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Kiruthika RAMANATHAN
Singapore Management University, kiruthikar@smu.edu.sg

Ning NING

Dhiviya DHANASEKAR

Guoqi LI

Luping SHI

See next page for additional authors

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Author

Kiruthika RAMANATHAN, Ning NING, Dhiviya DHANASEKAR, Guoqi LI, Luping SHI, and Prahlad VADAKKEPAT

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PRESYNAPTIC LEARNING AND MEMORY WITH A PERSISTENT FIRING NEURON AND A HABITUATING SYNAPSE: A MODEL OF SHORT TERM PERSISTENT HABITUATION

KIRUTHIKA RAMANATHAN, NING NING,
DHIVIYA DHANASEKAR, LI GUOQI and SHI LUPING
Data Storage Institute
Agency for Science, Technology and Research (A-STAR)
5 Engineering Drive 1, Singapore, 117608

PRAHLAD VADAKKEPAT
Department of Electrical and Computer Engineering
National University of Singapore
4 Engineering Drive 3, Singapore 117576

Our paper explores the interaction of persistent firing axonal and presynaptic processes in the generation of short term memory for habituation. We first propose a model of a sensory neuron whose axon is able to switch between passive conduction and persistent firing states, thereby triggering short term retention to the stimulus. Then we propose a model of a habituating synapse and explore all nine of the behavioral characteristics of short term habituation in a two neuron circuit. We couple the persistent firing neuron to the habituation synapse and investigate the behavior of short term retention of habituating response. Simulations show that, depending on the amount of synaptic resources, persistent firing either results in continued habituation or maintains the response, both leading to longer recovery times. The effectiveness of the model as an element in a bio-inspired memory system is discussed.

Keywords: Synaptic models; persistent neural activity; habituation; short term memory.

1. Introduction

Much research on spiking neuron models has focused on spike time dependent plasticity (STDP), almost to the exclusion of other learning methods.[1–6] STDP accounts for the pairing of pre- and post-synaptic firing times within a window to change the strength of the synapse, which can be either potentiated or depressed,[7–9] and it is thought to mediate associative learning in the brain.[10] However, recent evidence has proposed that STDP alone is not sufficient to explain associative conditioning in the brain.[11,12] Ito *et al.*,[13] for instance, showed that precise STDP-like learning rules do not apply for certain neurons even in Pavlovian conditioning procedure and that other neural mechanisms for learning are still waiting to be discovered.[11] Some of these neural mechanisms include the presence of a short term memory for the stimulus, especially in terms of persistent neural activity.[12,14] There is also a need to develop a comprehensive model of the neuron and synapse, including the simpler forms of synaptic learning such as habituation and sensitization.[15]

Habituation is the decrement in neural response due to the repeated exposure to an innocuous stimulus. With repeated stimulations of a neuron, the influx of Calcium ions at the presynaptic terminal largely reduces, causing a proportional decrement in the amount of neurotransmitters released into the synapse. This decrement results in reduction of the synaptic strength between the two neurons involved in learning, thus resulting in synaptic depression. This is followed by a reduced postsynaptic membrane

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potential. As this change in synaptic strength persists, the memory for habituation is generated.[16] The behavioral characteristics of habituation were defined in 1966 by Thomson and Spencer and the molecular characteristics were first specified by ER Kandel in the 1970s.[17,18] While habituation is termed as the simplest form of learning,[15] little is known about the neural mechanisms underlying habituation. However, as habituation allows animals to focus on important stimuli, researchers believe that it is a pre-requisite to other forms of learning,[19] and that understanding the building blocks of habituation is necessary to fully understand the mechanisms of more complex forms of learning. To this effect, Rankin *et al.*[19] published a revised and updated description of the behavioral characteristics of habituation.

Several computational models of habituation have been developed over the years. These include habituation with spiking and non-spiking inputs,[20] dynamic[21] and event driven synapses. The models display, to varying extents, the different characteristics of habituation as described by Thomson and Spencer.[16] To our knowledge, however, there have been no attempts at computationally modeling habituation after the publication of the revised and updated characteristics.[19]

The objective of this paper is two-fold. First, we propose a new model to describe the dynamic changes in synaptic conductance with respect to changing presynaptic membrane potential during the process of short term habituation. To this effect, we explore all nine of the revised behavioral characteristics of short term habituation as described by Rankin *et al.*[19] in a two neuron model. Second, we explore how persistent firing of action potentials in the presynaptic axon results in continued adaptation and retention of the habituated response after the removal of the stimulus. By combining the two components, we propose a model of element cell to be used in bio-inspired architectures, such as the proposal for artificial cognitive memory.[22]

The rest of our paper is organized as follows. Section 2 describes the neural model for the sensory and motor neurons. It also describes the equations of the habituating synapse. Section 3 presents a mathematical analysis of the model, including the conditions of persistent firing and operation modes. Section 4 benchmarks the model against the revised behavioral characteristics of habituation. Section 5 presents simulation results investigating the contribution of persistent firing to memory. Section 6 presents a discussion of the work, including how the element cell can be used as a part of bio-inspired artificial cognitive memory and Sec. 7 concludes the paper.

2. Mathematical Model of the Element Cell

Figure 1 shows the wiring diagram of the neural circuit. The element cell is represented by the neuron and synapse within the dotted lines. The persistent firing neuron[23] extends the Izhikevich neuron model[24] to describe the persistent firing behavior. The motor neuron is a classical implementation of the Hodgkin Huxley neuron.[25] The habituating synapse is described in Sec. 2.2.

2.1. *Presynaptic persistent firing sensory neuron*

Memory storage on short timescales is thought to be maintained by neuronal activity that persists after the stimulus is removed.[12] The positive-feedback processes or the feed forward mechanism are often hypothesized to explain the persistent activities in the neuronal network.[26] However, a recent experimental finding of slow integration induced persistent firing in distal axons of rodent hippocampal and neocortical interneurons,[14] has shed new light on identifying the mechanism of neuronal persistent activities. In addition to the prevalent observations of persistent neural activities in diverse brains and species,[27]

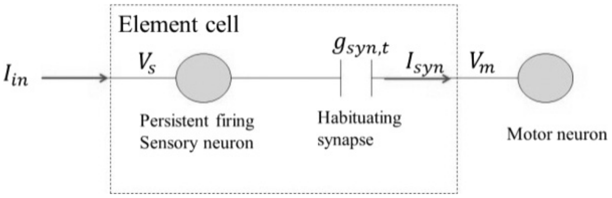

Fig. 1. Model of the element cell. The persistent firing sensory neuron receives external stimulus I_{in}. The membrane potential V_s of the sensory neuron is used to calculate the synaptic conductance g_{syn}, which influences the current I_{syn} to the Hodgkin Huxley motor neuron, whose response is measured using its postsynaptic membrane potential, V_m.

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it was found that as compared to classic somatic integration of synaptic currents on a timescale of milliseconds to seconds, the slow integration from tens of seconds to minutes in distal axon, leads to persistent firing of action potentials lasting for a similar duration. To trigger such persistent firing, axonal action potential firing was required but somatic depolarization was not, implying that axon may perform its own neural computations without any involvement from soma or dendrites.

We have recently proposed a model of persistent firing neuron,[23] in which the axon is considered as a slow leaky integrator capable of altering its operating states between passive conduction mode and persistent firing mode, through its own computation. The potential of the axonal leaky integrator controls the switching of axon operating states: in passive conduction mode, the axon acts as conventional transmission cable; and in persistent firing mode, it produces the persistent firing of action potentials, which do not require stimulus from dendrites to sustain. The model captures the essence of the persistent firing behavior of neurons and enables further study of the relationship between persistent firing activities and neural correlates of short term memory.

The sensory neuron in the model of Fig. 1 is a persistent firing neuron which can be mathematically described by Eqs. (1)–(6)[23]:

$$\frac{dV_s}{dt} = 0.04V_s^2 + 5V_s + 140 - u + I_{\text{in}}, \tag{1}$$

$$\frac{du}{dt} = a(bV_s - u), \tag{2}$$

$$\frac{dw}{dt} = -fw, \tag{3}$$

with the auxiliary resettings:

$$\text{if } V_s \geq 30\,\text{mV}, \quad \text{then } V_s \leftarrow c, \quad u \leftarrow u + d, \quad w \leftarrow w + e \tag{4}$$

$$\text{if } w \geq w_p, \quad \text{then } (a, b, c, d, e) \leftarrow (a, b, c, d, e)_p, \tag{5}$$

$$\text{if } w \leq w_n, \quad \text{then } (a, b, c, d, e) \leftarrow (a, b, c, d, e)_n, \tag{6}$$

where V_s, u, and w are dimensionless variables that describe the membrane potential, the membrane recovery, and the potential of the axonal leaky integrator, respectively. a, b, c, d, e, and f are dimensionless parameters. a describes the time scale of u, b represents the sensitivity of u to the subthreshold fluctuations of V_s, c is the after-spike reset value of membrane potential, d describes the after-spike reset of u. e represents the after-somatic-spike axonal accumulation in the passive conduction mode, and f is the value describing the rate of the axonal leak. w_p is the threshold value of w to trigger the persistent firing mode of axon, and w_n is the lower threshold value of w for the axon to return to passive conduction mode. $(a, b, c, d, e)_p$ describes the parameter set of a, b, c, d, e variables when the axon is in the persistent firing mode, chosen according to the analysis in Sec. 3.1, and $(a, b, c, d, e)_n$ describes the parameter set of a, b, c, d, e variables in the passive conduction mode.

The equations described above extend the Izhikevich neuron model[24] described by Eqs. (1) and (2). We have introduced a variable w, which represents the axonal potential that controls the switching of parameter set of a, b, c, d and e. The axonal firing states are determined by the current values of the parameter set. Table 1 shows the values of parameters of the persistent firing neuron model.

