# Modeling the Behavioral Substrates of Associate Learning and Memory: Adaptive Neuronal Models

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*ABSTRACT*

Three adaptive neuronal models based on neural analogs of behavior modification episodes are proposed, which attempt to bridge the gap between psychology and neurophysiology. The proposed models capture the predictive nature of Pavlovian conditioning, which is essential to the theory of adaptive systems. The models learn to anticipate the occurrence of a conditioned response before the presence of a reinforcing stimulus when training is complete. Further, each model can find the most nonredundant and earliest predictor of reinforcement. The behaviors of our models account for several aspects of basic animal learning phenomena in Pavlovian conditioning beyond previous related models. Computer simulations show how well our models fit empirical data from various animal learning paradigms.

## I. Introduction

Animal learning is inferred from observed behavior and constitutes carefully testified postulates regarding elemental processes of learning. Recent research into animal learning can be separated into two categories: the behavioral and neural substrates of learning, namely, the psychological and physiological levels of learning. One way to bridge such a gap is to postulate neural analogs of behavioral modification paradigms. Hebb's postulate [15] for synaptic plasticity was the first trial as a neural analog of associative learning, which attempted to bridge psychology and neurophysiology. The theory of adaptive networks originated with [15] and continues to be influenced by plausible neural analogs of behavioral conditioning [8,17,9,26,24,27,18,25,2, 19,11,20].

Contemporary artificial neural networks are frequently referred to as connectionist models, parallel distributed processing (PDP) models, and adaptive/self-organizing networks. Basically, it is a complex system of neuron-like processing units that operate asynchronously but in parallel and whose function is determined by the network topology of connectivity. Artificial neural networks provide a new computational structure, a plausible approach for information processing because of its adaptivity/learning as well as massive parallelism.

Although new learning algorithms and VLSI technologies have recently rejuvenated neural network research, many problems still exist. Among them, the comprehensibility of neural networks, theoretical parsimony/enormous cost, and few empirical successes are major issues of the limitations of current neural networks. The learning behavior of such networks cannot be well understood, and the role of generic elements and subnetworks is unclear. Furthermore, most of these networks lack a theoretical foundation. The time and effort required to develop neural network architectures (network topology) and training is very high. Researchers have been devoted to "modeling applications", while relatively few "fielded applications" have emerged [3]. Most of such applications are restricted to pattern recognition, categorization, and realizations of associative memory. They are still toy research problem, at the proof-of-concept stage. Among the few exceptions, the Adaptive Channel Equalizer (developed by Bernard Widrow) is perhaps the most commercially successful of all neural network applications to date. It is a single-neuron device used now in virtually all long-distance telephone systems to stablize voice signals [3].

Klopf [18] has postulated that, "An intelligent system will have to build on a foundation that amounts to a highly detailed, immense microscopic knowledge base, a knowledge base that can be interfaced effectively with higher functional levels." From this perspective, a neural substrate could develop into the microscopic knowledge base. The macroscopic capabilities of intelligence could then be built on top of this. Given the limitations of current neural networks, a plausible scheme is to incorporate capabilities previously found on the macroscopic, network level into the microscopic, neuronal level.

In this article, we introduce three adaptive neuronal models that coincide with existing animal learning theory. Each proposed model provides a basis for understanding and explaining Pavlovian (classical) conditioning, which is the best understood animal learning process. After discussing the taxonomy of learning and Pavlovian conditioning, we briefly review previous related works for modeling Pavlovian conditioning. We then present our models by using the theories of Pavlovian conditioning in animal learning studies and justify their convergent behavior. Finally, we report computer simulations of these models and show how well our models fit empirical data from various animal learning paradigms.

## II. Learning and Pavlovian conditioning

### A. Learning

Learning, the acquisition of knowledge about the real world, is the most fundamental environmental factor in altering behaviors in animals. Learning denotes changes in the system that are adaptive in the sense that they enable the system to do the same task or tasks drawn from the same population more effectively the next time [23]. It is one of the remarkable abilities that animals possess, and it has been fruitfully studied by psychologists, neurophysiologists, biologists, artificial intelligence and cognitive science researchers. Traditional theories of learning and behavioral conditioning have been associative in nature. From this perspective, it has been useful to distinguish two major classes of learning: nonassociative learning and associative learning. Nonassociative learning, which includes habituation and sensitization [5], is said to result from experience with a single type of event (stimulus). Associative learning, which includes Pavlovian conditioning and operant conditioning [22,5], is said to result from the relationship of one event (stimulus or response) to another (stimulus). The theory of Pavlovian conditioning is a landmark in the study of learning and is the most well understood animal learning process.

### B. Pavlovian Conditioning

Pavlovian conditioning was introduced by Ivan Pavlov in 1927. It involves establishing a contingency between a relative neural conditioned stimulus (CS) and a significant, response-eliciting, unconditioned stimulus (US), or more precisely, the learning of a predictive relationship between these two stimuli. The most well-known example of Pavlovian conditioning comes from Pavlov's own research: the study of the conditioned reflex of salivation by dogs. Prior to conditioning, when a dog hears the sound of a bell, it picks up its ears. And when food is presented to it, it salivates only after food has entered its mouth. If this sequence of events—ringing the bell before food is served to the dog—is repeated, the dog soon starts to salivate just by the sound of the bell. The dog has in effect been "conditioned." it salivated. Figure 1 illustrates the conditioning of Pavlov's dogs.

## Prior To Conditioning

```
                          leads to
Sound of a Bell    ─────────────────────→   Picking Ears up
Food in Mouth      ─────────────────────→   Salivation
```

## After Conditioning

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                          leads to
Sound of a Bell    ─────────────────────→   Salivation
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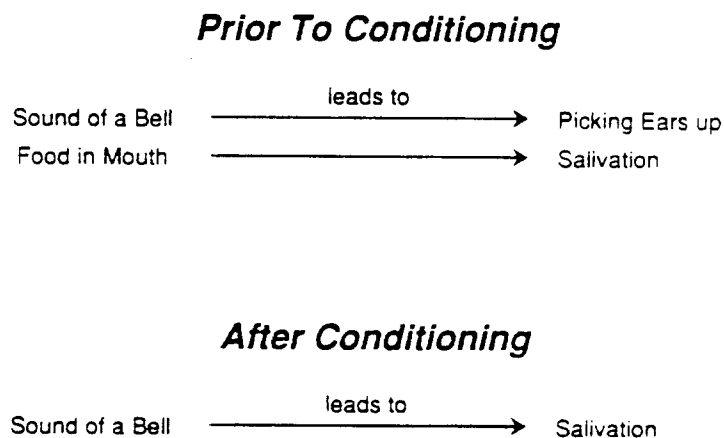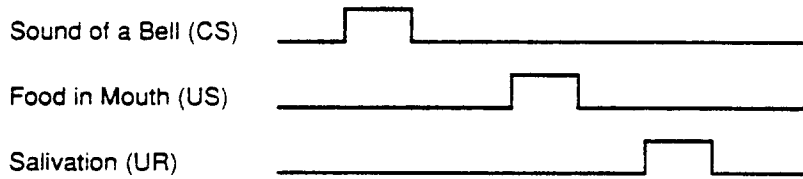
Fig. 1. Conditioning of Pavlov's dogs. Prior to conditioning, when the dog heard a bell sound, it picked up its ears. When food was in its mouth, it salivated. After conditioning, when the dog heard the bell sound, it salivated.

## Early Learning

Sound of a Bell (CS)

Food in Mouth (US)

Salivation (UR)

## After Learning

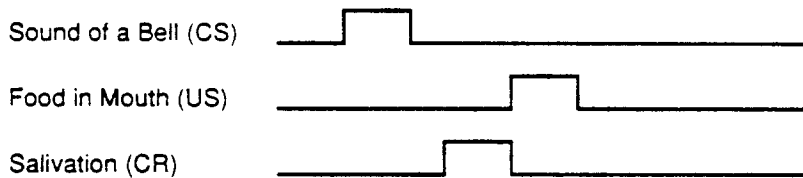Sound of a Bell (CS)

Food in Mouth (US)

Salivation (CR)

**Fig. 2.** Schematic paradigm of conditioning procedure. A US was repeatedly paired with the presence of a CS during early conditioning. After conditioning, the CS can predict the occurrence of the CR before the presence of the US.

Since food induces salivation by reflex, without training, the food is called the unconditioned stimulus (US) and the salivation is the unconditioned response (UR). Since, the sound of a bell elicits salivation after training, it is called the conditioned stimulus (CS). The salivation resulting from the sound of the bell is the conditioned response (CR). The paradigm for this learning procedure is illustrated in Figure 2. As can been seen, a CS can be used to predict the occurrence of a CR before the presence of a US. From animal learning theory literature, in addition to the the contiguity--temporal characteristics of the CS and the US during Pavlovian conditioning--we cannot overemphasize the importance of the contingency--a truly predictive relationship between the CS and the US.

