

A neuronal network for the logic of Limax learning

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Abstract We construct a neuronal network to model the logic of associative conditioning as revealed in experimental results using the terrestrial mollusk *Limax maximus*. We show, in particular, how blocking to a previously conditioned stimulus in the presence of the unconditional stimulus, can emerge as a dynamical property of the network. We also propose experiments to test the new model.

Keywords Limax · Associative conditioning · Logic of learning behavior · Neuronal network

1. Introduction

The synaptic substrates for computing the logic operations that enable higher order conditioning phenomena such as the Kamin blocking effect (Kamin, 1969) have not yet been identified (Hawkins, 1989; Fanselow, 1998; Jones and Gonzalez-Lima, 2001; Fanselow and Poulos, 2005). The search for these synaptic substrates is facilitated by the fact that the associative synaptic machinery to implement higher order conditioning phenomena such as second order conditioning, compound conditioning, overshadowing and blocking has proven to be present in both vertebrate and invertebrate nervous systems (Sahley et al., 1981b; Sekiguchi et al., 1999; Hosler and Smith, 2000; Brembs and Heisenberg, 2001; Brembs et al., 2004) but see (Farley et al., 2004; Guerrieri et al., 2005). An important step toward identification of synaptic mechanisms for higher order conditioning in a particular nervous system is the construction of a biologically plausible model that performs the relevant associative

computations and makes testable predictions. This is the goal of the present work.

The molluscan nervous system has proven to be fertile ground for illuminating the synaptic basis of several forms of associative conditioning (Balaban, 2002; Roberts and Glanzman, 2003; Antzoulatos and Byrne, 2004; Crow, 2004; Fulton et al., 2005; Kirino et al., 2005; McComb et al., 2005). These analyses have in some cases allowed identification of unique interneurons playing a causal role in acquisition or expression of a conditioned response (Balaban et al., 2004; Korneev et al., 2005; Sangha et al., 2005). These detailed cellular analyses (Crow, 2004) may allow experiments aimed at discriminating among the major classes of associative learning models for Pavlovian conditioning (Hawkins, 1989; Vogel et al., 2004).

The terrestrial mollusk *Limax maximus* exhibits a variety of higher-order conditioning phenomena within its olfactory information processing system, including second-order conditioning, blocking and a US pre-exposure effect (Sahley et al., 1981a, b; Sahley et al., 1990). *Limax* show both aversive and appetitive conditioning to odors (Gelperin, 1999) and multiple forms of memory storage (Sekiguchi et al., 1997). Early attempts to model *Limax* odor conditioning (Gelperin et al., 1986, 1989) led to predictions regarding stimulus conditions promoting either configural or elemental processing of odor mixtures. These predictions were borne out by subsequent experimental measurements (Hopfield and Gelperin, 1989). Given the more complete information currently available on the behavioral analysis of *Limax* odor learning (Sekiguchi et al., 1994; Teyke et al., 2000), the *Limax* odor processing network (Sakura et al., 2004; Gelperin, 2006; Kirino et al., 2005) and identification of several unique behavioral control neurons (Delaney and Gelperin, 1990b; Shimozono et al., 2001), it is appropriate to revisit models for the logic of *Limax* learning.

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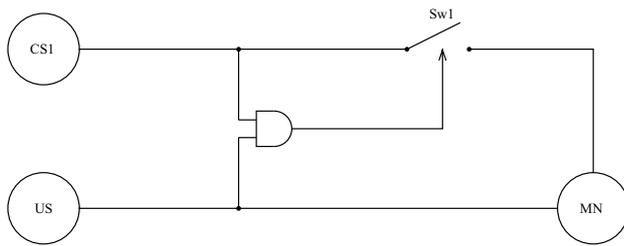


Fig. 1 The logic of first-order conditioning

2. A logic diagram expressing learning behavior

The logic of first order conditioning can be captured in the circuit shown in Fig. 1. We represent the inputs, Unconditioned Stimulus and Conditioned Stimulus, by US and CS1, respectively. A motor response, to the US prior to conditioning, to a combination of the US and CS1 during conditioning, and to the CS1 after conditioning, is reflected in the output, MN.

1. Figure 1 is used to represent first-order conditioning in the following way:

- (a) Prior to conditioning, the switch, Sw1 is assumed to be open. Thus, an input to the US activates the MN, but an input to CS1 does not.
- (b) The condition for the closure of Sw1 is: the input to Sw1 should be active. The output of an AND logic gate receiving input from CS1 and US is required to activate the switch. This means that unless both CS1 and US are active together, the switch remains open. Once the switch is closed, we assume that it remains closed, irreversibly. We do not deal with mechanisms for extinction in this model. Thus, if CS1 is presented simultaneously with the US, the CS1 acquires the ability to activate the MN independently.

2. Figure 2 shows second-order conditioning:

- (a) Once CS1 has been conditioned via association with US, Sw1 is closed i.e. either US or CS1 can independently activate the MN.
- (b) The logic of input states leading to the condition under which Sw2 is closed is $(CS1 \text{ XOR } US) \text{ AND } CS2$. This is because, in order to close Sw2, CS2 must be active together with either US or CS1, but not both. The case of CS2 paired with US is the usual first-order conditioning, as above. Similarly, CS2 can also be conditioned by association with a previously conditioned CS1 (second-order conditioning). The third case—CS2 active simultaneously with US and CS1—results in blocking: the switch Sw2 is not closed, and CS2 remains unconditioned. (The “trivial” case of the absence of CS2 altogether can be verified easily.)

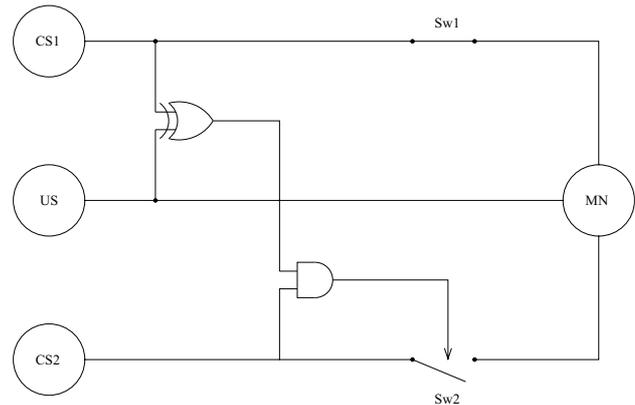


Fig. 2 The logic of second-order conditioning

We next show a neuronal architecture that implements the logic operations for the conditioning operations described above.

