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Neural dynamics of attentionally modulated Pavlovian conditioning: blocking, interstimulus interval, and secondary reinforcement

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Selective information processing in neural networks is studied through computer simulations of Pavlovian conditioning data. The model reproduces properties of blocking, inverted-U in learning as a function of interstimulus interval, anticipatory conditioned responses, secondary reinforcement, attentional focusing by conditioned motivational feedback, and limited capacity short-term memory processing. Conditioning occurs from sensory to drive representations (conditioned reinforcer learning), from drive to sensory representations (incentive motivational learning), and from sensory to motor representations (habit learning). The conditionable pathways contain long-term memory traces that obey a non-Hebbian associative law. The neural model embodies a solution to two key design problems of conditioning, the synchronization and persistence problems. This model of vertebrate learning is compared with data and models of invertebrate learning. Predictions derived from models of vertebrate learning are compared with data about invertebrate learning, including data from *Aplysia* about facilitator neurons and data from *Hermissenda* about voltage-dependent Ca^{2+} currents. A prediction is stated about classical conditioning in all species, called the secondary conditioning alternative, and if confirmed would constitute an evolutionary invariant of learning.

I. Introduction: the Problem of Selective Information Processing

An important problem for any information processing system, whether biological or artificial, is that of limited capacity. Amid the "blooming buzzing confusion" of experience, it is necessary to be able to process some events (the most significant) and ignore others, perhaps unmasking events at a later time when their significance changes with context. Hence we must ask: How can a limited-capacity information processing system that receives a constant stream of diverse inputs be designed to selectively process those inputs that are most significant to the objectives of the system?

Classical, or Pavlovian, conditioning provides a good simplified system for studying the selective information processing problem. For this reason, it has been an increasingly active area of recent study both by neural modelers (for example, Refs. 1–8) and neurophysiologists (for example Refs. 9–12).

In particular, classical conditioning is subject to numerous attentional modulations. An example is the blocking paradigm (Fig. 1). The basic blocking paradigm[13,14] (see also Refs. 15 and 16 for many variants) is as follows. First, a neutral stimulus (CS_1), such as a tone, is presented followed at a given time interval by an unconditioned stimulus (US), such as electric shock. The CS_1–US pairing occurs several times until a conditioned response (CR) is established to the CS_1. Then a series of trials is given in which CS_1 and another neutral stimulus (CS_2), such as a light, are presented simultaneously, followed at the same time interval by the US. Finally, the CS_2 is presented alone but not reinforced. On these recall trials, no CR occurs in response to the CS_2. (Kamin found that blocking may not occur if the CS_1–CS_2 combination is associated with a different level of shock from that associated with CS_1 alone—a point to which we will return in Sec. XIII.

In the blocking paradigm, the CS_1 which is selectively attended has more motivational significance than the CS_2 which is blocked. In this article, we use computer simulations to show that the same kind of selective attention can explain some data on temporal order effects in Pavlovian conditioning. For example, the strength of a conditioned response typically depends

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1. CS_1 US

$\longrightarrow$ CR

2. $CS_1 + CS_2$ — US

$CS_2 \not\longrightarrow$ CR

Fig. 1. Blocking paradigm; the two stages of the experiment are discussed in the text.

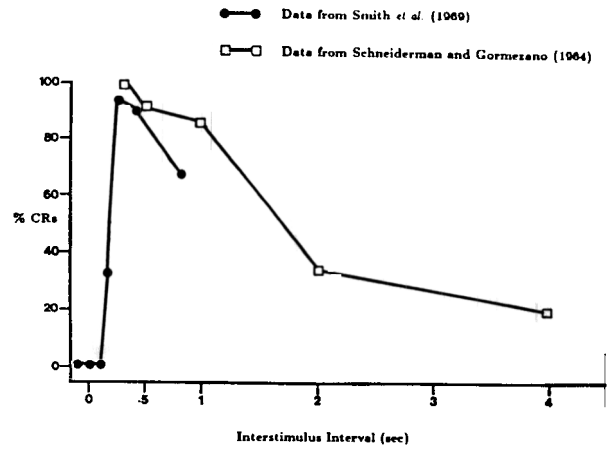

Fig. 2. Experimental relationship between conditioned response strength (measured by percentage of trials on which response occurs) and interstimulus interval in the rabbit nictitating membrane response. (From Sutton and Barto, 1981. Reprinted with permission from the American Psychological Association, Inc.)

in an inverted-U manner on the time interval, or interstimulus interval (ISI), between conditioned stimulus (CS) and unconditioned stimulus, as shown in the curve of Fig. 2. This curve is a composite of data on the nictitating membrane response of the rabbit,[17,18] where a conditioned eyeblink develops to a tone that has been paired with a puff of air to the cornea. The relationship between learning and ISI has also been studied in many other classical conditioning paradigms, such as salivation[19] and shock avoidance.[20] The length of the optimal interstimulus interval varies from one paradigm to another, and different measures are used for the strength of the response, but the qualitative relationship described in Fig. 2 holds in a variety of cases.

Our explanation of the curve of Fig. 2 is as follows. For interstimulus intervals (ISIs) that are too short, the CS and the US are processed nearly simultaneously. Since the US already has motivational significance for the organism and the CS does not, the organism selectively attends to the US and its processing of the CS is inhibited. For ISIs that are of intermediate length, this inhibition of CS processing by the US has less effect because the strength of the CS's neural representation can increase via a mechanism of short-term memory reverberation before the US is presented. For ISIs that are too long, the CS neural representation has decayed before the US is presented, because of competition from other incoming stimuli or passive decay.

In both blocking and the ISI effect, the stimulus of the pair which is selectively attended is the one that has more motivational significance. In the blocking paradigm, a CS_2 is blocked by a simultaneous CS_1 that has previously acquired reinforcing properties. In the ISI paradigm, a CS is blocked by a US. The US may be either a conditioned reinforcer, as is CS_1 in the blocking paradigm, or an unconditioned reinforcer, such as shock or the taste of food.

Thus the ISI effect can be regarded as a variant of blocking if one can understand how four types of process work: How does the pairing of CS_1 with US in the first phase of the blocking experiment endow the CS_1 cue with properties of a conditioned, or secondary, reinforcer? How do the reinforcing properties of a cue, whether primary (US) or secondary (CS_1), shift the focus of attention toward its own processing? How does the limited capacity of attentional resources arise, so that a shift of attention toward one set of cues (CS_1 or US) can prevent other cues (CS_2 or CS) from being attended? How does withdrawal of attention from a cue prevent that cue from entering into new conditioned relationships?

The present article provides a unified model, and computer simulations, of the ISI inverted-U and attentional blocking which is based on a neural model of how reinforcement, motivation, and limited capacity attention interact during classical conditioning. The model is a component of a conditioning theory that was originally developed to analyze classical and instrumental conditioning data other than the ISI curve, many of which are summarized by Grossberg.[5,6,21] The present article and that of Grossberg and Schmajuk[8] initiate a systematic program using quantitative computer simulations of conditioning phenomena to further develop this theory. The neural network architectures of these two articles combine four fundamental design principles: associative synaptic modification; competition between sensory representations; resonant feedback between two or more network levels; and opponent processing. The first three of these principles, and their implementation for the present application, will now be discussed. The fourth will be discussed at the end of this article in the context of related research.

II. Associative Synaptic Modification

Much attention has been paid recently by neural modelers, machine learning theorists, and neurophysiologists to rules for modifying connection strengths (or synaptic weights). The nodes in our neural network (and many others in the literature) may correspond either to populations of neurons or to individual neurons. In the former cases a connection between nodes

represents the average connection strength of a population of pathways. In the latter case it describes the strength of an individual synapse between neurons.

The modern era for analyzing neurophysiological correlates of Pavlovian conditioning can be dated to the well-known book of Hebb.[1] On p. 62, Hebb proposed the famous Hebb postulate: "When the axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased." The development of neural network models of conditioning since Hebb's work is discussed in detail by Levine.[22] Briefly, the hypothesis of an actual cellular growth process has largely been supplanted by other neurophysiological processes which could also alter synaptic efficacy. Mechanisms proposed have included, for example, correlated changes in the amounts of usable presynaptic transmitter substance and of postsynaptic protein synthesis;[23] changes in postsynaptic membrane receptor protein;[24] and changes in postsynaptic membrane resistance.[25] In this article we shall discuss the computational properties of a particular synaptic modification rule, without attempting to specify the biochemical mechanism leading to that rule. All the above biochemical mechanisms could generate such a formal conditioning rule.

