groups combined to Elements A, B, and X of AX and BX compounds as a function of the difference in the predictiveness of those compounds (AX–BX) for the occurrence of an allergic reaction.

A and B, respectively, perfectly predicted the occurrence or nonoccurrence of the allergic reaction, mean ratings of Element X gradually dropped far below its true predictive value of 50% (4 on the 0 to 8 rating scale) to a rating of 17% of the maximum possible score (a mean rating of 1.354) on the final day of the allergy test.

As in the Wasserman (1990a) study, these results demonstrate both a qualitative and a quantitative correspondence between humans' causal judgments and animals' conditioned responses. Although the *actual* correlation of Element X with the allergic reaction did not change across the three AX–BX correlations, the *perceived* causal efficacy of Element X decreased as Element A became a more reliable predictor of the allergic reaction and Element B became a more reliable predictor of its nonoccurrence.

Although the acquisition functions generally demonstrate the gradual development of accurate final judgments of correlation, these functions also show clear trial-by-trial fluctuations in the ratings of all stimulus elements. The next portion of our analysis attempted to determine how

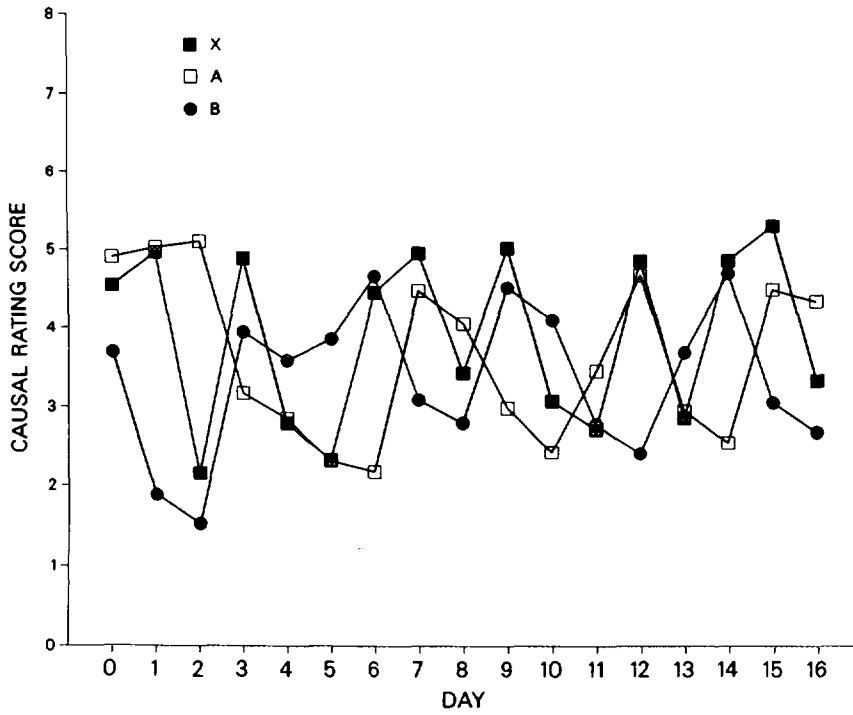

FIG. 3. Initial and daily mean causal rating scores of subjects in all groups combined to Elements A, B, and X of AX and BX compounds with the difference in the predictiveness of those compounds (AX–BX) for the occurrence of an allergic reaction equal to .00.

those *changes* in ratings were related to the trial types presented on specific days of the allergy test series. For each of the four trial types (AX+, AX−, BX+, and BX−), mean changes in ratings (from the previous trial) of each of the three stimulus elements (X, A, and B) were computed for each of the 48 subjects for each of the three AX–BX contingencies (e.g., the mean change in the rating of Element B across the four AX+ trials in the AX–BX = .00 contingency for Subject 1). A set of *t* tests was then conducted on these rating changes for each of the four trial types in each of the three AX–BX conditions for each of the three stimulus elements to see if the mean changes in ratings were significantly different from zero. Results of the *t* tests are shown in Tables 4, 5, and 6.

The purpose of the *t* tests was to evaluate the strength and direction of the changes in causal ratings for each of the four types of contingency table information and to evaluate the conformity of those rating changes with the predictions of the original and modified Rescorla–Wagner models. Normatively, Cells a and d represent positive contingency information and Cells b and c represent negative contingency information.