3. A neuronal architecture for implementing the learning logic

Even as co-activation between US and CS1 facilitates an association between CS1 and the motor output, there are other consequences of the CS1-US pairing that determine whether other CS's will be conditioned when subsequently paired with CS1. The preceding analysis, especially of second-order conditioning, suggests that there is an element in the neuronal architecture to perform an XOR transformation of the US and CS1 inputs during association with CS2.

One possibility is that there is an element for which both US and CS1 are inputs, and the XOR computation is performed by this element in a feed-forward way. This raises the question whether this element existed prior to first-order conditioning, or was created during the conditioning process. It is less satisfying to assume the existence of this “XOR element” ad hoc; rather, we confront how the XOR operation may be performed.

We present here a second, intriguing, possibility: we admit an intermediary facilitatory neuron (FN), but the XOR operation itself is “distributed” in the network. We claim that the FN exists “from the beginning” and is integral to each of the conditioning processes; an XOR operation emerges from the dynamical properties of the neurons and the connections that develop during conditioning. We will develop below the rules that define the evolution of the connectivities that are compatible with this learning behavior.

A neuronal architecture that coherently implements the logic of higher-order conditioning behavior is shown in Fig. 3. Excitatory connections are indicated by circles and inhibitory ones by short dashes. Prior to conditioning, with the circuit in its “naive” state, the animal only responds to

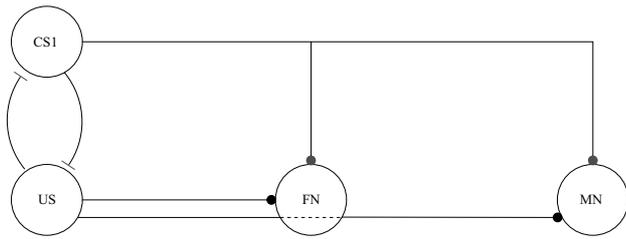


Fig. 3 Circuitry for the development of first-order conditioning in the neuronal network

the US. We indicate the connections to implement this in black. The synapses shown in red indicate that they are the ones whose connection strengths are modified during conditioning trials. Thus, before any conditioning, the CS1 is unassociated with this stimulus-response system, i.e. all of the synapses shown in red have zero or very low strength. The only effective connections are excitatory synapses from the US onto MN (responsible for the unconditioned response), and onto a facilitatory neuron, whose properties are revealed in higher-order conditioning.

We do not ascribe any further properties to the FN at this stage; although it is plausible that the FN may be involved in influencing the US-MN connection. We will examine this point in future work.

We first indicate the development of first-order conditioning:

The central claim here is that activating the FN results in strengthening all synapses coactive with it, via heterosynaptic facilitation, as studied extensively in *Aplysia* (Phares and Byrne, 2005; Sherff and Carew, 2004; Pittenger and Kandel, 2003). Thus, for example, co-activating the US with the CS1 results in strengthening of the naive synapse from the CS1 to MN. We model the FN neuron after dopaminergic (Baimoukhametova et al., 2004) or serotonergic (Udo et al., 2005; Marinesco et al., 2004a, b) neurons: activation of the neuron releases a neurotransmitter such as dopamine or serotonin which results in the strengthening of all the synapses active at that time. In the simulations presented below, this strengthening takes place only when the FN is firing.

The CS1 input neuron forms connections with all parts of the circuitry mediating the unconditioned response (US-FN-MN). Excitatory connections strengthen onto the FN and MN, but also, reciprocal inhibitory connections strengthen between CS1 and US. The significance of the excitatory connections is clear: if during first order conditioning CS1 is to acquire properties similar to the US so as to become effective in conditioning a CS2, then one might expect the connections activated by CS1 after first order conditioning are present in the US-FN-MN pathway. This may be interpreted as the CS1 acquiring the conditioning characteristics of the US. While it is reasonable that the CS1 also establish some relationship with the US, the full significance of the reciprocal in-

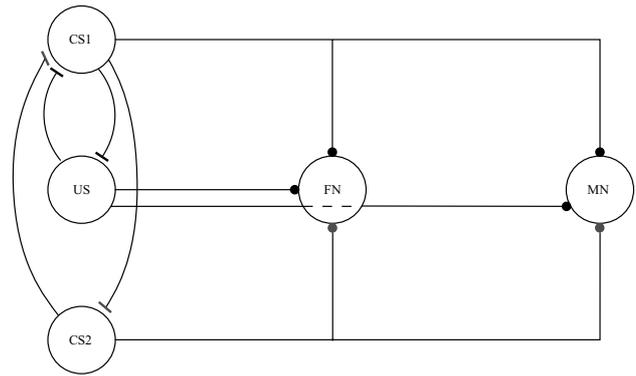


Fig. 4 Connections mediating the development of second-order conditioning in the neuronal network

hibitory connections will emerge when we examine blocking below.

Second-order conditioning: once a CS1 has been conditioned, pairing it with CS2 conditions CS2. It is easy to see why this is: CS1 engages FN, and FN firing is sufficient to strengthen synapses from the (coactive) CS2 onto MN and FN, as outlined above. Of course, CS2 can also be conditioned to a US. This is shown in Fig. 4, for ease of illustration: notice that the connections that develop significant strength depend on the history of presentation of pairing trials.

The most interesting case to consider is when CS1 and US are active together with CS2. In this case, we hypothesize that the inhibitory synapses between the CS1 and US suppress activity mutually, and their inputs into the FN cannot integrate sufficiently to depolarize FN above threshold. Thus the condition that explains blocking is: FN does not fire when US and CS1 are active together, so that CS2 remains unconditioned.

4. Simulations

4.1. First-order conditioning

Figure 5 shows responses of several elements in the “naive” circuit. Stimulating the CS1—in the absence of stimulating the US—does not produce any output: both the FN (facilitatory neuron), and the MN (motor neuron) do not fire.