## III. Previous Models of Pavlovian Conditioning

Modeling Pavlovian conditioning serves as an analytical tool for evaluating axiomatic principles of associate learning. It makes vague and complex ideas explicits, and it is precise enough to predict implications revealed through empirical research. Many researchers have explored modeling methods for this purpose since Hebb's work [15] . The development of models for Pavlovian conditioning can be classified into two main streams: neural and psychological (behavioral) substrates. The former includes the work of Hawkin and Kandel [13,14], Kleso and Brown [16], Gluck and Tompson [7]. The latter can be further subdivided into neural network models and single neuronal models. Network models were reported by S. Grossberg in a series of his work [8,9,10,11]. Single neuronal models, which rely less on detailed anatomies than

Grossberg's models and more on complex processing at the neuronal level, were proposed by Rescorla and Wagner [26,28], Frey and Sears [6], Pearce and Hall [24,12,25], Sutton and Barto [27,2], and Klopf [17,18,19,20]. These single neuron models have been a particularly active area for modeling Pavlovian conditioning. In this article, a special attention is paid to the single neuronal models, and a brief review of previous related works is given as follows.

### A. Rescorla-Wagner Model

Rescorla and Wagner [26] proposed that the associative strength of a CS depends in part on the differential effect of unexpected vs. expected responses. Their model confirms to various Pavlovian conditioning paradigms, such as blocking, conditioning with compound stimuli, and the other stimulus context effects. It extracts appropriate correlation between reliable, nonredundant CS and US. Also, Sutton and Barto [27] showed that the Widrow-Hoff (Adaline) model is essentially equivalent to this model. Despite many successes of this model, it has several well- known limitations and shortcomings. First, there is no evidence to support the extinction of conditioned inhibitors predicted by this model [29]. Second, it does not explain the retardation of conditioning produced by prior presentation of the CS alone (latent inhibition) [1]. Third, the simple acquisition curve is strictly negatively accelerated while the typical learning curve is sigmoid in shape [22]. Finally, the temporal dynamics of conditioning and decay terms are not included.

### B. Pearce-Hall Model

Pearce and Hall based their models on ideas originated by Mackintosh [21] and Wagner [28], namely that variations in a CS processing determine the course of conditioning and that predictive power is inversely proportionally to the "expectedness" of the events. They postulated that the associative strength of a CS is partly determined by an associability specific to that stimulus. The associability of a CS on one trial depends on the absolute value of the discrepancy experienced on the immediately preceding trial between the actual elicited and expected USs. One impetus for the Pearce-Hall model is that it more successfully describes the latent inhibition and surprising omission of the second shock [4], which could not be sufficiently addressed by the Rescorla-Wagner model. However, the simple acquisition curve is purely negatively accelerated. The intratrial temporal relationship between the CS and US was taken no further until [25]. In addition, while conditioning with compound stimuli, the Pearce-Hall model will be unstable once the sum of the associative strengths of all CSs is greater than the expected US. This is due to the use of absolute value in the associability for their model.

### C. Sutton-Barto Model

Sutton and Barto's [27] model is based on ideas from Rescorla and Wagner[26] and Klopf [17], that variations in a US processing determine the course of conditioning, and synaptic plasticity becomes eligible under a certain synaptic activity and remains eligible for a period of time. They proposed that the associative strength of a CS be dependent upon the discrepancy between the actual activity and expected activity responses plus an eligibility trace. This trace strengthens whenever a CS signal occurs and decays exponentially. Their model is in strong agreement with the behavior data regarding the effects of stimulus context, since it is a temporally refined extension of the Rescorla-Wagner model [27]. Hence, it shares the shortcomings of the Rescorla-Wagner model regarding the extinction of conditioned inhibitors and the shape of acquisition curve. One important aspect of the Sutton-Barto model is to express the

contingency, a predictive relationship between the CS and US in a formal way, and elicits a CR before the occurrence of the US. Also, it captures more temporal dynamics of Pavlovian conditioning than the Rescorla-Wagner model.


## IV. Adaptive Neuronal Models

In this section, we discuss the theories of our models and formulate those ideas mathematically. Then, the convergent behaviors of the proposed models are analyzed.


### A. Theory

Basically, the theories of our models originate from many animal learning literatures and previous related models. We abstract and adopt underlining principles from such literatures for which we consider essential for adaptive neural models with maximum consistency.

In [28], Wagner first postulated that the associative strength of a CS depends not only partly on the variations of a CS effectiveness but also on the variations of a US effectiveness, but this is taken no further. This will be known as the CS-US effective theory in this article. Before that, Rescorla and Wagner [26] emphasized on the variations in a US as a result of CS-US parings. We shall refer to this as the US effective theory. Alternately, Pearce and Hall [24] postulated that the variations in a CS processing (associability) determines the course of conditioning. Further, the associability of a CS is experienced on the immediately preceding trial. This will be referred to as the CS effective theory. After introducing these three fundamental theories, we would like to convey some properties of the associability from the Mackintosh attention theory [21], which are essential to adaptive neuronal models. That is, the associability depends on the nature of the stimulus (the correlation of a stimulus with reinforcements), and each associability varies independently. These have been implicitly adopted by various related models [26,27], especially in the conditioning of compound stimuli, including ours. A parameter is usually set to capture these properties, such as the stimulus salience in [26] and the learning rate in [27]. One important aspect of this theory is that the associability may change with a subject's experience. We propose that this is not limited only by the immediately preceding trial. We shall call it the experienced effective theory. As mentioned above, Sutton and Barto [27] incorporated two essential features of Pavlovian conditioning into their model, namely, the predictive nature and temporal intratrial dynamics of the CS and US. These features were implemented using the notion of the eligibility trace and the predictive version of the adaptive/learning rules. This shall be known as the temporal prediction theory. Finally, we abstract an idea from Klopf [19], Klopf's postulate, that a change in the associative strength of a CS is proportional to its current associative strength.

After introducing the theoretical background for our models, we can now describe three proposed models employing these theories. Basically, the theory behind Model I originates from the CS-US effective theory, experienced effective theory, temporal prediction theory, and Klopf's postulate. The theory behind Model II is from the US effective theory, experienced effective theory, temporal prediction theory, and Klopf's postulate. This model can be viewed as a simplification of the Model I or an extension of the Sutton-Barto model [27]. The theory behind Model III is similar to that of Model I, except the CS effective theory is used instead of the US-CS effective theory. It is a simplification of Model I or an extension of the Pearce-Hall model [24].
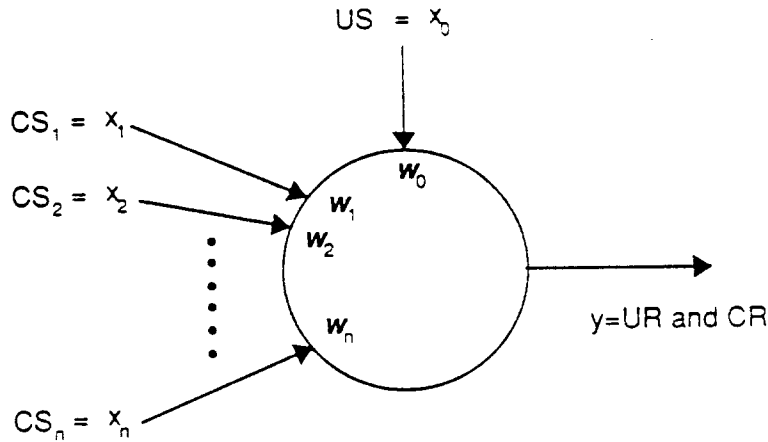
**Fig. 3.** A generic adaptive neuronal model with n conditioned stimuli $CS_i$ with associative strengths $w_i$, $i=1,2,...,n$, a unconditioned stimulus US with its fixed strength $w_0$, and an output $y$.

## B. Mathematical Formulation

To summarize the theories behind the proposed models discussed thus far, it is helpful to define these ideas in mathematical terms. Figure 3 illustrates a generic adaptive neuronal model which is used as a reference for describing the proposed models. It shows a model with an input pathway for each CS, an input pathway for the US, and an output pathway for the UR and CR. Basically, the generic adaptive neuronal model is characterized by an activation rule and a learning rule. The former combines the impinging inputs to produce a new level of activation. The latter is used to modify the associative strength of each input CS using its own experience. The dynamic behavior of this model with these rules simulates functions of Pavlovian conditioning paradigms.

### Activation Rule

Before defining the activation rule, a neural analog of Pavlovian conditioning needs to be addressed. The presence or absence of each $CS_i$ at time t, $i=1,2,...,n$, is represented by the activity on the corresponding signal $x_i(t)$. Similarly, the presence or absence of a US at time t is denoted by $x_0(t)$. The associative strength of each $CS_i$ with respect to the US is indicated by $\omega_i(t)$, CS-US synaptic weights. The fixed strength for the US is denoted as $\omega_0$. The strength of the CR at time t is represented by $y(t)$. In particular, $y(t)$ is a special combination strength of the CR and UR during various conditioning episodes. An activation rule is then characterized by the following equations:

$$y(t) = f(\sum_{i=0}^{n} \omega_i(t)x_i(t)),$$

where $f$ is an activation mapping function. In this article, an identity function is adopted. However, a hard limiter, a threshold logic, or a sigmoid-shaped function is a possible candidate.

