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Assessment of the Rescorla–Wagner Model

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The Rescorla–Wagner model has been the most influential theory of associative learning to emerge from the study of animal behavior over the last 25 years. Recently, equivalence to this model has become a benchmark in assessing connectionist models, with such equivalence often achieved by incorporating the Widrow–Hoff delta rule. This article presents the Rescorla–Wagner model's basic assumptions, reviews some of the model's predictive successes and failures, relates the failures to the model's assumptions, and discusses the model's heuristic value. It is concluded that the model has had a positive influence on the study of simple associative learning by stimulating research and contributing to new model development. However, this benefit should neither lead to the model being regarded as inherently "correct" nor imply that its predictions can be profitably used to assess other models.

After more than a decade of relative quiescence in the study of animal learning, illuminating experiments conducted by Kamin (e.g., 1969), Rescorla (e.g., 1968), and Wagner (e.g., Wagner, Logan, Haberlandt, & Price, 1968) concerning cue competition culminated in the highly influential Rescorla–Wagner model of Pavlovian conditioning (Rescorla & Wagner, 1972; Wagner & Rescorla, 1972). Over the last 20 years, the Rescorla–Wagner model has been the primary export of traditional learning theory to other areas of psychology. The model was initially met with wide acceptance because of its elegance, simplicity, and, particularly, ability to predict the associative and behavioral consequences of presenting multiple predictive cues together. Earlier models treated each cue of a multicue compound independently, erroneously assuming no interaction between cues. In contrast, the Rescorla–Wagner model was formulated primarily to provide a trial-by-trial description of how the associative status of a conditioned stimulus (CS) changes when the stimulus is trained (e.g., paired with an unconditioned stimulus [US]) in the presence of other CSs. Thus, it is not surprising that among the model's greatest successes is its ability to predict overshadowing (i.e., the deficit in conditioned responding to Stimulus X after AX → US pairings in which both A and X are initially neutral) and blocking (i.e., the deficit in conditioned responding to Stimulus X after AX → US pairings in which A alone was previously paired with the US).

The hallmark of the Rescorla–Wagner model is that, as the discrepancy between the current associative value of the CS and the maximum strength of association that the US can support decreases, less conditioning occurs. Consequently, there is a decrease in the trial-by-trial change in the CS–US association. This aspect of the model has been interpreted by some researchers to imply that the amount of learning that occurs on each trial decreases as the US comes to be fully expected on the basis of the CS (i.e., as the difference between the actual and expected US decreases). One characterization of this process is that different discrepancies between the actual and expected US as a function of training procedures result in differential processing of the US representation. By contrast, consistent processing of the CS representation is assumed. The model's position concerning differential processing of the US thus contrasts with so-called "attentional" models of Pavlovian conditioning, which presume differential processing of a given CS representation and consistent processing of a given US representation (e.g., Mackintosh, 1975; Pearce & Hall, 1980). It is important to note that such descriptions of the model using the language of "expectancy" and "representation" are interpretations; the model itself does not demand that language.

Formally stated, the Rescorla–Wagner model assumes that

$$\Delta V_X^{n+1} = \alpha_X \beta_1 (\lambda_1 - V_{total}^n) \quad (1)$$

and

$$V_X^{n+1} = V_X^n + \Delta V_X^{n+1}, \quad (2)$$

where ΔV_X^{n+1} is the change in the associative strength (V) of CS X as a result of a pairing with US_1 on Trial $n + 1$ (i.e., the superscript denotes the trial number, not an exponent); α_X is the associability of CS X (range of 0 to 1), which is closely related to the intensity of CS X; β_1 is the associability of US_1

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(range of 0 to 1), which is closely related to the intensity of the US; λ_1 is the maximum associative strength that US_1 can support in any single situation; and V^n_{total} is the sum of associative strengths of all CSs (including X) that are present on Trial $n + 1$. The associative strength of each of the CSs that are present is determined on the last trial on which each CS occurred (ordinarily trial n). In Equation 2, V^{n+1}_X is the associative strength of CS X after Trial $n + 1$; V^n_X is the associative strength of CS X immediately before Trial $n + 1$ (i.e., after trial n); and ΔV^{n+1}_X is the change in the associate strength of CS X as a result of Trial $n + 1$ and is determined by Equation 1. The associative strengths of each CS before Trial 1 (i.e., initial values of V) are ordinarily assumed to be zero.

A central tenet of the Rescorla–Wagner model is that associative learning is determined by the extent to which a US is surprising. Surprise is represented in the model by the difference between the US that is actually presented on the trial in question and the US that is expected on the basis of the summed predictive value of all of the cues that are present on the trial. Surprise is quantified in Equation 1 as the absolute value of the parenthetical term, $\lambda - V_{total}$. As the result of a trial, ΔV_X for that trial modifies V_X and consequently changes V_{total} so that, on the next trial, V_{total} more accurately anticipates the US (i.e., is closer to λ) provided the same CSs and US are again presented together. This reduces the value of the parenthetical term, $\lambda - V_{total}$, with a corresponding decrease in US surprise over repeated trials. Through this mechanism, the degree of associative learning that occurs on each trial is successively smaller.

Assumptions of the Rescorla–Wagner Model

The Rescorla–Wagner model rests on five basic assumptions. Two of these five assumptions are unique to the model. First, the amount of associative strength that can be acquired as a result of a single presentation of a US is assumed to be limited by the summed associative value of all the CSs present on that trial, V_{total}, rather than by the associative value of the CS alone, V_X, as was assumed in prior models (e.g., Bush & Mosteller's [1951] model, which, with its linear operator equations, is the immediate intellectual ancestor of the Rescorla–Wagner model; see also Hull, 1943).

The second unique assumption of the model is that conditioned inhibition is the opposite of conditioned excitation, which, in Equation 2, is represented by negative values of V. That is, conditioned excitation and conditioned inhibition are represented by opposite signs on a single dimension of associative strength. Consequently, for a single CS, conditioned excitation and conditioned inhibition are assumed to be mutually exclusive. This contrasts with prior models in which a CS could simultaneously acquire both excitatory and inhibitory associations (e.g., Hull, 1943; Pavlov, 1927). In the calculation of V_{total}, inhibitory CSs have negative values of associative strength that algebraically summate with the positive values of associative strength possessed by excitatory CSs.

The Rescorla–Wagner model makes three additional assumptions that are not original and are less central to the theory but are nevertheless important to the model. First, the model assumes that the associability of a given stimulus (i.e., α) is constant, that is, a parameter not subject to change as a function of experience. This is an important aspect of the model because if α were allowed to change as a function of experience, many of the behavioral effects that the Rescorla–Wagner model takes credit for explaining through differences in the parenthetical term in Equation 1 could potentially be explained in terms of appropriate changes in α (e.g., variation in CS processing; Mackintosh, 1975; Pearce & Hall, 1980; Wagner, 1978). However, it should be recognized that some revisions of the model (e.g., Frey & Sears, 1978) have, in fact, permitted α to vary while sustaining an emphasis on the importance of variation in the value of the parenthetical term of Equation 1. Although such revisions may well seem reasonable, it is equally recognized that they represent important departures from the model's initial formulation (see Cluster 3 in the Underlying Bases for Failures section for further analysis of this assumption).

Second, the Rescorla–Wagner model assumes that new learning is independent of the associative history of any stimulus present on a given trial. That is, change in the associative status of a stimulus depends solely on current associative strength and the outcome of the present trial, not on how the current associative strength was reached (i.e., the associative path). This is called the assumption of path independence. Both of these assumptions were also incorporated into the Bush and Mosteller (1951) model, which is an antecedent of the Rescorla–Wagner model.

A third important assumption of the Rescorla–Wagner model is a presumed monotonic relationship between learning and performance. Rescorla and Wagner (1972) themselves noted that acquired responding "would necessarily depend on a large number of 'performance' variables" (p. 77). In practice, the monotonicity assumption has provided a basis for generating testable, ordinal predictions about acquired behavior. Although neither Rescorla nor Wagner ever took a strong position against the role of performance variables, it seems clear that ordinal differences in behavior were taken in the model to reflect ordinal differences in associative strength. After describing some of the successes and failures of the model, we assess the validity of each of these five basic assumptions.

The only prior review to focus primarily on the Rescorla–Wagner model was that of Walkenbach and Haddid (1980). That review made a number of excellent points (which have been incorporated into the present review). However, the Walkenbach and Haddid review is now out of date, and it was not comprehensive even in 1980. Limited reviews not focused primarily on the Rescorla–Wagner model but evaluating select features of the model include Dickinson and Mackintosh (1978), Durlach (1989), and Frey and Sears (1978). To the extent that Dickinson and Mackintosh (1978) and Frey and Sears (1978) evaluated the same phenomena that we examine, we arrive at similar conclusions. Durlach (1989) was concerned with the learning–performance distinction in analyzing behavioral deficits observed in such phenomena as blocking, overshadowing, and the US–preexposure effect. She arrived at conclusions (which emphasized acquisition failures) somewhat discrepant with our views (which emphasize performance failures). We address her concerns in our discussion of the specific behavioral deficits that she examined.

Some Successes of the Rescorla–Wagner Model

Since 1972, the Rescorla–Wagner model has been cited with high frequency in the animal learning literature. Some of these citations have been in the context of post hoc explanations of experimental results well established before 1972 (overshadowing and blocking, as well as acquisition, extinction, stimulus generalization, and discrimination, fit into this category). In other instances, the model has been cited as a source of a priori predictions that have inspired experiments. These latter studies sometimes confirmed the predictions, thereby providing the Rescorla–Wagner model with predictive successes. In other instances, a priori predictions of the model were disconfirmed, thereby providing investigators of animal learning with new behavioral knowledge thanks to the heuristic value, if not the predictive accuracy, of the model. Here we briefly summarize a few of the more commonly cited successes of the Rescorla–Wagner model. Later, in our detailed list of failures of the model, we review data suggesting that some of these apparent successes are mediated by processes other than those posited by the model. With both successes and failures, citations tend to be representative of the literature rather than exhaustive. We note phenomena for which further demonstrations would be desirable to better establish reliability. In addition, because most of these successes and failures have been demonstrated across species (at least rats and pigeons) with appetitive and aversive reinforcers, we do not fully describe the details of the experimental reports that we cite. When there are grounds to question the generality of a phenomenon, we draw this to the reader's attention.

Success 1: Acquisition Curves

The first job of any theory of Pavlovian learning is to account for acquisition curves as a function of the number of training trials. That is, the rate of change in conditioned responding to a CS should decrease as the number of training trials increases, until finally, at asymptote, there is no further change in responding to the CS. The Rescorla–Wagner model successfully accounts for this decelerating rate of change by predicting that the difference between λ and V_{total} (see Equation 1) will decrease across successive training trials. In addition, the typically beneficial effect of greater CS intensity on rate of associative acquisition is addressed in the model by a correspondingly larger value of α, and the beneficial effect of greater US intensity on rate of acquisition is addressed by a greater corresponding value of β. Intensity of the US, as represented by λ, determines the final associative asymptote achieved as a result of simple CS–US pairings and also influences the rate at which the association is acquired.

Success 2: Extinction Curves

The next most reliable phenomenon that a model of learning must accommodate is extinction, which is the loss of responding to a trained CS seen across a series of nonreinforced presentations of that CS (Pavlov, 1927). The Rescorla–Wagner model assumes that λ during extinction is zero and that β for extinction (β_2) is a number smaller than β for acquisition (β_1) but larger than zero. With these additional assumptions, the model anticipates that the associative value of a CS, and hence conditioned responding to that CS, will decrease over nonreinforced trials. Associative strength is predicted to converge asymptotically on zero. Thus, the Rescorla–Wagner model explains extinction in terms of an absolute loss of associative strength. Symmetrical with acquisition, associative change in extinction is represented by decrements in V and is similarly proportional to the associability of the CS (i.e., α, which is closely related to CS intensity). Notably, both the extinction curves and the acquisition curves predicted over successive trials by the Rescorla–Wagner model are negatively accelerated.