Figure 6 shows conditioning progressing when both CS1 and US are stimulated together. Neurons CS1 and US were coactivated (see Appendix) repeatedly at intervals of 500 ms. The top row in the figure shows an early episode in conditioning, the middle row curves are after two CS1-US pairings. Notice that as conditioning progresses, reciprocal inhibitory synapses between the US and CS1 begin to strengthen (mediated by the firing in the FN). Thus the burst durations in CS1 and US are shorter, and firing in the FN is reduced. Bottom

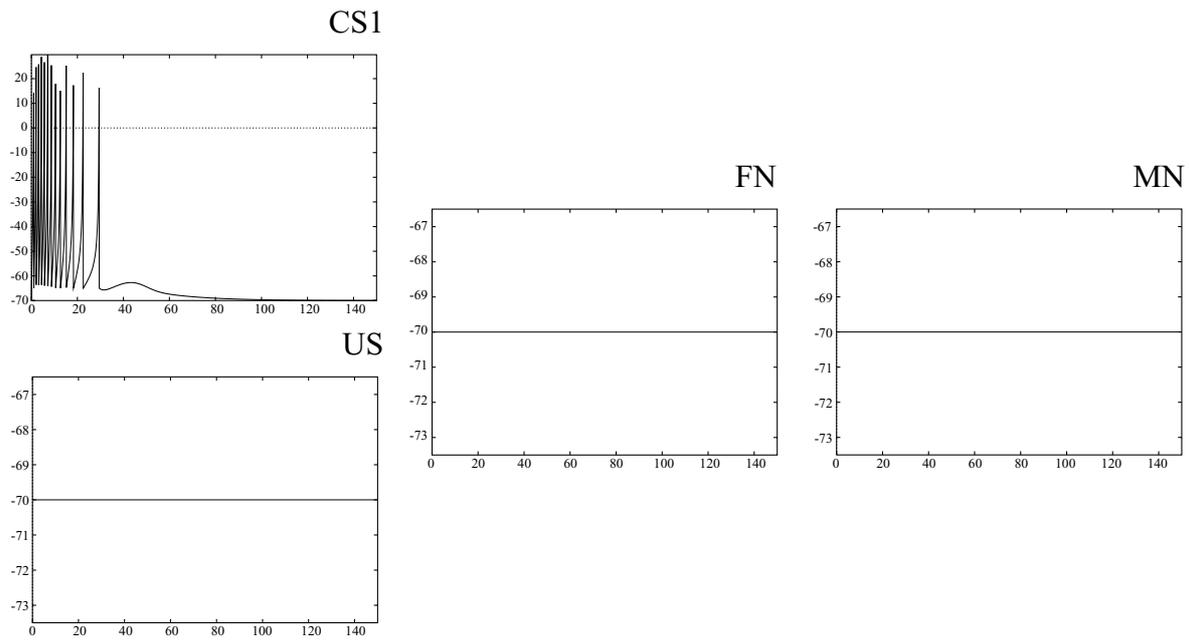


Fig. 5 Responses in the circuit before conditioning

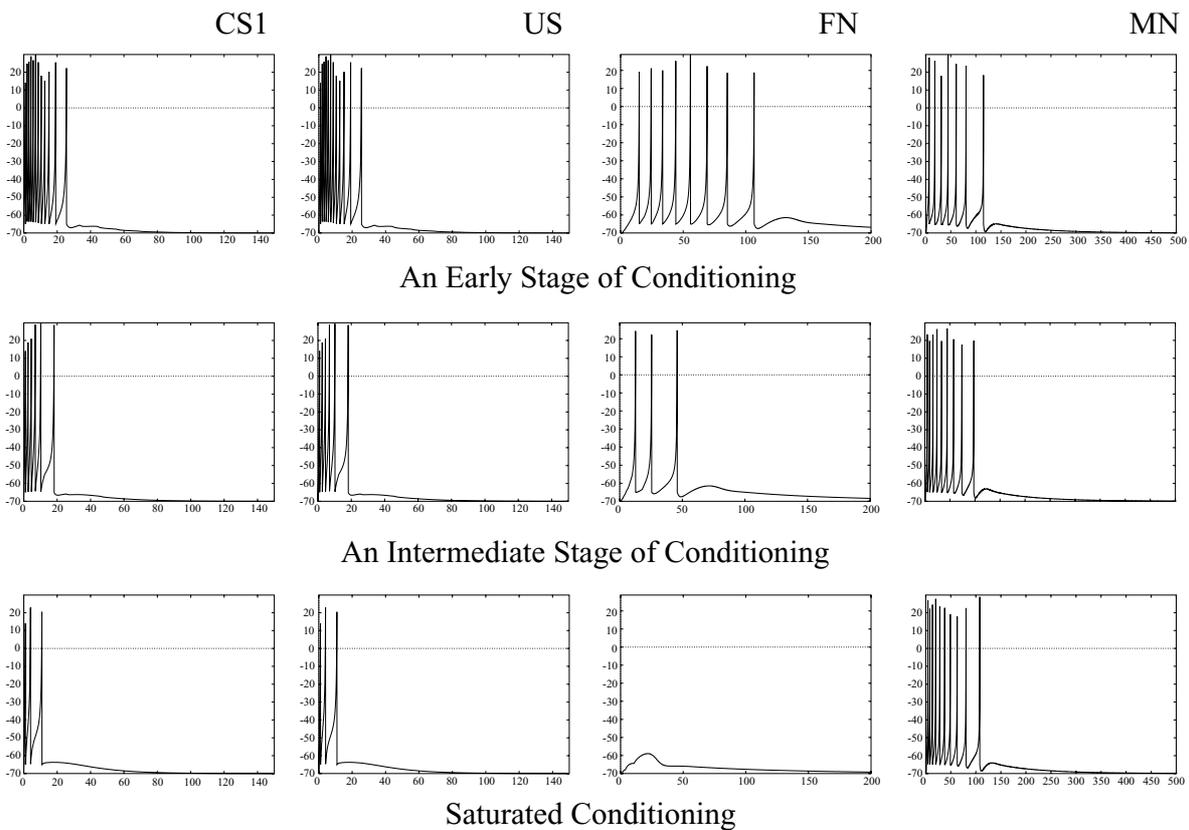


Fig. 6 Activity in four circuit elements at three stages of conditioning

row: upon seven more repetitions of CS1-US pairings, CS1 and US fire very briefly, and the FN is silent; conditioning is saturated.