*Learning Rule*

The learning rule plays an important role in our models, since it captures many essential features of the Pavlovian conditioning paradigm. Some other variables are introduced to characterize this rule. The associability of each CS, *conceptually* similar to the eligibility in [17,27], initiates a local prolonged trace by the presence of that stimulus, which captures the notion of the CS effectiveness. We also incorporate the idea from Klopf's postulate. The associability $\alpha_i(t)$ of each $CS_i$ at time t is then implemented by an averaging theory, an exponentially weighted moving average which calculates the weighted average of the values of $x_i(t)\omega_i(t)$ during a time interval immediately preceding t. Further, we acknowledge the possibility that the nature of the US may influence the CS associability (the term $y(t)-\bar{y}(t)$ as follows). Similarly, an averaged expected activity response $\bar{y}(t)$ is computed by the values of $y(t)$ over some time period preceding t. Furthermore, the nature of the associability of each $CS_i$ is separatedly denoted by a constant parameter $c_i$, a learning rate parameter. Therefore, the learning rules of our models are defined by the following equations:

Model I:

$$\bar{y}(t) = \beta y(t-1) + (1-\beta)\bar{y}(t-1),$$

$$\alpha_i(t) = \gamma|\omega_i(t-1)x_i(t-1)| |y(t)-\bar{y}(t)| + (1-\gamma)\alpha_i(t-1),$$

$$\omega_i(t+1) = \omega_i(t) + c_i\alpha_i(t)(y(t)-\bar{y}(y)),$$

Model II:

$$\bar{y}(t) = \beta y(t-1) + (1-\beta)\bar{y}(t-1),$$

$$\alpha_i(t) = \gamma|\omega_i(t-1)x_i(t-1)| + (1-\gamma)\alpha_i(t-1),$$

$$\omega_i(t+1) = \omega_i(t) + c_i\alpha_i(t)(y(t)-\bar{y}(y)),$$

Model III:

$$\bar{y}(t) = \beta y(t-1) + (1-\beta)\bar{y}(t-1),$$

$$\alpha_i(t) = \gamma|\omega_i(t-1)x_i(t-1)|(y(t)-\bar{y}(t)) + (1-\gamma)\alpha_i(t-1),$$

$$\omega_i(t+1) = \omega_i(t) + c_i\alpha_i(t)y(t),$$

where $\beta$ and $\gamma$ are constants with the values of $0 <\beta, \gamma\leq 1$. We require $\omega_i(t)$, to be nonzero initially, though it can be chosen arbitrarily close to zero.

In general, the activity $x_i$ and associative strength $\omega_i$ on an input pathway directly affect the output activity $y$. At the same time, they elicit a local prolonged trace (associability) on that pathway for possible synaptic modification. Particularly, for Model I and III, the associability also depends on the discrepancy of the actual activity $y$ versus averaged expected activity $\bar{y}$. A CS-US synaptic modification of Model III is possible whenever it has non-zero values for the associability $\alpha_i$ and output activity $y$. A CS-US synaptic weight of Model I or Model II is modifiable whenever it has a nonzero associability and a difference between $y$ and $\bar{y}$.

*C. Convergent Behavior of Simple Acquisition*

In this section, we show the convergent behaviors of our models with application to simple acquisition (with only one input CS). For simplicity, we shall take the value of $\beta$ equal to 1 which
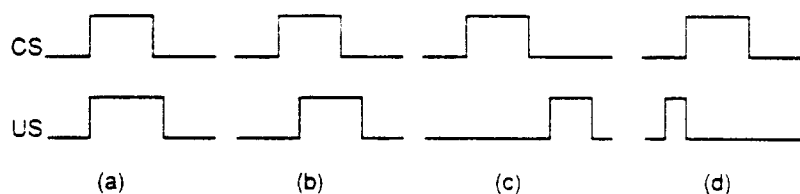
**Fig. 4.** Temporal relationship between the CS onset and US onset in basic conditioning paradigms. (a) simultaneously, (b) delay, (c) trace, and (d) backward conditioning.

results in $y(t)-\overline{y}(t)=y(t)-y(t-1)$, first difference of $y$ at time t. We also assume rectangular signals for the CS and US. Furthermore, the duration of the US is assumed long enough to have the CS lose its associability by the time of the US offsets. Finally, the initial value of the associability is set to zero. These assumptions will apply to the following discussion and the simulation in the next section.

As known in Pavlovian conditioning, the temporal dynamics of the CS and US are essential to forming a predictive relation. The CS-US temporal relationship is usually termed by the interstimulus interval (ISI), which is the time interval between the CS onset and US onset. Depending on the time course of CS-US pairings, conditioning is identified as forward conditioning (when ISI is nonnegative) and backward conditioning (when ISI is negative). Here, we shall focus on forward conditioning since backward conditioning usually does not occur. Three types of forward conditioning are possible: the simultaneous conditioning, delay conditioning, and trace conditioning. Figure 4 shows a schematic digram of forward and backward conditioning.

### Simultaneous Conditioning

Simultaneous conditioning results when the CS and US occur concurrently (ISI=0). In this case, our models do not learn any association from conditioning since the associability is always zero when $y(t)-y(t-1)$ is nonzero (as $y(t)$ in Model III).

### Delay Conditioning

Delay conditioning results when the US onsets before the CS offsets. The variation of the CS strength is strictly increased until it reaches an asymptotic associative strength. This convergence is ensured by the differential term $y(t)-y(t-1)$. Once $y$ remains a constant, the differential term becomes zero and ceases the learning process. However, different values of the ISI in all models influence learning speed substantially.

### Trace Conditioning

Trace conditioning results when the US onsets after the CS offsets. Figure 5 illustrates a trace conditioning paradigm with related variables. Let the duration of the CS be $l_{opt}$ simulation time steps. Then the ISI is represented by $l_{opt}+k$ time steps. We note the associability at point $a$ can be represented by:

$$\alpha(a) = \alpha(l_{opt+1}) = \gamma[(1-\gamma)^{l_{opt}}m(0) + \cdots + m(l_{opt})],$$

where $m(t)$ represents the corresponding term in each model, such as the absolute value of $\omega_i(t-1)x_i(t-1)(y(t)-y(t-1))$ in Model I. Furthermore,
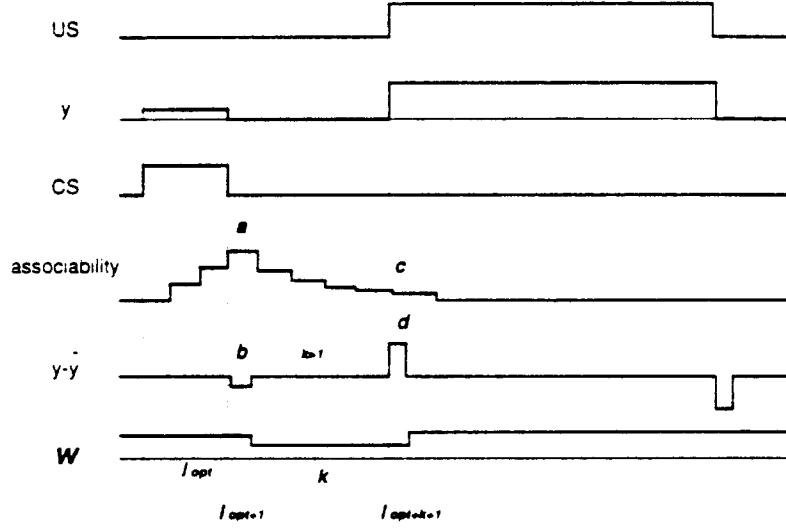
**Fig. 5.** Time courses of model variables for a conditioned trial in trace conditioning, in which a CS offset precedes a US onset by $k$ simulation time steps. The duration of the CS is $l_{opt}$ time steps and the US is of sufficient duration to ensure zero associability at the US offset. After some trials, the associative strength is positively strengthed. The new associative strength directly changes $y$ and hence $y-\bar{y}$. The CS offset with a positive associability causes a decrease in the associative strength, while an increase is caused by the US onset. At equilibrium, these two changes are balanced.

$$\alpha(b) = \alpha(l_{opt+k+1}) = (1-\gamma)^k \alpha(a),$$

since the values of $m(l_{opt+j})$, $j=1,2,\cdots$, are all zeros. In Model I and Model II, the dynamic equilibrium, $\omega_s$, of the associative strength CS is attained when the increase of its strength equals its decrease, namely,

$$\alpha(a)b + \alpha(c)d = 0,$$

$$\alpha(a)(0-\omega_s) + (1-\gamma)^k \alpha(a)1 = 0,$$

$$\omega_s = (1-\gamma)^k.$$

Hence, the asymptotic associative strength is proportional to $(1-\gamma)$ raised to the powers of $k$. However, the same property does not hold for Model III. Its asymptotic associative strength eventually reaches zero since negative values of the associability are always produced. If we take its absolute value, the asymptotic strength eventually becomes unbounded.

## V. Simulation Results

Our models were implemented on a Sun workstation, and some basic conditioning phenomena were simulated. After describing parameter conditions for our simulation, we investigate the effect of conditioning with a single stimulus, including the simple acquisition of a CR,

time courses of the signals during conditioning, and the ISI effect. We then observe the effects of stimulus context, such as overshadowing, blocking, and second-order conditioning.

## A. Parameter Conditions

In general, the behaviors of our models depend on the timing (ISI), magnitudes, shapes, and durations of the CS and US, the averaging theory for the associability $\alpha_i$ and expected response $\bar{y}$, and the initial value of the associative strength. For simplicity, we assumed rectangular signals for the CS and US. The duration of the CS and US is 3 and 30 simulation time steps, respectively. The duration of the US was long enough to assure zero associability by the time of the US offset. Furthermore, the initial value of an associability was set to zero and that of each associative strength was set to 0.001. The other parameters for conditioning with a single stimulus were $\beta = 1.0$, $\gamma = 0.8$, and $c_i = 0.01$. For conditioning with multiple stimuli, learning rate parameters, $c_i$, were set to 0.1 instead of 0.01. In addition, the initial associative strengths of the CS1 and CS2 in the second order conditioning were set to 1.0 and 0.1, respectively.