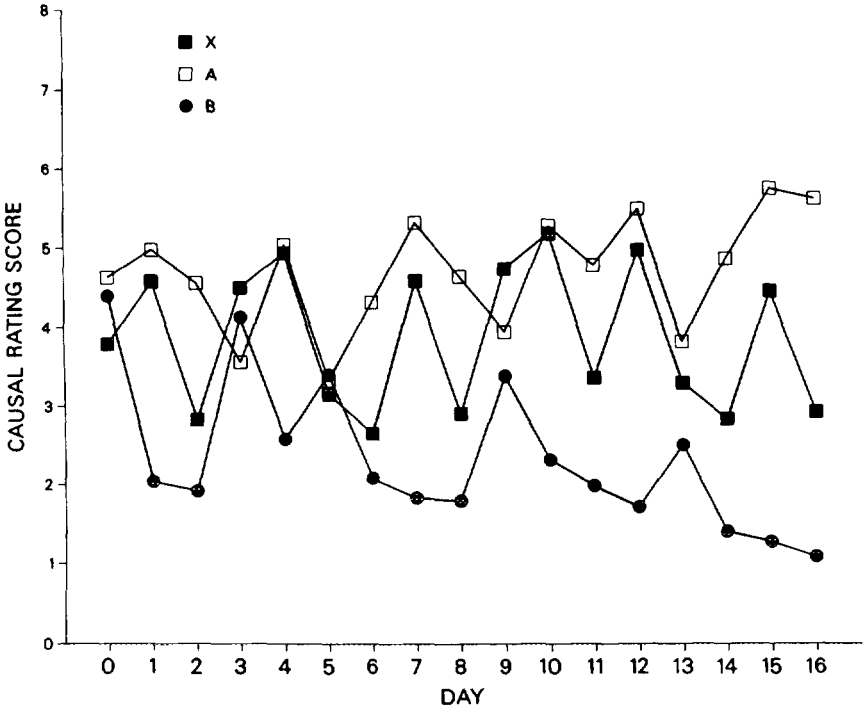

FIG. 4. Initial and daily mean causal rating scores of subjects in all groups combined to Elements A, B, and X of AX and BX compounds with the difference in the predictiveness of those compounds (AX–BX) for the occurrence of an allergic reaction equal to .50.

(Increases in the frequency of Cell a or Cell d result in increases in Delta P; increases in the frequency of Cell b or Cell c result in decreases in Delta P.)

The modified Rescorla–Wagner model predicts that the mean change in ratings following a particular trial type will be in the direction appropriate to the particular cell of the contingency table represented by the information presented—increases in ratings after Cell a or Cell d information and decreases in ratings after Cell b or Cell c information. (This prediction is based on the requirement that the α_2 parameter be assigned a negative value; the α_1, β_1, and β_2 parameters of the original Rescorla–Wagner model are always assigned positive values.) The original Rescorla–Wagner model predicts that ratings of stimulus elements presented with reinforcement (Cell a) will increase and that ratings of stimulus elements presented without reinforcement (Cell b) will decrease; ratings of nonpresented elements (Cell c or Cell d) are predicted not to change. The usual assumption in the use of the Rescorla–Wagner model that $\beta_1 > \beta_2$ also results in the prediction that increases in ratings on reinforced

