ASSOCIATIVE NEURAL NETWORK MODEL FOR THE
GENERATION OF TEMPORAL PATTERNS

Theory and Application to Central Pattern Generators

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ABSTRACT Cyclic patterns of motor neuron activity are involved in the production of many rhythmic movements, such as walking, swimming, and scratching. These movements are controlled by neural circuits referred to as central pattern generators (CPGs). Some of these circuits function in the absence of both internal pacemakers and external feedback. We describe an associative neural network model whose dynamic behavior is similar to that of CPGs. The theory predicts the strength of all possible connections between pairs of neurons on the basis of the outputs of the CPG. It also allows the mean operating levels of the neurons to be deduced from the measured synaptic strengths between the pairs of neurons. We apply our theory to the CPG controlling escape swimming in the mollusk Tritonia diomedea. The basic rhythmic behavior is shown to be consistent with a simplified model that approximates neurons as threshold units and slow synaptic responses as elementary time delays. The model we describe may have relevance to other fixed action behaviors, as well as to the learning, recall, and recognition of temporally ordered information.

INTRODUCTION

The collective properties of highly interconnected networks of model neurons have been the focus of much theoretical analysis. Recent work on this topic involves networks whose dynamics is governed by a cooperative relaxation process (1–5). Starting from an initial state, these networks will relax to one of a select number of stable states. Network models of this form have been used for associative memory (1) and for solving certain optimization problems (6–8). The final, stable states represent the retrieved information or the optimized configuration.

Despite some very suggestive analogies between the network models and biological computational processes, their application in biology is unclear. The difficulty in relating the models to experimental observations reflects, in part, the difficulty in identifying a cooperative relaxation process in large, complex nervous systems. Similarities between associative memory networks and central nervous functions, such as place learning in the hippocampus (9), olfaction (10–12), and visual processing (13) have been proposed. Yet the models remain untested at the level of neurophysiology.

Here we study an associative network model whose collective outputs consist of temporally coherent patterns of linear or cyclic sequences of states (14, 15). This model and its extensions may have a variety of implications for the learning and recall of temporally ordered information. Our objective here is to draw a connection between the properties of the model and biological nervous systems that produce fixed patterns of neural outputs. In particular, we focus on a class of biological systems known as central pattern generators (CPGs).

CPGs control the muscles involved in executing well defined rhythmic behaviors, such as breathing, chewing, walking, swimming, and scratching. Some networks forming CPGs are anatomically well localized and may contain small numbers of neurons. Their output consists of coherent, oscillatory patterns. These features make CPGs strong candidates for studying the relation between the output properties of a biological network and its underlying circuitry.

A number of basic principles about CPGs have emerged from studies on a wide variety of rhythmic behaviors (16–21): (a) A rhythmic neural output can occur in the absence of sensory feedback from the muscles and structures controlled by the CPG, and in the absence of control by higher neural centers. These features are clearly demonstrated with spinal preparations (22), i.e., isolated segments of spinal cord. The output activity of the motor neurons in these preparations is similar to the rhythmic firing pattern observed in the intact animal. (b) Some CPGs function without a pacemaker cell, i.e., a single neuron whose firing rate determines the output period of the network. This implies that the rhythmic output is a collective property of the network. Examples include the CPG that controls swimming in the mollusk Tritonia.
(T_{ij}^1 \neq T_{ij}^1), while the T_{ij}^S synapses, which depend only on the activity within the individual states, are symmetric.

The rule for forming the T_{ij}^1 synapses (Eq. 2) encodes transitions between consecutive pairs of embedded states. This allows the network to generate either linear sequences, cyclic sequences, or sequences down a tree structure. Several different patterns, as well as isolated, stable states, can be embedded in the same network. Patterns that involve ambiguous transitions, such as when two patterns share the same state, cannot be reliably produced by the present network. Such patterns can, however, be incorporated by forming synapses that encode transitions between distant states along the pattern (41–43).