## B. Results

### Simple Acquisition

Repeated reinforcement with a US resulted in the acquisition of a CR. Figure 6 shows simulated results for such a simple acquisition. All of these three models produced the sigmoid-shaped acquisition curves found in animal learning experiments.
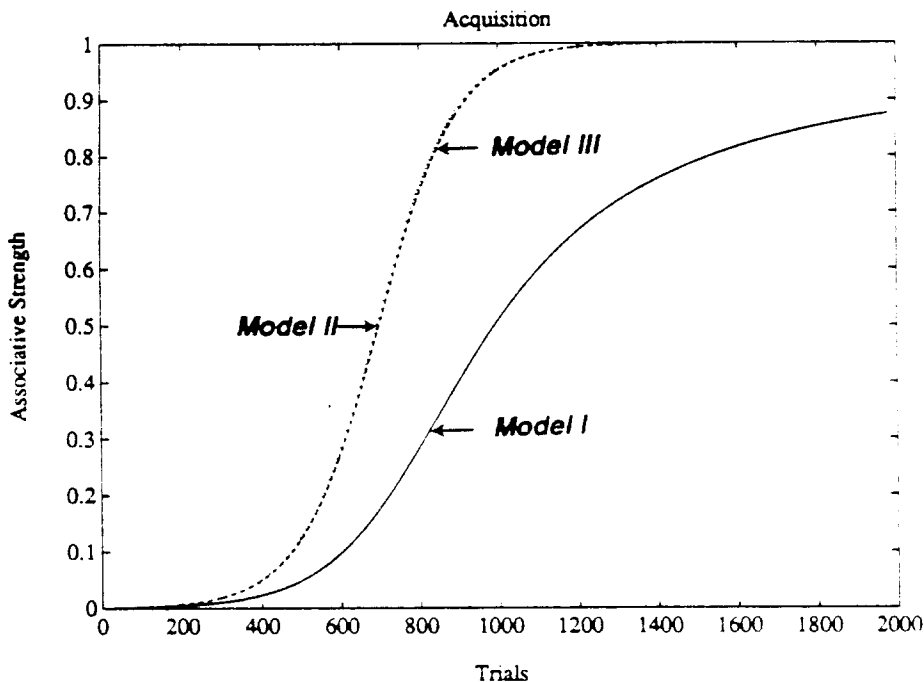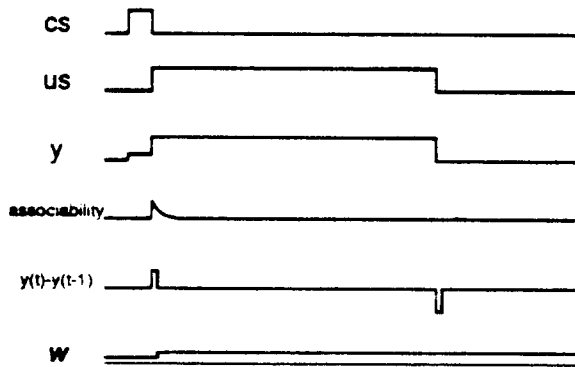


Fig. 6. Simulation of simple acquisition. The durations of the CS and US are 3 and 30 time steps, respectively. The time of the CS offset is the time of the US onset. A sigmoid-shaped acquisition curve is obtained by each model.

## First Trial of Conditioning
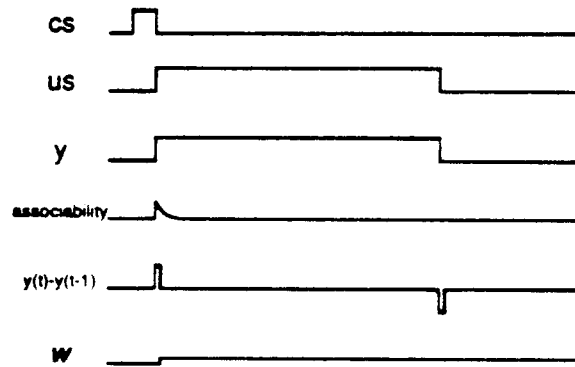


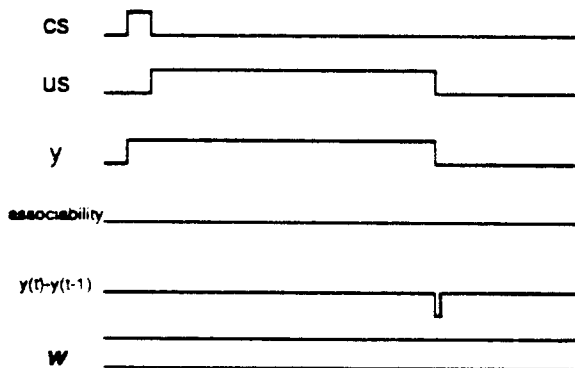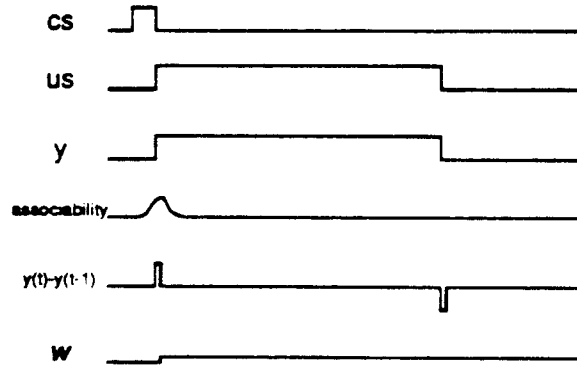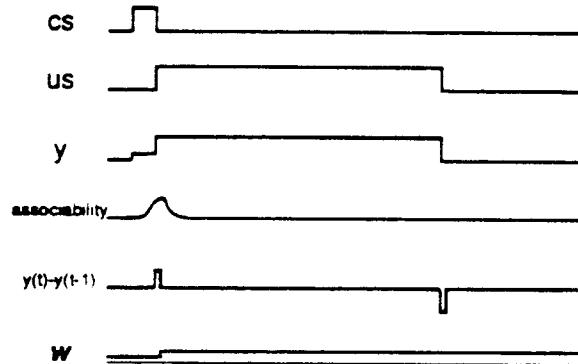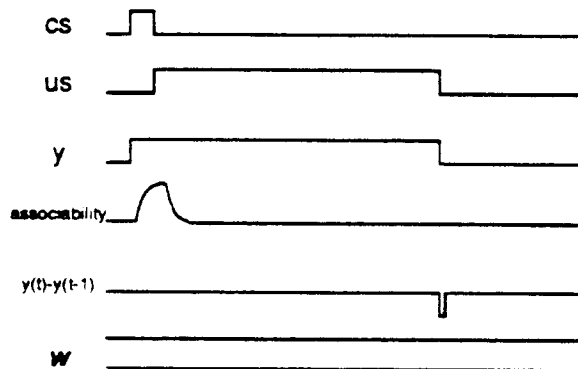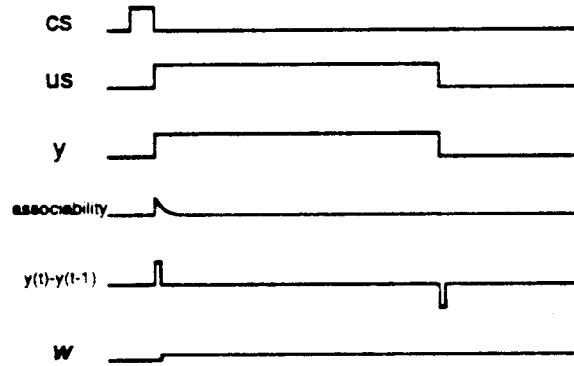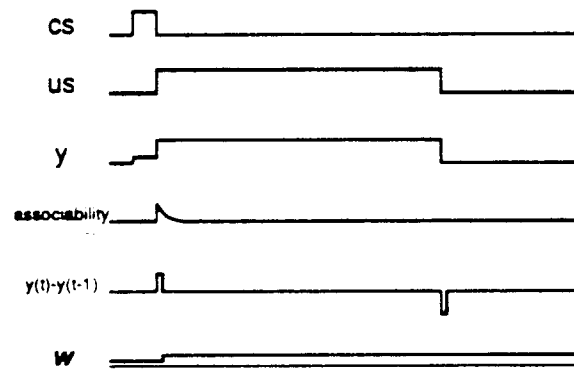## Second Trial of Conditioning



## After Conditioning



Fig. 7. Time courses of the signals of Model I in conditioning. (a) time courses of model signals during the first trial. The associative strength is increased due to the non-zero associability and positive difference of $y-\bar{y}$ caused by the US onset. (b) time courses of model signals during the second trial. An increased associative strength directly changes $y$ and then $y-\bar{y}$ as well as the associability. (c) time courses of model signals after sufficiently conditioning. Since there is no change in $y-\bar{y}$, the associability becomes zero and no further conditioning occurs.