Success 3: Stimulus Generalization

Stimulus generalization refers to the ability of one stimulus to elicit a response because it shares some properties with a different stimulus. The degree of stimulus generalization observed is thought to be a direct function of the similarity between the CS used in training and the test stimulus. Although the 1972 statement of the Rescorla–Wagner model says little about the ubiquitous phenomenon of stimulus generalization, Blough (1975) and Rescorla (1976b) suggested a means by which the original model can encompass such generalization. Specifically, they proposed a common elements approach in which generalization between two cues increases as the number of stimulus elements common to the two stimuli increases. Allowing this decomposition of integrated stimuli into elements, the Rescorla–Wagner model makes the following counterintuitive prediction. When a compound cue, AB, has been trained to asymptote (i.e., when $V_A + V_B = \lambda$), Cues A and B will each be partially conditioned, but continued AB compound training will not benefit either because the associative strength of the AB compound equals λ. However, because $V_A < \lambda$, Cue A will acquire further associative value through additional training of A in compound with a neutral cue C (AC → US). That is, the total associative value of AB ($V_A + V_B$) will increase by pairing AC with the US but not by pairing AB with the US. Rescorla (1976b) presented evidence from rats in a conditioned suppression situation that is consistent with this prediction of the model. Further corroboration was provided by Blough (1975) with pigeons in an operant preparation.

Success 4: Discrimination

When two similar CSs are presented separately, one paired with the US (CS+) and the other not paired with the US (CS−), subjects initially respond to both CSs and then begin to reduce responding to CS− while maintaining responding to CS+. The Rescorla–Wagner model is able to simulate the observed acquisition of behavioral discrimination between CS+ and CS−. The model assumes that there are stimulus elements unique to CS+ and to CS−, as well as some stimulus elements that are common to both CS+ and CS−. As a result of reinforcement, all of the elements of CS+ become excitatory. Those elements of CS+ that are also common to CS− are the vehicle for excitatory generalization, causing CS− to have the degree of excitation that it does. The nonreinforcement of CS−, including the excitatory common elements, results in inhibitory learning with respect to the stimulus elements unique to CS−. This inhibitory learning

eventually provides the unique elements of CS− with an inhibitory value that offsets the excitatory tendency of the common elements. Rescorla and Wagner's original 1972 paper provided an elegant example of this.

Successes 1–4 (acquisition, extinction, generalization, and discrimination in general) correspond to basic phenomena that were first reported by Pavlov (1927) and that all serious theories of acquisition have addressed. Hence, success at predicting these phenomena does not distinguish the Rescorla–Wagner model from competing models. Although each competing model also predicts some of the following phenomena, Successes 5–18 are at least relatively unique to the Rescorla–Wagner model in that the related phenomena are not consistently predicted by competing models.

Success 5: Tests for Conditioned Inhibition

Operationally speaking, conditioned inhibitors are CSs that pass negative summation and retardation tests for conditioned inhibition (Hearst, 1972; Rescorla, 1969b). A CS is said to pass a negative summation test for conditioned inhibition if, when it is presented in simultaneous compound with a conditioned excitor, it reduces the level of conditioned responding to that excitor that would otherwise occur. The Rescorla–Wagner model explains the summation test performance of an inhibitor in terms of the inhibitor possessing a negative associative strength that summates with (i.e., subtracts from) the positive associative strength of the excitor used in compound testing. This summation of inhibitory and excitatory value results in a smaller net associative strength for the compound than that of the test excitor alone. A CS is said to pass a retardation test for conditioned inhibition if it requires more pairings with the US to become an effective conditioned excitor than if the CS were novel (i.e., had not undergone inhibitory training). The Rescorla–Wagner model explains the retardation test performance of an inhibitor in terms of the inhibitor starting the sequence of CS → US pairings with a negative associative strength. The negative associative strength of the inhibitory stimulus must first be restored to zero, by CS → US pairings, before further CS → US pairings can result in a net positive associative strength for the CS. It is worth noting that, in the framework of the Rescorla–Wagner model, the inhibitory CS is retarded in coming to elicit excitatory behavior, not in the rate of change in its associative status (i.e., learning). Thus, the Rescorla–Wagner model successfully explains performance in summation and retardation tests for conditioned inhibition.

Success 6: Procedures for Producing Conditioned Inhibition

The two most widely used means of creating a conditioned inhibitor consist of not reinforcing the intended inhibitory CS in the presence of another cue that itself has been previously reinforced. In the procedure of Pavlov (1927), trials in which Stimulus A is paired with the US (thereby giving A excitatory value) are interspersed with trials in which Stimulus A and Stimulus X (the intended inhibitory cue) are presented together without the US (A → US/AX−). In the negative contingency procedure (Rescorla, 1969a), trials in which the US is presented without an accompanying discrete signal (thereby giving the context excitatory value) are interspersed with trials in which Stimulus X is presented without the US (i.e., presented together with the excitatory contextual cues). These two procedures are well documented to produce conditioned inhibition, defined by passage of the traditional summation and retardation tests for inhibition (Hearst, 1972; Rescorla, 1969b), in a large variety of preparations, including human eyelid conditioning, conditioned barpress suppression in rats, and autoshaping in pigeons. In both procedures, the Rescorla–Wagner model predicts that Stimulus X will become a conditioned inhibitor because the parenthetical term in Equation 1 for ΔV_X will be negative. This occurs because, on nonreinforced trials, λ is 0 and V_{total} is positive as a result of Stimulus A or the contextual cues being excitatory. Thus, the Rescorla–Wagner model readily explains why both procedures make Stimulus X a conditioned inhibitor.

Success 7: Patterning

One of the major questions in learning concerns identification of the effective stimuli. Rescorla and Wagner (1972) did not give as much attention to this issue as they did to the rules of learning. Their own concern for the question of effective stimuli can best be appreciated by considering their analysis of patterning. Patterning refers to a special kind of discrimination. In positive patterning, presentations of a compound of two stimuli are consistently followed by reinforcement (AB → US), but separate presentations of each stimulus alone are not followed by reinforcement (A− and B−). A subject is said to have solved the positive patterning problem when it responds to presentations of the AB compound but does not respond to presentations of either A or B alone. In negative patterning, presentations of the AB compound are not reinforced (AB−), but separate presentations of Stimulus A and Stimulus B are reinforced (A → US and B → US). The solution to the negative patterning problem is, of course, to respond to the separately presented stimuli but not to respond to the AB compound. With sufficient training, subjects can solve each of these patterning problems (e.g., Rescorla, 1973; Saavedra, 1975; Woodbury, 1943).

The Rescorla–Wagner model explains both types of patterning behavior by treating the AB compound as consisting of three stimuli: A, B, and the "unique configural cue" AB. Presentation of the AB compound presumably activates representations of all three stimuli, each of which can acquire associative strength. In the Rescorla–Wagner framework, after many positive patterning trials, the AB unique stimulus is thought to acquire all of the available associative strength, and Stimulus A and Stimulus B are left with associative strengths of zero. The situation is slightly more complex in the case of negative patterning. Stimulus A and Stimulus B are each assumed to acquire excitatory associative strength. After many negative patterning trials, the AB unique configural stimulus is left with negative associative strength (i.e., it is a conditioned inhibitor) that is equal and opposite to the total positive associative strengths acquired by Stimulus A and Stimulus B. Presentations of the AB compound are thus thought to activate the excitatory associative strengths of Stimuli A and B, as well as the inhibitory associative strength of the AB unique configural cue. Thus, the net associative

strength activated by presentation of the AB compound is zero. Consequently, and consistent with observed behavior, no responding to the AB compound is predicted. Note that the success of the Rescorla–Wagner model in this case depends on hypothesizing unique configural cues but not on other properties of the model. Consequently, this success does not differentiate the Rescorla–Wagner model from most other models that assume the existence of unique configural cues. Moreover, on the negative side of this issue, there is a growing body of evidence (e.g., Pearce & Wilson, 1991a) suggesting that, in some situations, the conjoint presentation of A and B is processed as a single configured AB stimulus without any appreciable representation of A alone or B alone.

Success 8: Overshadowing

If novel Stimulus A is presented simultaneously (i.e., in compound) with novel Stimulus X, reinforcement of the AX compound will result in less conditioned responding to Stimulus X than if Stimulus X had been reinforced in the absence of A (e.g., Kamin, 1969; Mackintosh, 1976; Pavlov, 1927). The Rescorla–Wagner model correctly anticipates this difference in responding, which is called overshadowing. It does so by assuming that the US will be less effective in entering into an association with X when A is present on the reinforced trial than when A is absent. On early compound AX → US trials, Stimulus A (as well as X) acquires associative strength that renders the US on subsequent AX → US trials less surprising than if A were not present on these later trials. Thus, the Rescorla–Wagner model predicts overshadowing not on the first AX → US trial but on subsequent AX → US trials. In mathematical terms, overshadowing is represented by V_{total} in the parenthetical term of Equation 1 (which, in the present case, is equal to $V_X + V_A$) being larger when training of X occurs in the presence of A, which has gained associative strength on earlier compound trials, than in the absence of A. The greater value of V_{total} reduces the magnitude of the parenthetical term, which in turn decreases the value of ΔV_X for that trial. As the rate of acquisition of associative strength by Stimulus A increases with the salience of A (i.e., α for A), greater overshadowing of X by A is anticipated.

Success 9: Relative Validity of Cues

If a compound of Stimuli A and X is consistently reinforced on some trials (AX → US), whereas, on other trials, a compound of Stimuli B and X is consistently not reinforced (BX−), X will become a weak elicitor of conditioned responding. In contrast, if AX and BX are each reinforced on 50% of their presentations, Stimulus X will become a strong elicitor of responding. This difference in responding to Stimulus X is counterintuitive in that, in both cases, X has been reinforced on 50% of its presentations. This phenomenon, called the relative validity effect, was first reported by Wagner et al. (1968). Those authors observed an effect of relative validity in both eyelid conditioning in rabbits and conditioned barpress suppression in rats. Subsequently, the effect has been demonstrated in pigeons (Wasserman, 1974) and humans (Wasserman, 1990). The Rescorla–Wagner model successfully predicts the relative validity effect, provided β_1 (for reinforced trials) is greater than β_2 (for nonreinforced trials, see Success 2). Without this provision, the model predicts that A would become highly excitatory, B would become highly inhibitory, and X would become moderately excitatory (with, at asymptote, the inhibitory status of B protecting X from losing further associative strength). In the condition in which AX is reinforced 100% of the time, Stimulus A is expected to eventually acquire much of the available associative strength because Stimulus X on nonreinforced BX trials should lose some of the excitatory associative strength it previously gained on reinforced AX trials. On subsequent reinforced AX trials, Stimulus A should gain part of the associative strength lost by Stimulus X on nonreinforced BX trials. In contrast, when both AX and BX are reinforced 50% of the time, Stimulus X (as well as A and B) should acquire and retain a moderate level of associative strength. The nonreinforced trials during partial reinforcement of AX (and BX) are predicted to prevent A (and B) from absorbing the vast majority of the available associative strength distributed on reinforced AX (and BX) trials. Thus, VX, VA, and VB are expected to oscillate around moderate values, rising slightly on reinforced trials and falling slightly on nonreinforced trials. These predictions are entirely consistent with the observed relative validity effect.

Success 10: Blocking

If Stimulus A is made excitatory through A → US pairings before a compound consisting of A and X is paired with the US (AX → US), resultant responding to X is diminished relative to that of control subjects that lack the A → US pretraining (Kamin, 1969). Blocking of X by pretraining of A is predicted by the Rescorla–Wagner model. The Rescorla–Wagner explanation of blocking is similar to that for overshadowing. Specifically, Phase 1 training with A (A → US) is thought to cause the US to become less effective at entering into associations with X during the later reinforced AX compound trials than without such prior A → US training. According to the model, Phase 1 training with A elevates V_{total} so that the parenthetical term in Equation 1, $\lambda - V_{total}$, is smaller for Stimulus X on the Phase 2 AX → US trials than if Stimulus A had not been pretrained. Notably, the Rescorla–Wagner model anticipates blocking even with a single reinforced AX compound trial, unlike some alternative explanations of blocking that require at least two compound trials for blocking to appear (e.g., Mackintosh, 1975; Pearce & Hall, 1980). Consistent with this prediction, Azorlosa and Cicala (1986); Balaz, Kasprow, and Miller (1982); and Gillan and Domjan (1977) have reported one-trial blocking.

Success 11: Unblocking With an Increased US

The blocking of Stimulus X just described can be attenuated (i.e., unblocked) if the magnitude of the US during the Phase 2 AX → US trials is increased relative to what it was during the A → US preconditioning trials of Phase 1 (Kamin, 1969). The Rescorla–Wagner model anticipates this unblocking effect. In terms of Equation 1, this unblocking effect arises because the increased US has a larger λ than the original US. This increment in λ results in a larger value of the parenthetical term, $\lambda - V_{total}$, on the AX → US trials and a resultant increase of conditioning (unblocking) to X.