The rules defined by Eqs. 1 and 2 for forming the synaptic components are applicable only when the overlaps between the embedded states are small, i.e.,

\[ \frac{1}{N} \sum_{j=1}^{N} (2V_j^{\mu'} - 1)(2V_j^{\nu'} - 1) = 0 \quad (\mu, \nu = (\mu', \nu')) \quad (3) \]

and when, on average, half of the neurons are active in each of the embedded states. Rules that are appropriate for embedding overlapping states in associative networks have been described (44–51).

The integrated synaptic input to each neuron is assumed to be a linear summation of the outputs of the pre-synaptic neurons. The total synaptic input to the i-th neuron via the fast components of its synapses, \( h_i^F(t) \), is

\[ h_i^F(t) = \sum_{j=1}^{N} T_{ij}^F V_j(t). \quad (4) \]

The total synaptic input via the slow components, \( h_i^S(t) \), corresponds to a weighted average over the histories of the neural activities, with a characteristic averaging time of \( \tau_L \). It is given by

\[ h_i^S(t) = \sum_{j=1}^{N} T_{ij}^S \bar{V}_j(t), \quad (5) \]

where \( \bar{V}_j(t) \) is the time-averaged output of the neuron,

\[ \bar{V}_j(t) = \int_0^t V(t - t') w(t') \, dt'. \quad (6) \]

The synaptic response function \( w(t) \) for the slow, \( T_{ij}^S \), components is a non-negative function that is normalized to unity and characterized by a mean time-constant \( \tau_L \). An example of the time-course of a postsynaptic response to a short presynaptic stimulus is illustrated in Fig. 1 A.

Network Dynamics

Before we define the detailed dynamics of the network, we present a qualitative description in terms of the time dependence of the neural inputs. For simplicity of notation we consider a network that produces a single pattern. Immediately after a transition from the \((\mu - 1)\)-th embedded state to the \(\mu\)-th state, the output of the network is \( V(t) = V^\mu \) and the time-averaged output is \( \bar{V}(t) \approx V^\mu \).

Figure 1 Schematic representation of the model network and its components. (A) The time dependent properties of the synaptic connection from the \( \mu \)-th to the \( i \)-th neuron. We illustrate the postsynaptic response observed after a short pulse (\( \Delta t = \tau_L \)) of activity in the presynaptic neuron. The area (shaded) under the fast synaptic response for a pulsed input is equal to \( T_{ij}^F \) (Eq. 1); in this example we take \( T_{ij}^F \) to be excitatory. The area (shaded) under the slow synaptic response for a pulsed input is equal to \( T_{ij}^S \) (Eq. 2); in this example \( T_{ij}^S \) is inhibitory. The ratio of these two areas, averaged over all pairs of synapses, equals the transition strength \( \lambda \) (Eqs. 2 and 3). The time-course of the slow synaptic response corresponds to the response function \( w(t) \) (Eq. 5); it has a characteristic time-constant of \( \tau_L \). (B) Illustration of a saturating gain function for a neuron. This function relates the output, or firing frequency of a neuron, \( V_i(t) \), to the value of its net input, \( u_i(t) \), and its mean operating level, \( \bar{u} \) (Eq. 8). The output of a neuron is most sensitive to changes in its inputs when \( u_i(t) - \bar{u} \). (C) The equivalent circuit describing the model network (Eq. 6). Neurons (\( N_i \)) are represented by saturating amplifiers, as in part B, with a charging time of \( RC = \tau_L \), where \( R \) represents the net input resistance of the neuron and \( C \) represents the input capacitance. Synaptic connections between each pair of neurons are represented by conductances (\( + \)-\( - \)) proportional to \( T_{ij}^F \) or \( T_{ij}^S \); their dynamic properties are illustrated in part A.

