SIMULATION OF THE CLASSICALLY CONDITIONED NICTITATING MEMBRANE RESPONSE BY A NEURON-LIKE ADAPTIVE ELEMENT: A REAL-TIME VARIANT OF THE SUTTON-BARTO MODEL

Diana E.J. Blazis, John E. Desmond, John W. Moore, and Nell E. Berthier

Department of Psychology
University of Massachusetts
Amherst, Massachusetts 01003

ABSTRACT

The Sutton-Barto (SB) model of learning is based on a neuron-like adaptive element. The model has computational features suitable for describing a variety of classical conditioning phenomena, including blocking, conditioned inhibition, and higher-order conditioning. However, it presently does not describe within-trial phenomena related to conditioned response (CR) topography. We here describe in detail an extension of the SB element, referred to as the Sutton-Barto-Desmond (SBD) model, which is capable of simulating topography of the conditioned nictitating membrane response (NMR) of the rabbit. The SBD model places certain constraints on the SB model's parameters and makes some additonal assumptions about the form of inputs to the element. The model describes (1) the gradually increasing amplitude of the CR within a trial with the peak amplitude at the temporal locus of the US, (2) the decrease in CR onset latency over training, and (3) appropriate interstimulus interval (ISI) functions, with optimal learning occurring with an ISI of .25 seconds. In addition, the model lends itself to descriptions of neuronal firing related to the CR. We believe the SBD model may have implications for neurobiological studies of learning and memory.

INTRODUCTION

Several extensions of the original Sutton and Barto (SB) model of connectionistic learning (Sutton & Barto,1981; Barto & Sutton, 1982) have been introduced in recent years. These connectionistic models have proven capable of supporting a wide variety of complicated learning control problems, providing plausible architectures for distributed processing in adaptive networks.

The success of the SB model and its extensions is apparent not only in the operation of adaptive networks, but also at the level of single elements. The original SB model was described in terms of single-neuron-like adaptive element operating under a learning rule similiar to that proposed by Rescorla and Wagner (1972), and was shown to be capable of simulating many features of classical conditioning, particularly of the rabbit nictitating membrane response (NMR). Among these features were the anticipatory nature of the conditioned response (CR), acquisition and extinction, blocking,higher-order conditioning, and interstimulus interval (ISI) effects upon trace conditioning of the rabbit NMR.

Fig.1 illustrates the original SB neuron-like adaptive element. Note that the US in the original model is signalled by a pathway of a fixed efficacy, denoted λ. Inputs for each conditioned stimulus (CS) vary in transmission efficacy according to the strength of a learned connection for each CS, the synaptic weight V_{CS}. Two memory processes contribute to changes in synaptic weight: $\bar{x}$, which

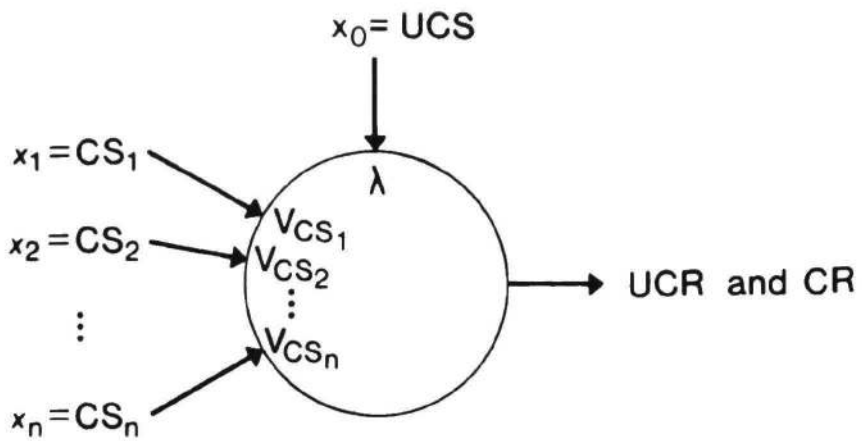

Figure 1: The original Sutton and Barto neuron-like adaptive element. Input pathway x_i has transmission efficacy V_{CS} corresponding to the associative strength of CS_i. The US (labeled UCS) is signalled via a pathway of fixed efficacy λ. Element output contributes to the UR (labeled UCR) prior to conditioning and to both the CR and the UR after conditioning. (from Barto and Sutton,1982).

determines the extent to which a given synapse is eligible for modification, and $\bar{x}$, a trace which represents the memory of the element's output in the preceding timestep. The single output of the element is computed as the weighted sum of all inputs. In the sense that the element receives several inputs, but transmits only one output, the SB element can be conceptualized as a "classic" neuron.