Many learning theorists, such as Pavlov, Guthrie, Hull, and Hebb, have noted the importance of changes in a contiguity trace, habit strength, or path connection as a function of learning. The book of Hebb[1] has been an enduring source of qualitative inspiration for such an associative rule, whereby synaptic efficacy changes as a function of the correlation between presynaptic and postsynaptic activities. Such conditioning rules are often called Hebbian rules, but we do not use this term because Hebb's postulate alone provides insufficient guidance for developing a quantitative model. Hebb's pioneering contribution did not, for example, suggest a quantitative framework or equation for specifying his postulate, and his qualitative postulate, as stated above, leads to serious difficulties when implemented formally. Indeed during the two decades subsequent to Hebb's book, modelers often used information theoretic algebraic operations to define an associative learning postulate. Grossberg[23,26-28] pioneered the development of a quantitative theory of associative pattern learning within a real-time neural network. In such a network, pairing of presynaptic and postsynaptic signals occurs but is counteracted by other network effects so that Hebb's postulate is not obeyed. These non-Hebbian associative rules have been used by other neural modelers[29-33] and verified by recent mammalian neural data.[10,11,34-36]

The distinction between Hebbian and non-Hebbian associative rules is illustrated in Fig. 3. The Hebb postulate seems plausible if one assumes that the unit of associative learning is a single cell's activity whose correlation with another cell's activity can increase the synaptic strength of a pathway between the cells [Fig. 3(a)]. A different conditioning rule is needed, however, if one agrees that the unit of associative learning is a spatial pattern of activity across a network of cells [Fig. 3(b)]. Then the correlation of this spatial pattern with another cell's activity enables the set of pathways from the network to the active cell to encode the entire spatial pattern of activity into long-term memory (LTM). In this situation, a conditioning rule is needed that can encode both increases and decreases of LTM strength as a function of the pairing of cell activities. The Hebb rule, in contrast, requires that only increases in strength be caused by associative pairing.

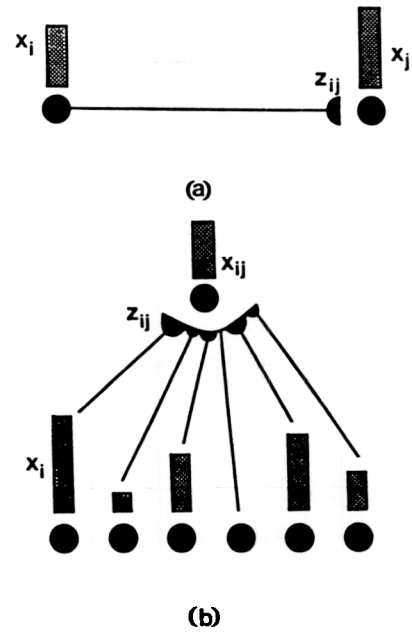

Fig. 3. Hebbian vs non-Hebbian associative rule: (a) In a Hebbian associative rule, correlation of the cell activities x_i and x_j always increases the long-term memory (LTM) trace, or synaptic strength, z_{ij} of the intervening pathway. (b) In our non-Hebbian associative rule, correlation of a spatial pattern of cell activities x_i with a cell activity x_j enables the LTM traces z_{ij} in the intervening pathways to either increase or decrease to match the spatial pattern.

In the simplest non-Hebbian associative rules, a model synapse as in Fig. 3(b) has a synaptic efficacy z that obeys a differential equation of the form

$$dz/dt = -Az + f_1(x)f_2(y), \quad (1)$$

or of the form

$$dz/dt = [-Az + f_2(y)]f_1(x), \quad (2)$$

where dz/dt denotes the time rate of change of the associative strength z, while x and y are the correlated cell activities, parameter A is a (slow) decay rate, and f_1 and f_2 are monotone nondecreasing, non-negative signal functions. Our equation therefore includes a Hebbian term $f_1(x)f_2(y)$ that increases with correlated cell activities, but Hebb's postulate is not obeyed because of the counteracting memory decay term and because of network interactions.

In the present simulations, we use Eq. (1), although similar properties derive from Eq. (2) using our nu-

merical parameters. If time is divided into discrete intervals, the difference equation form of (1) is

$$z(t+1) = (1 - A)z(t) + f_1[x(t)]f_2[y(t)]. \quad (3)$$

The signal functions f_1 and f_2 can, for example, be chosen linear above a threshold and zero below it (a threshold-linear signal function) or linear within a range but flat above and below it (a ramp or sigmoid signal function). The effects of different choices of signal functions on sensory pattern processing have been classified mathematically, including the important contrast enhancement and noise suppression properties of sigmoid signal functions.[37–39] The ramp function of Fig. 4 was used in our simulations because it has similar mathematical properties to the sigmoid but is simpler to compute.

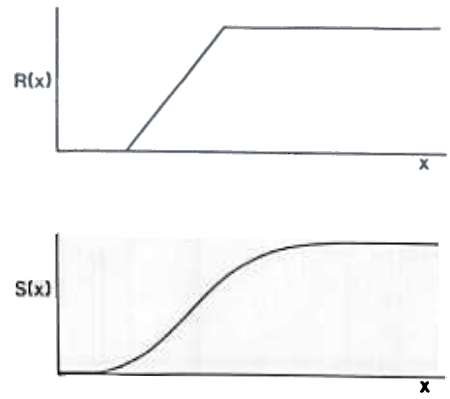

Fig. 4. Two signal functions: either a ramp function $R(x)$ or a sigmoid function $S(x)$ enables the network to suppress noise and contrast enhance the input pattern before storing it in short-term memory (STM).

III. Alternative Explanations of the ISI Effect: the Critical Role of the Conditioning Rule

Barto and Sutton[4,40] have argued that associative rules such as Eqs. (1) and (2) cannot account for the conditioning data of Fig. 2. They contended that a network with associative synapses should, to a first approximation, have an optimal ISI of zero, because cross correlation between two stimulus traces is strongest when the two stimuli occur simultaneously. To avoid this difficulty, other modelers[41,42] introduced a delay in the CS pathway that was equal to that of the optimal ISI. Hence, if the ISI between CS and US occurrences equaled the delay in the CS pathway, the CS and US signals would actually arrive simultaneously at the associative synapse between their representations. Such a delay, however, has other implications which are not easy to support. For one, delays within individual neural pathways, or axons, are typically very brief and cannot easily account for optimal ISIs that are hundreds of milliseconds long. More seriously, such a delay would also delay the conditioned response (CR) elicited in response to the CS by an equal amount, and hence is incompatible with the fact that a well-trained CR normally occurs earlier than the onset time of the US (the so-called anticipatory CR).

The alternative model simulated here reproduces both the ISI data and the anticipatory CR without invoking a long delay in the CS pathway. Such a delay is not needed to account for the ISI data, and moreover it violates symmetry properties between CS and US which are necessary for secondary conditioning to be possible. The need for such CS–US symmetry or path equivalence is discussed more fully in Refs. 2, 3, and 5.

The argument of Barto and Sutton was incomplete because their model did not incorporate certain network factors that are crucial to the model described here. These factors include the role of attention in regulating the conditioning rate, the role of reinforcing signals in shifting the focus of attention toward motivationally salient cues, and the role of inhibition between sensory representations in carrying out attention shifts by reallocating limited capacity short-term memory resources. Indeed, Barto and Sutton themselves stated (Ref. 40, p. 232): "The model clearly does not address higher order modulatory influences such as those produced by attentional or stimulus salience factors."

Because the Sutton-Barto model—and the more recent, related model of Blazis *et al.*[7]—did not incorporate attentional factors, their explanation of ISI effects and blocking relied on a particular law of synaptic modification that differs from ours. In their conditioning law, presynaptic activity is correlated with change in postsynaptic activity, rather than with postsynaptic activity itself. Other neural and psychological models which utilize some variant of this law are frequent (see, for example, Refs. 43–48). Grossberg[5] reviews a variety of context-sensitive conditioning data that such models have so far been unable to explain, primarily because of their lack of network mechanisms such as limited capacity competition and modulation of attention by motivational feedback. For example, Mackintosh[44] has stated that his own model cannot account for the fact that a more salient stimulus can block a less salient one but not vice versa, a fact that is easily explained by our model (see Sec. VII).

To understand our model, let us review the qualitative development[2,3,5,6] of the type of network shown schematically in Fig. 5. In addition to using conditionable synapses to encode associations in long-term memory (LTM), such a network uses modulatory mechanisms that were derived from three simple postulates: that CS–US associations can develop without catastrophic crosstalk even if the time lag separating the two stimuli is variable across trials; that the more we practice a task, the better we can learn it (practice makes perfect), other things being equal; and that freely attended inspection of multiple cues with different reinforcing properties does not, in itself, extinguish the reinforcing properties of these cues. Our review will provide just enough detail to define the model and explain the computer simulations. Extensive discussions of model derivations and of the large interdisciplinary data base clarifed by the model are found in the original articles.