Notice that the MN continues to respond throughout. Moreover, during the time that the FN fires, the strength of the CS1-MN synapse also increases.

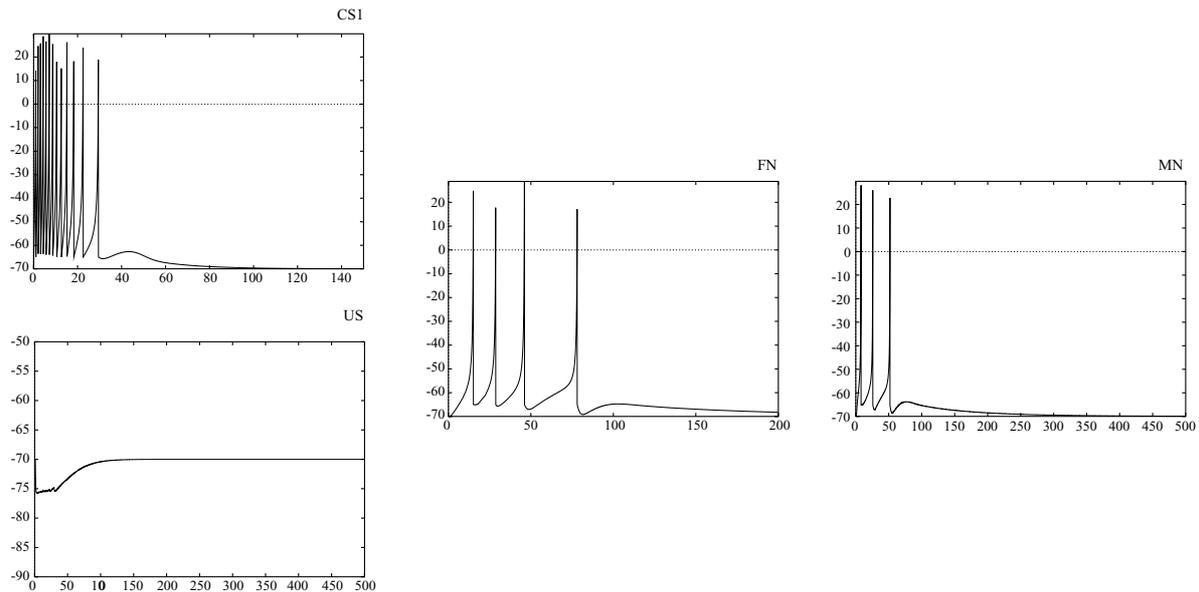


Fig. 7 Responses in the circuit once the CS1 has been conditioned

Post-conditioning: Fig. 7 shows, once again, the response of the MN to stimulating the CS1 alone. Now, the MN fires in response to a CS1 input .

Thus, these results show that:

1. If the US is not present, CS1 is not conditioned.
2. If the US is present, then FN fires, and subsequently strengthens the synapses from CS1 to the motor neuron and FN, and inhibitory connections between CS1 and US.
3. Once well-conditioned, the CS1 can evoke firing in the FN and MN independently.

4.2. Second-order conditioning

Figure 8 shows second order conditioning: a previously conditioned CS1 is paired with CS2 for several trials (three of these are shown in the red, blue, and green curves). Only CS1 and CS2 are active, US is silent. Here, the role of the US as the conditioning element is acquired by the CS1. Similar to the process of CS1-US association, as CS1-CS2 conditioning progresses, synapses from CS2 onto the FN and MN are strengthened, as well as the inhibitory CS1-CS2 synapses. Note that even as reciprocal inhibition strengths between the CS1 and CS2 upon successive trials, firing in the facilitator neuron diminishes.

Also notice, even though the US is inactive, CS1 firing induces an inhibitory response in the US cell. This is due to the earlier phase of first-order conditioning, when association formation between US and CS1 also resulted in inhibitory connections being strengthened between them.

Following second order conditioning, CS2 alone is able to evoke a response in the MN: as shown in Fig. 9. Notice that

activating the CS2 evokes a response in CS1 (from strengthened inhibitory connections), but not in US due to absence of any association formation between the two.

4.3. Blocking

To demonstrate blocking, we present a previously conditioned CS1 with a naive CS2, simultaneously with the US (Fig. 10). Despite repeated applications of these three stimuli, CS2 fails to be conditioned. Notice that the FN is not firing at all—an indication that the conditioning between CS1 and US is saturated. And, since firing in FN is necessary to strengthen any synapses, CS2 never acquires the capacity to evoke a response in either FN or MN, as seen in Fig. 11.

5. Discussion

In this work we propose neuronal circuitry to explain blocking to second-order stimuli in the Limax. The model is organized through the experimentally observed logic of the Limax conditioning behavior. We first propose a schematic circuit that captures these logic relationships succinctly. Such a logic diagram, however, does not indicate what mechanistic processes may underlie these logic functions, nor what circuit is capable of performing it. We therefore propose a neuronal circuit which implements this logic. Using reduced models of neurons and synaptic facilitation, we indicate the plasticity that is consistent with the desired behavior of this network. We demonstrate, through simulations, that strengthening excitatory and inhibitory connection throughout the circuit mediates association formation, including second-order