The inputs via the fast synaptic components are

\[ h_i^F(t) = \sum_{j=1}^{N} T_{ij}^F V_j^{\mu} = \frac{J_0}{2} (2V^\mu - 1), \quad (7) \]

where we used Eqs. 1 and 4 and assumed that the overlap between the \( V^\mu \) 's are small. The synaptic input \( h_i^F(t) \) is negative, i.e., inhibitory, if \( V^\mu = 0 \) (quiescent) and is positive, i.e., excitatory, if \( V^\mu = 1 \) (maximally firing). The inputs via the slow synaptic components are (Eqs. 2, 5, and 6)

\[ h_i^S(t) = \sum_{j=1}^{N} T_{ij}^S V_j^{\mu - 1} = \frac{J_0}{2} (2V^\mu - 1). \quad (8) \]
Fig. 2 A depicts the output pattern from 8 of the 100 neurons. The output is presented in the form of a spike pattern; the individual spikes were generated by a stochastic process in which the output $V_i(t)$ represented the probability that the $i$th neuron fired in an interval $\tau_s$. This stochastic process may represent rapid fluctuations in cellular or synaptic parameters that control the precise timing of the spike generation. The details of the temporal relation between the synaptic inputs and the output for a particular ($i - 8$) neuron are illustrated in Fig. 2 B; the remainder of the neurons exhibited a similar pattern.

Biphasic Oscillations

A particularly simple pattern is one that oscillates between an embedded state $V^u = [V^u_i]_{i=1}^{N}$ and its antiphase, $(1 - V^u_i)$, in which the quiescent neurons are now firing and vice versa. Multiple patterns of this form can be embedded in our network. The resulting synaptic strengths are (Eqs. 1 and 2)

$$T_{ij}^u = \frac{J_0}{N} \sum_{\lambda=1}^{k} (2V_i^\tau - 1)(2V_j^\tau - 1), \quad i \neq j$$  \hspace{1cm} (14)

and

$$T_{ij}^r - \lambda \frac{J_0}{N} \sum_{\lambda=1}^{k} [2(1 - V_i^\tau) - 1](2V_j^\tau - 1)$$

$$- - \lambda T_{ij}^u, \quad i \neq j,$$  \hspace{1cm} (15)

where $k$ is the number of patterns and $T_{ij}^u - T_{ij}^r = 0$.

Although the synaptic components $T_{ij}^u$ are, in general, asymmetric (i.e., $T_{ij}^u \neq T_{ji}^u$) they are symmetric for the special case of biphasic oscillations (cf. Eqs. 2 and 15). The relation $T_{ij}^r = -\lambda T_{ij}^u$ implies that the connections correspond to either short-term reciprocal inhibition followed by delayed excitation, or to short-term reciprocal excitation followed by delayed inhibition. Note that the symme-

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**Figure 2** Simulation of a network containing 100 neurons with nine embedded states. These states were arranged as a single isolated state, a cyclic pattern among five states, and a cyclic pattern among three states. In this simulation the output of each neuron is either quiescent or firing near its maximum rate. This feature of the output behavior results from the saturation characteristics of the neuron gain function. Other choices for a gain function can lead to stable output patterns in which the firing rate of the neurons does not saturate. (A) The firing pattern calculated from the outputs $V(t)$ of 8 of the 100 neurons in the network; the remainder of the neurons showed a similar firing pattern. The network was initially in the isolated, stable state $V^{12}$. At time $t$, an external input, $I(t)$, was applied for a time $\tau_t$. This input drove the network into state $V^{12}$ and thus initiated the $(r = 2)$-th pattern. The heavy lines at the top of the figure correspond to the output period of each pattern. (B) Details of the dynamic behavior of the $(i = 8)$-th neuron for the period of time delineated by the box in part A. Shown are the inputs from the fast synaptic components, $h^r_i(t)$, the inputs from the slow synaptic components, $h^u_i(t)$, the net synaptic input, $u_i(t)$, and the output of the neuron, $V_i(t)$. The dynamic equations for the network (Eqs. 4 and 8) were approximated using finite difference techniques. The firing rate of the neurons was taken to be described by a sigmoid gain function, i.e., $V_i(t) = 1/2(1 + \tanh [G_i(u_i(t) - \theta)])$, where $\theta$ given by Eq. 8 with $\Delta_i = 0$ and $G$ is the gain constant. In this simulation we used $G_i = J$ and chose $\theta = 0$.