2.2. *Mathematical description of the habituating synapse*

Habituation occurs at the presynaptic terminal i.e. the axon terminal of the sensory neuron. The response of the motor neuron is based on the amount of neurotransmitters received across the synapse from the presynaptic terminal. The synapse is modeled by the synaptic conductance, which influences the output current of the synapse, denoted as I_{syn}. This output is also the input current to the motor neuron.

Habituation begins when the sensory neuron spikes at least twice. Let $\{t_{AP,i}\}_{i=1}^{k}$, where i is the index of the spike and $k(k \geq 2)$ denotes the current spike index, be the spike time sequence from the first spike time $t_{AP,1}$ to the nearest spike time $t_{Ap,k}$. Let AP_t denote the current synaptic firing rate. Let $\{AP_{t'}\}_{t'=0}^{t'=t}$ be the sequence of $AP_{t'}$ from the time $t' = 0$ to the time $t' = t$. Based on above definitions,

Table 1. Parameters of the persistent firing neuron.

$(a, b, c, d, e)_p$	$(0.1, 0.267, -55, 0, 0)$
$(a, b, c, d, e)_n$	$(0.1, 0.2, -65, 2, 0.02)$
f	0.0005
(w_p, w_n)	$(0.2, 1.9)$

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the habituating synapse can be modeled as a nonlinear system $\Gamma(\cdot)$ presented as follows:

$$I_{\text{syn},t} = \Gamma(V_{s,t}, \{AP_{t'}\}_{t'=0}^{t'=t}, \{t_{AP,i}\}_{i=1}^{i=k}), \tag{7}$$

where $V_{s,t}$ is the output of the sensory neuron. We now describe the derivation of (7).

We characterize the synaptic connection by its maximum amount of resources, activated by the maximal firing rate of the neuron. In this sense, there is a maximum value for the firing rate AP_t, denoted as $AP_{\max}$. Let τ_d and τ_o be the average decay and onset time constants of V_s and α_t is a normalization parameter given by

$$\alpha_t = \frac{\tau_d}{(\tau_d - \tau_o)(AP_{\max} - AP_t)}. \tag{8}$$

The resulting synaptic conductance $g_{\text{syn},t}$ is computed as

$$g_{\text{syn},t} = w\alpha_t SE_t, \tag{9}$$

where w is the maximum synaptic conductance and SE_t is an introduced variable. It is known that each presynaptic action potential, arriving at time t_{AP} activates a fraction of the resources and deactivates them to the resting state of the synapse. The aggregate of this process is denoted by the variable SE_t of the synapse at time t. SE_t is evaluated as a function of the onset and decay processes involved in the action potential denoted by $SE_{o,t}$ and $SE_{d,t}$. In the habituating synapse, it is modeled that

$$SE_t = \frac{SE_{d,t} - SE_{o,t}}{g_{\text{syn},t-\Delta t} - e^{(-t_{AP,k} + t_{AP,k-1})}}, \tag{10}$$

where $g_{\text{syn},t-\Delta t}$ is the synaptic conductance at the time $t - \Delta t$ with Δt being the sampling time step, $t_{AP,k-1}$ and $t_{AP,k}$ denote the respective $(k-1)$th and the current spike time, and $SE_{\varphi,t}$ for $\varphi = d$ or o is updated by

$$SE_{\varphi,t} = \begin{cases} SE_{\varphi,t-\Delta t} e^{-\frac{\Delta t}{\tau_\varphi}} \\ \quad + e^{\frac{(t_{AP,k-1} - t)}{\tau_\varphi}} & \text{if } t - \Delta t < t_{AP,k} \le t \\ SE_{\varphi,t-\Delta t} e^{-\frac{\Delta t}{\tau_\varphi}} & \text{otherwise} \end{cases} \tag{11}$$

The above condition $t - \Delta t < t_{AP,k} \le t$ implies that a new spike has occurred between $t - \Delta t$ and t. By updating t by $t - \Delta t$, (10) and (11) can be obtained iteratively. For simulation, all the initial values of $SE_t, SE_{d,t}, SE_{o,t}$ and $g_{\text{syn},t}$ are set as zero.

Finally, the synaptic current $I_{\text{syn},t}$, which is also the input of the motor neuron, is calculated as

$$I_{\text{syn},t} = (E_{\text{syn}} - V_{s,t})g_{\text{syn},t}, \tag{12}$$

where E_{syn} is the synaptic reversal potential. $I_{\text{syn},t}$ modeled in Eq. (12) using a Hodgkin Huxley equation is to obtain the postsynaptic potential of the motor neuron, as discussed in the following section.

In Eqs. (8)–(12), $AP_{\max}, \tau_d, \tau_o, w, \Delta t$, and E_{syn} are user specified or biologically inspired constants, while $\text{SE}_t, SE_{\varphi,t} (\varphi = d$ or $o)$, α_t and $g_{\text{syn},t}$ are introduced intermediate variables. Then $I_{\text{syn},t}$ is dependent on the historical output of the sensory neuron. Combining (8)–(12) allows us to represent the computation of $I_{\text{syn},t}$ as a nonlinear function $V_{s,t}, \{AP_{t'}\}_{t'=0}^{t'=t}$ and $\{t_{AP,i}\}_{i=1}^{i=k}$. Such a nonlinear function can be represented as the nonlinear system $\Gamma(\cdot)$ presented in (7).

The constants employed in the above equations and their experimental values are discussed below:

1. The decay and onset time constants τ_d and τ_o are set to 40 ms and 60 ms, respectively.
2. $AP_{\max}$, the maximum number of action potentials per second that can be fired by the neuron, is between 100 and 1000 AP/s biologically.[28,29]
3. Biologically the synaptic reverse potential E_{syn} is between 0 mV and 20 mV for excitatory neurons.[30] We set the value at 10 mV.
4. The maximum synaptic conductance w is set to 720 pS.
5. The sampling time step Δt is set to 0.02 ms.

It is to be noted that the characterization of the synapse by its amount of resources is an approach inspired by Tsodyks and Markram.[21] The synaptic model proposed here is computationally more complex in that the value of EPSP at a given time depends on the membrane potential of the sensory neuron, instead of the spike time alone, as is the case in Markram's model. The analysis in our model is therefore at the neurotransmitter and the ion channel level and, at the expense of computational complexity, is more specific as compared to Markram's

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model, which combines all the parameters together as the "synaptic resources."

2.3. *Hodgkin Huxley motor neuron*

The motor neuron is modeled following the Hodgkin Huxley model[25]:

$$C_m \frac{dV_m}{dt} = (I_{\text{leak}} + I_{\text{Na}} + I_{\text{K}} + I_{\text{syn}}), \tag{13}$$

where

V_m is the potential across the membrane in the neuron,
C_m is the capacitance of membrane patch of unit area in the neuron,
$I_{\text{leak}} = g_{\text{leak}}[E_{\text{leak}} - V_{m,t-1}]$ is the leakage current in the neuron,
$I_{\text{Na}} = g_{\text{Na}} m^3 h [E_{\text{Na}} - V_{m,t-1}]$ is Sodium ion current,
$I_{\text{K}} = g_{\text{K}} n^4 [E_{\text{K}} - V_{m,t-1}]$ is the Potassium ion current,
I_{syn} is the synaptic current, which is the output of the habituating synapse.

$E_{\text{leak}}, E_{\text{Na}}$ and E_{K} are the reversal potentials associated with each membrane current for the neuron and related to the ionic distributions inside and outside the neuronal membrane. $g_{\text{leak}}, g_{\text{Na}}$ and g_{K} are the maximal conductance of their respective ion channels. m, n and h describe the probability of opening of the three ion channels in the Hodgkin Huxley model for the sensory and motor neurons. Table 2 shows the parameters of the Hodgkin Huxley neuron, where x is Na, K or L. The membrane capacitance is set as 1 uF/cm^2.

3. Model Analysis

In this section, we will analyze the conditions of the persistent firing of the sensory neuron and the operation modes of the element cell. Examples of the model will be illustrated and discussed further in the simulations in next section.

Table 2. Parameters of the Hodgkin Huxley neuron.

x	E$_x$ (mV)	g$_x$ (mS/cm^2)
Na	115	120
K	−12	36
L	10.6	0.3

3.1. *On the conditions of persistent firing*

Let $X = [V_s u w]'$. Equations (1)–(3) can be rewritten as:

$$\dot{X} = F(X) + HI_{\text{in}} = X'AX + BX + C + HI_{\text{in}}, \tag{14}$$

where I_{in} is the input current, $H = [1\ 0\ 0]'$ and $F(X) = [f_1(X) f_2(X) f_3(X)]' = X'AX + BX + C$ with

$$A = \begin{bmatrix} 0.04 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad B = \begin{bmatrix} 5 & 0 & 0 \\ ab & -a & 0 \\ 0 & 0 & -f \end{bmatrix}, \quad C = \begin{bmatrix} 140 \\ 0 \\ 0 \end{bmatrix}. \tag{15}$$

When the input current I_{in} is not presented, the equilibrium points of the dynamic system in (14) are the solutions of the equation $F(X) = 0$, i.e.

$$0.04V_s^2 + 5V_s + 140 - u = 0, \tag{16}$$

$$a(bV_s - u) = 0, \tag{17}$$

$$-fw = 0. \tag{18}$$

One can get that $u = bV_s$ and $w = 0$ from (17) and (18) and then (16) becomes

$$0.04V_s^2 + (5 - b)V_s + 140 = 0. \tag{19}$$

Finally, the two equilibrium points are obtained as

$$X_1 = [-12.5(5 - b + \sqrt{\Delta}) - 12.5b(5 - b + \sqrt{\Delta})0]'.$$

$$X_2 = [-12.5(5 - b - \sqrt{\Delta}) - 12.5b(5 - b - \sqrt{\Delta})0]'.$$

Here we assume that $\Delta = 2.6 + b^2 - 10b > 0$ and it is noted that X_1 is a stable equilibrium point but X_2 is an unstable one. Based on the analysis of stability of the equilibrium points and the nonlinear dynamic characteristic of Eq. (14), the following conditions can be summarized to guarantee the existence of the persistent firing of the sensory neuron.