One of the research objectives of this laboratory is to examine the validity of single-neuron schemas of classical conditioning of the rabbit NMR. To that end we have taken a variey of approaches: electrophysiological, anatomical and behavioral among them. Our interest in the SB neuron-like adaptive element is the generation and development of a variant which simulates the topography of the NM CR as it appears in real rabbits in real time. The NMR is the sweeping of the nictitating membrane over the surface of the eyeball; it is a protective response that is a passive consequence of the retraction of the eyeball into the orbit by the retractor bulbi and extraocular muscles. This response has been studied within-trials and over trials in both single and multiple-CS protocols, thus offering an extensive database for the assessment of any model (Gormezano, Kehoe & Marshall, 1983). Recently, we demonstrated that the algorithm subserving the original SB element, though silent with respect to the generation of CR topographies, can be extended to the simulation of the neurophysiological and behavioral features of the rabbit NMR, predicting the ontogeny of the conditioned response (CR) over trials as well as within trials (Moore,Desmond,Berthier,Blazis,Sutton & Barto,1985; Moore, Desmond, Berthier, Blazis,Sutton & Barto,in press).

The CR topography characteristics that we sought to model included (1) gradually-increasing neuronal firing within a trial with attainment of peak CR amplitude at the temporal locus of the US; (2) a decrease in CR onset latency over training; and (3) an appropriate ISI function, with optimal conditioned strength accruing at an ISI of 250 milliseconds (ms). In order to produce these real-time phenomena, additional assumptions and constraints upon the variables of the original

SB model became necessary. We modified the SB element to yield appropriate topography in a single-CS forward-delay training paradigm in real-time. The question then became whether the model could retain the success of the original model's predictions in the domain of multiple-CS conditioning without further modification of the parameters.

This report will present the equations and assumptions of the constrained SB element, referred to as the Sutton-Barto-Desmond (SBD) model. We will show that the SBD element extends the SB model into real time while retaining the predictions of the SB element in multiple-CS domains.

THE MODEL: ASSUMPTIONS, EQUATIONS AND CONSTRAINTS

We begin our discussion of the SBD model by addressing the assumptions we have made about conditioning, stating the equations which make up the model, and justifying the constraints we placed upon the rules of the original SB model.

An assumption fundamental to the SBD model is that computations affecting synaptic weights occur before, during, and after the US. The original SB element assumed a 300 ms US, a duration sufficient to complete all computations within a trial before US offset. In rabbit NM conditioning, a more realistic US duration is 50 ms; under the conditions of the original model, a 50 ms US does not allow sufficient time for $\bar{x}$, the eligibility trace, to return to zero. Implementing post-US computations into the SB model required constraining the decay of various traces and including a mechanism whereby the effectiveness of the US changes over trials. These changes in the model are discussed more fully below.

Another assumption of the model concerned the form of the CS input to the adaptive element. Extending the SB element to model CR topography was accomplished in part by treating the CS input, designated as x in the model, as a continuous function. In the original model x was a step function equal to 1 at CS onset and 0 at CS offset. Although a step function defines the onset and offset of the external stimulus, it does not allow the SB model to predict rabbit NMR topography. For the purposes of the adaptive element, we assumed that the functions describing x mimic the behavioral CR. Thus, while the CS is on, x is shaped by a function which begins to rise gradually in an S-shaped fashion soon after CS onset, eventually maximizing at the temporal locus of the unconditioned stimulus (US). Assuming that t is a timestep representing 10 ms of real time:

$$x_i(t) = [arctan(mt + b) + 90]/(180 * h)) \tag{1}$$

for time $t = 1$ to CS offset. We selected the arctangent function because it is sigmoidal in shape and because it is a convenient one to implement in FORTRAN programming. The x-shaping parameters m, b, and h are specified as input variables with current default values equal to 0.35,-12.5 and 1.0, respectively. Simulation experiments involving systematic variations of the x-shaping parameters revealed that the model is very sensitive to their values; the default values represent those which yield the best topography for the 250 ms CS.