## First Trial of Conditioning



## Second Trial of Conditioning
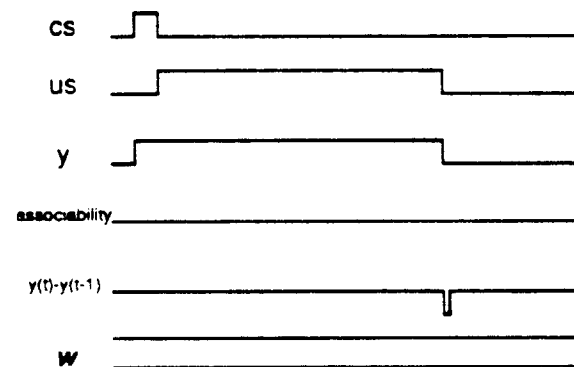


## After Conditioning



**Fig. 8.** Time courses of the signals of Model II in conditioning. (a) time courses of model signals during the first trial. The associative strength is increased due to the non-zero associability and positive difference of $y-\bar{y}$ caused by the US onset. (b) time courses of model signals during the second trial. An increased associative strength directly changes $y$ and then $y-\bar{y}$ as well as the associability. (c) time courses of model signals after sufficiently conditioning. The associability reaches its maximum since the associative strength is at the asymptotic level. There is no change in $y-\bar{y}$ and then no further conditioning occurs.

## First Trial of Conditioning



## Second Trial of Conditioning



## After Conditioning



Fig. 9. Time courses of the signals of Model III in conditioning. (a) time courses of model signals during the first trial. The associative strength is increased due to the non-zero associability and positive difference of $y-\bar{y}$ caused by the US onset. (b) time courses of model signals during the second trial. An increased associative strength directly changes $y$ and then $y-\bar{y}$ as well as the associability. (c) time courses of model signals after sufficiently conditioning. Since there is no change in $y-\bar{y}$, the associability becomes zero and no further conditioning occurs.

## Time Courses of Signals

The model behavior in classical conditioning with a single CS is traced by computer simulation. Figure 7, 8, and 9 illustrate the time courses of model variables corresponding to each model. It is worth noting that the variation of the associability differs not only from trial to trial but also from model to model.

## ISI Effects

Conditioning depends critically on the temporal relationship of the CS and US (ISI). Figure 10 shows the asymptotic associative strength for a series of simulation varying the ISI. It shows an ISI dependency with an inverted U-shaped curve, which is consistent with experiment data. Furthermore, the learning speed of each model is strongly affected by this variation of the ISI.

## Stimulus Configuration:

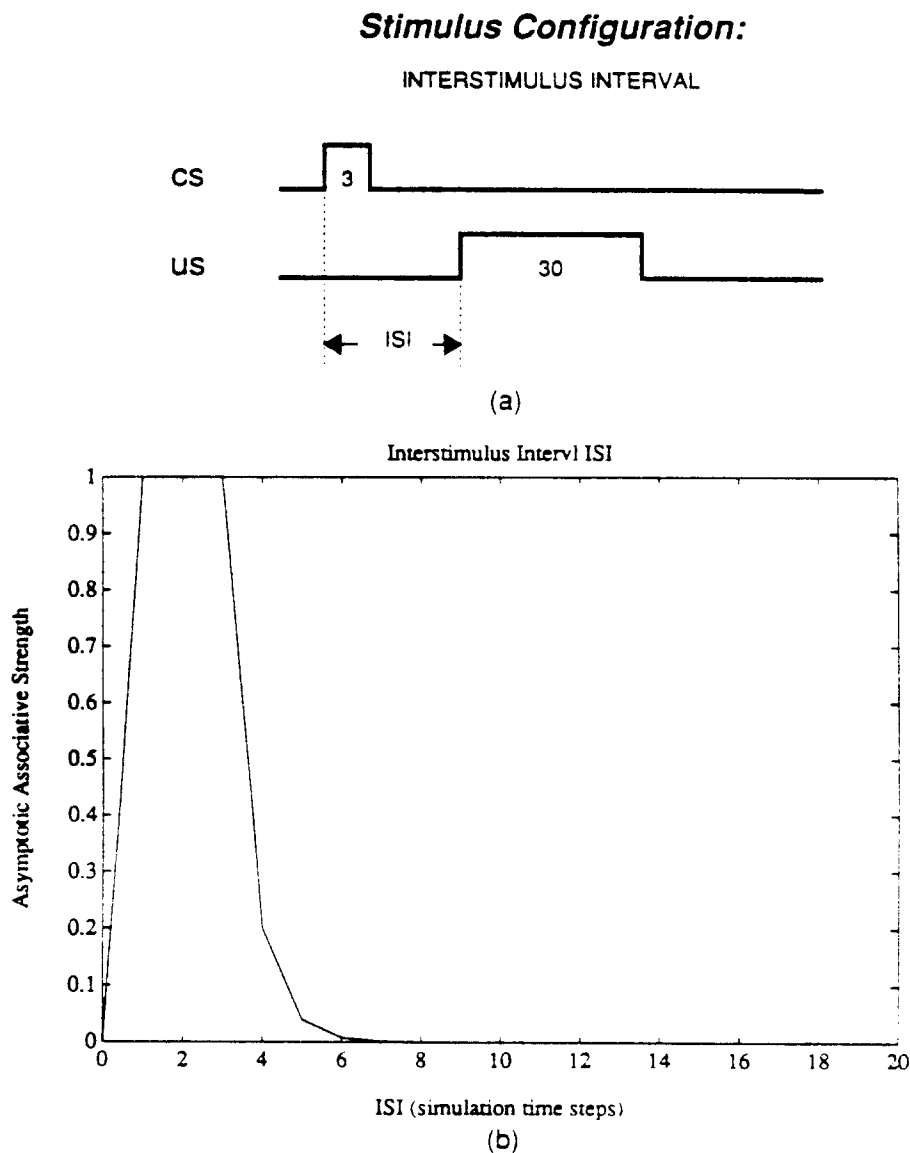### INTERSTIMULUS INTERVAL



(a)



(b)

Fig. 10. (a) A stimulus configuration of the interstimulus interval (ISI). The durations of the CS and US are 3 and 30 time steps, respectively. (b) Asymptotic associative strength vs. the ISI in a simulated conditioning. An inverted U-shaped curve results from Model I and Model II. No conditioning occurs in trace conditioning of Model III.

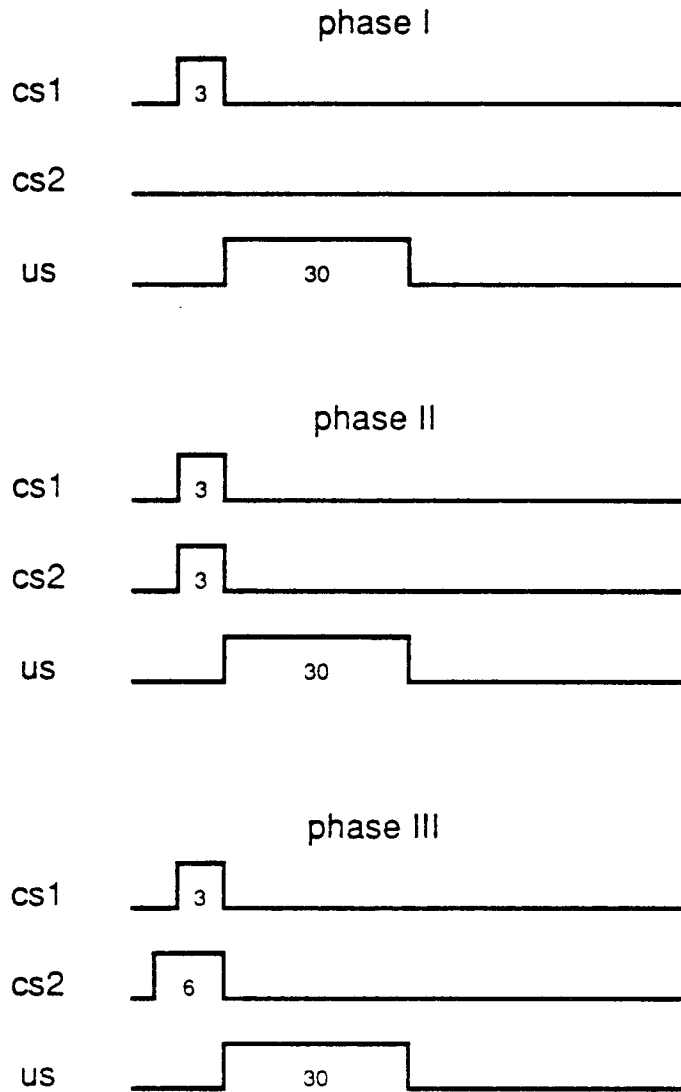## *Stimulus Configuration:*

BLOCKING and OVERSHADOWING

### phase I

cs1

cs2

us

### phase II

cs1

cs2

us

### phase III

cs1

cs2

us

**Fig. 11.** A stimulus configuration of blocking and overshadowing. A blocking experiment consists of phase I and II. In the phase I, $CS_1$ is sufficiently conditioned as usual. In the phase II, $CS_1$ and $CS_2$ are paired with the US. The phase III is a possible stimulus configuration of an overshadowing experiment.