IV. Review: the Synchronization and Persistence Problems of Classical Conditioning

Grossberg (Ref. 2, pp. 227–237) posed the problem, called the synchronization problem, of how CS–US associations can develop in a stable fashion in spite of the variability of time lag between CS and US on successive learning trials. In previous work,[49,50] each elementary sensory representation or motor command was interpreted mathematically as a spatial pattern of activation across a network of cell populations. If activity at a population coding a CS was followed repeatedly by the same US, the LTM traces activated by the CS population could cumulatively learn the spatial pattern corresponding to that US. However, if the CS was followed at different time intervals by two or more events, among which only a single US occurred, the CS-activated LTM traces would not learn the spatial pattern corresponding to the US. Instead, they would learn a mixture of the spatial patterns corresponding to all the events which occurred when the CS was active, whether meaningful to the organism or not. Such a mixture would typically encode little useful information about the environment and would certainly not resemble the US pattern.

Thus the synchronization problem brought into focus two related problems of fundamental importance: How does an organism know how to distinguish significant events for encoding in LTM among all the irrelevant environmental fluctuations that never cease to occur? How are conditioning systems designed to be capable of stably operating in continuous, or real, time, despite the fact that meaningful events, such as novel events or USs, occur at irregular and discrete time intervals?

Analysis of the synchronization problem led to the proposal that populations of cells, called D for drive representations, exist that are separate from the sensory representations of particular stimuli but are related to particular drives and emotions. Later workers have called such a drive representation an "emotion node"[51,52] or an "adaptive critic element."[53] These drive nodes are not drive reduction areas but are areas analogous to loci in emotional regions of the brain, such as the hypothalamus, where mechanisms of conditioning, reinforcement, homeostasis, and competition interact to select pathways for conditioned reinforcer learning and attentional feedback. (See Refs. 5 and 6 for further discussion of this concept.) A food US, for example, unconditionally activates the D population corresponding to the hunger drive if the hunger drive level is sufficiently high. Repeated pairing of a CS with a food US thus causes pairing of stimulation of the CS sensory representation, which we denote by S_{CS}, with that of the D representation for the hunger drive, which we denote by D_H. If the $S_{CS} \rightarrow D_H$ synapses are assumed to be modifiable according to an associative rule, the pairing $S_{CS} \rightarrow D_H$ can become strengthened, so that eventually the CS by itself will be able to activate the drive representation D_H and thereby becomes a conditioned, or secondary, reinforcer for food. Once a neutral CS (call it CS_1) has been conditioned, it can be used as a US to reinforce responses to another CS (call it CS_2) in a later experiment. Thus, after the $S_{CS_1} \rightarrow D_H$ synapses have been strengthened, repeated presentation of CS_2 followed by CS_1 can, in turn, strengthen the associative $S_{CS_2} \rightarrow D_H$ synapses.

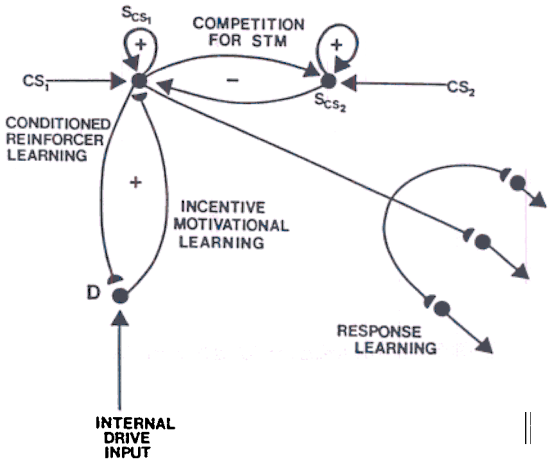

Fig. 5. Schematic conditioning circuit: Conditioned stimuli (CS_i) activate sensory representations (S_{CS_i}) which compete among themselves for limited capacity short-term memory activation and storage. The activated S_{CS_i} elicit conditionable signals to drive representations and motor command representations. Learning from an S_{CS_i} to a drive representation D is called conditioned reinforcer learning. Learning from D to an S_{CS_i} is called incentive motivational learning. Signals from D to S_{CS_i} are elicited when the combination of external sensory plus internal drive inputs is sufficiently large. In the simulations reported herein, the drive level is assumed to be large and constant.

Further study revealed that $S \rightarrow D$ conditioning must be supplemented by $D \rightarrow S$ conditioning. This became apparent through the definition and analysis of the persistence problem of classical conditioning.[3]

In Fig. 6(b), the cues CS_1 and CS_2 have previously been conditioned to responses CR_1 and CR_2. Responses CR_1 and CR_2 are assumed to be motivationally incompatible, such as eating and sex. A catastrophic problem could occur in an improperly designed learning circuit if CS_1 and CS_2 were then alternately scanned in rapid succession. If only one of the cues had previously been conditioned to a response, no difficulty would occur [Fig. 6(a)]. However, if both cues were already conditioned and if classical conditioning were merely a feed forward process which associatively links cues with simultaneously active responses, rapid cross conditioning from CS_1 to CR_2 and CS_2 to CR_1 could occur. This example identifies the core issue: When many cues are processed in parallel, and some of the cues are already conditioned to motivationally incompatible responses, why are these associations not quickly degraded by cross conditioning? How can the ubiquity of parallel cue processing be reconciled with the persistence of learned meanings?

The above paradigm identifies the persistence problem, which is also occasionally called the turkey-love fiasco, to dramatize the absurd world to which it would lead if not actively prevented. During an otherwise

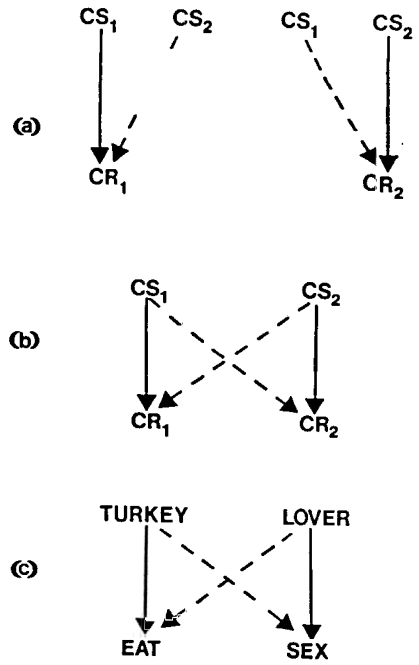

Fig. 6. Persistence problem of classical conditioning: (a) a CS_j can be quickly associated with the CR_i of a distinct CS_i (b) when each of the conditioned stimuli CS_1 and CS_2 is already conditioned to a distinct conditioned response CR_1 and CR_2, respectively, at the beginning of an experiment, alternative scanning of CS_1 and CS_2 does not always cause rapid cross conditioning of CS_1 to CR_2 and CS_2 to CR_1, as is clear by consideration of the absurd consequence depicted in (c) that would arise after dining with one's lover.

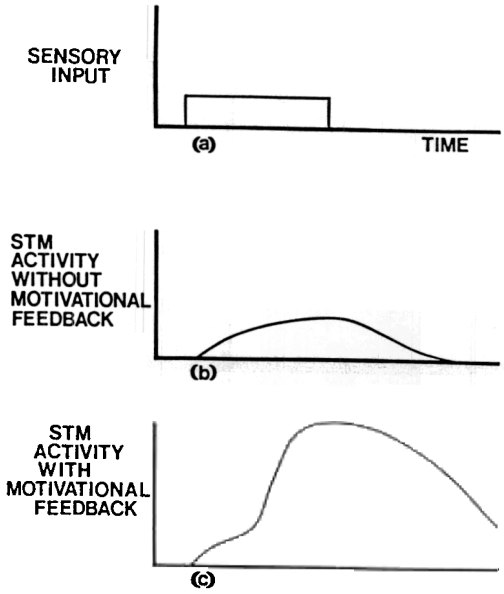

Fig. 7. Augmentation of STM activation at a sensory representation S_{CS} by feedback signaling through the pathway $S_{CS} \rightarrow D \rightarrow S_{CS}$. In response to the sensory input (a) received by S_{CS}, the STM activation profile before learning is schematized in (b). After learning within the $S_{CS} \rightarrow D \rightarrow S_{CS}$ pathway takes place, the initial activation remains as in (b). However, as the feedback signals are registered, the STM activation of S_{CS} can be greatly amplified and prolonged, as schematized in (c).

uneventful turkey dinner with one's lover, suppose that one alternately looks at lover and turkey, where lover is associated with sexual responses (among others) and turkey is associated with eating responses. Why do we not come away from dinner wanting to eat our lover and have sex with turkeys? That we do not illustrates that the persistence of learned meanings can endure despite the fact that sensory cues which are processed in parallel often control motivationally incompatible responses. Further discussion of the persistence problem is provided in Ref. 8.