Condition 1. The input current I_{in} satisfies that $I_{\text{in}} \geq |\min\{0.04V_S^2 + (5 - b)V_s + 140\}|$ where $|\cdot|$ denotes an absolute value function.

Condition 2. The condition $w > w_p$ is satisfied no later than the time when I_{in} is removed.

Condition 3. When $w > w_p$, the parameter c denoted as c_p in (5) (during the persistent firing

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period) is chosen to be value such that $c > -12.5(5 - b - \sqrt{\Delta})$.

Condition 1 means that there is no equilibrium point for the equation $F(X) + HI_{\text{in}} = 0$, i.e. $X'AX + BX + C + HI_{\text{in}} = 0$. Otherwise, X may approach the equilibrium point and not change any more even when I_{in} is presented. In this case, it is impossible for persistent firing to happen. Condition 2 requires that the sensory neuron spikes a sufficient number of times to ensure that $w > w_p$ before I_{in} is removed. Condition 3 implies that, during the persistent firing period, $0.04V_S^2 + (5 - b)V_s + 140 - u > 0$ should be guaranteed at the interval $-12.5(5 - b - \sqrt{\Delta}) \leq V_S \leq c_p$.

In the above analysis, we choose b to be such that $\Delta > 0$. Now we analyze the case that $\Delta \leq 0$. It can be shown that there is no real equilibrium point or at most an unstable equilibrium point for the equation $F(X) = 0$. Then Condition 1 is achieved even if $I_{\text{in}} = 0$. The persistent firing can be always observed no matter whether the Conditions 2 and 3 are satisfied or not. However, in this case, the nonlinear system $\dot{X} = F(X) + HI_{\text{in}}$ is unstable. This is the reason that generally b is suggested to be chosen as a value such that $\Delta > 0$.

3.2. *Modes of the working process analysis of the element cell*

The element cell in Fig. 1 is a cascade system consisting of three subsystems, persistent firing sensory neuron, habituating synapse and the motor neuron, which can be represented by Eqs. (1)–(6), (7) and (13), respectively. The synapse begins to habituate when sensory neuron spikes at least twice. In addition, the conductance of the habituating synapse is dependent on all the historical output of the sensory neuron as we discussed in Sec. 2.2. Based on our previous analysis, the status of the working process of the element cell now can be summarized as follows:

Case 1: *No input or a very small input current* I_{in}

We assume that $\Delta = 2.6 + b^2 - 10b > 0$ as discussed. In this case, Condition 1 $I_{\text{in}} \geq |\min\{0.04V_S^2 + (5 - b)V_s + 140\}|$ cannot be guaranteed under the presented I_{in} (I_{in} can be zero). Then there are two equilibrium points for the equation $\dot{X} = X'AX + BX + C + HI_{\text{in}} = 0$. One is a stable point and the other one is unstable. After an initial value of X is given, the first subsystem will converge to the stable equilibrium point. Conditions 1–3 are not satisfied and both sensory and motor neurons remain in resting state.

When I_{in} is greater than a threshold value such that $I_{\text{in}} \geq |\min\{0.04V_S^2 + (5 - b)V_s + 140\}|$ and the Conditions 2 and 3 are satisfied, the sensory neuron can be persistent firing after I_{in} is removed, which will be discussed in the following Cases 2 and 3.

Case 2: *Somatic firing without persistent activity*

This corresponds to the case where Condition 1 is satisfied but the Conditions 2 and 3 are not satisfied. Since Condition 1 is satisfied, somatic spiking of the sensory neuron exists when the neuron is stimulated. No new spikes are generated after the removal of the stimulus current. Then SE_t and $SE_{\varphi,t}$ will approach zero asymptotically and the habituating synapse recovers its response.

Case 3: *Persistent firing exists before* I_{in} *is removed.*

This case corresponds to the condition where $w > w_p$ is satisfied before I_{in} is removed. The persistent firing will exist in advance before I_{in} is removed and will continue after the removal of I_{in}. When I_{in} is removed, the habituating synapse will still be active and the output of the of the habituating synapse conducts the input current to the motor neuron.

In addition, there are two states of the persistent firing in Case 2. State 1 is the persistent firing when the input current I_{in} is presented to the sensory neuron. State 2 is the case when I_{in} is removed and only persistent firing contributes to synaptic change.

The model predicts that the frequency of sensory neuron spikes in State 1is higher than the frequency in State 2 due to the combined effect of somatic firing and persistent firing. Note that if the input stimulus is oscillatory, the sensory neuron switches states between State 1 and State 2. To the habituating synapse, this corresponds to the effect of observing alternating stimuli of varying frequency

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and therefore the synapse habituates and dishabituates in this phase.

Case 4: *Persistent firing exists at the time* I_{in} *is removed.*

This case corresponds to the case where $w > w_p$ is satisfied at the time I_{in} is removed. At the time I_{in} is removed, the persistent firing exists and the dynamic behavior of the whole system is the similar to what we analyzed in Case 2. The key difference lies in that there is no State 1 for the persistent firing in this case.

4. Simulations on the Behavior of the Habituating Synapse

In this section, we validate the model against the biological properties of the synapse. In Sec. 4.1, the model is validated against the biological properties of habituation. In Sec. 4.2, a two input case is presented where one of the inputs is sensitizing. In order to better tie in our results with the biological observations, all the neurons (sensory and motor) in this section are Hodgkin Huxley implementations.

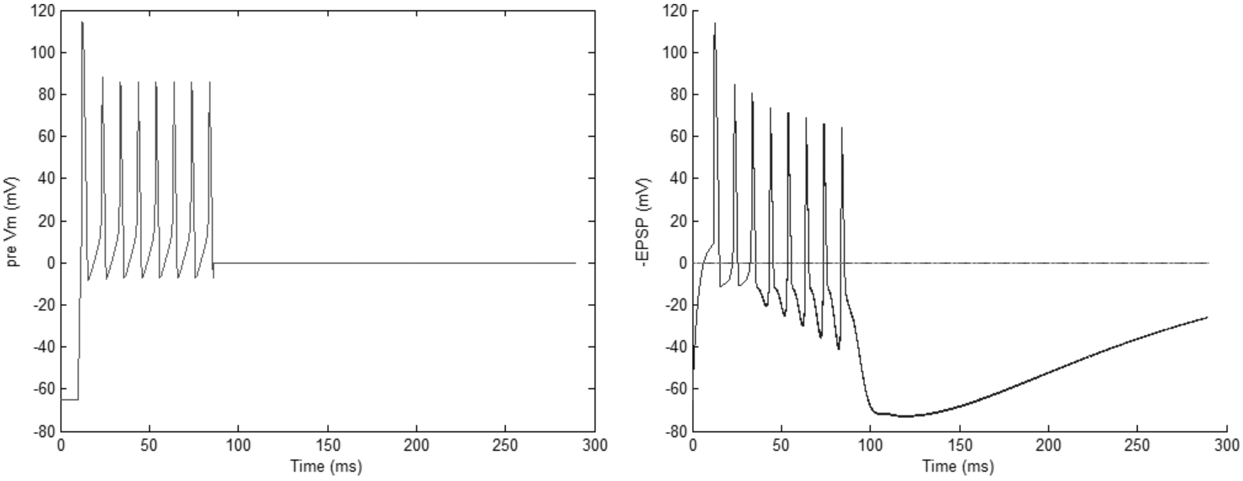
4.1. *Tests against the biological properties of habituation*

In this section we evaluate the model of the habituating synapse described in Sec. 2 against the behavioral properties described by Rankin.[19] Table 3 provides a comparison of the habituation model proposed in this paper with other models of habituation.

Table 3. A comparison between habituation models.

Models proposed	Brief description of model and characteristics achieved
Stanley[20]	3-neuron model. Characteristics 1,2,5
Wang[31] and Arbib	3-neuron model. Characteristics 1,2,3,5. Dishabituation in a hierarchy
Baxter and Byrne[32]	Modified Stanley's equations on 4 neuron circuit
Marsland[33]	Modification of Wang and Arbib's model
Habituation in element cell in this work	2 neuron circuit achieving 9 characteristics of habituation.

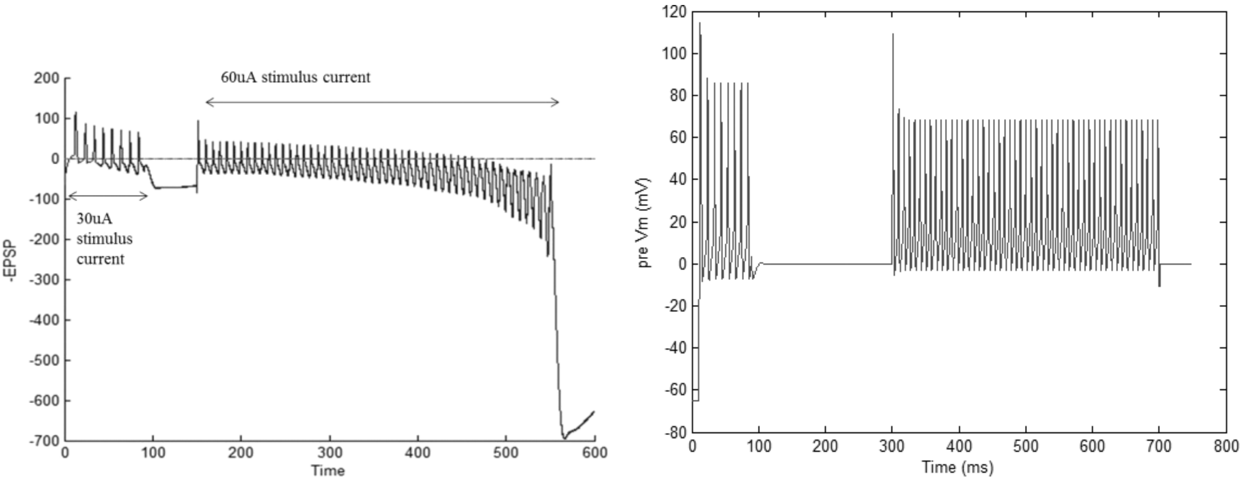
Characteristic 1 of habituation is as follows: Repeated applications of an innocuous stimulus to the sensory neuron results in a negative, usually exponential decrease in the response of the motor neuron. Figure 2 shows the pre- and post-synaptic membrane potentials of the model when the sensory neuron is stimulated using an input current of 30 μA. The postsynaptic membrane potential is predicted from Eqs. (7) and (8) and the response decreases exponentially over time. The model also shows the



(a)

(b)

Fig. 2. Application of a 30 μA stimulus leads to a presynaptic membrane potential as given in (a). The postsynaptic habituation response is given in (b).