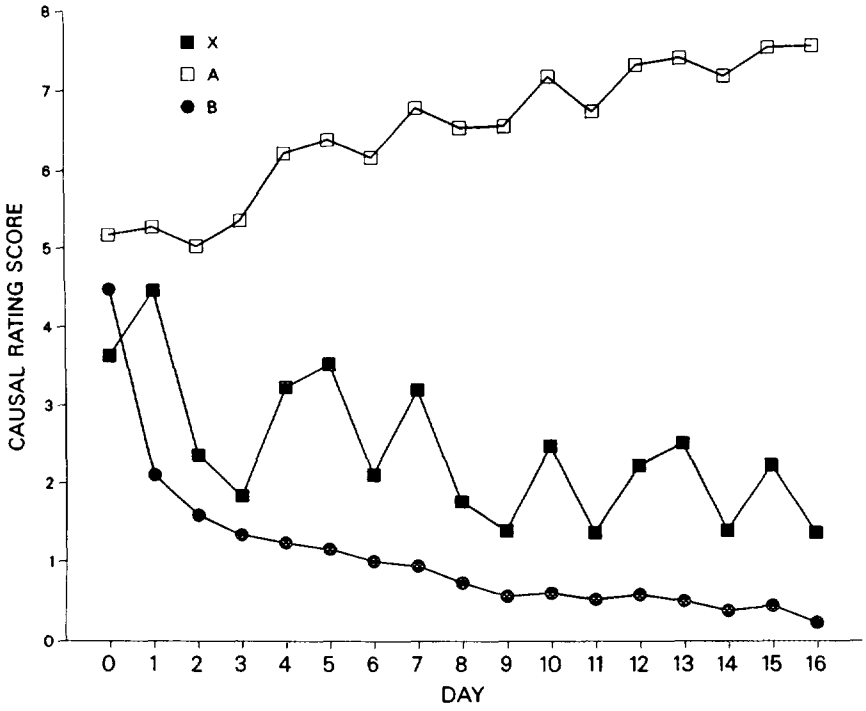

FIG. 5. Initial and daily mean causal rating scores of subjects in all groups combined to Elements A, B, and X of AX and BX compounds with the difference in the predictiveness of those compounds (AX–BX) for the occurrence of an allergic reaction equal to 1.00.

trials will be larger than decreases in ratings on nonreinforced trials (Cell a > Cell b).[3] This final prediction also accords with the results of contingency judgment research indicating that information from the four different cells is not weighted equally (Kao & Wasserman, 1993; Levin *et al.*, 1993; Wasserman *et al.*, 1990).

We first consider the rating changes for trial types representing Cell a and Cell b information. Tables 4, 5, and 6 show that the results for Cell a (AX+ trials for Elements A and X, and BX+ trials for Elements B and X) and Cell b (AX− trials for Elements A and X, and BX− trials for Elements B and X) conform with predictions of the original and modified Rescorla–Wagner models and Delta P concerning the direction of the rating changes. For all of the relevant trial types, ratings of stimulus

[3] Rescorla and Wagner (1972, pp. 85–86) have noted that their model can account for the cue-competition effect demonstrated by Wagner *et al.* (1968) *only* if this assumption is made. The results of Wasserman (1974) and those reported here—that ratings of Element X were larger in Condition .00 than in Condition 1.00—also require the assumption that $\beta_1 > \beta_2$, since each of these experiments used procedures based on those of Wagner *et al.*

TABLE 4
Results of t Tests for AX–BX = .00

	Stimulus element		
	X	A	B
AX+			
Mean	.870[a]	1.385[a]	**−1.344**[c]
t	7.26	8.41	**−8.28**
p	.000	.000	**.000**
AX−			
Mean	−1.615[b]	−.781[b]	**.255**[d]
t	−8.09	−5.97	**1.70**
p	.000	.000	**.096**
BX+			
Mean	2.099[a]	**−.885**[c]	1.479[a]
t	8.78	**−4.65**	9.12
p	.000	**.000**	.000
BX−			
Mean	−1.661[b]	**.135**[d]	−.583[b]
t	−9.14	**.63**	−3.20
p	.000	**.53**	.003

Note. Degrees of freedom for all t tests = 47. Means are mean rating changes for each stimulus element for each trial type. Boldface indicates instances where the original Rescorla–Wagner model predicts that change scores will not differ significantly from zero.

[a] Cell a.
[b] Cell b.
[c] Cell c.
[d] Cell d.

elements presented with reinforcement (Cell a) increased and ratings of stimulus elements presented without reinforcement (Cell b) decreased. In order to see if Cell a information was weighted more heavily than Cell b information, it was necessary to inspect the means for individual stimulus elements and to compare them across trial types. This analysis can only appropriately be done for the AX–BX = .00 contingency, because it is only in this condition that: (a) there are an equal number of trials of each type and (b) there is Cell a and Cell b information for all three of the stimulus elements. Table 4 shows that, for both Elements A and B, the absolute value of the rating changes for Cell a information was greater than the absolute value of the rating changes for Cell b information (Element A: Cell a = 1.385 and Cell b = .781; Element B: Cell a = 1.479 and Cell b = .583). This result did not hold for Element X, however; the absolute value of the mean rating changes averaged across AX+ and BX+ trials (1.485, Cell a) was slightly less than the equivalent average across AX− and BX− trials (1.638, Cell b). Nevertheless, across all