The second function mimics the decay of the CR in post-US timebins. It is implemented at CS offset, decays geometrically and is computed by multiplying successive values of x by a scalar:

$$\begin{aligned} x_i(t+1) &= k(x_i(t)) \\ k &= 0.85. \end{aligned} \tag{2}$$

Equations 1 and 2 specify, then, that the value of the input trace to the adaptive element begins to rise 70 ms after CS onset at the periphery,a latency justified by reports indicating that the minimal conditionable ISI for rabbits is 70 ms (Salafia, Lambert, Host, Chiaia & Ramirez, 1980). The input trace asymptotically reaches the value of one and decays to zero following CS offset.

The output of the SBD element $s(t)$, is defined as:

$$\begin{aligned} s(t) &= \sum_{i=1}^{n} V_i(t)x_i(t) + \lambda'(t) \\ 0.0 &\leq s \leq 1.0 \end{aligned} \tag{3}$$

where $\lambda'(t) = 0$ prior to the occurrence of the US. During US presentation, $\lambda'(t)$ is calculated as follows:

$$\lambda'(t) = \begin{cases} \lambda - V_i(t), & \text{if } 0 \leq V_i(t) \leq \lambda; \\ 0, & \text{if } V_i(t) > \lambda; \\ \lambda, & \text{if } V_i(t) \leq 0. \end{cases} \tag{4}$$

where V_{max} = the largest starting weight for all CSs present on a given trial, and λ = a constant that reflects US intensity.

Following the US, λ' is decremented :

$$\lambda'(t+1) = 0.9 * \lambda'(t) \tag{5}$$

The computation of $\lambda'(t)$ in equation (4) assumes that the contribution of the US to the element's output decreases as CS synaptic strength increases. In addition, λ' allows the element to predict diminution of the UR as conditioning progresses, a phenomenon previously observed in rabbits by Donegan (1981). Equation (5) reflects the assumption that the US input to the element is not equal to zero at US offset but instead, decays progressively, thereby influencing post-US decrements in V.

In the present implementation, CR topography is defined by the sliding arithmetic mean of the values of the current output s and that of the two preceding timesteps. This was done to smooth the transition from CR to UR. We further assume that the output of the element is bounded between 0.1 and 1.0. A negative s was deemed inappropriate for modelling NMR topography since a negative output implies NM retraction and exopthalmus, CR-opposing responses which are generally not observed in the rabbit NMR. The value of 0.1 reflects a threshold between the SBD element's output and the motoneurons which generate the peripherally observed response. The upper limit of 1.0 is derived from evidence indicating an upper limit for the amplitude of the behavioral NMR. Bounding s in this way also facilitates the computations relating s to neuronal firing rates of 10 to 100 Hertz needed for peristimulus– time histograms which cumulate neuronal firing over trials in the simulations.

Thus, equation (3) indicates that the output of the element at any given time t is equal to the weighted sum of its inputs. In a single–CS forward–delay paradigm, the output prior to the US is identical in form to the input, x, and the magnitude of the output is proportional to the synaptic weight.

The equation which dictates changes in synaptic weight (associative strength) is retained from the original SB model. Synaptic weights can be positive, negative or zero, which we interpret as

corresponding to excitatory, inhibitory or neutral inputs, respectively. $V_i(t)$,the synaptic weight of the ith conditioned stimulus, is modified as follows:

$$V_i(t+1) = V_i(t) + c[s(t) - \bar{s}(t)]\bar{x}_i(t) \tag{6}$$

where c is a learning rate parameter equal to 0.15, s is the element's output, and $\bar{s}$ is the element's prediction of its output based on its prior activity:

$$\begin{aligned} \bar{s}(t+1) &= \beta[\bar{s}(t) + (1-\beta)s(t)] \\ \beta &= 0.6 \end{aligned} \tag{7}$$

Note that the values of the learning rate parameter c, and of β in Equation 5 exert important influences upon the ability of the SBD model to accurately portray rabbit NM conditioning. For example, c can decrease or hasten the rate of learning and affect CR topography, although asymptotic synaptic weights are not affected.