(a) (b)

Fig. 3. (a) A 60 μA input pulse following habituation to a 30 μA input pulse shows dishabituation first, and then habituation to the 60 μA stimulus. (b) The presynaptic membrane potential due to the stimulus.

dual process theory of response habituation,[34] where animals show an increase in responsiveness, followed by a decrease in responsiveness to repeated stimulation. Figure 3 shows such a response of a neuron, initially habituated to a 30 μA input stimulus, when a 60 μA input stimulus is presented to it. The neuron shows a slight dishabituation to the new stimulus, followed by continued habituation.

Characteristic 2 of habituation describes the recovery of the response when the stimulus is withheld, at least partially over the observation time. This mechanism of spontaneous recovery is observed in both Figs. 2 and 3.

Characteristic 3 describes how, after multiple series of stimulus repetitions and spontaneous recoveries, the response decrement becomes successively more rapid — leading to a phenomenon called potentiation of habituation. Figure 4 shows the postsynaptic membrane potential of the motor neuron when the sensory neuron is stimulated with a 30 μA input stimulus. The stimulus is presented each time for 400 ms followed by 40 ms of spontaneous recovery. The response decrement of the postsynaptic membrane potential becomes successively more rapid with each repetition of the stimulus.

Characteristic 4 states that when all other things are equal, more frequent stimulation results in more rapid and/ or more pronounced response decrement and more rapid spontaneous recovery. This behavioral characteristic was revised from original following the research by Rankin and Bostner,[35] which showed that the frequency of stimulation during habituation determined the rate of spontaneous recovery only if habituation has reached asymptotic levels. We analyze this behavioral characteristic of habituation using two simulations.

In the first simulation, we set the neuron to habituate to asymptotic levels by setting the value of $AP_{\max}$ in Eq. (8) to a small value of 50 Hz. The synapse then habituates to asymptotic levels as shown in the gray plot in Fig. 5. The recovery rate of the synapse, once habituated to asymptotic levels, is much higher (slope of 9.818) than the synapse which is not habituated to asymptotic levels (slope of 2.531 for a neuron whose value of $AP_{\max}$ is set to 125 Hz) (shown by the black plot in Fig. 5).

In the second simulation, the synapses are not set to reach asymptotic levels, with $AP_{\max}$ set to 500 Hz. Figure 6 shows the postsynaptic membrane potential of the motor neuron when the sensory neuron is simulated using a more frequent stimulus (Average frequency: 200 Hz) in Fig. 6(a) versus a less frequent stimulus (Average Frequency: 100 Hz) in Fig. 6(b). The 200 Hz stimulus shows a more pronounced habituation response, with the postsynaptic membrane potential reaching a value of ~ -80 mV, while the minimum postsynaptic potential of the 100 Hz stimulus is -20 mV.

Characteristic 5 states that the weaker the stimulus, the more rapid and pronounced the habituation,

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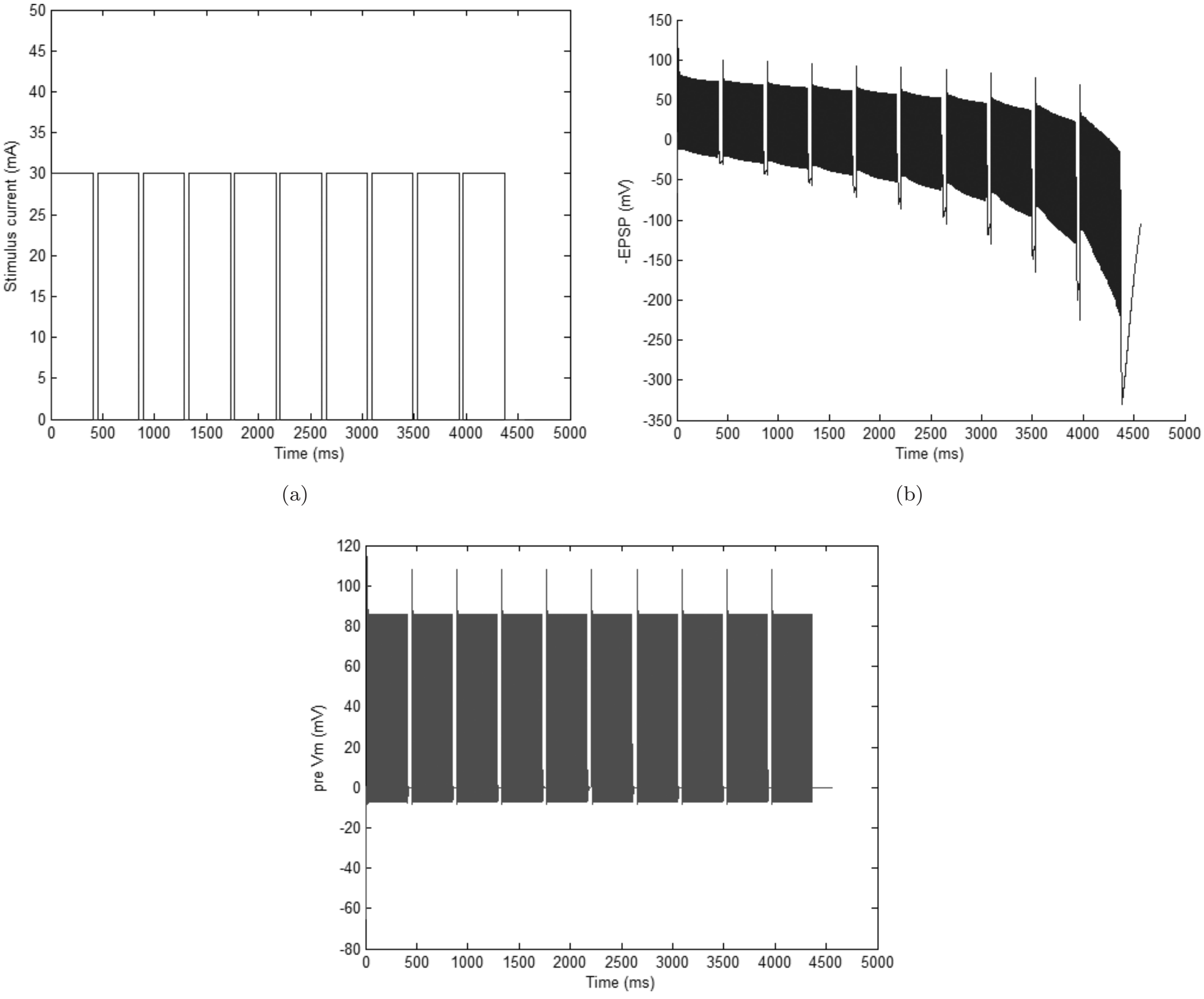

(a)

(b)

(c)

Fig. 4. Successive applications of a $30\,\mu$A stimulus current (a) results in habituation becoming successively more rapid and profound, showing habituation potentiation[a] (b). (c) shows the presynaptic membrane potential.

while strong stimuli yield no significant habituation. This is shown in Fig. 7 where a $90\,\mu$A stimulation shows very little habituation when compared to the $30\,\mu$A stimulation.

Characteristic 6 describes the effects of habituation as accumulating even after the response has reached asymptotic levels. These effects of accumulation of habituation include a delay in the onset of spontaneous recovery.[35] This is shown in Fig. 8 where a 200 ms stimulus causes a 4.1 ms delay in recovery of a synapse set to learn until asymptotic level $AP_{\max} = 75\,\text{Hz}$, when compared to a synapse whose habituation has not yet reached asymptotic level, $AP_{\max} = 175\,\text{Hz}$. In general, the delay in the onset of recovery is inversely proportional to the value of $AP_{\max}$.

Characteristic 7 refers to the stimulus generalization of the habituation response. The habituation

---

[a] For the purpose of analyzing how habituation to asymptotic levels behaves in the model, the values of g_{syn} have been unbounded. This causes the values of V_m to reach unbiological values of $-700\,\text{mV}$ or less. One way of getting around this problem is to bound the synaptic conductance by using a sigmoid function. However, this will not allow us to explore the behavior of the model in response to the beyond asymptotic habituation that is observed in biology. At the loss of Characteristic 5, setting $AP_{\max}$ to a large value will keep keep V_m within a bioplausible range.