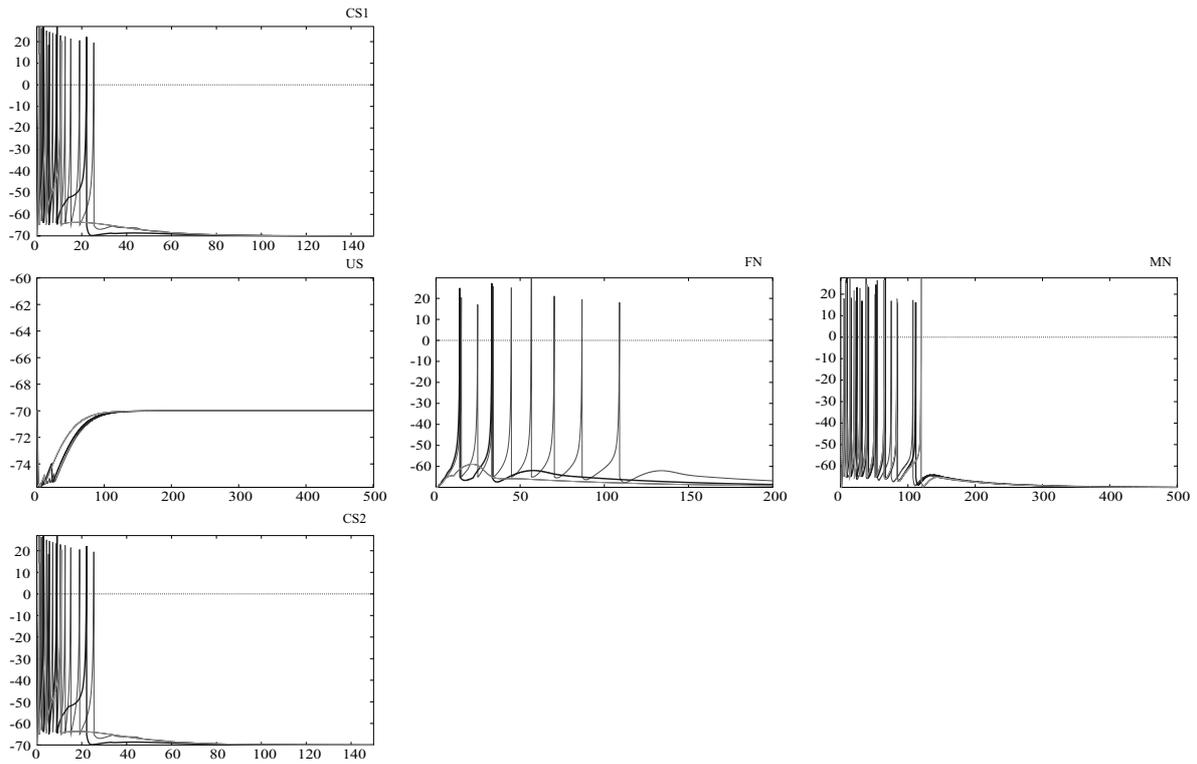


Fig. 8 Conditioning of CS2 to CS1

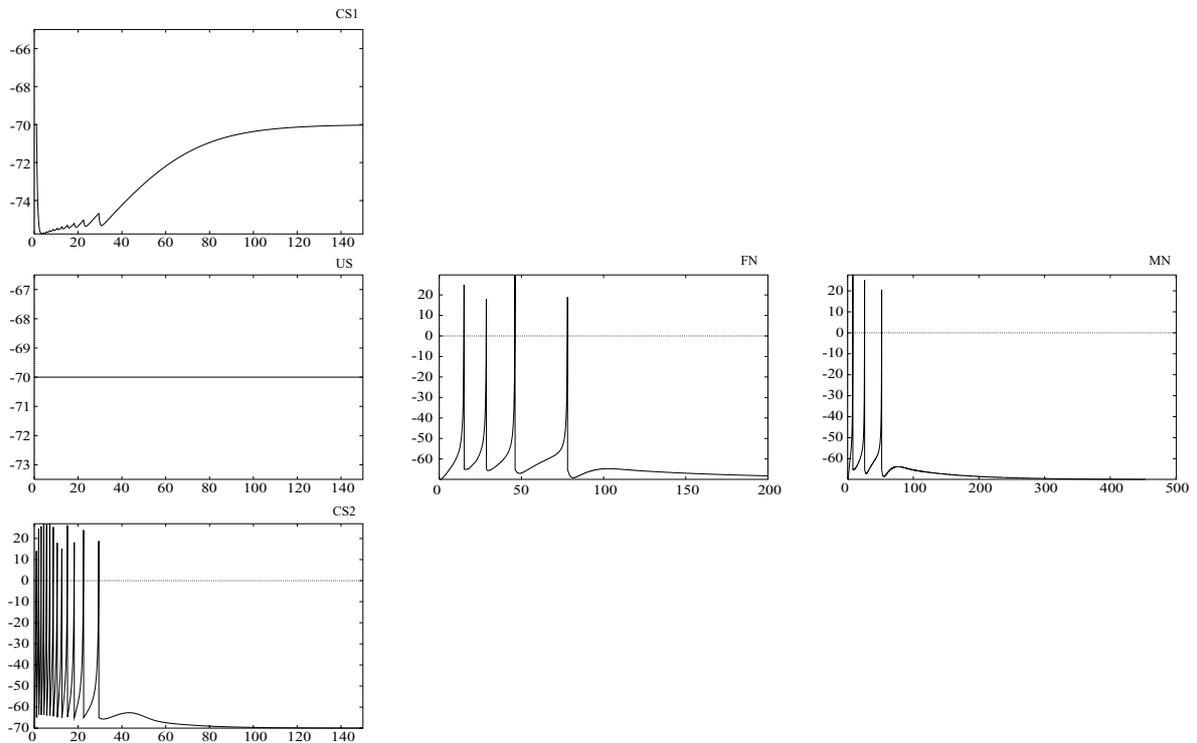


Fig. 9 Response to a conditioned CS2

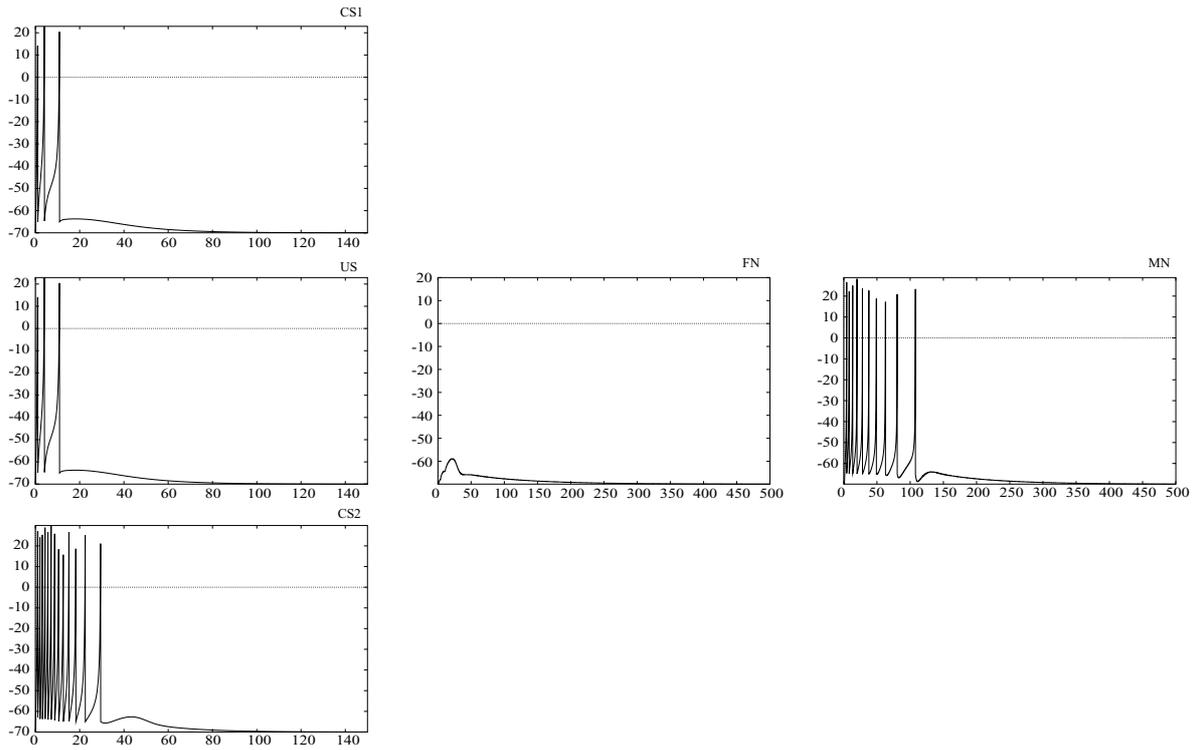


Fig. 10 US + CS1 blocks CS2 conditioning