Variable $\bar{x}$ is a CS duration-dependent stimulus trace which defines the extent to which a given synapse or connection is eligible for modification. In SBD, unlike SB, the eligibility trace mirrors and lags 30 ms behind the input trace.

$$\bar{x}(t+1) = x_i(t-2) \tag{8}$$

during the time that the CS is on. When the CS is off,

$$\bar{x}_i(t+1) = \delta * \bar{x}_i(t) \tag{9}$$

where $\delta = e^{-3/d}$, d = CS duration in timesteps; $d \geq 25$. These computations define a period of eligibility which begins some time after CS onset and persists beyond CS offset.

The goal of simulating appropriate ISI functions was accomplished in part through our specification of the decay of $\bar{x}$. Note that δ becomes larger for relatively longer CSs, thus slowing the decay of $\bar{x}$ and allowing greater opportunity for decrements in V following CS offset. Thus asymptotic synaptic weights are lower for CSs of relatively long duration.

As mentioned above, one goal in the development of the SBD model was to extend the SB model to encompass the simulation of neuronal firing. The current implementation of the SBD model lends itself readily to the computation of neuronal firing on single trials and accumulation of spikes over trials to form peristimulus-time histograms (PSTHs). Space limitations preclude a complete description of this portion of the model; see Moore et al (in press) for details. An example of neuronal firing is shown in Fig. 2, which shows a PSTH of neuronal firing to a 250 ms CS followed immediately by 30 ms US after 50 trials.

PREDICTIONS OF THE SBD MODEL

We have examined the behavior of the model at many levels: within- trials and over trials in single and multiple–CS conditioning. What follows is a brief account of our findings, beginning with topography for single–CS conditioning and interstimulus- interval functions, and progressing to multiple–CS procedures.

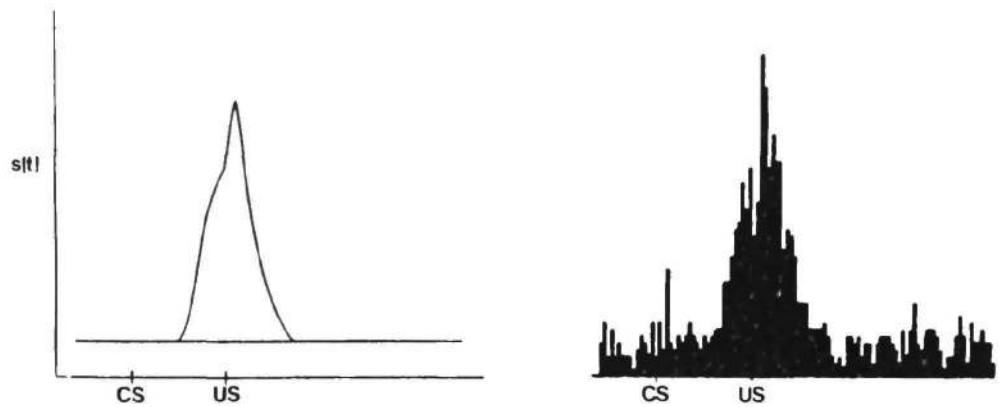

Figure 2: Simulated CR/UR complex and a PSTH for single–CS forward– delay training with a 250–ms CS terminating simultaneously with the onset of a 30–ms US after 50 trials. $\lambda = 0.9$, $c = 0.15$.

NMR Topography

The default values for the parameters shaping the input trace x (m= 0.35, b= -12.5 and h =1.0) allow the model to appropriately simulate many topographical features characteristic of the rabbit NMR. One, mentioned earlier, is a decrease in the latency of the onset of the CR as conditioning proceeds. The amplitude of the CR increases over training, with maximal amplitude occurring at or near the temporal locus of the US. In addition, the amplitude of the UR decreases over training, a phenomenon shown in real rabbits by Donegan (1981). During simulated extinction trials, the model produces the increase in the latency of CR onset and the diminution of the CR amplitude seen in the laboratory.