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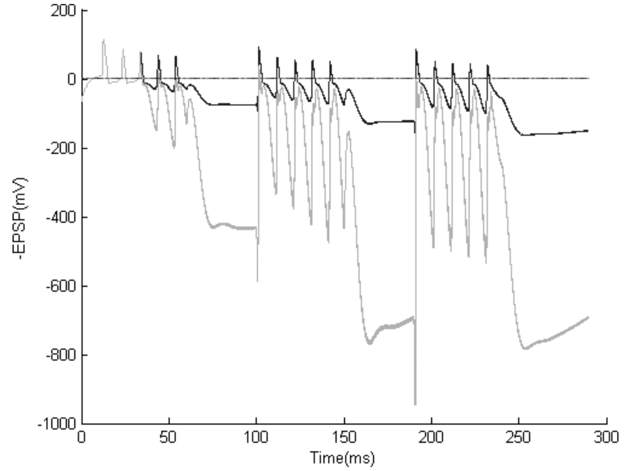


Fig. 5. The recovery rate a synapse habituated to asymptotic levels (gray) is higher than the recovery rate of a synapse whose habituation has not reached asymptotic levels (black).

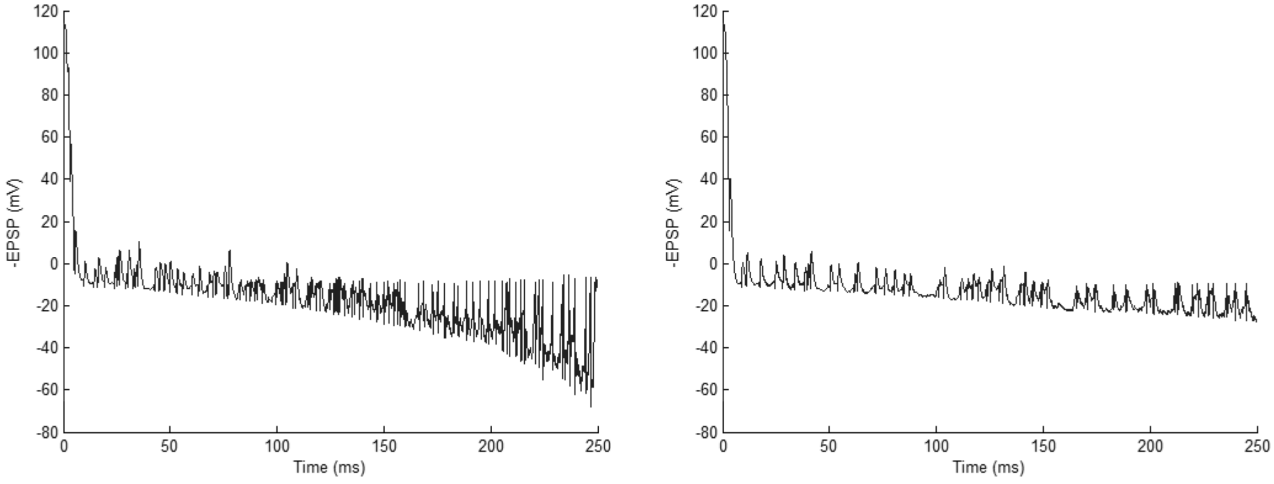
response to a given stimulus exhibits stimulus generalization to other stimuli. While this characteristic implies that stimulus generalization is a central process,[18] our model shows that some generalization also occurs in the presynaptic terminal of the sensory neuron. When a neuron ($AP_{\max} = 100\,\text{Hz}$), is stimulated using a $30\,\mu$A stimulus, followed by a $50\,\mu$A stimulus, a continued habituation to the $50\,\mu$A stimulus is observed and the habituation is more pronounced (Fig. 9(b)) with a minimum postsynaptic potential of $-400\,$mV. Whereas, when the sensory neuron is stimulated with two $30\,\mu$A stimuli, the habituation is less pronounced, with a minimum postsynaptic potential of $-350\,$mV (Fig. 9(a)). When the habituation to the $50\,\mu$A stimulus in Fig. 9(b) is compared to the habituation to a $50\,\mu$A stimulus presented alone (the two postsynaptic responses are compared in Fig. 9(c) on the black and gray plots, respectively), the effect of generalization overrides the effect of intensity as described in characteristic 5.

Characteristic 8 refers to dishabituation, where the presentation of a stronger stimulus results in the recovery of a habituated response. This is shown in Fig. 10(b), where the presentation of a $90\,\mu$A stimulation results in the dishabituation of the response of the original $30\,\mu$A stimulus. The application of the second $30\,\mu$A stimulus results in a minimum habituation response of $-94\,$mV without dishabituation (Fig. 10(a)) and $-55\,$mA with dishabituation.

The final characteristic described by Rankin *et al.*, with reference to short term habituation is that of habituation of dishabituation. The phenomenon of habituation of a dishabituating response, as it applies to this model was discussed in characteristic 1 (Fig. 3(b)).

4.2. *Simulations using multiple inputs*

Sensitization[15] occurs when an amplification of response follows the repeated administration of a stimulus.[36] As a result of sensitization, the behavioral response of the synapse is enhanced to



(a) (b)

Fig. 6. A stimulus with an average spike rate of 200 Hz (a) shows more profound habituation than a stimulus with an average spike rate of 100 Hz (b). Both stimuli were Poisson spike trains to the habituating synapse.

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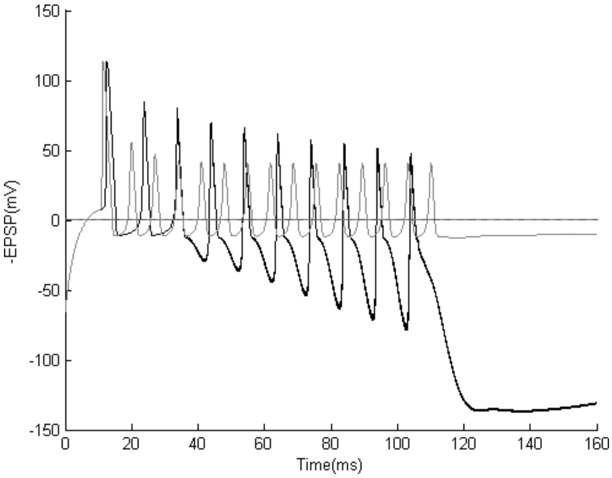

Fig. 7. A 90 μA input (gray) stimulus results is less (even negligible) habituation, when compared to a 30 μA stimulus.

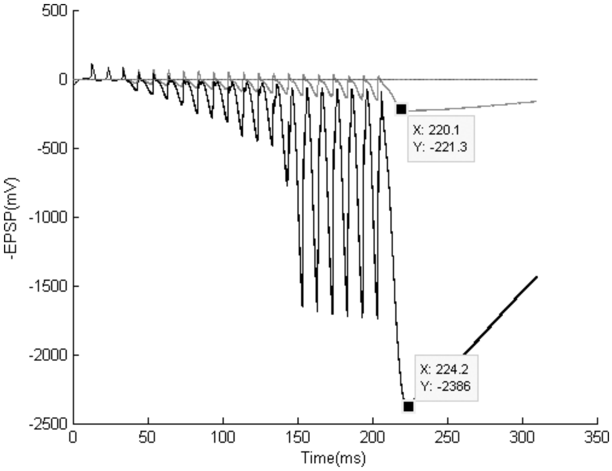

Fig. 8. Habituation continues even after asymptotic levels have been reached, resulting in a slight delay in the start of response recovery (black) when compared to the case when asymptotic levels have not been reached (gray).

any stimulus presented shortly after the sensitizing stimulus. In this section we describe a preliminary simulation when the proposed synaptic model is connected to two inputs, one of which is sensitizing. The circuit diagram used in the simulation is given below.

The spike effect $SE_{t,s}$ of the sensitization neuron is computed similar to Eq. (10) as given below:

$$SE_{t,s} = \frac{SE_{d,t} - SE_{o,t}}{g_{\mathrm{syn},t-\Delta t} - e^{(-t_{AP,k}+t)}} \tag{20}$$

Figure 11(b) shows the postsynaptic membrane potential and verifies biological behavior in that the sensitization response takes priority over the habituation response.

5. Simulations on the Short Term Memory of Habituation Resulting Due to the Connection Between the Persistent Firing Neuron and the Habituating Synapse

The process of working memory is, as mentioned in Sec. 2, thought to be related to persistent activity in the neural network. We therefore attempted to model the change in the synaptic parameters that led to retention of memory when the persistent firing neuron was connected to the habituating synapse. The model makes two predictions about the contribution of persistent activity to the retention of habituation response. The first prediction is that when the synapse has not reached asymptotic levels of habituation, persistent firing leads to continued habituation. The resulting synapse shows a more profound habituation. Figure 12(c) shows the continued decrease in synaptic conductance even after the stop of the stimulus, due to the onset of persistent firing ($AP_{\max} = 100\,\mathrm{Hz}$). This, in turn leads to a lower postsynaptic response (Fig. 12(d)) at the end of the persistent firing process.

When all other conditions are equal and $AP_{\max} = 300\,\mathrm{Hz}$, while the synaptic conductance continues to decrease such that the postsynaptic membrane potential reaches $-20\,\mathrm{mV}$, its effect is not as profound as that for $AP_{\max} = 100\,\mathrm{Hz}$ ($-150\,\mathrm{mV}$) (Figs. 12(e) and 12(f)).

On the other hand, when $AP_{\max} = 25\,\mathrm{Hz}$, the synapse shows the retention of the habituation response, but does not continue learning, as it has already reached asymptotic values (Figs. 12(g) and 12(h)).

With reference to Fig. 12(c), we observe changes in the synaptic conductance through the various stages of operation as discussed in Sec. 3. From the start of the simulation to 2200 ms, the input to the sensory neuron consists of pulses. This corresponds to Case 2 in the analysis presented in Sec. 3.2. Here, the synapse displays potentiation of habituation (Characteristic 3 of Sec. 4).