## Overshadowing / Blocking

Overshadowing of one CS by another is affected not only by their relative stimulus salience, but also by their relative predictive power [22]. The former one is simulated by using different learning rate parameters in our models. The latter could be interpreted as follows: a better predictor of the US will overshadow a less predictive one. A possible stimulus configuration is depicted in the third phase of Figure 11. In Figures 12, 13, and 14, the third phase of conditioning demonstrates this phenomenon for the three models. It implies that our models can find the most non-redundant and earliest predictor of reinforcement.
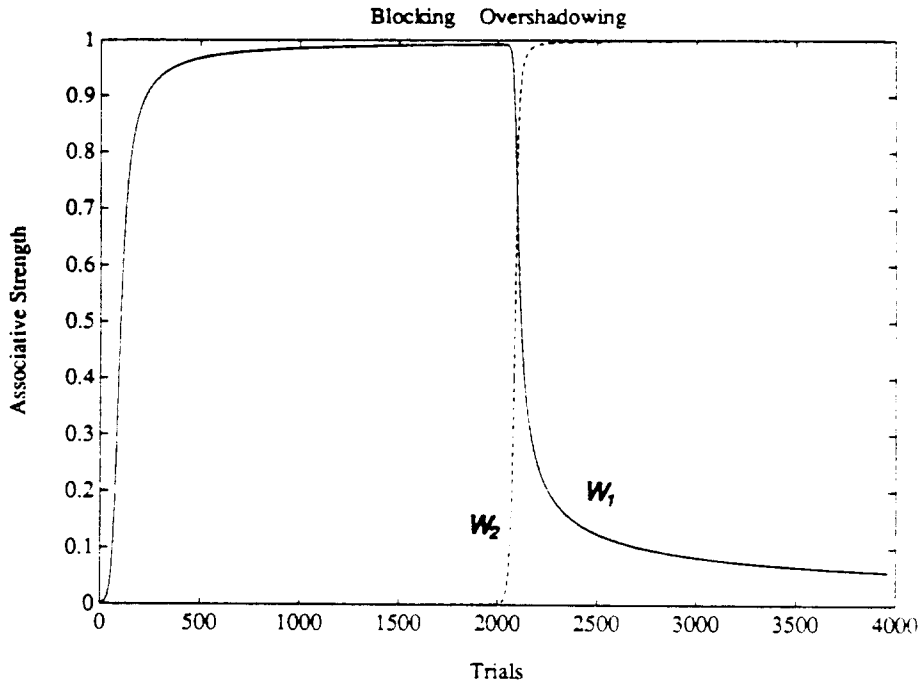


**Fig.12.** Simulation of a blocking and overshadowing experiment by Model I. The associative strengths at the end of each trial are illustrated. *Blocking.* In trials of 0-1000, a $CS_1$ is sufficiently conditioned by a US at its asymptotic level. In trials of 1000-2000, $CS_1$ and $CS_2$ present simultaneously and are paired with the US. No conditioning occurs to the $CS_2$. *Overshadowing.* In trials of 2000-4000, the earlier predictor $CS_2$ of the US overshadows the less predictive $CS_1$.
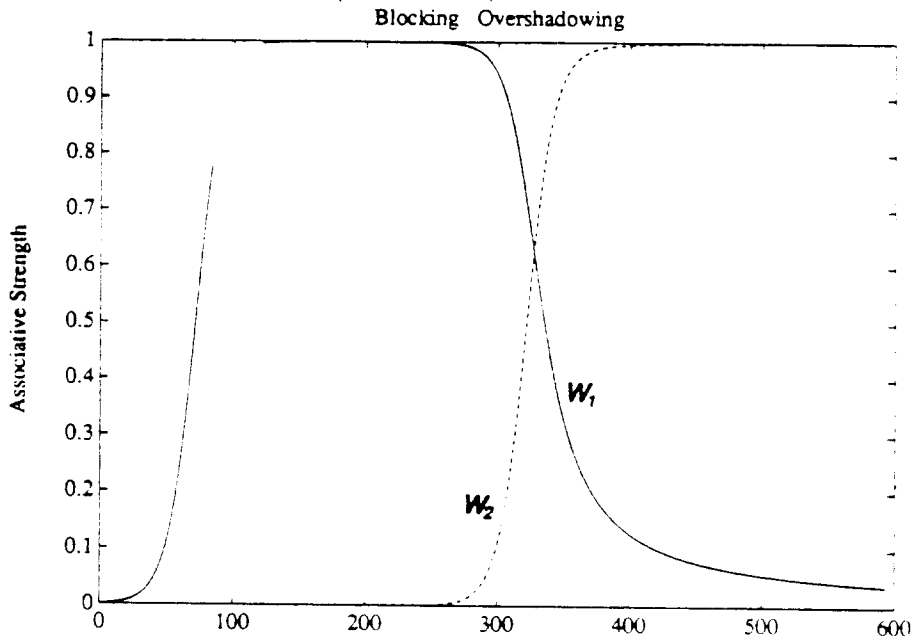


**Fig. 13.** Simulation of a blocking and overshadowing experiment by Model II. The associative strengths at the end of each trial are illustrated. *Blocking.* In trials of 0-200, a $CS_1$ is sufficiently conditioned by a US at its asymptotic level. In trials of 200-250, $CS_1$ and $CS_2$ present simultaneously and are paired with the US. No conditioning occurs to the $CS_2$. *Overshadowing.* In trials of 250-600, the earlier predictor $CS_2$ of the US overshadows the less predictive $CS_1$.
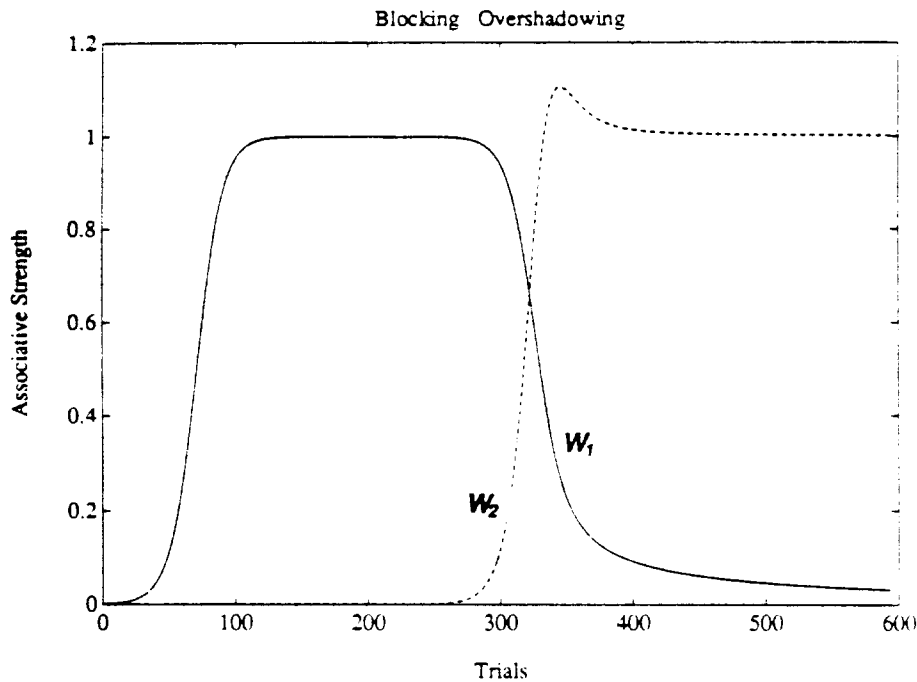
**Fig. 14.** Simulation of a blocking and overshadowing experiment by Model III. The associative strengths at the end of each trial are illustrated. *Blocking.* In trials of 0-200, a $CS_1$ is sufficiently conditioned by a US at its asymptotic level. In trials of 200-250, $CS_1$ and $CS_2$ present simultaneously and are paired with the US. No conditioning occurs to the $CS_2$. *Overshadowing.* In trials of 250-600, the earlier predictor $CS_2$ of the US overshadows the less predictive $CS_1$.

Blocking is the most well-known overshadowing. A stimulus configuration is shown as phase I and II in Figure 11. In the first phase of a blocking experiment, a CS is sufficiently conditioned as usual. In the second phase, a second CS is added to the previous CS and the compound stimuli is paired with the US. This paradigm results in no conditioning occurring to the second CS since the second CS is redundant. Phase I and Phase II in Figures 12, 13, and 14 show simulated results of blocking for each of the three models.