Success 12: Blocking With a Reduced US

If the US in the AX compound conditioning phase of a blocking procedure is of lower intensity (i.e., lesser λ) than the US was in the Stimulus A pretraining phase, the blocked stimulus appears not only to acquire less excitatory control of behavior than it would if Stimulus A had not been previously paired with the US, but it also can gain inhibitory control over behavior (Wagner, Mazur, Donegan, & Pfautz, 1980). The Rescorla–Wagner model predicts acquisition of inhibition by a novel stimulus whenever the delivered US is weaker than that predicted by all of the cues present on that trial. In terms of Equation 1, inhibition is expected when λ for the attenuated US is less than the acquired associative strength of V_A. Under these conditions, the parenthetical term, $\lambda - V_{total}$, will assume a negative value, which in turn will cause Stimulus X to acquire negative (i.e., inhibitory) associative strength. This successful prediction of the Rescorla–Wagner model is counterintuitive because, in the ongoing example, Stimulus X has a history of consistent reinforcement. Moreover, this prediction is contrary to what might be expected on the basis of Kamin's (1969) notion that surprise is necessary for learning to occur.[1] A reduction in US intensity going into the compound conditioning phase in Kamin's framework might be expected to surprise the subject and therefore stimulate excitatory acquisition about the new, weak US. Consistent with the Rescorla–Wagner model and problematic for Kamin's view, Wagner et al. observed inhibition rather than excitation in this situation (see Cotton, Goodall, & Mackintosh, 1982 [discussed in Failure 6], for illumination and qualification of this finding).

Success 13: Overexpectation Resulting From Two Excitors

After separate asymptotic conditioning of Stimuli A and X with a common US (A → US and X → US), reinforcement of A and X in compound with the same US (AX → US) decreases the level of responding to A and to X (e.g., Kremer, 1978, Experiment 2; Levitan, 1975; Rescorla, 1970; Wagner, 1971, p. 201). The Rescorla–Wagner model anticipates this outcome. In terms of Equation 1, V_{total} approaches twice the value of λ at the beginning of compound AX training (one λ resulting from V_A and one λ resulting from V_X). Thus, the parenthetical term, $\lambda - V_{total}$, assumes a negative value, which makes ΔV for Stimulus A and ΔV for Stimulus X negative numbers, with a resultant loss of excitatory associative strength.

Success 14: Superconditioning

In a variation of the blocking procedure, Stimulus A may be trained in Phase 1 as an inhibitor instead of as an excitor, before reinforced compound training (AX → US) in Phase 2. Given this procedure, the Rescorla–Wagner model anticipates that Stimulus X will become a stronger excitor than if A had not been previously trained as an inhibitor. This prediction for what has been called "superconditioning" arises because the value of the parenthetical term of Equation 1, $\lambda - V_{total}$, is enhanced by inhibitory pretraining. This enhancement occurs because V_{total} in Phase 2 is negative as a result of Stimulus A having been previously trained as an inhibitor. Rescorla (1971) and Wagner (1971) have confirmed this prediction.

Success 15: US–Preexposure Effect

Pairings of a CS and a US in a training context in which prior unsignaled USs were administered result in less conditioned responding to the CS than if unsignaled USs had not been administered (Randich, 1981; Randich & LoLordo, 1979; Tomie, 1976). The Rescorla–Wagner model explains this US–preexposure effect through blocking (see Success 10) of the CS by the training context. An important aspect of the Rescorla–Wagner analysis of the US–preexposure effect is that contextual stimuli act as any short-duration "standard" CS might (which is also the case in Successes 16 and 17 that follow). The model also correctly predicts that if the context is extinguished or switched between the unsignaled US presentations and the subsequent CS → US pairings, the deficit in responding to the CS as a result of US preexposure will be attenuated (Hinson, 1982; Randich & Ross, 1984). Both of these latter manipulations in the framework of the model are viewed as decreasing the contribution of $V_{context}$ to V_{total} before the reinforced CS–context "compound" trials. This presumably undermines the ability of the context to block the CS.

Success 16: Contingency Effect

The contingency effect refers to impaired responding to a target CS seen when extra unsignaled US presentations occur between target CS–US training trials (e.g., Rescorla, 1968). The Rescorla–Wagner model is able to explain the contingency effect through the same mechanism that it uses to explain the US–preexposure effect. That is, conditioning of the training context is assumed to allow the context to successfully compete with the target CS for associative strength (but see Jenkins & Shattuck, 1981). Moreover, the Rescorla–Wagner model correctly predicts that signaling the extra USs with a cue other than the target CS will alleviate the contingency effect deficit in conditioned responding (e.g., Dickinson & Charnock, 1985; Durlach, 1983; Goddard & Jenkins, 1987).

Success 17: Trial Spacing Effect

Training trials that are spaced in time are well known to be more effective than massed training trials (e.g., Jenkins, Barnes, & Barrera, 1981; Papini & Dudley, 1993; Prokasy, Grant, & Myers, 1958). The Rescorla–Wagner model is able to address this common observation by assuming that the training context acquires associative strength with each CS–US pairing (by virtue of the US presentation) and loses associative strength

[1] Kamin's (1969) view of surprise is different from surprise as it is embodied in the $\lambda - V_{total}$ parenthetical term of the Rescorla–Wagner model (see Equation 1). In Rescorla–Wagner terms, Kamin conceptualized surprise as a deviation between λ and V_{total} independent of its sign (i.e., the absolute value of $\lambda - V_{total}$). In contrast, the sign of the difference is critical to the predictions of the Rescorla–Wagner model because the sign differentiates increases from decreases in V resulting from a specific trial.

through extinction during each intertrial interval (in which the US is not presented). With short intertrial intervals, the context should undergo relatively little extinction and, consequently, is expected to partially overshadow the CS on subsequent trials. However, with longer intertrial intervals, the contextual cues should undergo relatively more extinction between CS–US pairings and, consequently, should be less able to overshadow the CS on subsequent trials. Mustaca, Gabelli, Papini, and Balsam (1991) presented extensive data supportive of this interpretation of the trial spacing effect. Thus, the Rescorla–Wagner model successfully explains the basic trial spacing effect by assuming that the training context cues are less effective in competing with the CS for associative strength in spaced conditions than in massed conditions.

Success 18: Instrumental Behavior

The Rescorla–Wagner model was designed to account for Pavlovian conditioning phenomena. However, if one assumes that a response can play an associative role equivalent to that of a CS, the model can be applied to instrumental learning tasks. This has been done in numerous instances with considerable success. One example is Hall, Channell, and Pearce's (1981) demonstration of overshadowing of an instrumental response by a simultaneously presented CS.[2] In this study, a CS that was presented whenever the subject responded attenuated the rate of operant responding. This occurred, presumably, because the CS successfully competed with the response for associations to the reinforcer. Another example was provided by Garrud, Goodall, and Mackintosh (1981), who found that a potential CS could be overshadowed by an instrumental response. This interchangeability of responses and CSs is consistent with the view that reinforcers can be equally well predicted by stimuli or responses.

Prior Emphasis of the Model's Successes

The Rescorla–Wagner model successfully predicts a number of phenomena central to the analysis of Pavlovian conditioning and, as just suggested, some instrumental learning phenomena. For purposes of presentation to individuals other than animal behavior researchers (e.g., cognitive psychologists, psychobiologists, and college undergraduates), these successes have been packaged in textbooks (e.g., Lieberman, 1991) in an oversimplified picture of the Rescorla–Wagner model that deemphasizes the model's many failures (but see Domjan, 1993, pp. 114–115). This is not surprising because educators commonly instruct by first presenting rules and confirming examples and only then muddying the waters with exceptions to the rules. If something has to be omitted for the sake of brevity, it is usually an exception. The Rescorla–Wagner model has proven successful and popular because it has (a) the ability to generate clear, ordinal predictions; (b) a number of predictive successes; (c) intuitive appeal of its central point that event representations are processed to the degree that the events are (up to a point) intense (i.e., λ in Equation 1) and unexpected (i.e., $\lambda - V_{total}$); (d) considerable heuristic value; (e) relatively few free parameters and independent variables; and (f) little competition from other theories that possess all of the preceding virtues.

Most investigators of animal learning are well aware of many of the failings of the Rescorla–Wagner model as well as its successes. Rescorla and Wagner themselves were, in fact, among the first to point out failures of the model. For example, Rescorla has reported much of the data problematic to the model (e.g., Zimmer-Hart & Rescorla, 1974). Moreover, Wagner has been in the forefront of those who are trying to develop improved theories that address the difficulties with the model (e.g., Wagner, 1981; Wagner & Brandon, 1989). In contrast, as just mentioned, researchers outside of animal learning are often exposed to a simplified picture of the model that includes only its successes. A realistic assessment of the model requires acknowledgment of the failures as well as the successes. Without minimizing the many successes of the Rescorla–Wagner model, the forthcoming focus of the present review is on unsuccessful predictions and underlying erroneous assumptions of the model because the shortcomings rather than the strengths of the model previously have been overlooked. We allude only briefly to alternative models here because there is no other tractable model that manages to explain all or even most of the phenomena that are troublesome to the Rescorla–Wagner model.

Some Failures of the Model

The following phenomena are contrary to explicit assumptions of the Rescorla–Wagner (1972) model or to predictions derived from these assumptions. Our list of failures of the model is not meant to be comprehensive (e.g., Pearce & Wilson, 1991a, provided additional data problematic for the model). Instead, we have limited our list largely to instances in which the Rescorla–Wagner prediction is obvious without long discussion or the need for computational simulation. Despite this type of selection, the evidence for some of these failures might be viewed as arguable by some researchers. Yet, even a somewhat shortened list of troublesome observations would testify that the model does not always correctly predict behavior. Failures of the model are listed in descending order of what we regard as their importance, with some deviation to list behaviorally related phenomena together. Within the list, we attempt to indicate when failures represent truly serious flaws to the model and when failures can potentially be accommodated by the model. Our list of failures is lengthy, which might be interpreted to reflect poorly on the Rescorla–Wagner model. However, many of the following behavioral phenomena would not have come to light without the model suggesting the critical experiments. In this heuristic sense, even its failures reflect well on the model.

The following list of failed assumptions and predictions of the model is restricted to errors of commission. We review only those phenomena that refute clear assumptions and predictions of the model rather than cite phenomena that the model overlooks. These latter phenomena are errors of omission and have been excluded because they lie outside the domain of the model

[2] The success of the Rescorla–Wagner model in addressing select instrumental phenomena is not to suggest that the model is applicable to all instrumental phenomena. For example, it is unable to explain the paradoxical effects of reward magnitude or the overlearning reversal effect (Mackintosh, 1974; but see Daly & Daly, 1982).

(e.g., occasion setting, perceptual learning, and effects of varying the CS–US interval). Differentiating errors of commission from errors of omission is not always easy. Some readers may view one or another of what we identify as an error of commission as actually being an error of omission. Nevertheless, the line for inclusion in such a review as this must be drawn somewhere, and our conclusions concerning the Rescorla–Wagner model do not rest on the acceptance of all of the itemized failures as being errors of commission. Rather, our point would be made even if only a fraction of these failures are viewed as errors of commission. At the end of this list of failures of the Rescorla–Wagner model, we review which initial assumptions give rise to each of the observed failures. As shown later, each of the five assumptions of the model discussed earlier falls into question as a result of one or more of the following observations.

Failure 1: Spontaneous Recovery, External Disinhibition, and Reminder-Induced Recovery From Extinction

The Rescorla–Wagner model views extinction as a loss of associative strength, that is, unlearning. Unlearning is represented in the model by V_X decreasing toward zero. Consequently, the model explicitly predicts no recovery from extinction in the absence of further training. However, there are several examples of the effects of extinction being reversible in the absence of additional training. One such instance is spontaneous recovery from extinction, which is often seen over extended retention intervals (e.g., Pavlov, 1927; Robbins, 1990).

A second instance of recovery from extinction is the phenomenon of external disinhibition, which is a temporary recovery of conditioned responding after extinction when a physically intense but associatively neutral cue immediately precedes the test CS (e.g., Bottjer, 1982; Pavlov, 1927). One possible addendum to the Rescorla–Wagner model that might begin to accommodate these types of recovery from extinction would be to decompose each CS into its component elements and to assume that not all elements of a stimulus are sampled on each trial. Therefore, extinction might affect some elements more than others. Either a long retention interval or presentation of an external disinhibitor at test could result in a shift in the stimulus elements sampled from those that were well extinguished to those that were less well extinguished. Such an approach might be viewed as reconciling the Rescorla–Wagner model with spontaneous recovery from extinction and external disinhibition. However, the problem with this approach is that limited sampling of stimulus elements is equivalent to allowing α for each attribute to vary across trials, a position that is contrary to the Rescorla–Wagner model (see Cluster 3 in the Underlying Bases for Failures section). Thus, this sort of reconciliation is not fully successful. Perhaps a more viable reconciliation of spontaneous recovery and external disinhibition with the Rescorla–Wagner model could be achieved by viewing memory traces of recent past trials as part of the immediate effective CS. Indeed, this kind of possibility has been suggested by Wagner and Rescorla (1972, p. 309). Thus, active memories of nonreinforced trials might serve as a cue for nonreinforcement. However, a long retention interval or a distracting cue (as is presented in the external disinhibition procedure) could result in the deactivation of this memory trace. Hence, these problems for the Rescorla–Wagner model can be resolved by the addendum that active memory traces can serve as CSs. Although this assumption is not contrary to the spirit of the model, it is not embodied in the model's formal statement.