TABLE 5
Results of t Tests for AX–BX = .50

	Stimulus element		
	X	A	B
AX+			
Mean	1.132[a]	.958[a]	**−.934**[c]
t	7.96	6.40	**−6.51**
p	.000	.000	**.000**
AX−			
Mean	−1.729[b]	−.740[b]	**.802**[d]
t	−7.55	−5.07	**3.27**
p	.000	.000	**.002**
BX+			
Mean	1.750[a]	**−.844**[c]	1.896[a]
t	8.79	**−3.66**	8.94
p	.000	**.001**	.000
BX−			
Mean	−1.215[b]	**.049**[d]	−.517[b]
t	−9.25	**.21**	−3.34
p	.000	**.84**	.002

Note. Degrees of freedom for all t tests = 47. Means are mean rating changes for each stimulus element for each trial type. Boldface indicates instances where the original Rescorla–Wagner model predicts that change scores will not differ significantly from zero.

[a] Cell a.
[b] Cell b.
[c] Cell c.
[d] Cell d.

three stimulus elements, the absolute value of the mean rating change for Cell a information (appropriately weighted for the number of trials on which each stimulus element was presented) was 1.458 compared with 1.160 for Cell b information. Thus, overall, Cell a information did result in larger changes in ratings than Cell b information, as predicted by both associative models and consistent with previous contingency judgment research.

The values of most interest in Tables 4, 5, and 6 (shown in italics) are the t test scores for changes in ratings of Element A on BX trials and Element B on AX trials (Cell c and Cell d information). The original Rescorla–Wagner model predicts that the means of these rating changes should not differ from zero, as the stimulus being rated is not presented on those trials. However, the revised model—which utilizes information from all four cells of the appropriate contingency tables—makes different predictions. The AX+ and BX+ trials represent Cell c information for Elements B and A, respectively, which is negative contingency infor-

TABLE 6
Results of t Tests for AX–BX = 1.00

	Stimulus element		
	X	A	B
AX+			
Mean	.836[a]	.422[a]	**−.318**[c]
t	5.46	2.58	**−3.91**
p	.000	.013	**.000**
BX−			
Mean	−1.117[b]	**−.128**[d]	−.214[b]
t	−7.61	**−.71**	−2.71
p	.000	**.48**	.009

Note. Degrees of freedom for all t tests = 47. Means are mean rating changes for each stimulus element for each trial type. Boldface indicates instances where the original Rescorla–Wagner model predicts that change scores will not differ significantly from zero.

[a] Cell a.
[b] Cell b.
[c] Cell c.
[d] Cell d.

mation. The modified Rescorla–Wagner model thus predicts that AX+ trials should result in deflation of ratings of Element B, and BX+ trials should result in deflation of ratings of Element A. As can be seen in Tables 4, 5, and 6, the means for all five of these rating changes are negative and the t test scores all differ significantly from zero.

The AX− and BX− trials represent Cell d information for Elements B and A, respectively, which is positive contingency information.[4] The revised model, therefore, predicts that AX− trials should result in inflation of ratings of Element B and BX− trials should result in inflation of ratings of Element A. And, because previous contingency judgment research has indicated that the weight used in the model for Cell d information should be substantially lower than the weights used for all other cell information, the model also predicts that the inflation results predicted for Cell d information should be weaker than the deflation results predicted for Cell C information. Tables 4, 5, and 6 show that, although four of the five rating changes are in the predicted positive direction, only one differs significantly from zero by t test.

Overall, 29 of the 30 mean rating changes shown in Tables 4, 5, and 6 are in the direction predicted by the modified Rescorla–Wagner model.

[4] Empirical assessments of subjects' utilization of Cell d information have usually shown that this information is accorded a positive value (Levin *et al.*, 1992; Wasserman *et al.*, 1990). An exception, however, is the Arkes and Harkness (1983, Experiment 7) study, in which Cell d data had a small negative impact on subjects' contingency judgments.