*Compound Stimulus Conditioning*

A special experiment with compound stimulus conditioning is designed to demonstrate the capability of appropriate association with nonredundant CSs. Trials for one CS1 are paired with a US with strength 0.6. Trials for compound stimuli CS1 and CS2 are paired with a US with strength 1.0. Two schemes are trained alternatively. Figures 15, 16, and 17 show that the asymptotic associative strength of the CS1 and CS2 reaches 0.6 and 0.4, respectively.
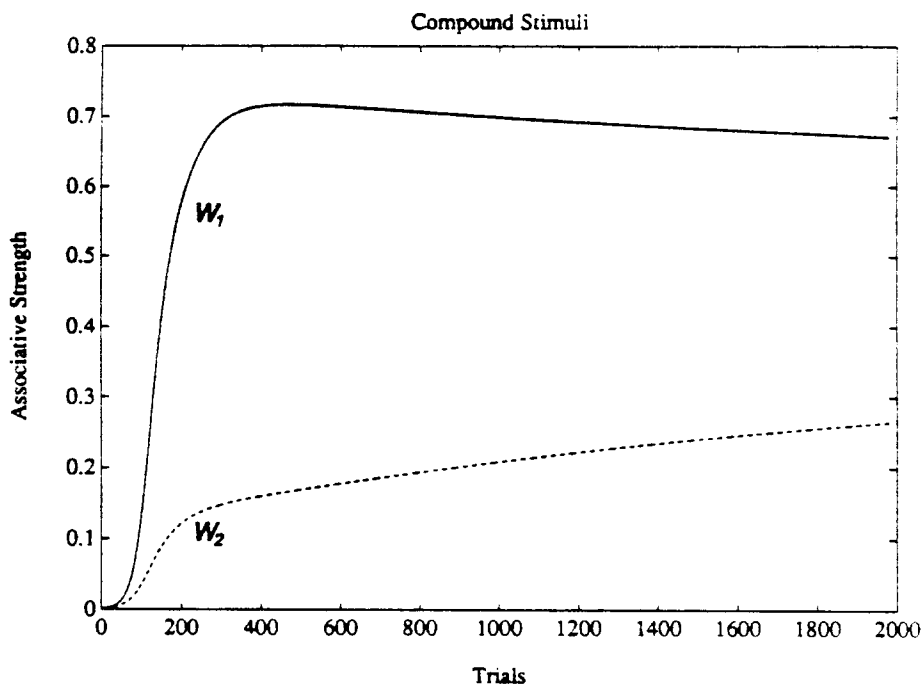
Compound Stimuli



**Fig. 15.** Simulation of a compound stimuli conditioning by Model I. Trials for one $CS_1$ are reinforced by a US with the strength 0.6. Trials for compound stimuli $CS_1$ and $CS_2$ are reinforced by a US with the strength 1.0. Two schemes are alternatively conditioned.
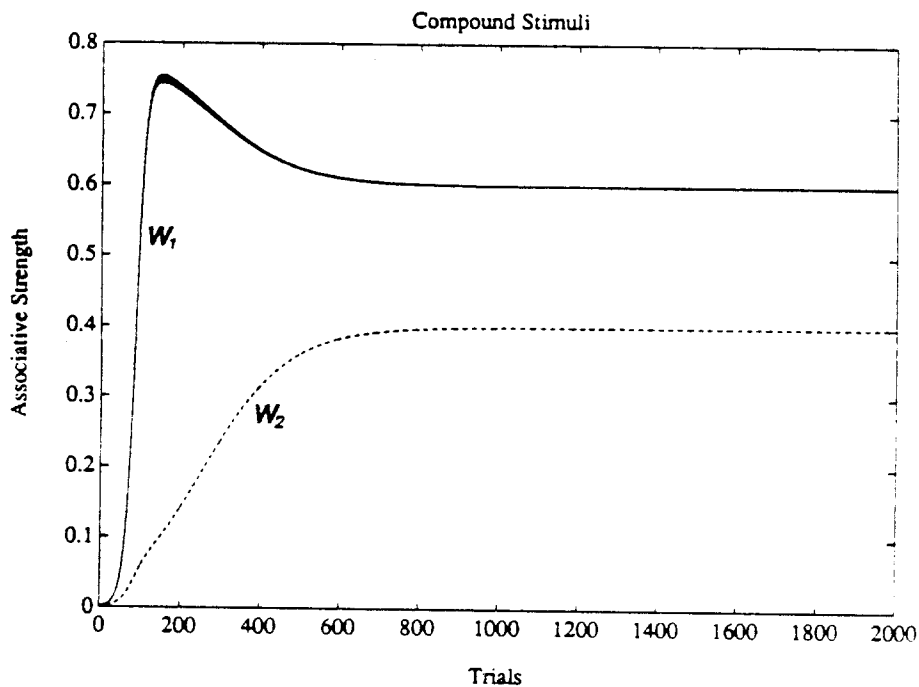
Compound Stimuli



**Fig. 16.** Simulation of a compound stimuli conditioning by Model II. Trials for one $CS_1$ are reinforced by a US with the strength 0.6. Trials for compound stimuli $CS_1$ and $CS_2$ are reinforced by a US with the strength 1.0. Two schemes are alternatively conditioned.
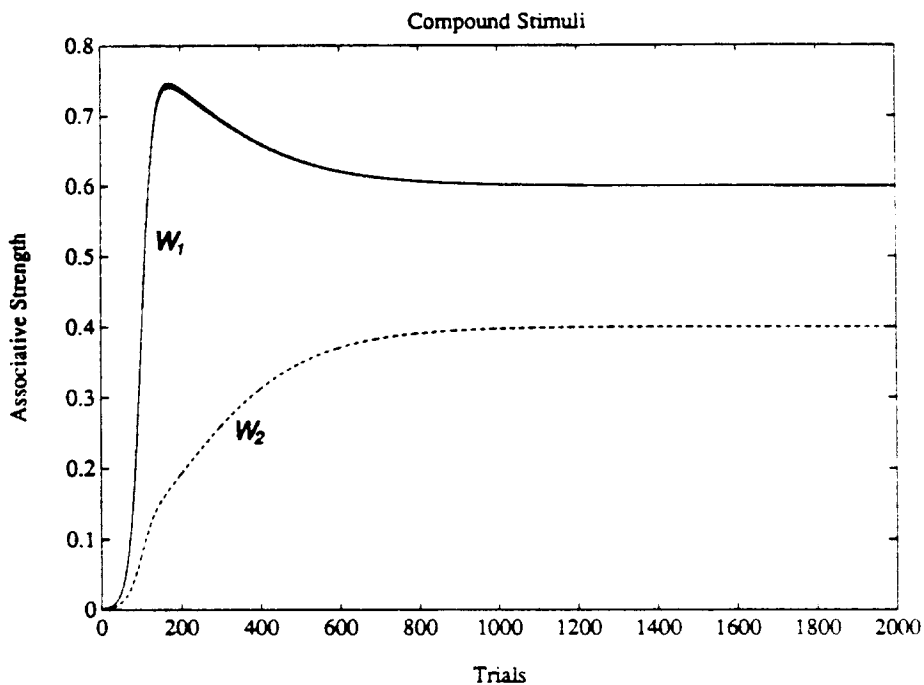
**Fig. 17.** Simulation of a compound stimuli conditioning by Model III. Trials for one $CS_1$ are reinforced by a US with the strength 0.6. Trials for compound stimuli $CS_1$ and $CS_2$ are reinforced by a US with the strength 1.0. Two schemes are alternatively conditioned.

## Second Order Conditioning

Figure 18 illustrates the stimulus configuration of a second-order conditioning. In the first phase, an effective US is used to reinforce the associative strength of an initially ineffective CS1. In the second phase, the CS1 served as a new US is used to strength a new CS2. Evidence of conditioning is provided by the occurrence of CRs to CS2. Simulated results of the second-order conditioning are depicted in Figures 19, 20, and 21 for Models I, II, and III, respectively.
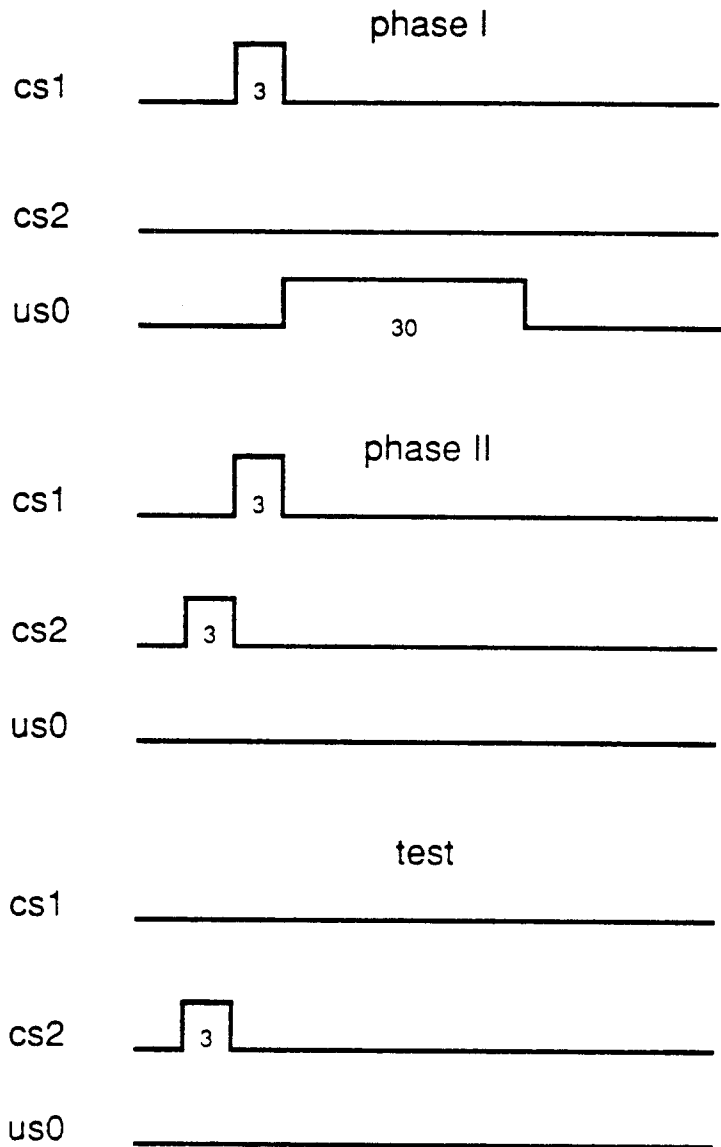
## *Stimulus Configuration:*

### SECOND ORDER CONDITIONING



Fig. 18. A stimulus configuration of second-order conditioning. $CS_1$ is paired with a US, $CS_2$ is then paired with the $CS_1$, and evidence of conditioning is provided by the occurrence of CRs to $CS_2$.