A third instance of recovery from extinction is provided by "reminder" treatments, which consist of presenting some cue from training (i.e., the CS or the US) without providing another complete training trial. Under appropriate circumstances, a reminder treatment between extinction and testing restores the extinguished response to the CS. One of the most effective reminder cues appears to be the US from training (e.g., Rescorla & Heth, 1975). When achieved with the US from training, such a reminder-induced recovery of responding is sometimes called a reinstatement effect. Not all postextinction reinstatement effects are problematic for the Rescorla–Wagner model. Bouton and Bolles (1979) have suggested that reinstatement can arise from context–US associations formed during the US-alone reinstatement presentations. In this view, the context–US associations later summate with postextinction residual components of the CS–US association, thus producing the reinstatement effect. Such residual CS–US components probably survive extinction treatment because no amount of extinction is apt to completely eradicate associative strength. It is important to recognize that this summation explanation of postextinction reinstatement is plausible whenever testing occurs in the same context that was used for the US reminder. To the extent that this summative mechanism obtains, improved responding after reinstatement reflects further learning (about the test context) and is not problematic for the Rescorla–Wagner model.

Although the summation mechanism can explain some instances of reinstatement, other demonstrations of reinstatement have carefully avoided testing the CS in the same context in which the reminder treatment had been given (e.g., Schachtman, Brown, & Miller, 1985). Moreover, Bouton (1984) has demonstrated that extinguished associations are more prone to reinstatement-induced enhancement than are initially weak associations that were never extinguished. This could occur only if extinguished associations survived in some latent form. Thus, at least under some conditions, the reinstatement effect appears to arise from reactivation of the extinguished CS–US association rather than from further learning about the test context. This is contrary to the Rescorla–Wagner model's view that extinction reflects a permanent loss of an association. Although these recoveries from extinction are rarely complete, that they occur to any appreciable degree is problematic for the Rescorla–Wagner model. To reconcile this phenomenon with the model, one might view the active trace of the reminder treatment as part of the effective CS on the subsequent test trial. Many reminder–reinstatement effects can be understood in this framework, but there are instances in which reminder–reinstatement effects have been observed on test trials that occur several days after the reminder treatment (e.g., Schachtman, Brown, & Miller, 1985). This latter observation calls into question the usefulness of the view that reminder effects can be explained by the active memory trace of the reminder treatment serving as part of the effective CS on a later test trial (which presumably makes the test CS more effective). If the test trial occurs several days after the reminder treatment, the active

memory trace for the reminder treatment would have decayed, taking any potential recovery benefit with it.

In contrast to the Rescorla–Wagner model view of extinction as unlearning, interference theory (e.g., Bouton, 1993; Miller, Kasprow, & Schachtman, 1986) views behavioral extinction as a consequence of the memory of extinction treatment interfering with retrieval of the memory of acquisition training. Increased retention intervals (i.e., as in spontaneous recovery) and reminder treatments presumably bias the competition in retrieval between memories of extinction treatment and memories of acquisition training to favor retrieval of memories of acquisition training (see Bouton, 1993, for details as to why this is predicted by interference theory).

Failure 2: Facilitated and Retarded Reacquisition After Extinction

After extinction of Pavlovian conditioned responding, reacquisition to asymptotic levels of responding ordinarily occurs in far fewer trials than initial acquisition (e.g., Frey & Ross, 1968; Hoeler, Kirschenbaum, & Leonard, 1973; Smith & Gormezano, 1965). A particularly well-controlled demonstration of this effect was provided by Napier, Macrae, and Kehoe (1992). Using the nictitating membrane response in rabbits, they reported facilitated reacquisition after extinction even after they controlled for incomplete extinction, generalization decrement between extinction and reacquisition, and spontaneous recovery. As noted earlier, after extinction is empirically complete, the absence of responding could reflect a residual CS–US association that is below some threshold for responding. Such subthreshold associations could contribute to the typically observed facilitated reacquisition. Indeed, associative thresholds for responding were suggested by Rescorla and Wagner as an ancillary feature of the model and today are widely viewed as a necessary modification. However, Napier et al. used several procedures to eliminate subthreshold CS–US associative strength. Thus, relearning about the CS during retraining should have occurred at the same rate as initial training. In contradiction to this prediction, they still observed facilitated reacquisition.

This research indicates that facilitated reacquisition is not always the consequence of residual associative strength (at least in the Rescorla–Wagner sense) surviving extinction treatment. The observed facilitated reacquisition after extinction leads to the same conclusion as recovery from extinction without retraining (see Failure 1). That is, contrary to the Rescorla–Wagner model, extinction of conditioned responding does not reflect a permanent loss of an association. One possible means of reconciling rapid reacquisition after extinction with the Rescorla–Wagner model would again be to regard active memory traces of immediately prior reinforced trials as part of the stimulus elements constituting the effective CS (see Success 4). A test of this position might consist of making the reacquisition trials widely spaced so that memory traces would not be likely to stay active between trials. To our knowledge, this prediction has not been examined with proper control groups.

In contrast to the ordinarily observed facilitated reacquisition, there are also some reports of retarded reacquisition after extinction (e.g., Bouton, 1986). Retarded reacquisition appears to depend on massive overextinction treatment, that is, continued extinction treatment even after responding has ceased (e.g., Pavlov's [1927] "extinction below zero"). This dependence suggests that facilitated reacquisition, with less extinction treatment, might arise from residual excitatory associative strength below the threshold for eliciting conditioned responding. On this possibility, massive overextinction might eliminate the residual subthreshold associations, thus eliminating their contribution to facilitated reacquisition. Thus, as previously suggested, facilitated reacquisition might be viewed as compatible with the Rescorla–Wagner model. However, retarded reacquisition observed after protracted extinction would be inconsistent with the prediction of the Rescorla–Wagner model that, after complete extinction, reacquisition should be indistinguishable from initial acquisition. Moreover, unlike the case of facilitated reacquisition, adding the concept of a threshold for responding to the Rescorla–Wagner model fails to reconcile the model with reports of retarded reacquisition after extinction. Although the basis of retarded reacquisition after extinction of a CS is not yet fully understood, it may arise from the same processes that are responsible for the retarded acquisition seen in the CS–preexposure effect (see Failure 7).

Failure 3: Failure to Extinguish a Conditioned Inhibitor

One of the most elementary predictions of the Rescorla–Wagner model concerns the effect of presenting an inhibitory stimulus alone (i.e., operational extinction) after the completion of conditioned inhibition training. The model assumes that conditioned excitation and inhibition reflect positive and negative values, respectively, of a common variable representing associative strength. Consequently, symmetry between excitation and inhibition is expected. On an extinction trial, λ in Equation 1 is zero. Consequently, the parenthetical term, $\lambda - V_{total}$, will be negative for excitatory CSs and positive for inhibitory CSs. Therefore, the model predicts that V for both types of CSs should move toward zero as a result of nonreinforced presentation of the CS. Just as CS-alone presentations after excitatory training reduce the CS's excitatory response potential (i.e., conventional extinction as reported by Pavlov, 1927), so too should CS-alone presentations after inhibitory training reduce the CS's inhibitory response potential.

There are many reports documenting the fallacy of this latter prediction, the first of which was by Zimmer-Hart and Rescorla (1974). CS-alone presentations of a conditioned inhibitor either have no effect on the CS's inhibitory potential or increase the CS's inhibitory potential (e.g., DeVito & Fowler, 1986, 1987; Hallam, Grahame, Harris, & Miller, 1992; Williams, Travis, & Overmier, 1986; but see Robbins, 1990). These observations are among the more problematic for the Rescorla–Wagner model. Several alternative accounts of conditioned inhibition have been formulated. For example, Rescorla (1975; also see Konorski, 1948) has proposed that inhibitory cues raise memory activation thresholds necessary for a response to appear, and Rescorla (1985; also see Konorski, 1967) has proposed that a conditioned inhibitor is a negative conditional discriminative stimulus. However, each of these accounts of conditioned inhibition represents a significant departure from the Rescorla–Wagner model.

Failure 4: Nonreinforcement of a Novel Cue in the Presence of a Conditioned Inhibitor

Nonreinforcement of an associatively neutral cue (X) in the presence of a previously established conditioned excitor (A) is known to transform Stimulus X into a Pavlovian conditioned inhibitor (especially when the reinforced A → US trials and nonreinforced AX− trials are interspersed [but see Failure 8]). This observation is consistent with the Rescorla–Wagner model. However, because of the presumed symmetry between excitation and inhibition, the Rescorla–Wagner model also predicts that nonreinforcement of a neutral cue in the presence of a previously established conditioned inhibitor will transform the neutral cue into an excitatory CS. This counterintuitive prediction of a cue becoming excitatory without reinforcement stems from the parenthetical term in Equation 1, $\lambda - V_{total}$, having a positive value because λ is zero and V_{total} is negative. Rescorla (1971) initially reported support for this prediction, but in 1976 (Rescorla, 1976a) he reinterpreted his 1971 data in light of a potential confound by excitatory second-order conditioning and retracted his earlier conclusion. Baker (1974; also see Soltysik, 1985) subsequently tested this prediction, avoiding Rescorla's earlier confound by using a procedure that minimized excitatory second-order conditioning. In contradiction with the Rescorla–Wagner model, Baker found no evidence of the neutral cue having become excitatory.

Failure 5: Nonexclusiveness of Conditioned Excitation and Conditioned Inhibition

As previously stated, one of the principal tenets of the Rescorla–Wagner model is that conditioned excitation and inhibition are opposite sides of a common dimension of associative strength. Positive values of V correspond to excitation, and negative values of V correspond to inhibition. Consequently, a CS should not be able to simultaneously serve as both a conditioned excitor and a conditioned inhibitor for the same US. Notably, this exclusiveness of excitation and inhibition contradicts earlier views of excitation and inhibition (e.g., Hull, 1943; Pavlov, 1927), as well as some more contemporary models (e.g., Pearce & Hall, 1980). However, the Rescorla–Wagner position was so widely accepted that a number of years passed before anyone critically examined the validity of the assumption that inhibition and excitation were mutually exclusive.

In 1986, Tait and Saladin reported that a CS that was backward paired with a US (US → CS) became excitatory but was subsequently retarded in acquiring further excitatory strength during forward pairings with the same US (CS → US). Specifically, after backward pairings with a paraorbital shock US, an auditory CS came to elicit excitatory suppression of licking in trained subjects (rabbits) relative to appropriate control subjects (the CS acted as an excitor). Now recall that retardation in acquiring excitatory control is one of the tests commonly used to assess conditioned inhibition (see Success 5). In a second phase of the study, the subjects that had previously experienced backward pairings were found to require more forward pairings (CS → US) before they exhibited a conditioned eyeblink than were required for otherwise equivalent control subjects that lacked the initial backward pairings. This suggests that the CS was an inhibitor. These observations suggest that a stimulus can simultaneously be both an excitor and an inhibitor. However, Tait and Saladin did not include a summation test for inhibition to fully document that their CS was an inhibitor as conventionally defined. In addition, it remains possible that the dissimilar response systems used (lick suppression and eyeblink) may have tapped into different memory systems (e.g., consummatory vs. preparatory).

Subsequently, Matzel, Gladstein, and Miller (1988) conducted related studies using conditioned lick suppression to measure both the excitation and inhibition supported by a single CS. They exposed rats to negative contingency treatment with a low density of partial reinforcement of the CS. The CS functioned as an excitor relative to controls when it was presented alone and also passed both summation and retardation tests for inhibition (see also Pearce & Wilson, 1991b). The observations of both Tait and Saladin (1986) and Matzel, Gladstein, and Miller (1988; see also Droungas & LoLordo, 1994; Williams & Overmier, 1988) are contrary to the incompatibility of conditioned excitation and inhibition postulated by the Rescorla–Wagner model. The idea that a stimulus can have both excitatory and inhibitory properties is consistent with many models of learning (e.g., Hull, 1943; Pearce & Hall, 1980; Wagner, 1981). The conditions under which those properties are expressed in behavior need to be determined more fully.