The stimulus current is removed at 3250 ms. From 2200 ms to 3250 ms, the behavior of the system can

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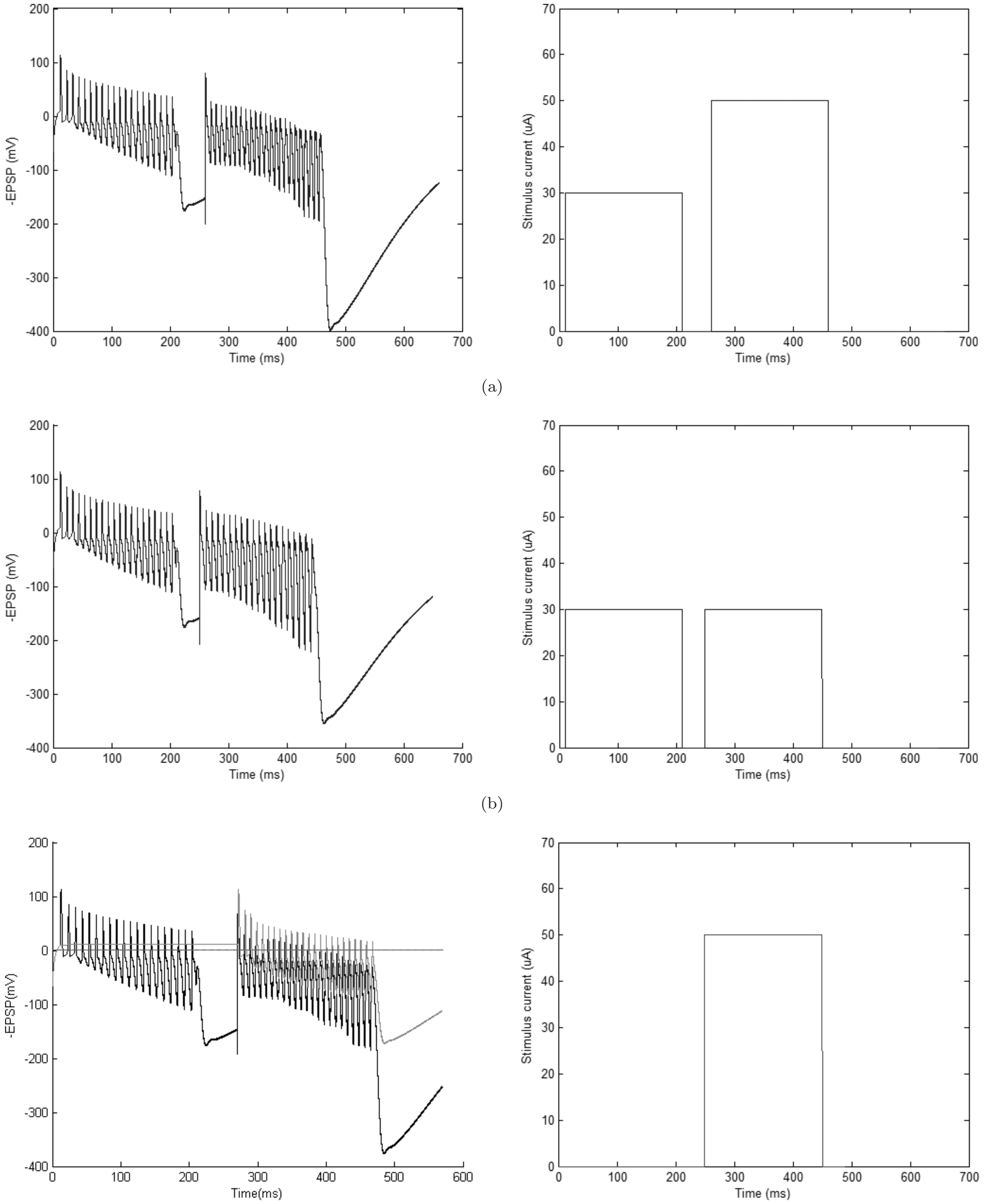

(a)

(b)

(c)

Fig. 9. Habituation to a 30 μA stimulus shows a more profound habituation to a 50 μA stimulus (a). The habituation to the new stimulus is more profound than the habituation to another 30 μA stimulus (b) and does not display the intensity effects associated with habituation characteristic 5 (c).

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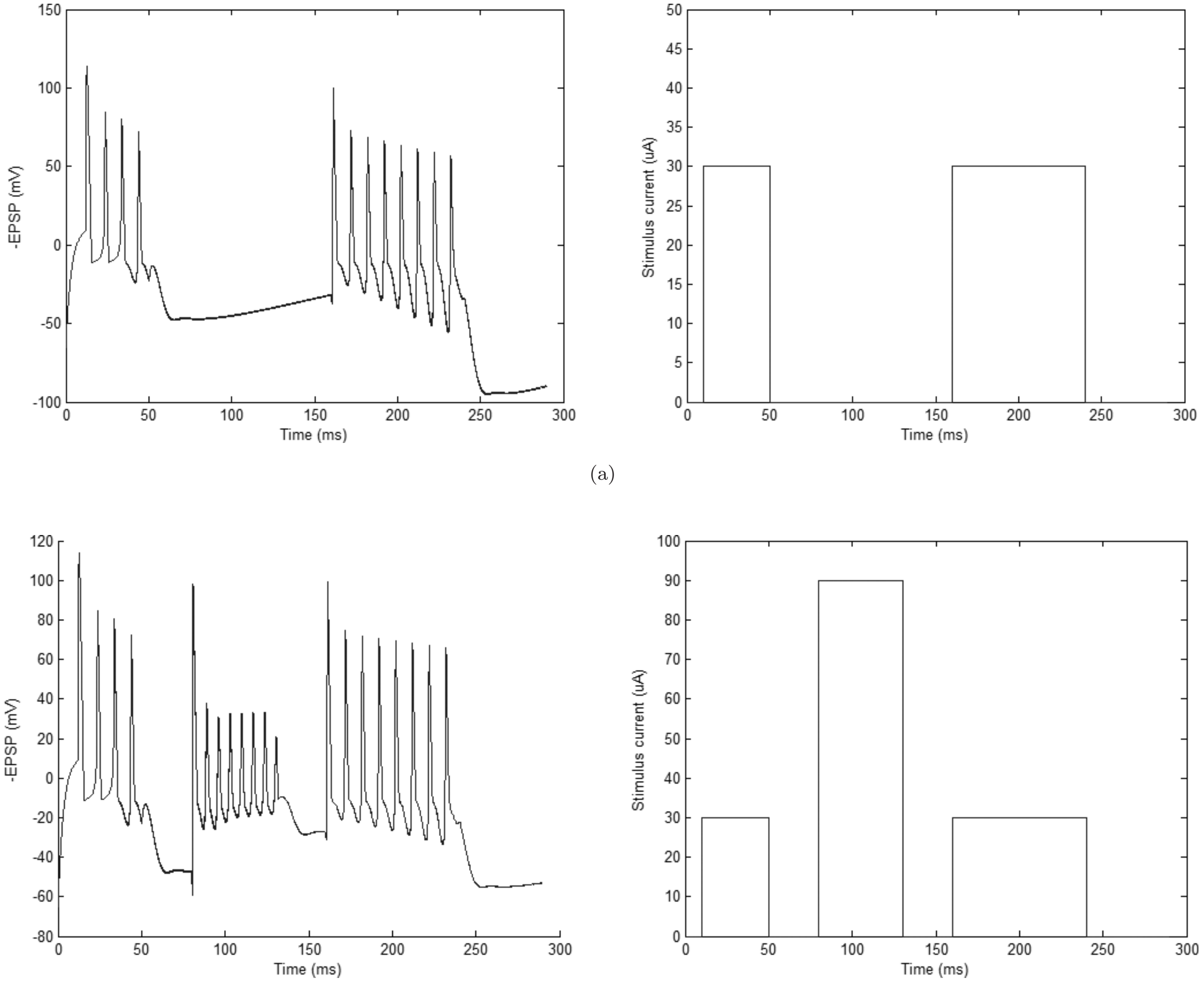

(a)

(b)

Fig. 10. There is no dishabituation in the absence of a stimulus (a) but dishabituation of the habituated response results in the presence of a stronger stimulus (b).

be explained by Case 3 in Sec. 3.2. The presynaptic input is a series of spikes, whose frequency oscillates between high and low depending on the presence or absence of stimulus. The change in synaptic conductance and the change in presynaptic membrane potential are shown in Fig. 13. A few points should be noted here. The model behaves to this change in stimulus frequency in a way which reflects characteristics 1, 3 and 4 of habituation, showing more rapid habituation during the period of high frequency stimulus (indicated by a marked reduction in synaptic conductance during the period). It also shows dishabituation to the novel stimulus before continued habituation, where the synaptic conductance increases in response to the low frequency persistent firing stimulus before continuing to decrease. Finally, the model shows habituation potentiation as the habituation and dishabituation during the phase of oscillating input frequency result in an overall reduction in synaptic conductance.

Once the stimulus current is removed, the behavior of the model switches to Case 4 of the analysis in Sec. 3.2.