Second Order Conditioing



Fig. 19. Simulation of second-order conditioning by Model I. The associative strengths at the end of each trial are plotted. $CS_1$ is first conditioned by a US and reaches its asymptotic value. Then, $CS_1$ is served as a US for reinforcing $CS_2$ in the absence of the previous US. Since $CS_1$ is not reinforced by the US, its strength is weakened and hence the strength of the $CS_2$ is decreased.
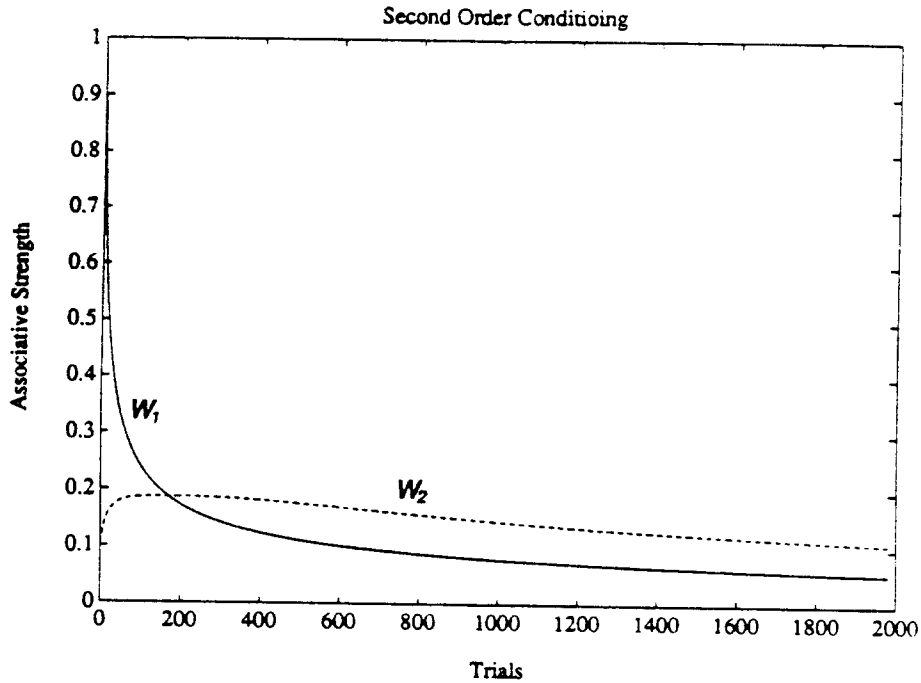
Second Order Conditioing



Fig. 20. Simulation of second-order conditioning by Model II. The associative strengths at the end of each trial are plotted. $CS_1$ is first conditioned by a US and reaches its asymptotic value. Then, $CS_1$ is served as a US for reinforcing $CS_2$ in the absence of the previous US. Since $CS_1$ is not reinforced by the US, its strength is weakened and hence the strength of the $CS_2$ is decreased.
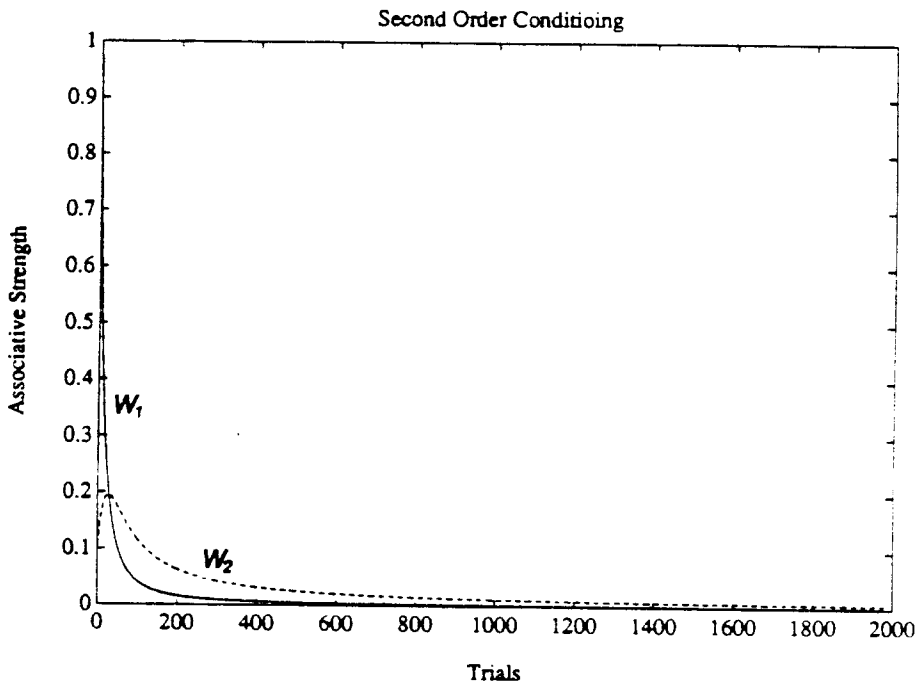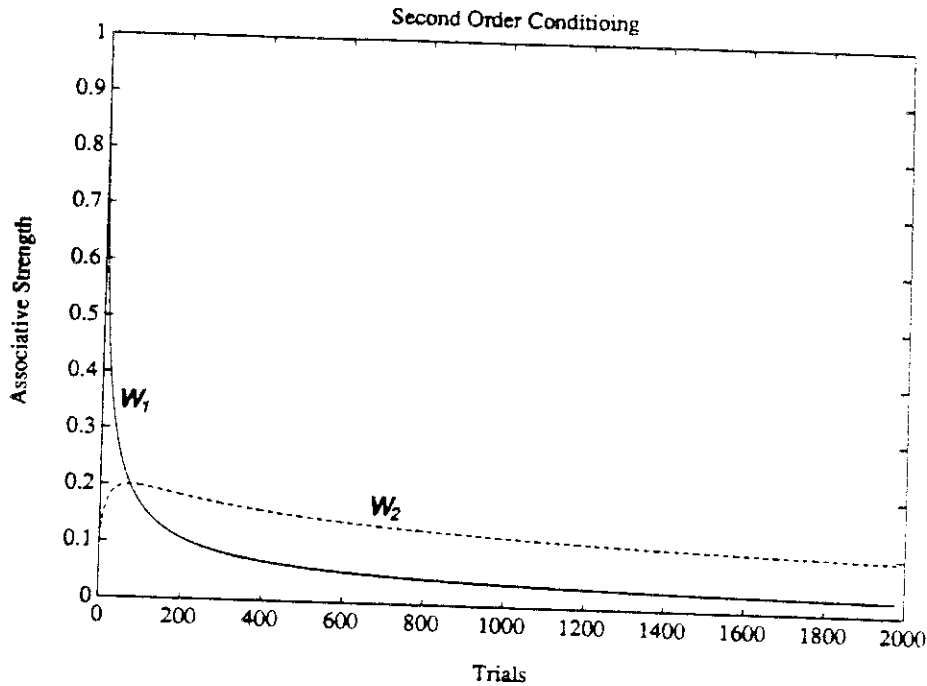
**Fig. 21.** Simulation of second-order conditioning by Model III. The associative strengths at the end of each trial are plotted. $CS_1$ is first conditioned by a US and reaches its asymptotic value. Then, $CS_1$ is served as a US for reinforcing $CS_2$ in the absence of the previous US. Since $CS_1$ is not reinforced by the US, its strength is weakened and hence the strength of the $CS_2$ is decreased.

## VI. Conclusion

In the preceding sections, we developed our adaptive neuronal models and investigated their behaviors by computer simulations. We postulated neural analogs of behavior modification episodes to bridge the gap between psychology and neurophysiology. Given the limitations of contemporary neural networks, a plausible trend is to shift the emphasis placed on the macroscopic capabilities from the network level into the neuronal level. Our models present one step toward this direction and no attempt is made to be exhaustive.

The behaviors of our models strongly agree with animal behavior data for Pavlovian conditioning, although our models are not complete for modeling Pavlovian conditioning. Our models account not only for the effects of conditioning with a single stimulus, including simple acquisition with a sigmoid-shaped curve, conditioned inhibition, extinction, forward conditioning, and the ISI effects, but also for the effects of conditioning with multiple stimulus context. Examples are blocking, overshadowing, latent inhibition, second-order conditioning, and higher order conditioning. The development of latent inhibition predicted by our models would be much faster than that in the case of animals since no learning would occur on the first conditioning trial. Further, Model I can explain the surprising omission of the second shock[4] by acknowledging that the nature of the US may influence the CS associability. We also incorporate the predictive nature of Pavlovian conditioning in our models, which is essential to the theory of adaptive systems. Further, our models capture the temporal intertrial relationship of the CS and US beyond the Rescorla-Wagner, Pearce-Hall, and Sutton-Barto models.

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