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We made a crude classification of the observed synaptic strengths in *Tritonia* based on the pairwise measurements of Getting (23, 25) and on Getting’s detailed analysis (57) of the time dependence of the synaptic response. The observed response was classified as either a fast component, $T_0^{s}$, or a slow component, $T_0^{l}$, according to the time-scale of the decay of the observed synaptic response. Synaptic components that decayed on a time-scale $< 1$ s were designated as fast whereas synaptic components that decayed on a time-scale substantially $> 1$ s were designated as slow.

In our simple analysis, we have considered primarily the sign of the measured post-synaptic response. Thus detailed variations between the values of the individual $T_0^{s}$ connection strengths and between the $T_0^{l}$ connection strengths were neglected. For example, the synaptic connection from $C2$ to $DSI$ (Fig. 3B) was parameterized by the values $T_0^{s} = +J_0/4$ and $T_0^{l} = -\lambda J_0/4$. Nevertheless we have not included synaptic components whose strengths are considerably weaker than the rest. The complete set of connection strengths $T_0^{s}$ and $T_0^{l}$ are summarized in Table I; a more detailed discussion of the assignments is given later. These synaptic strengths were used to construct the equivalent circuit shown in Fig. 4A.

The determination of the value of the transition strength, $\lambda$, involves a considerable degree of uncertainty. This uncertainty reflects, in part, the difficulty in separating the fast and slow components that contribute to the measured synaptic response. We have considered values of $\lambda$ in the range $\lambda = 5$–10 in our simulations. A large value for $\lambda$ appears to be consistent with the magnitude of the slow versus the fast response observed in some of the synapses, e.g., Fig. 3B.

The signs of the experimentally observed synaptic

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**Table 1**

<table>
<thead>
<tr>
<th>SYNAPTIC CONNECTION STRENGTHS FOR TRITonia</th>
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<tr>
<td><strong>Fast synaptic components, $T_0^s$</strong></td>
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<tr>
<td>------------------------------------------</td>
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<tr>
<td>$0 \quad +1 \quad -1 \quad -1$</td>
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<tr>
<td>$+1 \quad 0 \quad -1 \quad -1$</td>
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<tr>
<td>$-1 \quad -1 \quad +1 \quad 0$</td>
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<tr>
<td>$J_0$</td>
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<tr>
<td>$\frac{4}{6}$</td>
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<td><strong>Theory</strong></td>
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<tr>
<td>$0 \quad +1 \quad \bullet \quad -1$</td>
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<tr>
<td>$+1 \quad 0 \quad -1 \quad -1$</td>
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<tr>
<td>$-1 \quad -1 \quad +1 \quad 0$</td>
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<tr>
<td>$\bullet \quad -1 \quad \bullet \quad 0$</td>
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The synaptic connection strengths for *Tritonia*. The theoretical values were found using Eqs. 1, 2, and 10. The observed values were abstracted from the data of Getting (23,25); filled circles indicate connections that are not present in *Tritonia*.
The simplified analysis presented above suggests that the mechanism for rhythmic output proposed by the model may be applicable to the CPG in Tritonia. To further ascertain the correspondence between the model and the observed properties of this CPG, we studied the dynamics of the model network using parameters appropriate for Tritonia. Eqs. 11–13 were simulated using the observed connection strengths (Fig. 4 A), analog neurons (Fig. 1 B) and a synaptic response function \( w(t) \) that decays exponentially over time; this function approximates the response observed in Tritonia (57). Stable oscillations of the form described by the previous simplified analysis (Eqs. 17–19) were observed. The output activity for the transition strength \( \lambda = 10 \) is shown in Fig. 4 B.