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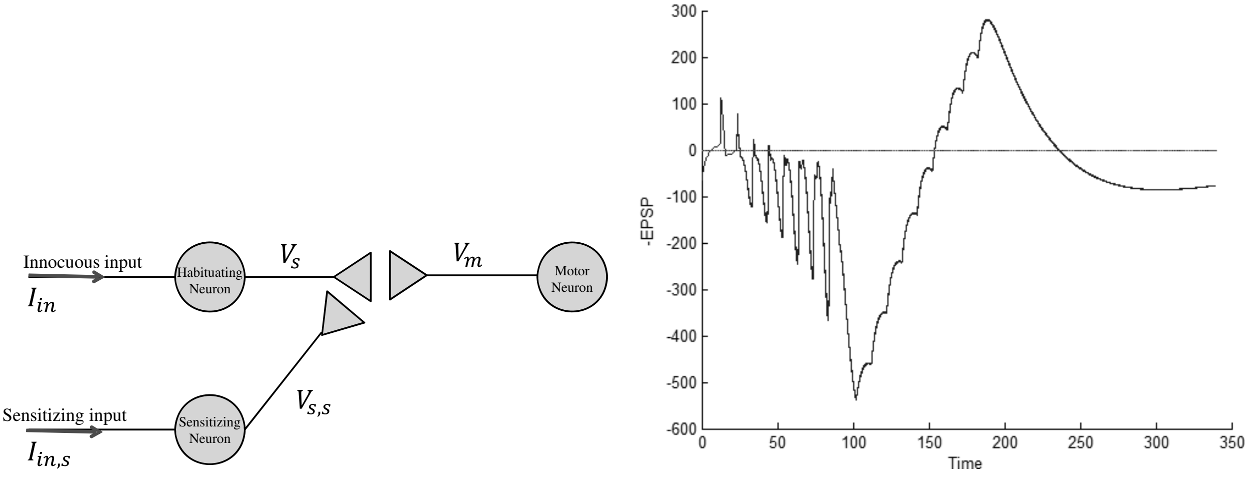
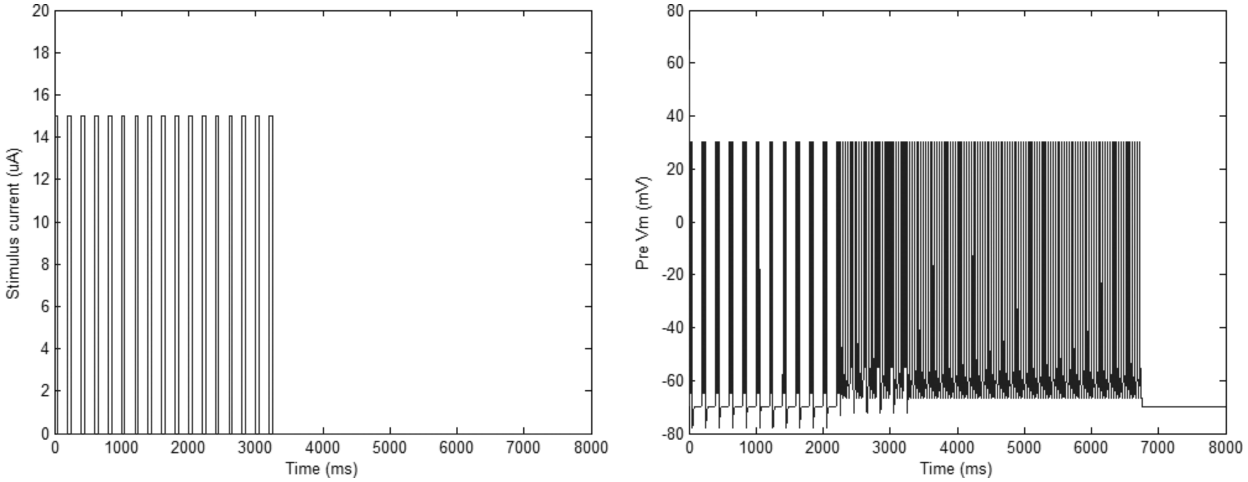


(a) (b)

Fig. 11. (a) Model of the synapse with multiple inputs. $I_{\text{in},s}$ is the strong sensitizing input and $V_{s,s}$ is the resulting membrane potential of the sensory neuron. The sensory neuron makes a synapse with the presynaptic terminal of the habituating neuron. (b) Postsynaptic membrane potential showing response of the synapse to a $50\,\mu$A, 80 ms stimulus to the habituating synapse followed by a 100 ms, $30\,\mu$A stimulus to the sensitization neuron.



(a) (b)

Fig. 12. Retention and continued training of habituation response due to persistent firing in the axon. (a) Input stimulus is 17 repetitions of an input pulse of $15\,\mu$A. (b) The presynaptic membrane potential of the sensory neuron, as computed using the persistent firing neuron model. (c) The conductance of the habituating synapse ($AP_{\max} = 100$ Hz) showing the conductance decreasing as a result of the persistent firing. (d) The postsynaptic membrane potential (PSP) for the motor neuron connected to the neuron in (c). (e) The conductance of the habituating synapse ($AP_{\max} = 300$ Hz) showing the conductance decreasing as a result of the persistent firing. (f) The PSP for the motor neuron connected to the neuron in (e). (g) The conductance of the habituating synapse ($AP_{\max} = 25$ Hz) showing the retention of habituation response at asymptotic level due to the persistent firing. (h) The PSP for the motor neuron connected to the neuron in (g).[b]

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[b] As reflected in Fig. 12(h), the model has a singular point when $AP_t \to AP_{\max}$, $g_{\text{syn}} \to -\infty$. This can be overcome by setting $AP_{\max}$ to a large value, as is shown in Figs. 12(d) and 12(f).

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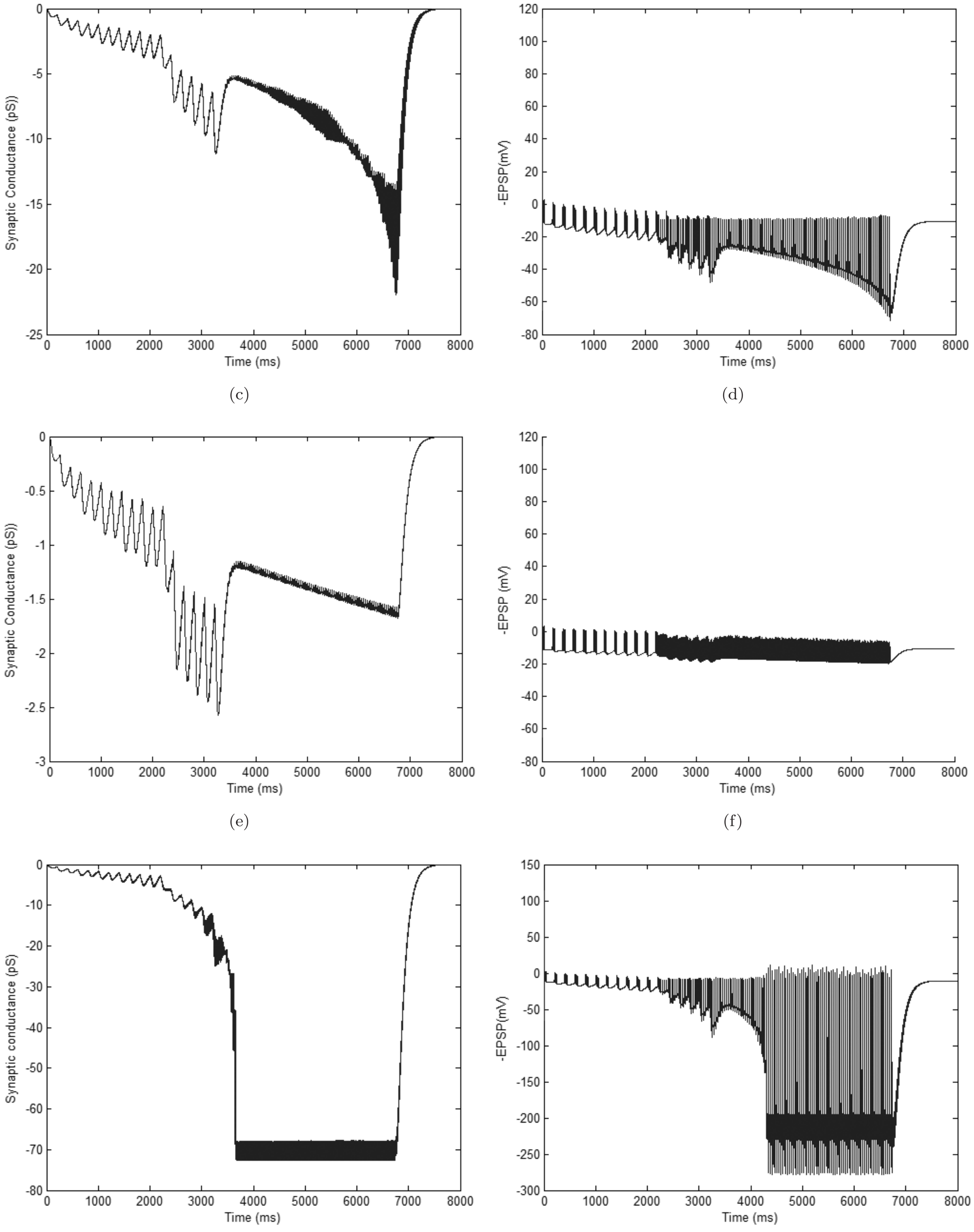

(c)

(d)

(e)

(f)

(g)

(h)

Fig. 12. (*Continued*)

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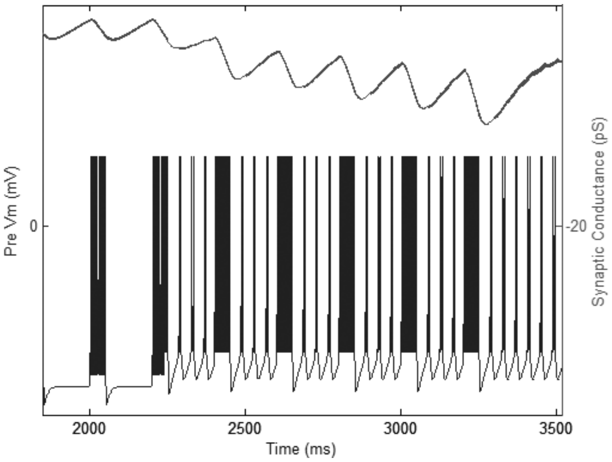

Fig. 13. Change in synaptic conductance corresponding to the change in membrane potential when the sensory neuron oscillates between two firing frequencies corresponding to persistent firing and persistent and somatic firing. This figure is a zoomed in view of Fig. 12(c) from 1800 ms to 3550 ms.