The solution of the persistence problem offered in Ref. 3 led to explanations of a wide variety of difficult conditioning data, including blocking, unblocking, latent inhibition, overshadowing on a single trial, and the enhanced short-term memory (STM) encoding of novel cues.[3,5,21] More generally, this solution suggested how incentive motivational feedback due to conditionable $D \rightarrow S$ pathways could shift an organism's sensory attentional focus to process preferentially reinforcing cues and other motivationally salient cues. That is, conditioning in $S \rightarrow D$ pathways endows a sensory cue with conditioned reinforcer, or secondary reinforcing, properties. Conditioning in $D \rightarrow S$ pathways endows a sensory cue with incentive motivational properties. A sensory cue which possesses a large conditioned $S \rightarrow D \rightarrow S$ feedback pathway can quickly augment the STM activity of its sensory representation (Fig. 7). In other words, reinforcing cues can draw attention to themselves via self-generated incentive motivational feedback signals.

V. Competition Between Sensory Representations: Limited Capacity Short-Term Memory Activity

The sensory representations which emit conditioned reinforcer signals and receive incentive motivational signals also compete among themselves for a limited capacity STM resource. The ubiquitous occurrence of limited capacity STM was traced[3,38,54] to a more basic processing requirement: the ability of cell networks to process spatially distributed input patterns without irreparably distorting these input patterns due to either internal noise or saturation effects. This noise-saturation dilemma can be prevented by an on-center off-surround anatomy through which the cells interact via mass action (or shunting) laws (Fig. 8). Within a robust parameter range, the off-surround competition of such a network interaction implies that the total suprathreshold activation of the network tends to be conserved, and thus that the network has a limited capacity.

When a shunting on-center off-surround network is also designed to accomplish STM storage, its on-center off-surround interactions are recurrent, or feedback, interactions in which the nodes excite themselves and inhibit other nodes via feedback pathways [Fig. 8(b)]. In addition to its noise-saturation and limited capacity properties, such a recurrent on-center off-surround network contrast enhances an input pattern before storing the contrast-enhanced activation pattern

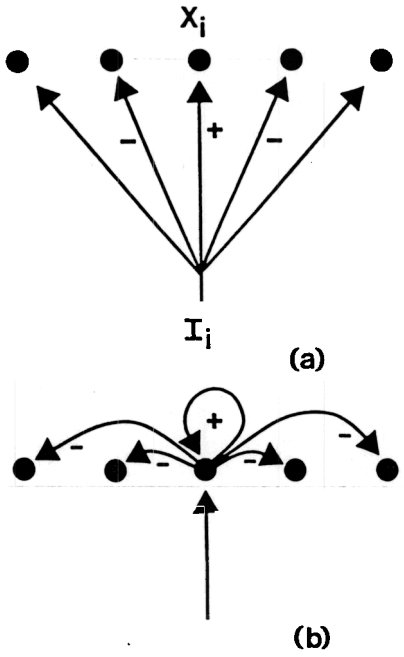

Fig. 8. Noise-saturation dilemma is solved by mass action on-center off-surround networks. (a) A feed forward, or nonrecurrent, network with inputs I_i activating and inhibiting STM activities x_j. (b) In a feedback, or recurrent, network excitatory and inhibitory signals are distributed among the network cells.

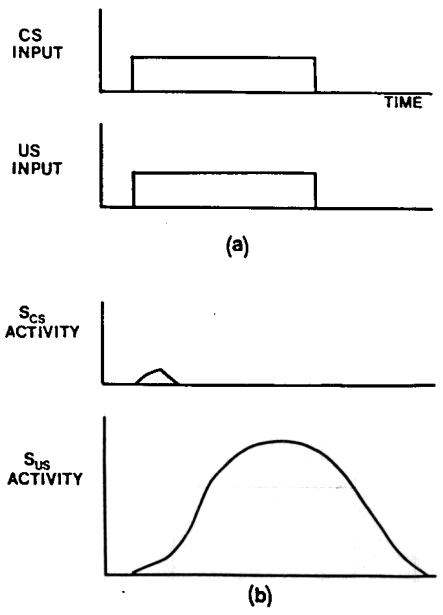

Fig. 9. Schematic of STM activation at zero ISI: (a) simultaneous onset of CS and US; (b) rapid amplification of S_{US} by $S_{US} \rightarrow D \rightarrow S_{US}$ feedback signals enables S_{US} to quickly inhibit the S_{CS} representation.

which emerges across the cells in STM (also called working memory). Thus one must distinguish between the input pattern and the more focal STM activity pattern that it generates. Attention is paid to those sensory representations whose cells receive a positive level of *stored* STM activity.

When incentive motivational feedback signals form part of the total input pattern to the sensory representations (Fig. 7), these signals can bias the competition for STM activity toward motivationally salient cues. Due to the limited capacity of STM, primary and secondary reinforcers can draw attention to themselves via their strong conditioned $S \rightarrow D \rightarrow S$ feedback loops. To initiate such an attention shift, such cues must first start to be processed due to their sensory properties. After sensory processing is initiated, it can activate the learned reinforcing $S \rightarrow D$ and motivating $D \rightarrow S$ pathways of the cues, and can thereby help to direct the ultimate allocation of sensory and attentional resources.

Once attention shifts away from a sensory represenation, its activity can become subthreshold. In both Eqs. (1) and (2), a subthreshold activity x or y prevents new growth of associative strength. In particular, if $f_1(x) = 0$ in Eq. (2), then $dz/dt = 0$, so that no associative change whatsoever can occur. If $f_2(y) = 0$ in Eq. (1) or (2), then $dz/dt \leq 0$, so that no increase of associative strength can occur.

VI. Effects of ISI on Conditioning: Inverted-U

These mechanisms are sufficient to describe the relationship which we claim to exist between attentional blocking and the ISI effect. To explain the inverted-U as a function of ISI, consider first a CS and a US that are simultaneously presented. Each of these sensory cues will initially receive less STM activity than either one presented separately, due to the competition that they elicit for limited capacity STM resources. The US can nonetheless quickly activate a strong $S_{US} \rightarrow D \rightarrow S_{US}$ feedback pathway. Delivery of this large positive feedback signal to S_{US} signifies that the US is a motivationally important cue. Due to this large feedback signal, the STM activity of the sensory representation S_{US} is amplified as attention is drawn to S_{US} (Fig. 9). As a result of competition for STM resources [Fig. 8(b)], the sensory representation S_{CS} of the CS is quickly inhibited before it can elicit significant conditioning in the $S_{CS} \rightarrow D$ conditioned reinforcer pathway or the $D \rightarrow S_{CS}$ incentive motivational pathway (Fig. 9).

In contrast, suppose that CS onset precedes US onset by a duration sufficient to enable the CS to generate, in the absence of US competition, a fully developed STM activation of its sensory representation S_{CS} (Fig. 10). When the US then does occur, it must from the outset compete for STM activity with an active S_{CS} representation. The initial activation of S_{US} therefore proceeds less vigorously than during simultaneous CS–US presentation. Consequently, the activation of the feedback loop $S_{US} \rightarrow D \rightarrow S_{US}$ also builds up more slowly. Throughout the time interval when the S_{US} is activating the $S_{US} \rightarrow D \rightarrow S_{US}$ feedback pathway, growing competition from S_{US} to S_{CS} begins to develop. However, the S_{CS} is also sending the S_{US} large competitive signals during this interval due to its large STM activity when the US is presented. All these

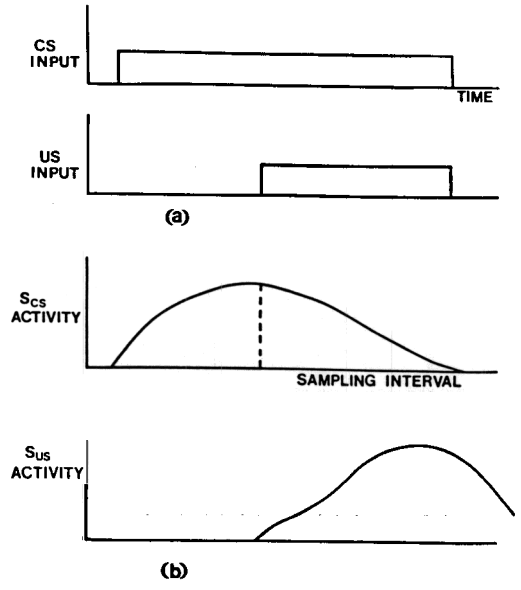

Fig. 10 Schematic of STM activation at positive ISI: (a) CS is presented before US onset; (b) large S_{CS} activation prior to US onset enables the S_{CS} to drive learning during the sampling interval after US onset before the S_{US} can inhibit the S_{CS}.

factors conspire to enable the S_{CS} to remain intensely active for a time interval after US onset. Throughout this time interval, large sampling signals in the $S_{CS} \rightarrow D$ pathway and the $D \rightarrow S_{CS}$ pathway enable the LTM traces in these pathways to grow.