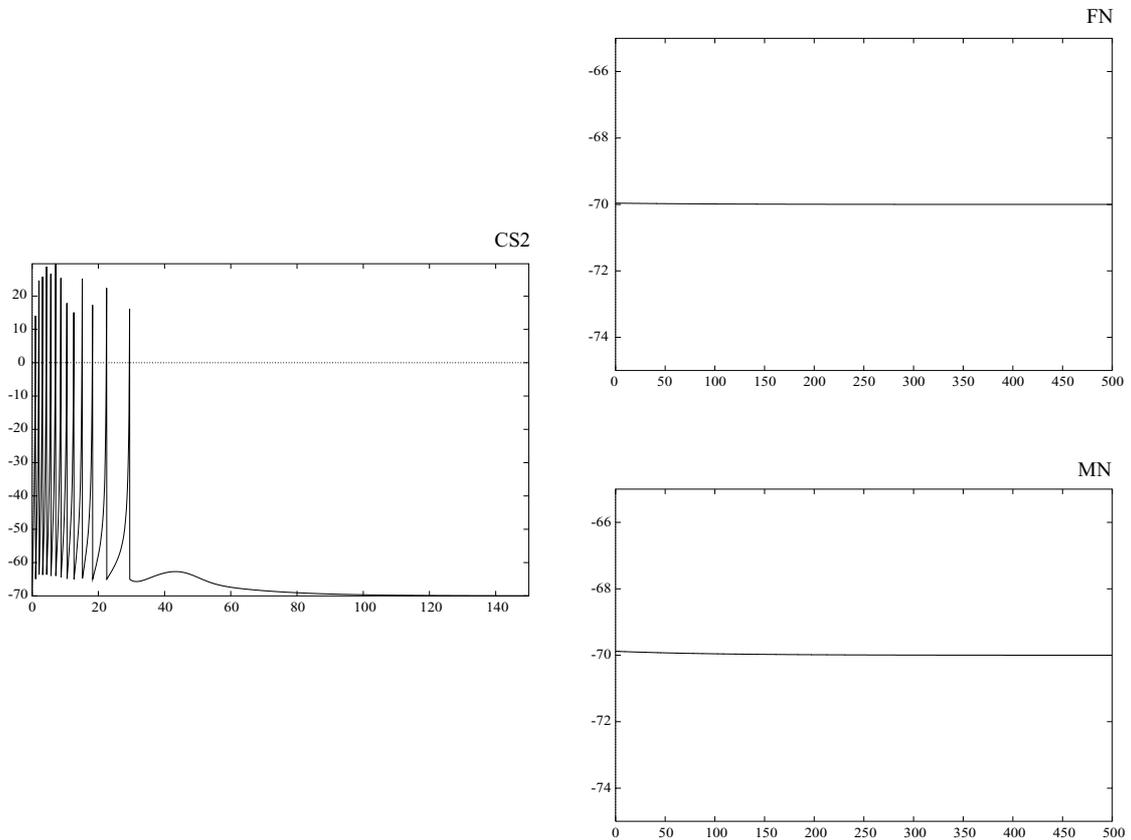


Fig. 11 CS2 remains unconditioned due to blocking

conditioning. We show, in particular, that a second conditioned stimulus in compound with pre-conditioned CS1 and US is indeed blocked.