Failure 6: Dependence of Negative Summation on the Degree to Which the Transfer Excitor Is Excitatory

In the Rescorla–Wagner framework, an inhibitory CS should pass a summation test for inhibition whenever it is paired with any (transfer) excitor that signals the same qualitative US that was used in inhibitory training (Wagner & Rescorla, 1972). Exactly how the transfer excitor is made excitatory should be irrelevant to the outcome of the summation test as long as V for the transfer excitor is positive. As one test of this assumption, Cotton et al. (1982; see also Mackintosh & Cotton, 1985) trained a target CS (X) by presenting it simultaneously with a signal (A) for an intense US and reinforced this stimulus compound with a weak US (A → strong US, followed by AX → weak US). Stimulus X subsequently passed a negative summation test for inhibition (i.e., the inhibitor reduced responding below that to a test trial excitor [B] alone) when the test trial excitor had itself previously been trained with a more intense US (B → strong US) than that used during target training. However, Stimulus X yielded positive summation (i.e., the inhibitor enhanced responding above that to the test trial excitor [B] alone) when the test trial excitor had been trained with a US less intense than that used during target training (B → weak US).

Congruent with this finding, Nelson (1987) reported work in which a partially reinforced CS was trained with a negative contingency (i.e., the US was more likely in the absence than in the presence of the CS). When this target CS was subsequently presented simultaneously with a test trial excitor that itself had previously been trained with a higher percentage reinforcement than the target CS, the CS passed a negative summation test for inhibition. However, the identically trained target CS yielded positive summation when the summation test was with a test trial excitor that had been produced with a lower percentage

reinforcement than was used in training the target CS. Again, the potential of a conditioned inhibitor to pass a summation test for inhibition depended on the excitatory strength of the excitor used on the test.

Both of these observations indicate that associative summation of two CSs independently trained with the same qualitative US is not simply a sum of the associative strengths of the two CSs, as is predicted by the Rescorla–Wagner model. This conclusion raises questions about the adequacy of the model's explanation of how a putative conditioned inhibitor comes to pass a summation test for conditioned inhibition (see Success 6) and qualifies the success (Success 12) of the model in predicting that the blocked stimulus will become an inhibitor if the US in Phase 2 (AX+) is weaker than the US in Phase 1 (A++).

Failure 7: CS–Preexposure Effect

The CS–preexposure effect, sometimes called "latent inhibition," is the retarded behavioral control acquired by a CS during CS → US training that occurs as a result of prior nonreinforced exposure to the CS (Lubow & Moore, 1959). This retardation is evident with either excitatory or inhibitory training and, consequently, does not reflect acquisition of inhibition during the preexposure to the CS. Because no US is present during pretraining exposure to the CS, the Rescorla–Wagner model predicts that nothing should be learned about the CS at this time. Consequently, a preexposed and nonpreexposed CS should have the same associative status at the initiation of later reinforced training. As a result, the subjects preexposed to the CS should acquire the CS–US association at the same rate as control subjects lacking the CS preexposure experience. This prediction is incompatible with the retarded acquisition of excitatory responding ordinarily observed in preexposed subjects.

The most common explanation of the CS preexposure effect is a loss of attention to the CS as a result of the nonreinforced pretraining exposures to the CS (e.g., Lubow, Weiner, & Schnur, 1981), which, in the Rescorla–Wagner framework, might be conceptualized as a decrease in α. Although Wagner and Rescorla (1972) admitted that the CS–preexposure effect is best explained in terms of decreasing α, they chose not to incorporate the changing of α values into their model. The formal Rescorla–Wagner model states that α is a constant for a given CS. This position is important to the model for reasons that we discuss later (see Cluster 3).

Failure 8: Second-Order Conditioning

Conventional second-order conditioning (e.g., Pavlov, 1927; Rizley & Rescorla, 1972) consists of first pairing CS1 with a US (CS1 → US) until CS1 becomes a conditioned excitor and then pairing CS2 with CS1 (CS2 → CS1) until CS2 elicits a conditioned response. Although, in our view, no contemporary model adequately explains second-order conditioning, the Rescorla–Wagner model does make specific predictions concerning what should happen as a result of exposure to a second-order conditioning procedure. Because the CS2 → CS1 pairings are ordinarily not reinforced, the Rescorla–Wagner model predicts that CS2 should become a conditioned inhibitor rather than a conditioned excitor (see Failure 4 for a symmetrical prediction). This prediction arises from the subject, during the CS2 → CS1 pairings, expecting the US on the basis of the presence of CS1 and then not receiving the US. According to the model, the parenthetical term in Equation 1, $\lambda - V_{total}$, becomes negative on the CS2 → CS1 trials because λ is zero (as a result of the lack of a US) and V_{total} is positive (as a result of prior conditioning of CS1). Although conditioned inhibition to CS2 is often seen when the CS1 → US and CS2 → CS1 trials are numerous or interspersed, or both, the conventional second-order conditioning procedure with a few CS2 → CS1 trials following all of the CS1 → US trials usually results in CS2 acquiring excitatory value (e.g., Holland & Rescorla, 1975). Thus, excitatory second-order conditioning is contrary to the inhibition anticipated by the Rescorla–Wagner model.

Reconciliation of the Rescorla–Wagner model with second-order conditioning might be achieved by emphasizing within-compound associations or acquired increases in the λ value of CS1. That is, CS1 could be allowed to serve as a "US," with its own associated λ, during CS2 → CS1 trials. There is nothing in the Rescorla–Wagner model that would deny this possibility. However, such a modification would potentially undermine the ability of the model to predict conditioned inhibition in those cases in which it is observed. In fact, small numbers of CS2 → CS1 pairings appear to make CS2 an effective excitor, whereas large numbers of CS2 → CS1 pairings make CS2 an effective inhibitor (Rashotte, Marshall, & O'Connell, 1981). The Rescorla–Wagner model predicts that CS2 should become a conditioned inhibitor starting with the first CS2 → CS1 pairing, which is contrary to what actually is observed. Interestingly, Rashotte (1981) has presented a variant of the Rescorla–Wagner model that is able to predict both second-order conditioning (with few CS2–CS1 trials) and conditioned inhibition (with many CS2–CS1 trials). Rashotte's model assumes that the effective US for CS2 is a compound of the associative values of CS1 and the trace of CS2. This variant of the Rescorla–Wagner model works to explain both second-order conditioning and conditioned inhibition only if a "weighted-sum rule" is used. This rule gives more weight to inhibitory (negative) associations than to excitatory (positive) associations in determining the effective associative strength of two CSs that are presented in compound. Although Rashotte's model is successful in its intent, its weighted-sum rule is a distinct departure from the (unweighted) rule for summing associative strengths that is built into the Rescorla–Wagner model. Thus, the success of Rashotte's model in predicting both second-order conditioning and conditioned inhibition cannot be viewed as a success of the original Rescorla–Wagner model.

Failure 9: Cue-to-Consequence Effects

Garcia and Koelling (1966) first reported that, in rats, a gustatory cue is more readily associated with a US consisting of gastric upset than is an audiovisual compound cue. Conversely, they found that an audiovisual compound cue is more readily associated with a US consisting of footshock than is a gustatory cue. Such favored associations between specific CSs and USs are quite robust (e.g., Domjan & Wilson, 1972) and suggest that α and β in Equation 1 are not independent. Instead, it appears that each CS–US dyad has its own associability that is not re-

ducible to the product of an independent α and an independent β, as is assumed by the Rescorla–Wagner model. This appears to pose a major challenge to the model.

Failure 10: Dependence of Asymptotic Responding on CS Intensity and US Intensity

In the Rescorla–Wagner framework, asymptotic responding to a CS depends exclusively on λ, which is a function of US intensity alone (see Equation 1); α and β determine only the number of trials needed to reach an asymptotic level of conditioned responding. However, there are numerous examples (e.g., Kamin, 1965; Kessen, 1953; Scavio & Gormezano, 1974) of more intense CSs (which presumably have higher αs) supporting higher asymptotic levels of conditioned responding when US intensity is held constant (CS intensity is also known to influence the magnitude of unconditioned responding; Young, Cegaveske, & Thompson, 1976). This indicates that λ is not the sole determinant of asymptotic responding. The Rescorla–Wagner model might accommodate this observation by treating the more intense CS as being composed of several independent but coterminus weaker CSs, each of which asymptotes at λ (which therefore could summate), but this modification would appear to open a Pandora's box of ambiguities that would probably undermine the clear successes of the model.

Failure 11: Learned Irrelevance

Learned irrelevance refers to the retarded behavioral control acquired by a CS during CS → US pairings as a result of prior random exposure to the CS and the US (e.g., Baker & Mackintosh, 1976, 1977; Kremer, 1971; Overmier & Wielkiewicz, 1983). Part of the observed learned irrelevance deficit appears to be due to summation of the CS–preexposure effect (e.g., Lubow & Moore, 1959) and the US–preexposure effect (e.g., Randich & LoLordo, 1979). The Rescorla–Wagner model has no trouble explaining the component that arises from the US–preexposure effect (Success 15; but see Failure 14c). However, Baker and Mackintosh (1979); Bennett, Maldonado, and Mackintosh (in press); Dess and Overmier (1989); and Matzel, Schachtman, and Miller (1988) have all demonstrated that even after eliminating the contributions of the CS–preexposure effect and the US–preexposure effect, responding to the CS is still impaired. This impairment is generally presumed to arise from the subject "learning" that the CS is not informative concerning the US (e.g., Baker & Mackintosh, 1979). Such learning appears contrary to the Rescorla–Wagner model, which assumes that associative strength approaches a nonzero asymptote under the conditions of positive correlation (e.g., excitation) and negative correlation (e.g., inhibition), but not when the CS and a US are explicitly uncorrelated.

Failure 12: Potentiation

Potentiation is the opposite of overshadowing (see Success 8). When a less salient CS is reinforced in the presence of a more salient CS, conditioned responding to the less salient CS is sometimes enhanced (i.e., potentiated) rather than attenuated (i.e., overshadowed). This phenomenon was first reported by Clarke, Westbrook, and Irwin (1979) and Palmerino, Rusiniak, and Garcia (1980). It appears to be most readily obtained when gustation is the modality of the potentiating CS and olfaction is the modality of the potentiated CS. Nevertheless, potentiation has been obtained with other modalities (e.g., Best, Batson, Meachum, Brown, & Ringer, 1985; Mellon, Kraemer, & Spear, 1991). The Rescorla–Wagner model would appear to predict overshadowing whenever two novel CSs are compounded and paired with a US. Thus, any instance of potentiation might be viewed as inconsistent with the Rescorla–Wagner model.

Several explanations of behavioral potentiation have been proposed. Palmerino et al. (1980) hypothesized that the presence of the potentiating CS during training results in a stronger association between the potentiated CS and the US. Such potentiation of associative strength is in direct contradiction to the Rescorla–Wagner model's basic prediction of overshadowing. Alternatively, Rescorla and Durlach (1981) suggested that potentiation is a consequence of a within-compound association between the potentiated CS and the potentiating CS that is formed during the reinforced compound trials. Because the potentiating CS acquires associative strength through its direct association to the US, the within-compound association might result in the potentiated CS becoming an effective second-order cue. In this view, excitatory control by the second-order (potentiated) CS might summate with excitatory control by the first-order (potentiating) CS to produce the potentiation effect. Supporting this view, Durlach and Rescorla (1980) reported that potentiation can be attenuated by posttraining extinction of the potentiating CS, presumably because that extinction attenuated the excitatory summative contribution of the association between the potentiating CS and US. With some modifications, the Rescorla–Wagner model might be tailored to accommodate within-compound associations (see Failures 8 and 9 for details and problems). However, Lett (1984; see also Droungas & LoLordo, 1991) has shown that posttraining extinction of the potentiating stimulus does not consistently attenuate potentiation. Thus, not all cases of potentiation appear explicable in terms of within-compound associations. The presence of one CS in a compound of two CSs does not always diminish what is learned about the other CS. However, more data are needed concerning the generality of potentiation that cannot be explained by within-compound associations (for related discussions, see Kurcharski & Spear, 1985; Rescorla, 1981).