Finally, suppose that the CS occurs so long before the US that S_{CS} is already inactive before the drive representation D gets activated by the US. Then no conditioning can occur within the $S_{CS} \rightarrow D$ pathway and the $D \rightarrow S_{US}$ pathway, so that the CS does not acquire reinforcing or motivating properties.

These considerations suggest how incentive motivational feedback from a drive representation D can give rise to the ISI inverted-U. In other words, the ISI effect is a consequence of the feedback mechanisms which identify a US sensory cue as a motivationally salient, and therefore differentially attended, event. The argument above can now be readily applied to stimulus-response, or S-R, conditioning (Fig. 5). An inverted-U in S-R conditioning is also explained if the conditioning rate is a function of S_{CS} activity, as it is in the associative Eqs. (1) and (2).

VII. Effects of ISI on Conditioning: Blocking, Secondary Conditioning, Anticipatory CRs, Overshadowing

One remark now suffices to relate our explanation of the ISI inverted-U to phenomena of attentional blocking and secondary conditioning. In the above explanation, simply replace CS by CS_2 and US by CS_1. In a typical blocking experiment, CS_1 is paired with a US until conditioning enables CS_1 to control a strong feedback pathway $S_{CS_1} \rightarrow D \rightarrow S_{CS_1}$. Such conditioning operationally defines CS_1 as a secondary, or conditioned, reinforcer that can draw attention to itself. Later simultaneous presentation of CS_1 and CS_2 can now be seen to be mechanistically similar to simultaneous presentation of a CS and a US. Simultaneous CS–US presentation in an ISI paradigm prevents conditioning to the CS due to a combination of large $S_{US} \rightarrow D \rightarrow S_{US}$ feedback and $S_{US} \rightarrow S_{CS}$ competition which quickly drives the activity of S_{CS} below threshold. After CS_1 becomes a conditioned reinforcer in a blocking paradigm, simultaneous presentation of CS_1 and CS_2 prevents conditioning to CS_2 due to a combination of large S_{CS_1} feedback and $S_{CS_1} \rightarrow S_{CS_2}$ competition which quickly drives the activity of S_{CS_2} below threshold.

In summary, our explanation of secondary conditioning enables us to show how suppression of CS conditioning during zero ISI and the blocking of a CS by a US can both be caused by the same mechanisms. The existence of anticipatory CRs is also easily explained within the network of Fig. 5. The pathways $S \rightarrow D$, $D \rightarrow S$, and $S \rightarrow R$ do not create significant delays in the network. The times needed for STM activities to grow, for the competition between sensory representations to take effect, and for the firing thresholds in $D \rightarrow S$ pathways to be exceeded by $S \rightarrow D$ reinforcing inputs are the rate-limiting times within the network. As the LTM traces within the $S \rightarrow D$, $D \rightarrow S$, $S \rightarrow R$ pathways grow larger, the STM activity of S_{CS} can grow more quickly and can more quickly and strongly read out a CR via the $S \rightarrow R$ pathway, thereby leading to an anticipatory CR.

The unified explanation of ISI, blocking, and anticipatory CRs depends critically on the manner in which attention can be shifted by conditioned $S \rightarrow D \rightarrow S$ feedback. Attentional factors are particularly important for a thorough understanding of blocking. Mackintosh[44] has, for example, stated that his own model cannot account for the fact that a more salient stimulus can block a less salient one, but not vice versa. Since our model of blocking is based on a limited capacity competitive interaction between stimulus representations, it can easily account for this fact. A similar argument suggests how overshadowing occurs;[16] that is, how a more intense stimulus can dominate learning when it is presented with a less intense stimulus. Any factor which enables the sensory representation of a cue to better compete for limited capacity STM resources increases its chance to emit effective learning signals to the drive and motor representations of the network.

VIII. Quantitative Model

The networks that we have simulated are depicted in Fig. 11. The network of Fig. 11 includes on-center off-surround competition between sensory representations for two CSs (with activities x_{11} and x_{21}) and a single US (with activity x_{31}), all projecting to the same drive representation D with activity y via conditioned reinforcer pathways with LTM strengths z_{11}, z_{21}, and z_{31}, respectively. In Fig. 5, the drive representation D projects back to the same sensory representations via incentive motivational pathways that possess their own LTM strengths. The sensory representations also project via conditionable sensory-motor pathways

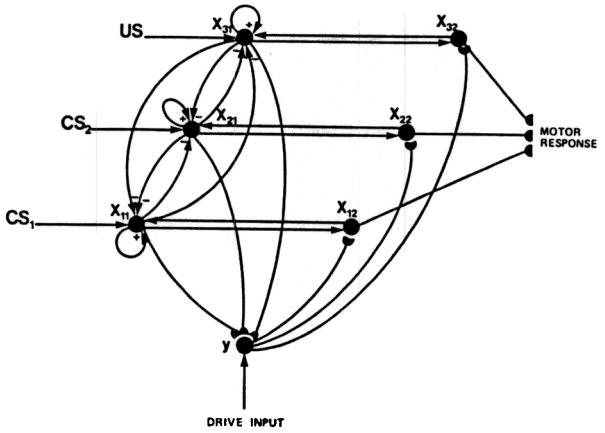

Fig. 11. Simulated network. Each sensory representation possesses two stages with STM activities x_{i1} and x_{i2}, respectively. A CS or US input activates its corresponding x_{i1}. Activation of x_{i1} generates unconditionable signals to x_{i2} and conditioned reinforcer signals to D, whose activity is denoted by y. Conditionable incentive motivational feedback signals from D activate the second stage potentials x_{i2}, which then deliver feedback signals to x_{i1}. Motor learning is elicited by sensory-motor signals from the x_{i2} to the motor command representations. Long-term memory traces are designated by semidisks at the ends of conditionable pathways.

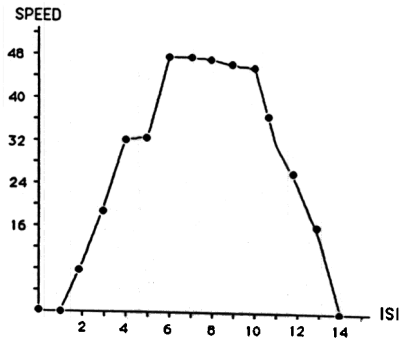

Fig. 12. Plot of CR acquisition speed as a function of ISI. This speed was computed by the formula 100 × (number of time units per trial)/(number of time units to first CR).

to representations of motor commands. Figure 11 describes a variant of Fig. 5 (Refs. 3 and 5) in which the sensory representations are divided into two successive stages. The activity x_{i1} of the ith first stage can activate conditioned reinforcer pathways, whereas the activity x_{i2} of the ith second stage receives conditioned incentive motivational pathways from D. The x_{i2} loci, in turn, project back to the x_{i1}, thereby closing the $S \rightarrow D \rightarrow S$ feedback loop, and also project forward to the motor representations.

An additional mechanism was used to regulate STM decay of the sensory representations of CSs and USs. Short-term memory can be weakened through time due to habituation, competition from other incoming stimuli, and nonspecific gain changes that occur, for example, when attention shifts to a different modality. (See Refs. 8 and 55–58 for theories and simulations incorporating some of these effects.) These multiple influences on STM decay are replaced here by a simple rule which suffices for our present purposes. We assume that the self-excitatory feedback term that maintains STM storage is multiplied by a factor that equals 1 for a short time after the initiation of STM activation and decays exponentially thereafter. The self-excitation term is also supplemented by feedback due to x_{i2}, so that STM decay is slower for motivationally significant stimuli.

IX. Computer Simulation Results

Figures 12–14 show some representative computer simulations of the ISI effects using the network in Fig. 11. Various measures of the strength of conditioning were plotted against the ISI, all other parameters being equal. The first measure is the speed of CR acquisition, as indicated by the reciprocal of the number of trials to the first conditioned response. To generate this figure, a conditioned response was said to occur if the STM trace y of D was activated by the CS representation x_{11} in the absence of the US.