5.1. Comparison with other models for blocking

5.1.1. Competitive learning rules and blocking

Models such as the Sutton-Barto model (Sutton and Barto, 1990) rely on competitive learning rules to explain blocking. In that scheme, CSs compete for the attention of the adaptive unit that determines a reinforcement signal. In the Sutton-Barto model, for example, both a CS and the US have reinforcing capacity. A compound stimulus consisting of a conditioned CS1, a US, and a CS2 does not provide any reinforcement to the CS2 because a preconditioned stimulus acquires an ability to signal an exhaustion of reinforcement while precluding the US. No reinforcement is available in a compound trial stimulus because of the presence of the previously conditioned CS1. And since no reinforcement is present when a fully conditioned CS1 is delivered individually it follows as a corollary that reinforcement is available in the system conditional to presence of CS1 regardless of the context of presentation, whether by itself, or in compound with a US (and CS2). In our model, reinforcement can be characterized through two components:

1. The firing activity in the FN which releases dopamine (Wieland and Gelperin, 1983) (or its equivalent, e.g., serotonin (Gelperin, 1981; Yamane et al., 1989)). It may be—realistically—that repeated stimulation of the FN eventually does not release any more dopamine, but we neglect this possibility in our model by claiming that this is a very slow process.
2. The released dopamine facilitates synapses. We claim this facilitation saturates at some level.

Translating the Sutton-Barto hypotheses into our model, once a CS1 is fully conditioned there is no further reinforcement available to other CSs. We claim, however, that if the CS1 is active alone dopamine continues to be released even though the synapses (corresponding to the CS1 association formation) may not facilitate much further. Thus reinforcement is exhausted from the CS1's point of view, while it is available to a CS2.

Experiment 1. Which of the two hypotheses is correct can potentially be determined by experimental measurement: by repeated pairings with a US a CS1 can be fully conditioned; then a single test stimulus that excites the CS1 alone may be used to determine whether the FN releases dopamine or not. According to the Sutton-Barto hypothesis applied to our model, if reinforcement is exhausted then FN cannot release any more dopamine.

5.1.2. Relevance of US to blocking

As noted above, in the CS1+US+CS2 compound pairing trials, reinforcement from the US—which is otherwise capable of providing reinforcement by itself—is precluded by the presence of CS1. This might mean that even though the input from the US to the FN remains essentially the same, the response is determined exclusively by the CS1 (and, possibly, the MN). In general, whatever the nature of the reinforcing signal in the Sutton-Barto, and similar, models the presence of the preconditioned CS1 signals the absence of reinforcement. The input provided by the US to the adaptive unit is unaltered, but simply excluded in determining the reinforcement. Thus, by determining the nature of the input from the US onto the FN in the various cases of conditioning, it should be possible to distinguish whether the US participates directly in shaping the outcome of the reinforcement. In our model, we have proposed that the reinforcing signal is the release of dopamine. In our model, if the input from the US in the CS1+US compound were unaltered, the release of dopamine could not be prevented since that input is sufficient to depolarize the FN. We claim that the CS1 and US interfere with the activity of each other to produce a complex input signal that fails to activate the FN. In principle, however, it might be argued that the input from the US (in compound) may remain the same and the FN yet precluded from firing if a feedback signal from the output is involved. To test our claim that the input into the FN from the US is significantly different in compound than otherwise, we propose the following experiment.

Experiment 2. The objective is to determine if the input into the FN is indeed different when the preconditioned CS1 and US are presented together than if they are presented individually. For simplicity, we have assumed that the dopamine releasing neuron is also the site of convergence of the CS and US pathways. In what follows, this might not be a serious difficulty, since the dopamine neuron, if distinct from the convergence neuron, is likely to be directly controlled by the convergence neuron. Note that for this experiment, it may not be sufficient to measure an output spiking response, but rather to determine the synaptic input to the FN.

1. The CS1 is activated, but not the US, and membrane voltage is recorded from the FN. If the FN neuron has previously been modeled in biophysical detail, then this is sufficient to determine the input to the neuron by inversion of the equations (see Goel and Röbenack, 2005).
2. A similar experiment with the US active and CS1 inactive will determine the input to the FN from the US alone.
3. If now, both the US and the CS1 are active together, and one determines the input, then extrapolating from the

claims of our model, the FN input should be significantly different from the sum of the inputs obtained in the two cases above. In fact, we claim that dopamine would fail to be released by US and preconditioned CS1 application. To fully resolve if the US plays any role at all in determining dopamine release during second-order conditioning, that is, whether the reinforcement signal is dependent on factors other than the CS, one further experiment might be done.

4. In this experiment, the CS and US are both activated, but the FN is simultaneously injected intracellularly with a current input that is the negative of that obtained in Part 2 above. In this strategy, even though the US was stimulated, the input from the US onto the FN is canceled. Then, if the CS and US pathways are indeed independent: the US was effectively never present at all, and dopamine should be released or not, exactly as in the case when the CS alone was present. In such a case, the US can be claimed to be ineffectual to the determination of association—the rules deciding association formation during blocking are determined by the CS, FN and MN alone, and the US enters passively. If, however, the CS and US pathways are indeed dependent on each other even before they converge onto FN, then activity in the US will influence the release of dopamine (indirectly, through the CS pathway) even if the experimentally applied current into the FN cancels the supposed input from the US.

It is interesting to note that there are various similarities between the neuronal circuitry we propose here, and the circuit that has been identified for Pavlovian conditioning in the marine slug *Hermissenda* (Crow, 2004). The synaptic and intrinsic excitability of the neurons along the CS and US pathways during conditioning has been investigated in some detail. It is known that 5-HT is responsible for mediating this plasticity. Moreover, a serotonergic neuron has been identified in that system, onto which both the US and CS pathways are known to converge. It is also notable that direct GABAergic inhibitory connections are known between the US and CS receptor cells. The topological organization of the network in the *Hermissenda* (see, for example, Fig. 3 in Crow, 2004) is suggestive that a circuit similar to the one we propose here may indeed be a crude representation of the circuitry in the *Limax*, and possibly, other similar invertebrates.