6. Discussions

6.1. *The model and results*

In this paper, a model of an element cell for short term memory was proposed. The model consists of two parts, a persistent firing sensory neuron and a habituating synapse. The sensory neuron was modeled by extending the Izhikevich neuron model so as to enable its operation is two states — somatic firing and axonal persistent firing. We have analyzed the model to provide an optimal range of parameters for the sensory neuron so as to enable persistent firing in a stable system. The second part of the model is the habituating synapse which is faithful to the revised behavioral characteristics of habituation.

The goal of proposing the model of synaptic habituation was to fit it with all the revised and updated characteristics of habituation. To that effect, the equations proposed have been successful. But, since we have limited the inputs of our model to the timings of the action potentials and the membrane potential of the presynaptic axon, the model exhibits non biological singularities when the average firing rate of the presynaptic neuron approaches the preset maximal firing rate. These have been discussed in Secs. 4.1 and 5.

Work is currently in progress to compensate this effect by adding new decisional features to the neuron model. The results presented in this paper constitute the basis for the modified neuron models as they have provided a good fit to the input–output behavior of the habituating synapse.

Further, the model predicts that if the persistent firing starts before the stimulus is removed, both somatic and axonal firing takes place, sending different frequencies of spikes to the synapse. The synapse distinguishes the stimuli as being different and accordingly displays habituation of dishabituation, where it goes through a phase of habituation and dishabituation, where the effect of each habituation phase is more marked than the effect of the previous phase.

6.2. *On the computational modeling of memory*

Memory, which is explained by a change in synaptic strength, is attributed to various factors, as shown in Fig. 14. It is known that both short term (working) and long term memories arise as a result of molecular processes involving a change in the amount of neurotransmitter across the synapse. However, other factors also contribute to the generation and the persistence of memory in time. These include the axonal process of persistent firing and the network level process involving reverberatory circuits[37,38] of neurons.

The intense complexity of the brain makes it difficult to study biologically. State of art technology only enables Neuroscientists to perform *in vitro* observations of the behavior and mechanisms of synapses. *In vivo* recordings, while possible, are highly invasive in nature. Computational Neuroscience, on the other hand, is an emerging field that explores the boundary between Neuroscience and Computation. Using computational modeling, it is possible to architect neural circuits that have, as yet, not been identified in biological systems.

The model described in this paper, though bio-inspired, has not been biologically validated and one aspect of future research involves the empirical validation of the model in wet lab studies. With the proposed theoretical model, it is now possible to design better bio-inspired systems as habituation is closely related to the concepts of novelty detection and attention, which, in humans, help to improve learning and filter out irrelevant information. The implementation of persistent firing adds a dimension of working memory to the circuit. Cognitively, the addition of working memory corresponds to better

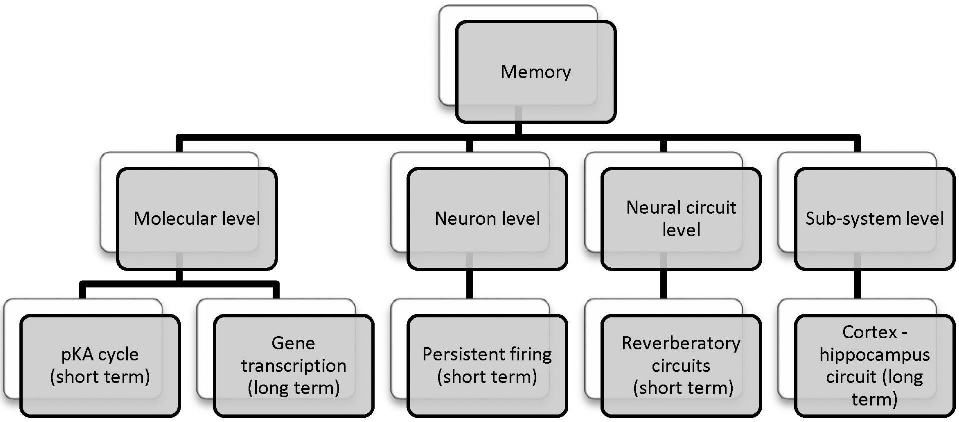

Fig. 14. The different processes involved in memory.

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habituation to similar stimuli and less dishabituation to the primary stimulus when the system is exposed to novel, innocuous stimuli. This enables the system to distinguish faster, the differences between two different stimuli.

6.3. *Towards the development of Artificial Cognitive Memory*

The recent upsurge of research into cognitive systems has prompted us to propose new data storage architecture and a new development trend that is different from the currently implemented density driven approaches to memory.[22] We are working towards the development of Artificial Cognitive Memory (ACM),[22] a system inspired by the versatile nature of human memory. In the brain, information is stored in a nonbinary, structure based, adaptive, and experience-dependent manner and is retrieved using contextual cues. Human memory is also encoded in a-modal, invariant representation format, storing information in a parallel distributed and hierarchical fashion. In our earlier paper,[22] we proposed the concept of element cells, fundamental information processing units, which can then be connected together to form a larger model of cognition, such as a model of semantic representation.[39] Such element cells are created as a combination of suitable material "molecular" synapses and neurons, as shown in Fig. 15. It is predicted that molecular level modeling of neurons and synapses in such devices can dramatically increase storage capabilities.[40]

This paper makes the following contributions to the design the element cell for ACM. First, it

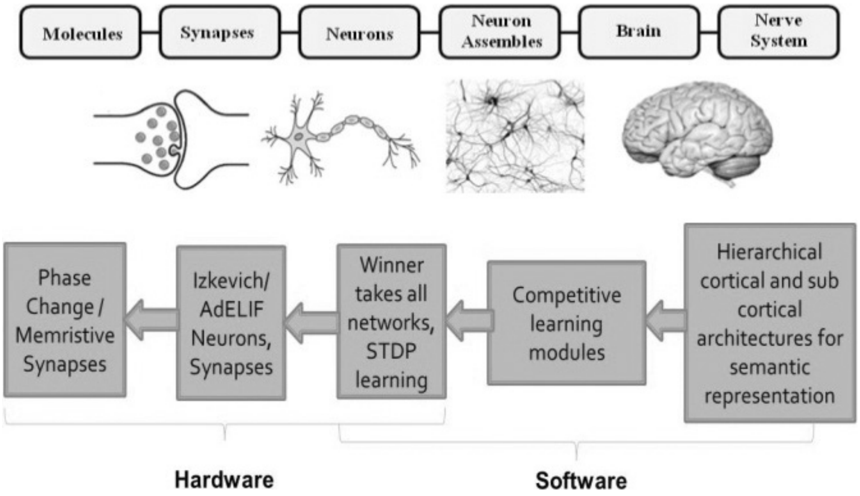

Fig. 15. Spatial organization of the human brain and the corresponding proposed framework for developing ACM.

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proposes a mathematical model of habituation to be implemented as a synapse. Second, it integrates the model of habituation with a persistent firing neuron which is thought to be important in the processes of working memory, thereby increasing the retention time of the synaptic conductance and therefore the recovery time, and continuing the learning of short term habituation for the duration of persistent firing. This, in turn, leads to a working memory for habituation.

ACM approaches the problem of a bio-inspired memory system based on the principles of cortical uniformity and hierarchy, under a materialistic assumption.[41] As such, work is in progress in different areas of research, including the architecture of the element cell,[23] the module and subsystems.[39] The implementation of working memory with a synaptic model in a single neuron-synapse element cell model is part of the cellular level modeling and the first step towards the realization of the element cell. At the hardware level, it remains for us to design the optimal circuit for realizing the element cell using the right combination of emerging materials and devices. At the system level, the authors are working on integrating habituation into the module and system architecture to perform cognitive tasks such as implicit representation and semantic memory.[42]

Further, spiking neural networks are increasingly being used to perform machine classification, knowledge extraction, and robotics.[33,43–47] In the context of spike-based networks for machine learning, modeling habituation is desirable for several reasons.

First, as mentioned earlier, habituation is an important part of novelty detection and therefore attention focus. Furthermore, like attention, habituation can also be classified into two kinds, stimulus-dependent (non-associative, bottom-up) habituation which is modeled in this paper, and context-dependent (associative, top down) habituation.[48] It is therefore possible to propose system level models of habituation,[42] similar and complementary to those of attention.[49] Habituation is therefore essential in filtering out irrelevant information and, together with attention; it influences learning and information processing in biological systems. We therefore expect that a bio-plausible, spike driven habituating synapse implementing short term habituation will be an asset in machine learning algorithms.[33]

7. Conclusion

Amongst ongoing developments to the element cell design, the work presented in this paper represents a significant contribution to research in the domain of spiking neural networks. In particular, we highlight that while STDP is a powerful concept that stores weights based on the causal relationship which is being learnt, other synaptic mechanisms are also in place to facilitate the process of learning and memory. Habituation is one such mechanism and in this paper, we have proposed and analyzed a model of short term persistent habituation, consisting of a persistent firing neuron and a habituating synapse. A mathematical model of habituation is described and further integrated with a persistent firing neuron. We have explored the model of habituation and made predictions on how persistent firing can contribute to a short term memory for habituation.

While the cascade model of the sensory neuron and habituating synapse proved successful in demonstrating the behavioral properties of short term habituation and as a tool for the analysis of the neural correlates of short term habituation, there are many subsequent steps to this work. These include the circuit level implementation of the element cell for its incorporation into ACM, and continued modeling of synaptic processes. Looming over these is the question of how a memory for habituation can help the ACM in filtering out redundant data and enable function driven memory storage.

We are confident that the proposed model of the interaction of the presynaptic processes of habituation and persistent firing paves a new way to explore information processing and memory encoding in the presynaptic neuron, and adds a building block to the research of ACM.

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