Failure 13: One-Trial Overshadowing

According to the Rescorla–Wagner model, overshadowing occurs because the associative strength of the overshadowing CS that is acquired on early compound CS → US trials degrades the effectiveness of the US on later trials (see Success 8). Because the overshadowing CS presumably has no associative strength until the end of the first training trial, the model predicts (as do most other models) that no overshadowing will occur on the first training trial. However, several researchers have reported that overshadowing can be obtained as a result of a single training trial (James & Wagner, 1980; Mackintosh & Reese, 1979). The observation of one-trial overshadowing encourages explanations (e.g., conservation of attention) that

might also explain multitrial overshadowing without recourse to the processes evoked by the Rescorla–Wagner model. That is, given the need for an explanation of one-trial overshadowing that is outside the scope of the Rescorla–Wagner model, why should that explanation not suffice to explain all overshadowing? Among the several likely candidate explanations of both one-trial and multitrial overshadowing is generalization decrement (Hull, 1943; Pearce, 1987). This account typically emphasizes the change in stimulus conditions between the compound CS → US training trial(s) and the test trial during which the putatively overshadowed CS is presented alone.

Failure 14: Recovery From Overshadowing, Blocking, and the US–Preexposure Effect

The Rescorla–Wagner model attributes both overshadowing and blocking of a CS to a failure to acquire an appreciable association between the overshadowed or blocked CS and the US. Thus, overshadowing and blocking should be irreversible without further training. However, a number of different treatments (other than further training) have been found to eliminate the deficits of overshadowing and blocking. Thus, it appears that at least some overshadowing and blocking behavioral deficits arise from the associations in question being latent rather than weak. In addition, the retarded conditioned responding to a CS observed when unsignaled USs are presented in the training context before CS → US pairings (i.e., the US–preexposure effect) is explained in the Rescorla–Wagner framework as blocking of the CS by the context that was presumably conditioned during the prior unsignaled US presentations. As a type of blocking, the US–preexposure effect also should be irreversible. However, there is evidence to the contrary. Here we describe three different treatments that have been found to reverse overshadowing, blocking, and the US–preexposure effect. Collectively, these observations converge on the conclusion that overshadowing, blocking, and the US–preexposure effect are not, in fact, acquisition failures. This conclusion constitutes one of the more serious challenges to the Rescorla–Wagner model.

Failure 14a: Spontaneous recovery from overshadowing. Kraemer, Lariviere, and Spear (1988), using a one-trial training procedure, and J. S. Miller, McKinzie, Kraebel, and Spear (1993), using a multitrial training procedure, found that when the retention interval between the termination of overshadowing treatment and testing is lengthened from days to weeks, overshadowing response deficits are attenuated. Spontaneous recovery from overshadowing is particularly difficult to attribute to covert learning between training and testing because the critical restorative "treatment" is the passage of time in the home cage in the absence of any specific cue from the training situation. Thus, spontaneous recovery from overshadowing poses some difficulty for the Rescorla–Wagner model explanation of overshadowing. In contrast, a generalization decrement view of overshadowing (see Failure 13) is quite able to explain this phenomenon because generalization gradients are well known to broaden over time (e.g., Thomas & Lopez, 1962).

Failure 14b: Reminder-induced recovery from overshadowing and blocking. A second treatment that has been successful at revealing overshadowed associations is the presentation of "reminder" cues between training and testing. Reminder cues refer to stimuli from the training situation that are constrained to preclude relevant new learning during the reminder treatment.[3] Kasprow, Cacheiro, Balaz, and Miller (1982) reported that a reminder treatment consisting of brief exposure to the overshadowed CS selectively enhanced responding to that CS. Notably, they administered their reminder treatment outside of the context that was used for training and testing. This presumably minimized the contribution of associations between the reminder context and the CS to the recovery effect that was observed (see Failure 1 for related remarks). The reminder-induced recovery of conditioned responding has also been obtained after blocking. Balaz, Gutsin, Cacheiro, and Miller (1982; see also Schachtman, Gee, Kasprow, & Miller, 1983) successfully used three different types of reminder treatments to attenuate the blocking deficit. These included exposure to either the blocked CS or the US (outside of the context used for training and testing) and exposure to the training context alone. In each case, the enhanced conditioned responding was specific to the blocked stimulus. Like spontaneous recovery from overshadowing, reminder-induced recovery from overshadowing and blocking is contrary to the Rescorla–Wagner view that these response deficits represent failures in associative acquisition.

Failure 14c: Posttraining extinction-induced recovery from overshadowing and the US–preexposure effect. A third means of revealing latent associations to an overshadowed CS consists of posttraining extinction of the overshadowing CS. Such restoration of responding to an overshadowed CS has been demonstrated both when overshadowing and overshadowed CSs were presented simultaneously during reinforced training (Kaufman & Bolles, 1981; Matzel, Schachtman, & Miller, 1985) and when the CSs were presented serially during reinforced training (Matzel, Shuster, & Miller, 1987). In each instance, recovery of responding to the overshadowed CS was specific to extinction of the overshadowing CS as opposed to extinction of another excitatory CS. A potential explanation of this phenomenon was provided by Miller and Schachtman (1985; see also Miller & Matzel, 1988). That recovery from overshadowing by any means short of further training occurs at all is contrary to the explanation of overshadowing provided by the Rescorla–Wagner model. In addition to overshadowing, the US–preexposure effect has been reversed by posttraining extinction of the training context (Barnet, Grahame, & Miller, 1993; Matzel, Brown, & Miller, 1987). Stimulus specificity of this restorative effect has similarly been demonstrated. As in the case of blocking by previously trained discrete cues (see Failure 14b), recovery from the US–preexposure effect achieved through any means other than further reinforced training with the target CS is inconsistent with the acquisition-based, blocking explanation of this deficit provided by the Rescorla–Wagner model.

A recent variant of the Rescorla–Wagner model proposed by Van Hamme and Wasserman (in press; see also Markman, 1989) cleverly manages to explain in terms of acquisition defi-

[3] Throughout these reminder studies, the duration of exposure to the reminder cues proved critical. Overexposure to the US resulted in pseudoconditioning of the control subjects (i.e., nonassociative responding), and overexposure to the CS resulted in less apparent recovery of the blocked association, presumably as a result of extinction of the CS–US association.

cits both overshadowing and the US–preexposure effect, as well as extinction-induced recovery from these two behavioral deficits. Van Hamme and Wasserman's model posits processing of a CS representation even on trials in which it is absent, provided the CS has been presented at least once on previous trials. This is achieved by postulating two α values, one α for trials on which the CS is present and a second, smaller α for trials on which the CS is absent. This arrangement creates symmetry between α and β because the original Rescorla–Wagner model (as well as Van Hamme and Wasserman's version) uses two β values for a US, one for trials on which the US is present and the other for trials on which the US is absent. This reformulation of the Rescorla–Wagner model is one of the exciting new directions in contemporary research in Pavlovian conditioning, but it still encounters problems with many of the other failures of the original Rescorla–Wagner model reviewed here.

Failure 15: Modulation of Conditioned Inhibition Through Posttraining Extinction of Other Cues That Were Present During Training

In Failure 14c, we described how posttraining extinction of other cues that were present during training of a target CS can reveal otherwise latent excitatory associations to that CS. That is, in the cases of overshadowing and the US–preexposure effect, posttraining extinction of the overshadowing CS and the background cues of the training context, respectively, increased excitatory responding to the target CS. The same manipulation, given appropriate parameters, has been found to decrease the inhibitory potential of a CS, as indexed by both summation and retardation tests for inhibition. After using Pavlovian inhibition training to establish CS X as a conditioned inhibitor (A → US/AX−), Hallam, Matzel, Sloat, and Miller (1990; see also Miller, Esposito, & Grahame, 1992) demonstrated this decrease in inhibitory potential as a consequence of posttraining extinction of Stimulus A. Analogously, after using negative contingency training to make CS X a conditioned inhibitor (US/X−), Kasprow, Schachtman, and Miller (1987; see also Schachtman, Brown, Gordon, Catterson, & Miller, 1987) observed a decrease in inhibitory potential as a result of posttraining extinction of the training context. The Rescorla–Wagner model posits that although other cues present during inhibitory training can modulate the formation of inhibitory associations to a CS, once these inhibitory associations are formed, the associative status of these other cues after training plays no further role in determining the response potential of the target CS. Seemingly, this assumption of the Rescorla–Wagner model is in error and converges with Failures 4, 5, and 6 in leading us to conclude that a new conceptualization of conditioned inhibition is needed.

Failure 16: Superconditioning

As we described among the successes of the Rescorla–Wagner model, superconditioning refers to enhanced excitatory conditioned responding to a CS (X) as a result of its being trained in the presence of a previously established conditioned inhibitor (A) for the same US (see Success 14 for a description of how the model accounts for this phenomenon). In the Rescorla–Wagner framework, superconditioning is assumed to reflect the target CS acquiring more associative strength in the presence than in the absence of Conditioned Inhibitor A. However, in the early published demonstrations of superconditioning (e.g., Rescorla, 1971; Wagner, 1971), superconditioning was ordinarily demonstrated by comparing responding to the target CS in the superconditioning group (which received AX → US trials after A had been established as an inhibitor) with responding in a control group for which both A and X were novel before reinforced compound training (i.e., AX → US). The superior conditioned responding to X by the superconditioning group relative to this control group was then assumed to reflect better learning about X when A was inhibitory than when A was novel.

An alternative interpretation of the observed difference in responding to the target CS is that, rather than enhanced performance in the superconditioning group, there was impaired performance in the control group. Navarro, Hallam, Matzel, and Miller (1989) compared these alternatives by performing a superconditioning study that included a control group for which the nontarget inhibitory stimulus (A) was omitted during training of the target CS (i.e., X → US). This was intended to serve as a baseline to assess impairment and facilitation in both the superconditioning group and the conventional control group. The superconditioning group did not differ in conditioned responding from this new control group. That is, no enhanced responding was observed in the putative superconditioning group, as would be predicted by the Rescorla–Wagner model. Rather, the conventional control group, for which Stimulus A was neutral at the beginning of the compound AX → US trials, responded less to Stimulus X than did either the superconditioning group or the new control group. This indicates that apparent superconditioning is not different from normal conditioning and appears "supernormal" only when inappropriately compared with the conventional control group.

One theoretical interpretation of why apparent superconditioning occurs is that there are differences between groups in A's potential to overshadow X on the basis of the associative history of A. Without any prior training, Stimulus A may overshadow Stimulus X (thus decreasing conditioned responding in the conventional control group). However, inhibitory training with Stimulus A may decrease A's potential to overshadow Stimulus X (possibly as a result of similar mechanisms that control the CS–preexposure effect; see Failure 7). There are data supporting this view. Navarro et al. (1989) obtained enhanced responding to Stimulus X (i.e., a superconditioning-like effect) when they gave simple preexposure to Stimulus A before the AX → US trials (see also Carr, 1974). This treatment reduced Stimulus A's potential to overshadow Stimulus X but could not be considered superconditioning because Stimulus A was not inhibitory. Thus, although the phenomenon of superconditioning appears to be reliable, the explanation for it provided by the Rescorla–Wagner model is probably incorrect.

Failure 17: Blocking With Two Blocking Stimuli

If Stimuli A and B are made excitatory through independent pairings with the US in Phase 1 of a blocking procedure, and then in Phase 2 a compound stimulus ABX is paired with the US, the Rescorla–Wagner model predicts that Stimulus X not only should acquire no excitatory strength, as in ordinary

blocking of excitation but should become a conditioned inhibitor (i.e., superblocking). The prediction that Stimulus X will become a conditioned inhibitor is counterintuitive because Stimulus X would have a consistent history of reinforcement. Although Kremer (1978, Experiment 3) reported observing inhibition with this procedure, he used only a retardation test to assess inhibition. Such a retardation effect might be due to mechanisms other than conditioned inhibition. Subsequently, Schachtman, Kasprow, Chee, and Miller (1985) used both retardation and summation tests to assess inhibition, as well as a conventional test for excitation. They found robust blocking of excitation but no evidence of the blocked stimulus having become a conditioned inhibitor. Thus, their observations (which no one to date has attempted to replicate) failed to confirm the inhibitory prediction of the Rescorla–Wagner model. This failure of the model could be addressed by relaxing the assumption that associative strengths of simultaneously presented cues are linearly summed. Instead, it could be assumed that the effective associative sum of simultaneous CSs is greater than either cue alone but less than their linear sum. However, such a modification would weaken the model's ability to explain numerous phenomena, such as overshadowing.