Several identified interneurons in the central ganglia of *Limax* are candidate loci for testing the synaptic consequences of CS1, CS2 and US application after second order conditioning. The metacerebral giant cell (Gelperin, 1981) is activated by both gustatory and olfactory inputs and is the largest cell in the cerebral ganglia, hence providing a technically convenient target for long-term intracellular recording. The recently identified parietal neuron, v-PN, shows

learning-induced changes in its firing in *in vitro* preparations related to mantle shortening elicited by aversive odors (Inoue et al., 2004). In addition, there is a set of command neurons for feeding which has been characterized anatomically and physiologically (Delaney and Gelperin, 1990 a, b, c) which might show learning-related changes to odor input since odor stimulation demonstrably affects feeding behavior (Sahley et al., 1992). Several nose-brain preparations retaining motor output pathways signaling decisions about odor aversion have been developed (Teyke and Gelperin, 1999; Teyke et al., 2000; Kirino et al., 2005). Based on earlier success obtaining taste-taste learning in an *in vitro* *Limax* lip-brain preparation (Chang and Gelperin, 1980) and *in vitro* learning in other molluscan preparations (Lukowiak and Sahley, 1981; Kemenes et al., 1997; Reyes et al., 2005) it is likely that first order, and perhaps second order, odor aversion conditioning can be obtained in the *Limax in vitro* nose-brain preparation while recording from one of the identified interneurons described above.

Appendix

All simulations were performed using the software XPPAUT (Ermentrout, 2002)—available at <http://www.pitt.edu/~phase>—running on a UNIX workstation.

We model the neurons with an integrate-and-fire model from Izhikevich (Izhikevich EM., Which model to use for cortical spiking neurons? IEEE Trans Neural Netw. 2004 Sep;15(5):1063–70.)

$$\begin{aligned} \frac{dV}{dt} &= 0.04 V^2 + 5 V + 140 - u + I^{syn} + I^{stim} \\ \frac{du}{dt} &= a(bV - u) \text{ if} \\ V &= 30 \text{ mV}, \quad V \leftarrow c, u \leftarrow u + d \end{aligned} \tag{1}$$

where the parameters $a = 0.1, b = 0.2, c = -65, d = 2$ are chosen to resemble the FS (fast-spiking) type of neuron (see Izhikevich, 2004)

Synaptic currents are modeled as:

$$I^{syn} = g_{syn} s (V_{syn} - V) \frac{ds}{dt} = -s/\tau_s \tag{2}$$

such that if $V_{pre} = 30, s = 1$. Details of the various synaptic connections are given below.

Neurons in the input layer: CS1, CS2 and US

The “stimulus” currents to CS1, CS2, US are taken to be of the form:

$$I^{stim} = I_0 e^{-t/t_i} \quad (3)$$

with $t_i = 20$ and $I_0 = 50$ if the stimulus is present, or 0 otherwise. Inhibitory connections that develop between these neurons are modeled with the synaptic terms:

$$I^{syn} = g_{inh} s_{inh} (V_{inh} - V) \quad (4)$$

with $V_{inh} = -80$. The maximal conductance g_{inh} of the synapse increases in value gated by heterosynaptic facilitation from the FN. The synapse is assumed to be facilitated (only) while the FN is active. Inhibitory connections between a pair i and j of neurons in the input layer are strengthened according to:

$$\text{if } V_{FN} = 0, \quad g_{inh} \leftarrow g_{inh} + \left(\frac{I_{0,i}}{50} \frac{I_{0,j}}{50} \right) g_{inh}^r (g_{inh}^m - g_{inh}) \quad (5)$$

with $g_{inh}^r = 0.05$, $g_{inh}^m = 2.0$. That is if both, the pre- and post-synaptic neurons, i and j , are active, the synapse is facilitated as a function of the firing activity in the FN. Note that we consider the symmetric facilitation of the reciprocal inhibitory connections, thus we take $g_{inh,US \rightarrow CS} = g_{inh,CS \rightarrow US}$.

Notice that the concave growth of synaptic conductance towards an asymptotic value is vaguely reminiscent of the Rescorla-Wagner law for the growth of associative conditioning.

The CS and US make the excitatory connections onto the MN with $g_{US \rightarrow MN} = 0.1$. The facilitation of the $CS \rightarrow MN$ synapses is also mediated by the FN and follows an equation similar to Eq. (5),

$$\text{if } V_{FN} = 0, \quad g_{inh} \leftarrow g_{inh} + \frac{I_{0,CS}}{50} g_{inh}^r (g_{inh}^m - g_{inh}) \quad (6)$$

with $g^r = 0.1$, $g^m = 0.1$.

The facilitator neuron

The FN is modeled with Eq. (1) with $I_{FN}^{stim} = 0$. The excitatory connection from the US is modeled using a potentiating synapse:

$$\frac{ds_{US \rightarrow FN}}{dt} = -s_{US \rightarrow FN} / \tau_{US \rightarrow FN}, \quad (7)$$

$$\text{if } V_{US} = 0, \quad s_{US \rightarrow FN} \leftarrow s_{US \rightarrow FN} + s_{US \rightarrow FN}^r (1 - s_{US \rightarrow FN}) \quad (8)$$

where $\tau_{US \rightarrow FN} = 100$, $g_{US \rightarrow FN} = 0.55$ and $s_{US \rightarrow FN}^r = 0.02$. Excitatory synapses from the CS's also obey a sim-

ilar expression with the additional rule for facilitation of the conductance:

$$\text{if } V_{FN} = 0, \quad g_{CS \rightarrow FN} \leftarrow g_{CS \rightarrow FN} + \frac{I_{0,CS}}{50} g_{CS \rightarrow FN}^r (g_{CS \rightarrow FN}^m - g_{CS \rightarrow FN}) \quad (9)$$

with $g_{CS \rightarrow FN}^r = 0.2$, $g_{CS \rightarrow FN}^m = 0.55$.

Notice that the slowly potentiating synapse onto the FN has the property that the summation of a US synaptic current with input from an active (fully-conditioned) CS1 synapse (say) does not integrate fast enough for the FN to depolarize and spike. This behavior is crucial to the US-CS-FN XOR operation described above.

The motor neuron

The MN is modeled using Eq. (1) with $I^{stim} = 0$, and excitatory synaptic connections from CS's and US. Note that the connections from the CS's onto MN may be facilitated; the US itself can excite the MN regardless of any conditioning. The synaptic facilitation of the synapses from the CS's follow the rule similar to Eq. (9), but with $g_{CS \rightarrow MN}^r = 0.1$ and $g_{CS \rightarrow MN}^m = 0.1$.

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