The presentation of Cell a information resulted in increased ratings in all instances; Cell b and Cell c trials produced decreased ratings in all instances; and, four of the five Cell d cases resulted in increased ratings.

DISCUSSION

The present results support the suggestion derived from a modification of the Rescorla–Wagner model that the judged efficacy of a potential causal factor *changes* following stimulus events in which that factor is *not* present. These rating changes appear to be appropriately related to the contingency information provided by the experienced events. One particularly clear way in which the revised model can be shown to differ from the original Rescorla–Wagner formulation is seen by examination of Tables 4, 5, and 6. Scrutiny of the data for Elements A and B reveals that pairing with outcome *occurrence* led to *increased* ratings of the *presented* element (in five of five cases) and to *decreased* ratings of the *nonpresented* element (in five of five cases); pairing with outcome *nonoccurrence* led to *decreased* ratings of the *presented* element (in five of five cases) and to *increased* ratings of the *nonpresented* element (in four of five cases). The upward and downward changes in ratings of the *presented* element correspond with Cell a (mean change = +1.228) and Cell b (mean change = −.567) contingency table information and are predicted by both the original and modified Rescorla–Wagner models; however, the downward and upward changes in ratings of the *nonpresented* element correspond with Cell c (mean change = −.865) and Cell d (mean change = +.223) contingency table information and are predicted *only* by the modified Rescorla–Wagner model.

In addition, the impact of particular cell information was consistent with previous work on contingency judgments (Cell a > Cell b <=> Cell c > Cell d). Impetus is therefore provided for the further development and testing of the modified associative model of causal/contingency judgments that—unlike the original Rescorla–Wagner account—directly and immediately utilizes and appropriately weights all of the contingency table information.

The revised associative model described here—in which information from all four cells of the contingency table is appropriately registered and weighted—is similar to the weighted Delta P model outlined by Wasserman *et al.* (1993). Both models involve an attempted integration of the Rescorla–Wagner and Delta P rules. Wasserman *et al.* (1993) proposed that, at asymptote in the Rescorla–Wagner model, with $\beta_1 > \beta_2$, the associative strength of a stimulus (V_i) is equal to a weighted version of Delta P:

$$\text{Weighted Delta P} = w_aA/(w_aA + w_bB) - w_cC/(w_cC + w_dD), \quad (3)$$

with $w_a = w_c > w_b = w_d$.

There are, however, some important differences between that model and the one proposed here. One of these differences lies in the treatment of Cell c and Cell d information. The Rescorla–Wagner model is stated in terms of increments and decrements in the associative strength of the elements of stimulus *compounds* as a result of reinforcement and nonreinforcement. But, animals are also sensitive to the correlation of a *single* CS with a US in situations that do not explicitly involve the manipulation of compound cues. This correlation is described in terms of the difference in the probability of the US in the presence and absence of the CS. In order to account for this single-stimulus sensitivity, Rescorla and Wagner (1972) introduced the notion of a *contextual* cue representing a background of uncontrolled stimuli present on every trial. This contextual cue is said to compete for associative strength in the same manner as the discrete cues manipulated by the experimenter, and it is incremented and decremented by the Rescorla–Wagner model in the same manner as any other causal factor. The weighted Delta P model utilizes Cell c and Cell d information in the same indirect manner as the original Rescorla–Wagner model, which does not increment or decrement any cue not present on a trial. As the Wasserman *et al.* (1993) model is concerned with the presence and absence of a single causal cue, the Cell c and Cell d information in the model actually represents the *presence* of the context (Cells a and b for the context), not the *absence* of the cue (Cells c and d for the cue). On trials when the cue is absent, both of those conditions (presence of the context and absence of the cue) occur, but statistically, in terms of the Delta P for each of the variables (context and cue), they do not represent the same event. However, the original Rescorla–Wagner model, as well as the weighted Delta P model, treats both conditions as a single event (presence of the context).