Failure 18: Elimination of Overshadowing and Blocking Deficits With Large Numbers of Compound Trials

The Rescorla–Wagner model assumes that overshadowing and blocking to a target CS occurs because the associative value of the overshadowing and blocking CSs degrades the ability of the target CS to acquire associative strength (see Successes 8 and 10, respectively). If the compound CS trials of overshadowing and blocking procedures continue to be reinforced, V for the overshadowing or blocking CS should retain (or increase) its associative strength. Therefore, overshadowing and blocking deficits should be sustained over additional reinforced compound trials. Consequently, once overshadowing or blocking has occurred, the model predicts that further pairings of the compound CS and the US should not reduce the degree of overshadowing or blocking. Contrary to this prediction, Bellingham and Gillette (1981) have found that overshadowing is reduced when large numbers of reinforced trials with the compound CS are administered. That is, with a large number of training trials, the overshadowing subjects came to respond to the overshadowed CS with the same frequency and vigor as did the overshadowing control subjects. Azorlosa and Cicala (1988) have reported a similar loss of blocking with large numbers of reinforced compound CS trials. Thus, contrary to a central assumption of the Rescorla–Wagner model, in certain situations λ does not appear to serve as an upper limit to the total amount of associative strength that a US can support across all CSs present on a particular trial.

Failure 19: Unblocking by Omission of a Second Expected US

In the traditional blocking procedure, the US is presented only once during each Phase 1 and Phase 2 trial (see Success 10, for the Rescorla–Wagner model's explanation of blocking). If the US is administered twice on each Phase 1 trial and each Phase 2 trial, the prediction of blocking of X is unchanged. Consider now the case in which the US is presented twice during each Phase 1 trial (A → US → US) but only once during each Phase 2 trial (AX → US). The Rescorla–Wagner model assumes that omission of the second US on the Phase 2 trials should result in X becoming a conditioned inhibitor. This is represented by the parenthetical term of Equation 1, $\lambda - V_{total}$, becoming negative in Phase 2 because V_{total} will exceed λ. In the framework of the model, negative values of V correspond to conditioned inhibition. To the extent that X acquires inhibition, it can also be viewed as acquiring less excitation than if both USs had been presented in Phase 2. Therefore, the model predicts more blocking (less excitation to X) when the second US is omitted on the AX compound trials than when the second US is present on the AX compound trials. Studies using these procedures have been conducted by Dickinson, Hall, and Mackintosh (1976); Dickinson and Mackintosh (1979); and Kremer (1979). These authors all found that Stimulus X actually supports less blocking (i.e., more excitation) when the second US is omitted than when only one US is presented on each trial of Phase 1 and Phase 2. Such a reduction in blocking is contrary to the results anticipated by the Rescorla–Wagner model and invites explanation in terms of changes in CS associability (e.g., α) rather than changes in the parenthetical term $\lambda - V_{total}$ (e.g., Pearce & Hall, 1980).

The observation of unblocking by omission of a second expected US is particularly interesting because it contrasts assumptions of the Rescorla–Wagner model with those of the model's immediate predecessor, Kamin's (1969) view of surprise. Kamin's position was that surprise, independent of its being induced by the unexpected presence or absence of a US, is necessary for excitatory learning to take place. In this view, like that of Rescorla–Wagner, omission of a second US on the Phase 2 compound trial is surprising because the presence of Stimulus A predicts two USs, but only one US is presented. However, according to the Kamin view of surprise, strong excitatory learning to X is predicted. In contrast, the parenthetical term that embodies surprise in the Rescorla–Wagner model will have a negative value, which leads to the prediction that negative excitation (i.e., inhibition) will be acquired to Stimulus X. The observed excitatory responding in this situation is supportive of Kamin's position rather than that of the Rescorla–Wagner model. Although damaging for the Rescorla–Wagner model, the ongoing discussion should not be taken to imply that Kamin's formulation is superior to the Rescorla–Wagner model (see Success 12 for an instance of the Rescorla–Wagner model succeeding when Kamin's position fails).

Failure 20: Retardation of Acquisition by a Previously Blocked Cue

The Rescorla–Wagner model explains blocking in terms of the blocked CS having weak (or possibly no) associative strength. Processing of the blocked CS is presumably unaltered by the blocking procedure because α is constant. Thus, after complete blocking of a CS, the Rescorla–Wagner model assumes that further presentations of the CS should result in the CS being processed just as if prior blocking of that CS had never occurred. Therefore, simply pairing the blocked CS with the

US after blocking treatment should result in the acquisition of excitatory behavioral control equivalent to that which would be seen if the previously blocked CS was novel. In contrast with this prediction, Mackintosh and Turner (1971; see also Mackintosh, 1978) found that a previously blocked cue was less effective in acquiring associative strength than the same cue that had not previously been blocked (i.e., the previously blocked CS was retarded). This observation suggests that blocking results, at least in part, from variations in α rather than entirely from variations in the value of the parenthetical term $\lambda - V_{total}$. Similar mechanisms may be suggested to operate in a different predictive failure of the model, namely the CS–preexposure effect (see Failure 7).

Failure 21: Overshadowing of a Pretrained CS

The Rescorla–Wagner model predicts that a more salient stimulus (e.g., A) will overshadow a less salient stimulus (X) if both stimuli are novel at the beginning of reinforced compound training (AX → US). However, the model makes very different predictions if the less salient X stimulus is pretrained (X → US) before reinforced training with the AX compound. Specifically, the model anticipates that acquisition to the novel A stimulus will be blocked, despite its being more salient. This occurs as in standard blocking (Success 10). In the present case, prior training with X enhances V_{total}. The result is to decrease the value of $\lambda - V_{total}$ for Stimulus A during AX → US training more than if X had not been previously trained. In addition, the model explicitly predicts that the pretrained less salient X will not lose behavioral control during compound conditioning. In contradiction of this prediction, Hall, Mackintosh, Goodall, and Dal Martello (1977) have reported that, in this situation, the more salient stimulus acquires behavioral control and the less salient pretrained stimulus actually loses behavioral control. Their study was well controlled, but, as with any important finding, a replication would be welcome.

Failure 22: Associative Processing During a Training Trial

The Rescorla–Wagner model assumes that only at the end of a trial are the new values of V determined; these values then influence behavior and information processing the next time that the same CSs are presented. This constraint is produced by the parenthetical term in Equation 1, $\lambda - V_{total}$, which cannot be calculated until the outcome of the trial is known (i.e., US or no-US presentation). In contrast to this simplifying end-of-trial view for associative determination, various researchers have suggested that the associative value of a CS may change during as opposed to only after a training trial (i.e., real-time processing of information; e.g., Ayres, Albert, & Bombace, 1987; Kehoe & Napier, 1991). One particularly compelling instance of this is the apparent change in attention to a stimulus during a single exposure demonstrated by DeVietti, Bauste, Nutt, Barrett, Daly, and Petree (1987). These authors found that the effect of CS preexposure in rats does not interfere with the CS's acquisition of behavioral control throughout its entire presentation but only during the later part of each reinforced CS presentation. Real-time information processing has been incorporated in several recent models of information processing (e.g., Kehoe, Schreurs, & Graham, 1987; Wagner, 1981). The Rescorla–Wagner model might be reworked into a real-time model by dividing the CS into a series of time bins (perhaps with α decreasing over successive bins; see Cluster 3), with each bin being treated as a separate trial (e.g., Ayres et al., 1987; Sutton & Barto, 1981). Alternatively, one might contend that associative revaluation during a trial is beyond the scope of the Rescorla–Wagner model and, consequently, that this failure is an error of omission rather than commission. It is nonetheless clear that the original statement of the model provides no mechanism for modifying the associative strength of the CS at various points within the trial; rather, such mechanisms are engaged at the end of the trial. However, we would not argue with anyone who chose to regard information processing before the end of a training trial as being beyond the scope of the Rescorla–Wagner model.

Failure 23: Recovery of Responding to a CS After Attenuation of Conditioned Responding by Random Exposure to a Previously Paired CS and US

The Rescorla–Wagner model assumes that associative value previously established by CS → US pairings can later be degraded by random exposure to the CS and US. Such associative degradation leads to the prediction of attenuated responding to the CS. These kinds of decrements in conditioned responding are commonly observed (e.g., Gamzu & Williams, 1971). According to the Rescorla–Wagner model, the loss of conditioned responding reflects a loss of the CS's associative strength that could not be reversed without further CS → US pairings. However, Lindblom and Jenkins (1981; see also Durlach, 1986) have reported that if the CS is presented alone after attenuation of conditioned responding by random (or explicitly unpaired) treatment, there is a temporary restoration of responding to the CS. This restoration appears contrary to the Rescorla–Wagner model. However, the effect might be reconciled with the model by regarding internal aftereffects of the US as additional cues that could acquire inhibitory value during random exposure to the CS and US. The removal of these cues during the subsequent CS-alone phase might reveal residual excitatory strength of the CS that was masked during the random treatment phase by the inhibitory aftereffects of the US. This possibility has not been directly tested, but Durlach's (1986) data raise doubts as to whether internal US aftereffect cues become inhibitory. Moreover, the only robust demonstrations of the Lindblom and Jenkins effect to date have been exclusively with autoshaping in pigeons. The generality of the phenomenon has yet to be determined.

Underlying Bases for Failures of the Rescorla–Wagner Model

A failure of a prediction can be seen in observed behavior, but the reason for the failure is only a hypothesis that depends on how the observer chooses to explain the behavior. Hence, there is room for argument as to which assumptions and rela-

tionships of the Rescorla–Wagner model are responsible for each of the failed predictions of the model. However, if we appeal to prevailing wisdom (however transient that may be), we can attempt to categorize the different sources of the preceding failures of the model. Most of the Rescorla–Wagner model's errors of commission appear to fall into one (and occasionally more) of five clusters, each characterized by a different erroneous underlying assumption (as described subsequently). The first two clusters of failures concern the unique assumptions of the model, that is, assumptions that differentiate the Rescorla–Wagner model from all earlier models. The next three clusters of failures arise from erroneous assumptions that have long been used to simplify theories of learning (e.g., Bush & Mosteller, 1951), thereby making such theories more tractable. There are a few problems that do not appear to fit easily into any of these five categories. We have arbitrarily placed them in a sixth cluster.

Cluster 1: Limit to the Total Amount of Associative Strength That a US Can Distribute on a Given Trial

The first cluster of problems for the Rescorla–Wagner model arises from the assumption of the model that, on a given trial, a US can support only a fixed amount of associative strength that is distributed across all stimuli present on that trial. In other words, the summed associative strength of all stimuli present on a specific trial will gravitate toward the λ of the US present on that trial. Potentiation (Failure 12), blocking with two excitatory stimuli not making the blocked cue into a conditioned inhibitor (Failure 17), elimination of overshadowing and blocking deficits with large numbers of trials (Failure 18), and unblocking by omission of a second expected US (Failure 19) contradict this assumption of the model. (So too do phenomena in which recovery from a deficit in acquired behavior is seen [i.e., Failures 1, 2, 14, 15, and 23]; we discuss these phenomena later as part of Cluster 5.) However, the common alternative (e.g., Bush & Mosteller, 1951; Couvillion & Bitterman, 1987) that the associative strength of each CS present on a specific trial will independently gravitate toward the λ of the US present on that trial appears to be equally or more incorrect. Reality appears to be somewhere between these two extremes. Mackintosh (1975) has provided one example of a model in which this issue is addressed. His model accounts for interactions in associative strength between simultaneously presented stimuli, but, unlike the Rescorla–Wagner model, it does not place a rigid limit on the total associative strength that can be acquired by all CSs that are simultaneously presented. However, in other arenas, Mackintosh's model is not as successful as the Rescorla–Wagner model. For example, in contrast with the Rescorla–Wagner model, it does not predict one-trial blocking (i.e., blocking with a single compound trial), which has been observed (see Success 10). Although Mackintosh (1975), Azorlosa and Cicala (1988), and Clarke et al. (1979), among others, have previously discussed the error of the Rescorla–Wagner model in assuming λ to be a rigid ceiling for simultaneously presented CSs, the problem with this assumption of the model has not been widely acknowledged. Among those who have acknowledged this problem, there is little consensus on proper redress. One solution that has been suggested is that cue competition does not occur at acquisition but at the time of testing (e.g., Miller & Matzel, 1988). Other researchers have proposed that in many situations cue competition simply does not occur and therefore does not require explanation (e.g., Couvillion & Bitterman, 1987). In summary, one can see that although the specific proposal of the Rescorla–Wagner model for stimulus interaction is not correct, it led researchers in the correct direction by providing the first quantitative theoretical framework that accounted for the interaction observed between simultaneously presented CSs in acquiring behavioral control.