Though the current version of the SBD credibly simulates many aspects of NMR topography at optimal ISIs, there are other features of rabbit nicitating membrane conditioning which the model does not portray successfully. For example, in protocols involving very long CS durations, rabbit NM CRs begin to rise about halfway through the ISI (Smith, 1968). However, the current specification of the input trace allows the NMR to begin far earlier than that. What the model needs, then, is a mechanism specifying what Pavlov referred to as "inhibition of delay".

Preliminary simulation experiments involving systematic variation of parameters which shape x indicate inhibition of delay can be built into the model by allowing m, the parameter which determines the rise time of x, to increase over conditioning in an s-shaped function which is determined partly by the ISI and partly by the number of trials. m is allowed to maximize at .35, the default value of the current implementation, and a prerequisite for proper asymptotic behavior and topography. Since m is recomputed at every trial, x is also computed at every trial. Note that x in the current version of the SBD model is computed only once. The idea is that the input to the element is not a static entity, but rather one which varies in its efficiency according to the optimality of the ISI and the amount of exposure to the CS that the element has received.

Changing the quality of the input trace over the course of training provides a theoretical framework for generating appropriate topography during trace conditioning. Like its parent model, the

SBD model predicts trace conditioning. The model extends SB by predicting that trace conditioning yields less conditioning than forward-delay for the same ISI. These predictions are borne out in real NMR conditioning. However, the CRs of real rabbits occur in the trace interval preceding the US, while the SBD model predicts that CRs occur during the CS.

Producing CRs in the trace interval rather than during the CS might be achieved by altering assumptions regarding the onset and timecourse of x. First, we can assume that the offset of x is simply extended beyond the offset of the nominal CS, and that the decay rates of x and $\bar{x}$ are prolonged for trace conditioning. However, simply extending x is insufficient because CRs would still rise rather early during the nominal CS, and short CSs would probably not yield conditioning even when the ISI is optimal by empirical standards. Another approach is to base the computation of x not upon the nominal CS duration, but upon the ISI, a tactic not taken in the present FORTRAN implementation of the SBD model. Such an approach is supported by an early study in rabbit NMR trace conditioning, wherein a 50 ms CS was presented at ISIs ranging from 0 to 4 seconds (Smith, 1968); the present version of SBD would not even begin to show conditioning until the nominal CS duration exceeds 70 ms.

Testing of an experimental program basing the computation of a dynamic x upon the true ISI (CS onset to US onset) is now underway. Preliminary results indicate that it may indeed be possible to simulate CRs which occur during the trace interval. However, defining x based upon ISI naturally yields the same topographies and asymptotic weights for both forward-delay and trace protocols, contrary to the laboratory evidence. Furthermore, this implementation does not account for evidence indicating that the *offset* of the nominal CS and the onset of the US set up a temporal CS for the animal (Liu and Moore, 1969). This finding suggests that x ought to be generated during the trace interval. What kind of x would a trace interval produce? Can we assume that the trace interval x is of a different character from that generated by a "real" CS? Lastly, how and when during conditioning would the element come to regard the trace interval as the true CS? Future simulation experiments will address these questions.

As the preceding comments indicate, there are areas where the ability of the model to provide an accurate description of CR topography is incomplete. However, we are encouraged not only by how easily changes in x can account for these problem areas, but also by how these changes in x suggest to us what the actual "CS" can become for an animal.

Interstimulus Interval (ISI) Functions

As we have shown, the present implementation of the SBD model can, with a few exceptions, generate reasonable descriptions of within-trial events. Given this ability, how does the model fare in its description of events which occur over trials in a number of paradigms? The simplest starting place is the examination of the relative synaptic weights at asymptote for CSs of a variety of durations, the ISI function. The rabbit NMR literature describes the ISI function as an inverted U-shaped function with peak conditioning occuring to CSs of 250 ms duration (Gormezano, Kehoe and Marshall,1983). As Fig. 3 shows, the default values of the SBD model produce ISI functions for acquisition of forward-delay and trace conditioning which are generally consistent with the literature, with the exception of the prediction of negative weights for CSs of 100 ms duration. Extinction (not shown) also proceeds in appropriate fashion, with more optimal CSs extinguishing more slowly.