The period of the rhythmic output, \( 2t_o \), depends on the values of \( \tau_L, \lambda \), and on the form of \( w(t) \). We estimated the value of \( t_o \), deduced from the theory by both analytical and numerical methods using \( \tau_S \leq 0.5 \text{ s} \), the observed range of \( \tau_L = 2 \text{ s} \) to \( 5 \text{ s} \) (57), \( \lambda = 5 \) to \( 10 \), and \( w(t) \) as described above. The calculated period was \( 2 < 2t_o/\tau_L < 4 \) for the range of parameters \( 5 \leq \lambda \leq 10 \) and \( 5 \leq \tau_L/\tau_S \leq 10 \); this implies \( 2t_o = 4 \text{ s} \) to \( 20 \text{ s} \). The lower estimate, corresponding to \( \lambda = 10 \), is in accord with the experimental value (25) of \( 2t_o = 6 \text{ s} \) to \( 10 \text{ s} \) (Fig. 3 A).

**Neuron Operating Levels**

We now consider the issue of the mean operating level of each neuron, \( \theta_i \). In order for the network to produce a stable, rhythmic pattern, the firing rate of each neuron must be sensitive to changes in the value of its input. The values of \( \theta_i \) that optimize this sensitivity are given by Eq. 13. For the connections in Tritonia, this relation becomes

\[
\theta_i = I_i + \frac{1}{2} \sum_{j=1}^{L} \left( T_{i,j}^h + T_{i,j}^l \right) - I_i + \frac{J_{i,j}}{8} \begin{pmatrix} 0 & -1 - \lambda \\ -1 + 2\lambda & -1 + \lambda \end{pmatrix}
\]

(20)

with \( \lambda > 3 \). Consider the DSI neuron first (\( i = 2 \)). Eq. 20 implies either that this neuron should be in a tonically excited state when it is functionally isolated from its synaptic inputs (\( \theta_2 < 0 \)), or that this neuron requires an external excitatory input for the CPG to be active (\( I_2 > 0 \)). A combination of both of these features is observed in vivo (24, 39). The DSI neurons fire tonically, although at a considerably reduced rate, in isolation (24). Activation of the CPG in Tritonia requires an effective excitatory input to the DSI neurons (39). After this input is removed, the output from the CPG gradually loses its temporal coherence and the CPG becomes inactive. We next consider the VSI neurons. In the absence of synaptic inputs and external inputs, the output of VSI-B is expected to be quiescent (\( \theta_4 > 0 \)). This result is in agreement with observation (25).

The problematic neuron is VSI-A. This neuron is not known to receive an external input while the CPG is producing oscillatory output. Thus, according to Eq. 20, VSI-A should have a positive operating level. In practice, VSI-A exhibits a weak tonic output when it is functionally isolated (24). Violation of Eq. 20 suggests that the oscillations in the output of VSI-A will be less robust than that of the other neurons. This conclusion is consistent with the observed outputs, i.e., the relative change in the firing rate of VSI-A during the oscillations is smaller than that of the other neurons (Fig. 4 B).

**Assumptions in Assigning the Synaptic Strengths**

Several assumptions were made in assigning the observed connection strengths. The connection from DSI to C2 exhibits short-term excitation followed by a much weaker long-term excitation. We ignored the weak long-term effect; thus \( T_{12}^h = +J_0/4 \) and \( T_{12}^l = 0 \). Similarly we have ignored the weak, extremely slow inhibition (\( r \sim 15 \text{ s} \)) that appeared in some measurements of the synaptic coupling from VSI-B to the DSI; thus \( T_{32}^h = -J_0/4 \) and \( T_{32}^l = 0 \). This component does not appear to play a significant role in controlling the dynamics of the network on the time-scale, \( t_o < 5 \text{ s} \), of the rhythmic output (P. A. Getting—private communication).

The observed synaptic connection from the DSI to VSI-A exhibits two short-term responses as well as a long-term response. Short-term inhibition is preceded by a relatively shorter period of excitation, with the pair followed by long-term excitation. We ignored the initial, relatively short excitation and assigned \( T_{32}^h = -J_0/4 \) and \( T_{32}^l = +J_0/4 \). A different choice for the sign of \( T_{32}^h \) does not significantly affect the output pattern of the network.