Cluster 2: Excitation and Inhibition as Symmetrical Opposites

The second cluster of problems for the Rescorla–Wagner model stems from the assumption that conditioned excitation and conditioned inhibition are symmetrically represented by opposite sides of a single continuous variable of associative strength. With the single exception of V taking positive values for conditioned excitation and negative values for conditioned inhibition (see Equation 1), excitation and inhibition are treated identically in the model and should obey the same rules for changes in associative status. The observations that an inhibitor does not lose behavioral control as a result of presentations of the inhibitor alone (extinction; Failure 3), that a CS can simultaneously have both excitatory and inhibitory potential (Failure 5), that an inhibitor's action on a summation test depends on the excitatory status of the transfer excitor (Failure 6), and that a novel cue presented simultaneously with an inhibitor in the absence of an excitor does not become an excitor (Failure 4) all refute this assumption of the Rescorla–Wagner model. The lack of symmetry between conditioned excitation and conditioned inhibition is widely recognized. Indeed, Wagner and Rescorla (1972) themselves expressed some doubts about their assertion. Rescorla (e.g., 1979) has proposed specific alternative ways of conceptualizing conditioned inhibition, as have Miller and Matzel (1988) and Wagner (1981). However, no single view has emerged as the preferred replacement for the Rescorla–Wagner position on this issue.

Cluster 3: Fixed Associability (i.e., α and β) of a Stimulus

The third cluster of problems arises from the model's assumption that α for a specific CS and β for a specific US are constants (i.e., parameters independent of experience). That is, neither past experience nor the presence of other stimuli is supposed to alter the associability of a stimulus. Several different phenomena argue against this view. These include the CS–preexposure effect (Failure 7), cue-to-consequence effects (Failure 9), one-trial overshadowing (Failure 13), superconditioning (Failure 16), retardation of a previously blocked cue (Failure 20), and, more questionably, learned irrelevance (Failure 11). Apparently, prior experience with a target CS as well as the presence of other CSs and the specific US present on a current training trial influence the effective α of the target CS on that trial. This difficulty with the Rescorla–Wagner model is well known and was noted in 1972 by Rescorla and Wagner. They suggested that insufficient data were available to specify the rules for vari-

ation of α for a given stimulus and argued for the utility of their simplifying assumption that α is a constant.

Frey and Sears (1978) and Wagner (1978) proposed models that preserve much of the Rescorla–Wagner model but use a dynamic attention variable. Those modifications of the model have not been incorporated by many researchers, perhaps because the resultant models are less tractable than the original. Indeed, the prevailing version of the Rescorla–Wagner model today is the original 1972 version, and in that version α is a constant (Rescorla and Wagner's expressed doubts notwithstanding). Although there is wide agreement that α and β are both variables that depend on prior experience, there is disagreement on exactly how they vary. For example, Mackintosh (1975) and Pearce and Hall (1980) presented two diametrically opposed proposals. One might view the failure of the Rescorla–Wagner model to allow for changes in α as a failure of omission that is beyond the scope of the model. Alternatively, it is not unreasonable to suggest that allowing α to vary simply means that some aspects of results explained by the Rescorla–Wagner model are partially, but not exclusively, due to a variable α. The bottom line, however, is that if α is allowed to vary, many of the successes of the model could be explained by changes in α as a function of experience, without recourse to the unique features of the model (e.g., Mackintosh, 1975; Pearce & Hall, 1980).

Cluster 4: Path Independence

The fourth cluster of problems stems from the assumption of the Rescorla–Wagner model that subjects possess only the current associative value of a cue and retain no knowledge of its prior associative history. In other words, past associative status of a cue (and the particular history of various combinations of reinforced and nonreinforced trials), per se, is assumed to influence neither behavior nor future changes in associative status. Contrary to this assumption, recovery from extinction without retraining (Failure 1), facilitated and retarded reacquisition after extinction (Failure 2), and recovery of responding after response elimination through random exposure to the CS and US (Failure 23; each of which also fits into Cluster 5) all indicate that changes in associative status are path dependent. Many of these failures of path independence (Failures 2 and 23) involve both reinforced and nonreinforced trials. If, contrary to the Rescorla–Wagner model, nonreinforcement after reinforcement results in inhibition without the loss of excitation, then these failures of path independence may reduce to conditioned excitation and inhibition not being mutually exclusive (see Cluster 2; for a detailed discussion of path dependence, see Miller & Matzel, 1987). This difficulty with the Rescorla–Wagner model is also evident in most other models of learning, including contemporary models as well as those that antedate the Rescorla–Wagner model. However, using a simple connectionist model, Kehoe (1988) described one way in which path dependence can be built into models of associative learning. Notably, his model uses the assumptions of the Rescorla–Wagner model at the level of individual connections between adaptive units in a network. Thus, the problem of path dependency can be addressed without totally rejecting the Rescorla–Wagner model. However, major revisions of its application appear to be necessary.

Cluster 5: The Learning–Performance Distinction

The fifth cluster of problems arises from the Rescorla–Wagner model's assumption that differences in behavior reflect differences in associative strength (perhaps after correcting associative strength for a behavioral threshold, which was mentioned by Rescorla and Wagner [1972] but not centrally installed in the model). The model posits only a monotonically positive relationship between associative value and a relevant response. Consequently, its behavioral predictions are of ordinal ranking. However, many ordinal differences in conditioned behavior do not seem to be mediated by ordinal differences in associative strength. For example, low levels of responding seen in the critical experimental groups of blocking, overshadowing, and US–preexposure experiments (see Failure 14) do not seem to be mediated by low associative strength. That is, they do not represent acquisition failures, and therefore the Rescorla–Wagner model's explanation of these effects appears to be flawed.

Rescorla and Wagner noted, in 1972, that behavior probably depends on a large number of performance variables. However, these variables are outside the domain of the model. When deficits in behavior that the Rescorla–Wagner model attributes to impaired acquisition of associative strength can be reversed, without further CS–US training, the simplest conclusion is that the association was present but masked when the behavioral deficit was observed, which is contrary to the spirit of the model. One potential defense of the Rescorla–Wagner views on performance is to state the model remains essentially silent on the issue because it is a theory of learning and, therefore, has no stand on the issue of performance. However, the Rescorla–Wagner model is a theory of behavior, and it seeks to directly explain behavioral phenomena through the mechanism of acquisition of knowledge about the US. Moreover, contemporary learning theorists use the model this way; for example, Durlach (1989) has used the model to explain failures of contiguity through a learning mechanism and directly contrasted the Rescorla–Wagner view with theories of performance, such as those of Gibbon and Balsam (1981) and Miller and Schachtman (1985).

Alternatively, the Rescorla–Wagner model might be defended with the view that the treatment manipulation (e.g., the operation of overshadowing) really did reduce associative strength and that the subsequent recovery of performance was due to compensation by a performance variable. However, there are two problems with this view, both of which are troubling but neither of which demands total rejection of the model. First, invoking two processes (i.e., attenuated acquisition and subsequent enhancement by performance variables) is less parsimonious than attributing both the initial behavioral deficit and the subsequently observed recovery of performance to a single variable (such as retrieval). Second, for many of the recovery manipulations, the recovery is evident days after the recovery manipulation (e.g., see Failures 14a–c and 15). The recovery manipulation might temporarily enhance a performance variable such as hunger or anxiety, but changes in performance variables

are presumably short lived. Yet, recovery treatments often have effects that are evident days after the treatment. If performance variables perturbed by the recovery manipulation have returned to their initial states, they are implausible explanations of the enhanced performance that is still seen. However, if the various recovery manipulations are assumed to enhance retrieval (as in Failures 14a and 14b), the initial behavioral deficit could be attributed to impaired retrieval more readily than impaired acquisition. Demonstrations that large discrepancies can exist between what has been learned and performance can be seen in recovery from extinction without retraining (Failure 1), facilitated and retarded reacquisition after extinction (Failure 2), and recovery of responding after response elimination through random exposure to the CS and US (Failure 23; each of which also fits into Cluster 4), as well as recovery from overshadowing, blocking, and the US–preexposure effect (Failure 14) and modulation of conditioned inhibition through posttraining extinction of other cues that were present during training (Failure 15).

In addition, one might also view data indicative of associative path dependence (see Cluster 4) as instances of prior associative states being encoded within the subject but having a behaviorally latent influence until further training occurs. In this framework, instances of path dependence are also demonstrations of the learning–performance distinction. Apparently, no simple monotonic relationship can consistently map knowledge into behavior. The learning–performance distinction has been addressed by many researchers working in the associative tradition (e.g., Miller et al., 1986; Miller & Matzel, 1988; Spear, 1971; Tolman & Honzik, 1930). However, it is in the domains of motivational and cognitive psychology that researchers appear to be most attuned to this problem.

Cluster 6: Remaining Failures of the Model

Not all of the problematic phenomena just listed represent apparent errors in one or more of the Rescorla–Wagner model's basic assumptions. The failures of the model in these cases appear to be erroneous outcomes of the full model. Phenomena in this class include second-order conditioning (Failure 8), asymptotic responding depending on CS intensity (Failure 10), associative processing within a trial (Failure 22), and overshadowing of a pretrained CS (Failure 21).

Underlying Clusters 1–5, which collectively encompass most of the failings of the Rescorla–Wagner model, are all of the major assumptions that went into the making of the model (see introduction). Thus, there does not appear to be any one assumption that could be revised or deleted from the model that might render it valid. Rather, all of the assumptions of the model appear suspect. Any serious attempt to align the model with the existing data would require a major revision of the model, perhaps at the expense of its simplicity and ease of application.

Notably, the assumptions underlying Clusters 1 and 2 are the features of the Rescorla–Wagner model that were unique when the model was first proposed in 1972. Therefore, one might take the position that the model should be judged strictly on how well these two assumptions fare. However, it can be seen that these two assumptions fare no better than the other, less unique assumptions of the model. Thus, such a narrowed criterion does not require a change in our overall assessment of the model.

Conclusions

Any given researcher will probably disagree with a few of the earlier-mentioned failings of the Rescorla–Wagner (1972) model; however, the disputed failings would differ from researcher to researcher. We can safely predict that no one would dispute all of the failings. Because acceptance of any one of the listed failures of the model would indicate that the model is flawed, the present list forces us to conclude that the Rescorla–Wagner model does not provide an accurate statement of how associative information is processed.

Today, most contemporary theories of acquired behavior are predicated on observations initially made to assess the Rescorla–Wagner model (e.g., Gibbon & Balsam, 1981; Miller & Matzel, 1988; Pearce, 1987; Pearce & Hall, 1980; Sutton & Barto, 1981, 1990; Van Hamme & Wasserman, in press; Wagner, 1981; Wagner & Brandon, 1989). Moreover, many of these models retain some of the features of the Rescorla–Wagner model.

Uniformly, all of these newer models are highly complex or have their own lists of failures at least as extensive as that of the Rescorla–Wagner model. Yet, each of these contemporary models makes strides in addressing one or more of the erroneous assumptions of the Rescorla–Wagner model. For example, Ayres et al. (1987), Frey and Sears (1978), and Pearce and Hall (1980) have addressed the problem of attention to a stimulus changing during conditioning; Ayres et al. (1987), Sutton and Barto (1981, 1990), and Wagner (1981) have focused on the dynamics of associative information processing during the occurrence of a single trial (i.e., within-trial processing); and Bouton (1993) and Miller and Matzel (1988) have addressed the learning–performance distinction. Although there is not yet a clear successor to the Rescorla–Wagner model, the 2 decades since the model appeared have been particularly fruitful in terms of an understanding of elementary learning processes. This progress was in large part stimulated by the Rescorla–Wagner model. For the time being, researchers would be well advised to continue using aspects of the Rescorla–Wagner model, along with those of other contemporary models, to help them design certain classes of experiments.

In regard to investigations of information processing outside the framework of traditional learning theory (e.g., connectionist modeling), it may be instructive to draw parallels and borrow concepts from the Rescorla–Wagner model. However, given the current status of the Rescorla–Wagner model, it is not appropriate for models outside the framework of traditional learning theory to claim any measure of success because their assumptions, processes, or predictions are concordant with those of the Rescorla–Wagner model. Theories are, of course, best judged by their approximation to observation. Approximation to another theory is at best only a shortcut and is acceptable only when the theory used as a benchmark has itself been highly successful. Surely, the Rescorla–Wagner model does not so accurately describe acquired behavior that another theory could meaningfully be judged by how well it approximates the

Rescorla–Wagner model rather than by how well it describes the observed behavior.

Nevertheless, the Rescorla–Wagner model has distinct merits (see introduction and list of successes). Investigators continue to use it for lack of a better model, which is neither inappropriate nor unusual in the history of science. The geocentric model of the heavens was known to have serious flaws for a long time before the heliocentric model was proposed. Yet, the geocentric model was widely retained during this period. Science needs models to serve as heuristic devices that direct research. Consequently, a flawed model is often better than no model at all. The Rescorla–Wagner model has stimulated much important research, the results of which will probably be central to the models of associative learning that will ultimately displace the Rescorla–Wagner model.

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