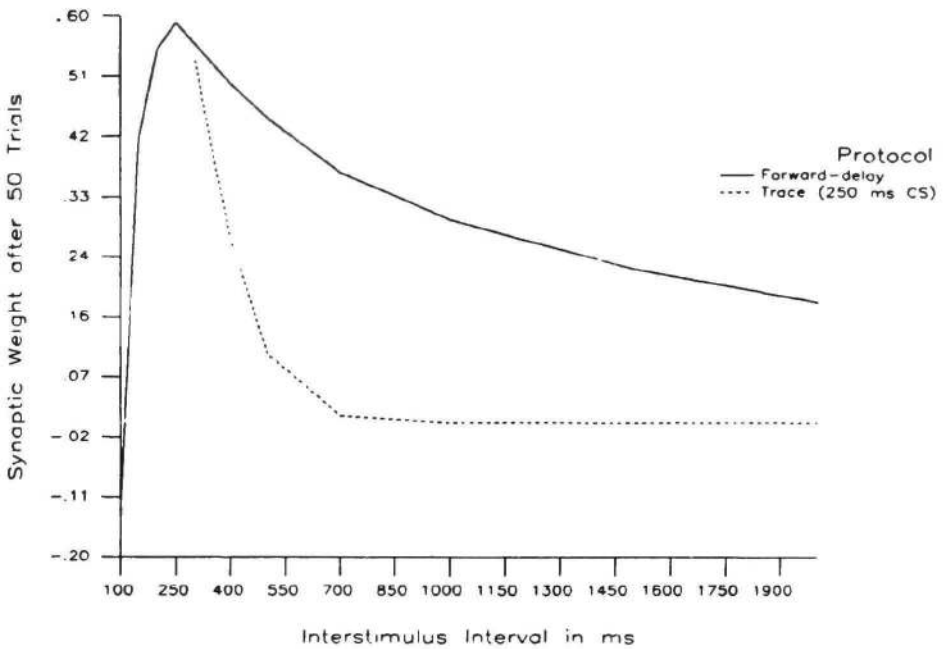

Figure 3: Synaptic weights after 50 trials for CS–US intervals ranging from 100 to 2000 ms for forward-delay conditioning and trace–conditioning protocols.

Conditioned Inhibition

The SBD model satisfactorily simulates Pavlovian conditioned inhibition (CI), a fairly difficult multiple-CS discrimination paradigm. A simulation of the CI paradigm is presented in Fig. 4. In CI, two trial types are presented in a pseudorandom sequence: the first trial type consists of CS_1 presented with a US; and the second consists of a compound of $CS_1$and CS_2 presented without the US. Fig. 4 shows that the synaptic weight for CS_1 becomes positive, while that for CS_2 becomes negative. Negative weights are interpreted as an indication of conditioned inhibition. The SBD model not only predicts CI, it also generates salient features of the paradigm including the extended number of trials required for asymptotic performance and the initial slightly excitatory character of the unreinforced compound. An unusual and untested prediction of the SB model is that the conditioned inhibitor will become excitatory if it precedes the conditioned excitor in the non-reinforced compound presentations.

As we mentioned earlier, the original Sutton-Barto learning rule is similiar to the Rescorla-Wagner model of associative learning. One of the weaknesses of both the SB and RW models is the prediction that a conditioned inhibitor will extinguish if presented alone, a prediction that has not been verified empirically. The SBD model predicts that a conditioned inhibitor presented alone will retain its inhibitory character and in this respect the model emulates the empirical evidence. Inhibition does not extinguish in the SBD model because the output of the element, s, is never less than zero. Consequently, the term $(s - \bar{s})$ in Equation 1 is also equal to zero and no changes in V for that inhibitory CS can occur in the absence of a US.