The curve of Fig. 12 shows that the speed of CR acquisition relates to ISI in a manner that is qualitatively compatible with the experimental data of Gormezano and his co-workers[17,18] on the nictitating membrane response. For ISIs of 1 time unit or less, the competition from the US representation x_{31} prevented the CS activity x_{11} from staying above the threshold in the $S_{CS} \rightarrow D$ pathway long enough while y at D was being activated by the US for the associative strength z_{11} to increase appreciably. At long ISIs, the decay of the STM trace x_{11} prevented z_{11} from sensing the later large values of y at D.

In Fig. 13, the STM activities of S_{CS}, S_{US}, and D (x_{11}, x_{31}, and y) and the LTM trace z_{11} of the $S_{CS} \rightarrow D$ pathway are plotted in real time given a choice of ISI = 6 that led to good learning in Fig. 12. Although the US [Fig. 13(b)] suppressed the x_{11} variable [Fig. 13(a)] after activating the y variable [Fig. 13(c)], the LTM trace z_{11} correlated positive x_{11} and y values well enough to achieve an S-shaped cumulative learning curve across trials [Fig. 13(d)]. As the LTM trace z_{11} grew, the CS elicited a progressively larger STM reaction x_{11} across trials due to the increasing size of the positive feedback signal which it generated in the $S_{CS} \rightarrow D \rightarrow S_{CS}$ pathway [Fig. 13(a)]. This reduced the STM activity x_{31} of the US [Fig. 13(b)] due to competition between CS and US sensory representations. Such a small decrement in the sensory activity of S_{US} during conditioning is an, as yet, untested prediction of the model. Figure 14 plots the asymptotic, or maximal, value of z_{11} as a function of ISI. An inverted-U function obtained even after many learning trials.

Figure 15 illustrates a computer simulation of a blocking experiment using the same parameters. The STM activities of the CS_1 and CS_2 representations (x_{11} and x_{21}) and the LTM traces of the $S_{CS} \rightarrow D$ pathway (z_{11} and z_{21}) were plotted in real time. Pairing of CS_1 with a delayed US enabled the LTM trace z_{11} to achieve a classical S-shaped learning curve [Fig. 15(c)]. After CS_1 became a conditioned reinforcer, it enhanced its own STM storage via x_{11} by generating a

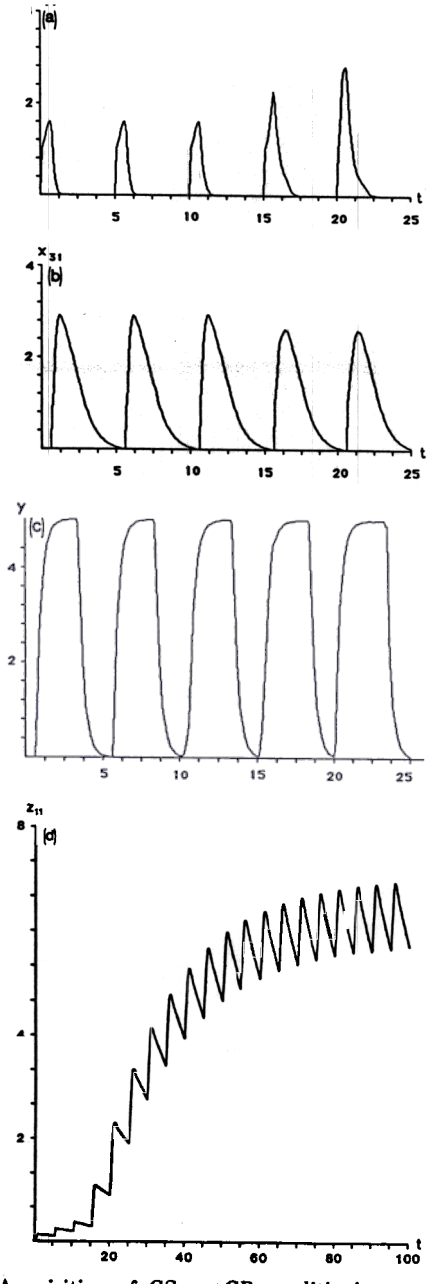

Fig. 13. Acquisition of $CS_1 - CR$ conditioning at a favorable ISI(=6): (a) plot of CS_1 activity x_{11} through time over five trials during which CS_1 is paired with US; (b) plot of US activity x_{31} through time over five trials; (c) plot of D activity y through time over five trials; As CS_1 becomes a conditioned reinforcer, it activates y before the US occurs; (d) plot of $CS_1 \rightarrow D$ LTM trace z_{11} over twenty trials during which CS_1 is paired with US.

large $S_{CS_1} \rightarrow D \rightarrow S_{CS_1}$ feedback signal [Fig. 15(a)]. As a result, when CS_1 and CS_2 were simultaneously presented, the STM activity x_{21} of S_{CS_2} [Fig. 15(b)] was suppressed by competition from x_{11}. Consequently, the LTM trace z_{21} [Fig. 15(d)] could not grow, and the CS_2 could not learn to elicit a CR.

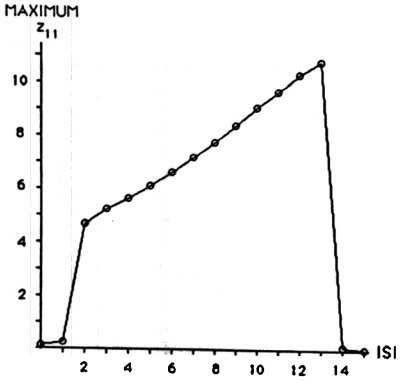

Fig. 14. Plot of maximal z_{11} over twenty paired trials as a function of ISI.

X. Comparison with Invertebrate Learning: an Evolutionary Invariant of Associative Learning?

Much discussion has recently focused on the relationships which may exist between vertebrate and invertebrate learning circuits to identify possible evolutionary invariants of associative learning. Hawkins and Kandel[59] have described neural data and a conditioning model based on studies of the invertebrate *Aplysia*. We compare their model with the model that we have here used to simulate mammalian learning data.

The Hawkins and Kandel[59] model was described from data reported in Refs. 60 and 61. These data suggested that each US activates a facilitator neuron that influences each pathway activated by a CS. Only when a CS and a US can simultaneously activate a CS pathway and a facilitator neuron can the LTM trace in the CS pathway grow.

The functional similarity between a facilitator neuron and a $D \rightarrow S$ incentive-motivational pathway (Fig. 5) was used in Ref. 59 to suggest a possible explanation of secondary conditioning and blocking much like our own.[2,3,5] The existence of a conditionable pathway from the CS to the facilitator neuron was postulated that is analogous to the $S \rightarrow D$ conditioned reinforcer pathway. This scheme was suggested as a possible model of secondary conditioning and blocking in *Aplysia* as well as higher organisms.

Despite these qualitative similarities between the two models, the model suggested in Ref. 59 cannot explain either secondary conditioning or blocking and is inconsistent with the data reported in Ref. 60. The analysis leading to these conclusions is provided below. In addition, we state an organizational principle, called the secondary conditioning alternative, that is consistent with these data, and that is predicted to hold across all species, both vertebrate and invertebrate.[62] If confirmed, the alternative would constitute an evolutionary invariant of associative learning.