The modification described here (Fig. 1) adopts a different analytical tactic by adding exact equations to the Rescorla–Wagner model that involve information about occasions on which a discrete cue is absent, rather than by considering those events to be equivalent to the presence of other discrete or contextual cues. Although the modified model does not, therefore, require the notion of a contextual cue to account for subjects' sensitivity to the probability of outcomes in both the presence and absence of a cue, logically, the occurrence of outcomes on trials in which none of the discrete, manipulated cues are present requires the assumption of a background of other unknown causes. In such a situation, the modified formulation would utilize a contextual cue in the same manner as does the original formulation of the Rescorla–Wagner model. The revised model does not differ from the original Rescorla–Wagner account in its treatment of *presented* cues (contextual or discrete); the difference lies in its treatment of *nonpresented* cues. There is, of course, the added implicit assumption that judgments about contingency or causality are

always relevant to a particular context; one event may be a reliable predictor of another only in one particular setting. This idea has been expressed in the areas of both human causal judgment and animal conditioning by constructs such as focal sets (Cheng & Novick, 1992), causal fields (Einhorn & Hogarth, 1986), decision frames (Tversky & Kahneman, 1981), and occasion setters or hierarchical cues (Holland, 1989).

Another difference between the Wasserman *et al.* model and the modified Rescorla–Wagner formulation is that the weighted Delta P model represents only an *asymptotic* formula for a single causal cue; it does not describe the *acquisition* of causality judgments or the *competition* among multiple causal cues. The weighted Delta P formula also incorporates the assumption that all entries in any particular cell of the contingency table represent equal increments (or decrements) in associative strength, regardless of *when* those entries occur—early or late in training. However, the modified Rescorla–Wagner model (Fig. 1) predicts that the actual amount of each increment (or decrement) is affected by trial order and, in the case of competitive-cue procedures, by which other cues are presented on a trial.

Associative models like those of Mackintosh (1975), Pearce and Hall (1980), and Rescorla and Wagner (1972) were developed to account for the competition of stimuli for predictive strength when compounds of more than one element are paired with reinforcement. These models assume that, during conditioning, each element enters separately into association with reinforcement. Rescorla and Wagner also suggested that combinations of stimuli may generate *configural* cues, and that these configural cues may separately acquire associative strength in the same manner as the individual elements. The development of associative strength by a configural cue would be analogous to a higher-order interaction accounting for a portion of the variance in a regression analysis. The Rescorla–Wagner model integrates configural cues by assigning an α parameter to the configuration on compound stimulus trials and then by applying the learning equation to all of the individual elements present on the trial as well as to the configural cue. The revised version of this model could accommodate configural cues in the same manner when necessary. Although some theories of associative conditioning have placed great importance on configural information (e.g., Pearce, 1987), Wagner and Rescorla (1972) suggested that the influence of configural cues is generally negligible, because their salience is quite low compared with that of the individual elements.

The present results and analysis represent only the initial stage in the assessment of an associative model of causality judgments, which would attempt to integrate and weight all of the information utilized by statistical models in a manner that reflects the sequential nature of information receipt. Although the present results do indicate that immediate changes

occur in the associative strength of nonpresented stimulus elements, it will be necessary to test the revised model using procedures that require such immediate changes in associative strength to provide an explanation of the behavioral effects observed. The present experimental results—in which a decrement in ratings of Element X occurred when Elements A and B were made increasingly predictive of outcome occurrence—are equally well predicted by the original Rescorla–Wagner model and by the modified version of it, because the target cue (X) was presented on every trial.[5] The two models differ only in their treatment of nonpresented cues.

The next stage will involve further research to assess the revised model's ability to predict the effects of trial order in stimulus selection procedures, such as forward and backward blocking and forward and backward conditioned inhibition. Both Chapman (1991) and Shanks (1991) have noted that providing an explanation for the occurrence of backward blocking is one of the major challenges to the application of current associative models to human causal and contingency judgments. The original Rescorla–Wagner model, because it does not provide for the immediate updating of the associative strength of a nonpresented cue, does not predict backward blocking. The revised model we have developed here has the potential to predict backward blocking as well as to account for its smaller size compared with forward blocking. Whether the behavior of human raters corroborates the unique predictions of the model remains to be seen.

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[5] Because Element X was presented on every trial, it is similar to a contextual cue. Also note that no explicit consideration outside of the experimental situation is generally given to such a stimulus. Thus, only Cell a and Cell b information is available for Element X; Cell c and Cell d information is unavailable.

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