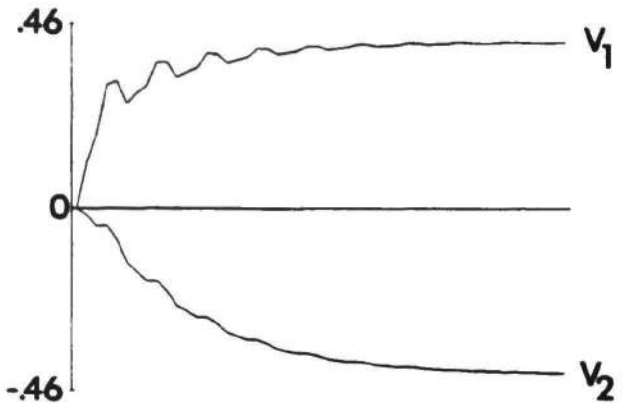

Figure 4: Synaptic weights (V_1 and V_2) for CS_1 and CS_2 for a simulated conditioned inhibition paradigm as a function of trials.See text for discussion.

Kamin Blocking

The model successfully predicts blocking of conditioning by a fully pre-trained CS to a novel added CS. Blocking is predicted by both the RW model and the original SB model. In Stage 1 of a blocking paradigm, CSB, the CS which will become the blocker, is presented with a US until an asymptotic level of responding is produced. In Stage 2, CSB is paired with an added, novel CS, denoted here as CSA. The compound of CSs A and B is reinforced. The added CS will fail to condition normally, a phenomenon demonstrated in the NMR preparation by Marchant and Moore (1973). The SBD model predicts that maximal blocking to CSA occurs when CSB is fully trained and when both CSs are presented simultaneously in Stage 2. Simulations of blocking with incompletely-trained blockers or with serial presentations of CSs A and B yield a variety of effects too numerous to describe here. However, one particularly interesting and novel prediction of both SB and SBD is that the blocker will become inhibitory in Stage 2 of a blocking paradigm if the added CS precedes and overlaps the blocking stimulus.

Higher-order conditioning

The SBD model recapitulates the success of the original model in its treatment of higher-order conditioning. In Stage 1 of a higher-order conditioning task, CS_1 is paired with a US and trained to an asymptotic level of responding. In the second stage, another CS, CS_2,is presented, followed by the fully-trained CS_1. Even though the usual US is not presented, the pairing of CSs 1 and 2 results in increased responding to CS_2. If refresher presentations of CS_1+US are not included, responding to CS_2 and eventually CS_1 falls off.

Other multiple-CS phenomena which are encompassed by the model include several untested predictions regarding within-trial timing of serial compounds. Discussion of these topics will be the focus of a future report.

CLOSING COMMENTS

Sutton and Barto (1981) recognized that their model places a heavy computational burden on a single neuron. However, they identified several possible cell-physiological mechanisms for components of the model, including the eligibility trace and the prediction of reinforcement. The SBD model places an additional burden on the neuron, in particular the computation of $\lambda'(t)$, which reduces the effectiveness of the US as associative strength increases. The justification for $\lambda'(t)$ arose from the assumptions that computations affecting synaptic weight occur not only during the CS, but after the US as well. Without the $\lambda'(t)$ rule, synaptic weights after extensive training are unreasonably low. Furthermore, the $\lambda'(t)$ rule enhances the model's performance regarding ISI functions and response topography.

There are features of classical conditioning of the rabbit NMR which the current implementation does not address. Among these are intertrial interval phenomena, stimulus salience effects, and attentional effects. Some of these phenomena can be easily encompassed within the framework of the SB and SBD models. Others, for instance, attentional phenomena, could be included, but perhaps at some cost in terms of our intuitions about the sheer number of computations that a single cell could perform.

Detailed descriptions of the behavior of the components of the SBD model and of the model's performance in a variety of simulations involving within-trial timing of stimulus events are now underway. Indeed, the strength of the model lies in its ability to generate predictions regarding within-trial events in rabbit NMR conditioning. To the neurophysiologist, such predictions provide hypotheses about the timing functions of the nervous system components involved in NM conditioning. For those working in adaptive architectures, the model's successes and failures in multiple–CS domains like compound conditioning can suggest the types of computation a single element can sustain when its inputs are assumed to model desired output. For animal learning theorists, empirical verification of the predictions of the SBD model can extend the sparse literature pertaining to within–trial events in rabbit NMR conditioning. For now, we are encouraged by the preliminary success of the model and suggest that its structures and constraints may have implications for the understanding of the physiology of learning and memory.

Acknowledgements

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