The hypothesis that a facilitator neuron mediates conditioning in *Aplysia* was based on data which showed that "the US produces substantially more facilitation of the synaptic potential from the sensory neuron to a motor neuron than if the US is not paired

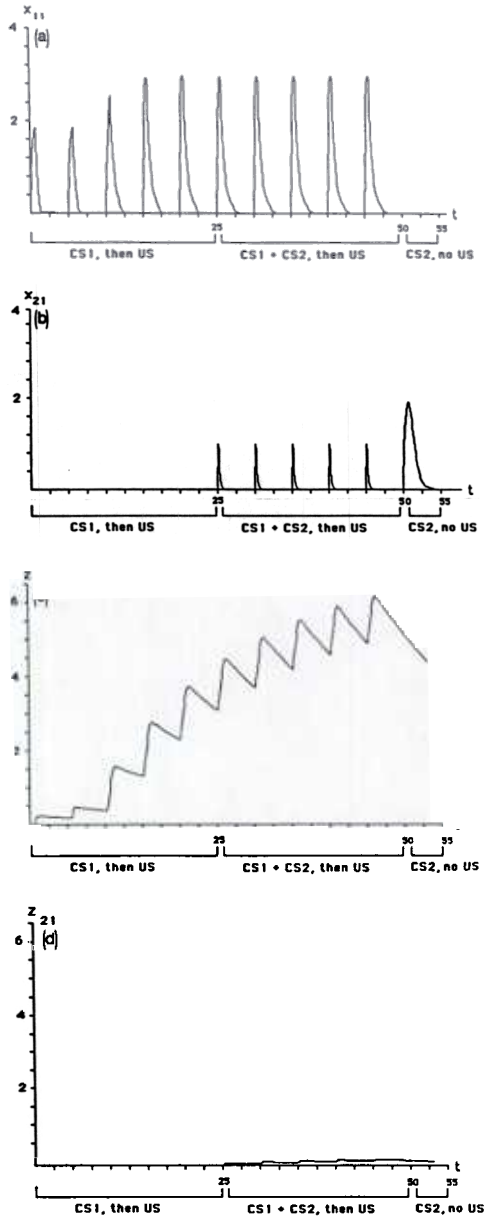

Fig. 15. Blocking simulation: in (a)–(d), the ISI = 6 between CS_1 and US onset. Five trials of CS_1–US pairing are followed by five trials of (CS_1 + CS_2)–US pairing. Then CS_2 is presented alone for one trial: (a) Activity x_{11} of S_{CS_1} through time; (b) activity x_{21} of S_{CS_2} through time; (c) LTM trace z_{11} from S_{CS_1} to D through time; (d) LTM trace z_{21} from S_{CS_2} to D through time. Conditioning of z_{21} is blocked by prior conditioning of z_{11}.

with activity in the sensory neuron" (Ref. 59, p. 379). However, Hawkins *et al.* (Ref. 60, p. 403) reported that "paired presentation of the CS and the US produced no more total activation of the facilitators than did unpaired presentation." These *Aplysia* data are inconsistent with the existence of a conditionable pathway from the CS to the facilitator neuron, and the circuits derived directly from the data in Refs. 60 and 61 did not contain such a pathway.

To reconcile these *Aplysia* experiments with data about vertebrate conditioning, it was suggested in Ref. 62 that an anatomical difference may exist between the conditioning circuits of certain invertebrates and vertebrates, and that this difference would influence the ability of different species to undergo secondary conditioning and to experience motivationally biased attention shifts. The following statement summarizes this possible difference in circuitry in a way that can be tested across all species.

The Secondary Conditioning Alternative: Either a neural system is incapable of secondary conditioning, or a CS will cause increased total firing of its facilitator neuron (read $D \rightarrow S$ pathway) as CS–US pairing continues.

The secondary conditioning alternative suggests that a modest modification of an invertebrate conditioning circuit, namely, adding on a conditionable $S \rightarrow D$ pathway, can permit the circuit to undergo secondary conditioning.

XI. Properties of an Invertebrate Conditioning Model

Hawkins and Kandel (Ref. 59, p. 385) suggested that blocking is due to the postulated property that "the output of the facilitator neurons decreases when they are stimulated continuously." Thus after a CS_1 is paired with a US on a number of trials, subsequent presentation of a compound stimulus $CS_1 + CS_2$ with a US would not condition CS_2 because the facilitator neuron could not fire adequately. Unfortunately, this explanation is incompatible with the phenomenon of unblocking, which is the counterpoint to blocking in vertebrates, as well as with the phenomenon of secondary conditioning.

This hypothesis cannot explain unblocking for the following reason. Blocking of a CS_2 occurs if a compound stimulus $CS_1 + CS_2$ occurs prior to the same US that was paired with CS_1. However, unblocking of CS_2 is observed if a compound cue $CS_1 + CS_2$ occurs prior to either a less or more intense US than did the previous CS_1 (Refs. 13 and 14); that is, in these conditions, the CS_2 can become conditioned to the subsequent US. If the facilitator neuron is fatigued by the previous US, it cannot enable a CS_2 which occurs prior to a different US to become conditioned for the same reason that it cannot allow such a CS_2 to become conditioned if it occurs prior to the same US.

Secondary conditioning cannot be explained for a similar reason. After a CS_1 becomes well enough conditioned to act as a US, it cannot activate the facilitator neuron during a subsequent time interval because the facilitator neuron has become depressed due to previous activation by the US. Consequently, pairing a CS_2 with the conditioned reinforcer CS_1 does not enable the CS_2 to become conditioned, and secondary conditioning does not occur.

XII. Modulation of Conditioning by a Ca^{2+} Current

Confirmation of the secondary conditioning alternative would provide an example of an evolutionary invariant of associative learning, while clarifying the role that variations and specializations of anatomical circuitry may play in endowing some species with a more

sophisticated repertoire of conditionable skills than others. Evolutionary invariants of associative learning have also been identified on the more microscopic level of biochemical learning mechanisms. For example, Alkon and his colleagues have studied the anatomy, physiology, and biochemistry of an associative learning circuit in the nudibranch mollusc *Hermissenda crassicornis*.[63-70] These experiments have identified postsynaptic membrane channels that mediate an association which is learned by *Hermissenda* when light (the CS) and rotation (the US) are paired.[68] The *Hermissenda* data thus support the existence of an associative rule, such as Eq. (1) or (2), wherein both presynaptic and postsynaptic influences are required during the learning process. In this learning situation, a sustained voltage-dependent inward Ca^{2+} current inactivates an outward K^+ current, thereby causing enhanced depolarization of the cell membrane and further inward flow of Ca^{2+}. In addition, the level of Ca^{2+} –dependent phosphorylation of specific cell proteins changes only in the cells of conditioned animals. Vertebrate conditioning studies are reviewed in Ref. 69 in which a voltage-dependent inward Ca^{2+} current is again implicated as a mediator of more long-lasting cellular changes due to learning.

These experiments support and significantly refine a prediction[23,26] (see also Ref. 71, Chap. 3) whose goal was to biochemically interpret the associative rules (1) and (2). The prediction was based on a comparison of these associative rules, which were derived from mammalian conditioning data, with the fragmentary biochemical evidence that was available in the 1960s. The prediction suggested that an inward Ca^{2+} current is synergetic with an inward Na^+ current during associative learning. This prediction was based on an analysis which suggested that an inward ionic current, other than Na^+, was needed which could act synergetically with the inward Na^+ current and the outward K^+ current that were well known to occur during cell depolarization. A functional analysis suggested that this extra ionic current should be able to accumulate intracellularly and thereby trigger more permanent associative changes. Based on what was known in the 1960s about biochemical regulation by synergetic ionic currents, an inward Ca^{2+} current was selected as the most likely candidate for the predicted current, and an increase in an inward Na^+ current was identified as a likely synergist. A decrease in an outward K^+ current can also augment cell depolarization, however, and that possibility is the one which conditioning models and recent data have supported.

This early prediction illustrates a convergence of models for vertebrate and invertebrate associative learning that has recently emerged, and highlights the way in which conditioning models, vertebrate behavioral studies, and invertebrate biochemical studies can complement and strengthen each other during their shared search for evolutionary invariants.

Not all studies of invertebrate conditioning have identified a postsynaptic influence on the site of adaptive biochemical change. *Aplysia* data[60,61] suggest that a US uses its facilitator neuron to activate a Ca^{2+} current which acts directly on the presynaptic terminals of a CS-activated pathway without postsynaptic mediation. A special feature of the relatively simple *Aplysia* circuitry may clarify the absence of a postsynaptic influence on presynaptic conditioning in this organism. In this *Aplysia* circuit, direct prewired pathways exist between sensory and motor neurons. Only a modest number of sensory pathways converge on each motor neuron. A postsynaptic influence on presynaptic conditioning is not functionally mandated in this type of simple circuit. A postsynaptic influence becomes more useful if a large number of neurons converge on each target cell. In such a circuit, a $D \rightarrow S$ pathway which acted presynaptically would have to send a separate signal to every sensory pathway converging on a target cell. It seems to be much simpler, other things being equal, to send a single $D \rightarrow S$ pathway to the target cell and to let the target cell communicate a postsynaptic learning signal—possibly in the form of a Ca^{2+} current—to all the synaptic terminals which converge on it.

In some learning situations it is, in principle, impossible to use exclusively presynaptic conditioning mechanisms. For example, to self-organize a spatial map from one sensory field to another, postsynaptic competition mediates the learning at interfield synapses.[28,31,32,55,56,71-74] A number of model circuits for mammalian adaptive sensory-motor control have also been proposed in which a presynaptic modulatory signal, such as a Ca^{2+} current, regulates a postsynaptic-to-presynaptic learning signal.[75] Thus the anatomies of specialized learning circuits may vary widely across neural systems and species, but the associative rules, such as Eqs. (1) and (2), and their biochemical substrates may be much more universal.

XIII. Gated Dipole Opponent Processing and Adaptive Resonance Cognitive Processing

The qualitative arguments and quantitative computer simulations reported herein show how blocking, overshadowing, nonzero optimal ISI, anticipatory conditioned responses, secondary reinforcement, attentional focusing by conditioned motivational feedback, and limited capacity short-term memory processing can be explained as emergent properties of a neural network model of Pavlovian conditioning. This model uses a type of associative learning law for which neurophysiological data have recently been reported,[10-12,34-36] thereby providing direct support for an early prediction of neural network theory.[26,28,49,50]

The interactive circuitry of the model, as in Figs. 5 and 11, is just as important as its microscopic cellular laws for explaining these data and their mutual relationships. In particular, the nonlinear positive feedback interactions between the model's distinct network levels—the sensory representations S and drive representations D—and the competitive interactions within each level S and D, respectively, constitute the first adaptive resonance theory (ART) circuit to have been modeled.[3] For simplicity, the present article has

omitted a number of circuit interactions that are not necessary to treat the data simulated herein, but that play a major role in the modeling of other conditioning phenomena.

This paper does not, for example, analyze opponent positive and negative drive representations. A model of positive and negative opponent interactions, called a gated dipole model,[58,3,5,21] was developed to explain how termination of a negative or aversive stimulus, such as an electric shock, can be positively reinforcing. The property that the sudden termination of a negative input can generate an internal positive reaction is called a temporal contrast or antagonistic rebound effect.

Many conditioning theorists[4,40,43,44,46] have modeled such contrast phenomena in terms of single synapse whose associative strength is a function of the change in the activity of that synapse's postsynaptic cell. Such a learning rule cannot, however, account for several important sets of data; for example, the amount of positive reinforcement associated with shock termination depends on the shock's duration; cutting shock level in half can be less reinforcing than shutting off a shock of half the size; a sudden increase in shock can be more punishing than a gradual increase; and reinforcement is an inverted-U function of arousal. All these phenomena can be explained in terms of emergent properties of a gated dipole circuit.[58,21]

Although the use of a gated dipole circuit to build up a drive representation expands the explanatory range of a conditioning theory, many conditioning data cannot be explained without the inclusion of cognitive mechanisms, notably the attentional and orienting mechanisms that regulate the learning of sensory and cognitive codes and expectations.[56,76] Conditioning data were in fact, a primary source of design constraints leading to the discovery of adaptive resonance theory.[5,21] Such an expanded conditioning theory can be viewed as an adaptive resonance theory in which two specialized types of ART circuit interact: a sensory-cognitive circuit and a cognitive-reinforcement circuit. In this expanded theory, which represents a computational synthesis of sensory, cognitive, learning, reinforcement, and homeostatic mechanisms, a very large body of conditioning data—including such subtle phenomena as unblocking and dishabituation due to novel cues—has been analyzed and predicted.[5,8,21,77,78] This paper and Ref. 8 together begin a new phase in the development of this conditioning theory by initiating a systematic program of parametric computer simulations whose goal is to explain finer quantitative details of behavioral conditioning data and of the functional anatomy and dynamics of their generative brain regions.

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Appendix: Simulated Equations and Parameters

The computer simulations reported in this paper are based on the network depicted in Fig. 11. The STM variables (x_{i1}, x_{i2}, and y) and LTM variables (z_{i1}) are intuitively described in Sec. VIII.

The equations for x_{i1} describe an on-center off-surround network undergoing mass action, or shunting, feedback interactions. In particular, x_{i1} obeys an equation of the form

$$\frac{d}{dt}x_{i1} = -Ax_{i1} + (B - x_{i1})I_{i1} - x_{i1}J_{i1}, \tag{A1}$$

$i = 1, 2, 3$. In Eq. (A1), the term $-Ax_{i1}$ represents passive decay of STM. The constant B is the maximum possible activity of each variable x_{i1}. If B is interpreted as the number of sites (cells or membrane patches) capable of being excited, $B - x_{i1}$ represents the number of inactive sites that can be excited, whereas x_{i1} represents the number of active sites that can be inhibited. Terms I_{i1} and J_{i1} are the total excitatory and inhibitory inputs, respectively, that influence x_{i1}. The term $(B - x_{i1})\, I_{i1}$ says that inactive sites are activated by I_{i1} via mass action. The term $-x_{i1}\, J_{i1}$ says that active sites are inhibited by J_{i1} via mass action.

The total excitatory input is a sum of an external CS or UCS activated signal, plus a positive feedback signal from x_{i1} to itself, plus a positive feedback signal from x_{i2} to x_{i1}. All feedback signal functions in the simulations are ramp functions (Fig. 4), which are the simplest type of sigmoid signal functions. A ramp function is defined in terms of parameters α and β by

$$R(x;\alpha,\beta) = \begin{cases} 0, & x \le \alpha, \\ x - \alpha, & \alpha < x \le \beta, \\ \beta - \alpha, & \beta < x \end{cases}. \tag{A2}$$

Parameter α is the threshold and parameter β is the saturation point of the signal function. When possibly distinct sets α_i,β_i of parameters are used, we write

$$R_i(x) = R(x;\alpha_i,\beta_i) \tag{A3}$$

for simplicity.

We assume, in addition, that the positive feedback signal from x_{i1} to itself triggers a process of habituation that steadily attenuates the net size of the feedback signal. For simplicity, we model this habituative process as an exponentially decaying function of time, rather than as a habituating transmitter gate. Thus

$$I_{i1} = I_i + C[R_1(x_{i1}) + R_2(x_{i2})] \exp(-D[t - T_i - E]^+), \tag{A4}$$

where $C, D, E, \alpha_i,\beta_i$ are positive constants, I_i is the ith externally activated input, T_i is the onset time of I_i after each input presentation, and $[w]^+ = \max(\omega,0)$.

The total inhibitory input J_{i1} is a sum of inhibitory feedback signals activated by the other activities x_{j1}. Thus

$$J_{i1} = F\sum_{j \ne i} R_1(x_{j1}). \tag{A5}$$

In all

$$\frac{d}{dt}x_{i1} = -Ax_{i1} + (B - x_{i1})\{I_i + C[R_1(x_{i1}) + R_2(x_{i2}$$

$$\times \exp(-D[t - T_i - E]^+\} - Fx_{i1}\sum_{j\neq i} R_1(x_{j1}), \qquad \text{(A6)}$$

$i = 1, 2, 3.$

We assume that each activity x_{i1} reads out a signal $GR_1(x_{i1})$ toward the drive representation D, where G, is a positive constant. This signal is gated by the LTM trace z_{i1} before the net LTM-gated signal influences D. The drive representation D is activated only if the total input due to all x_{i1} exceeds a threshold α_3. Thus we let

$$\frac{d}{dt}y = -Hy + KR_3\left[\sum_{j=1}^{3} GR_1(x_{j1})z_{j1}\right]. \qquad \text{(A7)}$$

Simultaneous signals from x_{i1} and y are needed to activate x_{i2}. The signal from y is gated by an LTM trace, which is assumed for simplicity to equal z_{i1} due to the fact that x_{i1} and x_{i2} are both activated at similar times during the conditioning process. We also assume, for simplicity, that these inputs do not drive x_{i2} into its saturation range. In all, we let

$$\frac{d}{dt}x_{i2} = -Lx_{i2} + MR_4(x_{i1})R_5(y)z_{i1}, \qquad \text{(A8)}$$

$i = 1,2,3.$

Finally, each LTM trace corresponding to a CS is assumed to obey a law such as Eq. (1):

$$\frac{d}{dt}z_{i1} = -Nz_{i1} + PR_1(x_{i1})R_6(y), \qquad \text{(A9)}$$

$i = 1, 2.$ The LTM trace corresponding to the UCS was assumed to be constant:

$$z_{31} = Q. \qquad \text{(A10)}$$

The following numerical parameters were used in the reported simulations. Several other sets of parameters were identified which are capable of generating the same qualitative results: $A = 2, B = 4, C = 2, D = 1.5, E = 0.4, F = 4, G = 4, H = 3, K = 10, L = 3, M = 10, N = 0.05, P = 1.25, Q = 10; \alpha_1 = 0.5, \beta_1 = 2, \alpha_2 = 0.2, \beta_2 = 2, \alpha_3 = 0.5, \beta_3 = 2, \alpha_4 = 0.25, \beta_4 = 2, \alpha_5 = 0.05, \beta_5 = 1, \alpha_6 = 0.5, \beta_6 = 1.5.$

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