The synaptic coupling between VSI-A and VSI-B could not be measured under conditions that suppressed possible indirect interactions, i.e., poly-synaptic pathways, between these neurons (25). Intracellular excitation of VSI-B caused VSI-A to weakly fire; we assigned \( T_{43}^h = +J_0/4 \) and \( T_{43}^l = 0 \). Excitation of VSI-A caused a slow depolarization in VSI-B, but did not cause it to fire. We chose \( T_{43}^h = T_{43}^l = 0 \), but one cannot rule out the possibility \( T_{43}^h = 0 \) and \( T_{43}^l > 0 \). An analysis of the network dynamics showed that stable oscillations persist if \( T_{43}^h \) is excitatory, so long as it is weaker (by ~25% or more) than the other slow synaptic components.

**Interactions Among the DSI Neurons**

There are three ipsilateral DSI neurons, connected to each other via excitatory connections (23). Noting that these cells fire in synchrony with each other when the CPG is active (23) and that functional removal of some of these cells does not affect the basic rhythmic output (39), we grouped all three DSI as a single neuron. The role of the
neurons in the CPG controlling feeding in the snail *Helisoma* cause their response time to be an order of magnitude slower than other synapses in the network (62). The converse situation occurs in the circuit controlling feeding in the mollusc *Navanax*, where the electrotonic couplings act rapidly compared with the chemically-mediated synapses (63). Synaptic delays can also result from the delays inherent in active propagation along a relatively long process and when the synaptic connections $T^I_q$ between pairs of neurons are mediated by interneurons.

Neurons may contain cellular as well as synaptic delays. Cellular delays can affect the response time of a neuron to many or all of its synaptic inputs. When the response time of the cellular delay is short compared with the slow synaptic response time, $\tau_s$, the separation of the time-scales between $\tau_s$ and $\tau_t$ is maintained and the output properties of the network model are unaffected. On the other hand, some well characterized cellular delays can be considered in terms of an effective synaptic delay. For example, the outward potassium current $I_A$ (64, 65) is partially responsible for the delayed response of the VSI-B neuron in *Tritonia* (25, 57). This current has the effect of allowing only slow excitatory inputs into VSI-B, but does not affect the time-scale of the inhibitory connections.

Lastly, our model is capable of producing rhythmic output in large networks that contain only monophasic connections. In this case, a synapse has either a fast response time or a slow response time, but not both. The strength of each synapse is chosen according to the formalized Hebb rules (Eqs. 1 and 2), but the minimum value of the transition strength, $\lambda$, depends on the relative number of fast versus slow connections (Eq. 10). This suggests that our model may be appropriate for analyzing CPGs that do not contain multiphasic synapses.

**Modulation of the Output**

The output activity of many CPGs can be initiated and modulated by external inputs from command neurons (66, 67) or from circulating neurohormones (68–70). Large changes in the period of the output can occur if the external inputs or neurohormones affect either the time-constant of the slow synaptic response, $\tau_s$, or the transition strength, $\lambda$. For example, a neuromodulator that selectively augments the strength of the slow synaptic components, or diminishes that of the fast components, will shorten the period of the output. It will be interesting to see if neurophysiological correlates for these and related predictions are found.

It should be emphasized that we have considered so far only networks with parameters, e.g., synaptic strengths, neuron operating levels, and external inputs, that do not change in time. Biologically these parameters undergo slow changes, such as increases (facilitation) or decreases (fatigue) in the values of the synaptic strengths. This slow change may modulate the overall behavior of the network.

For example, a gradual change in the mean operating levels or an external input will dephase the output pattern of a CPG. This will eventually terminate the oscillatory output, similar to the effect of the slowly decreasing tonic input to the CPG in *Tritonia* (Fig. 3 A) (39, 71).

**Learning and Plasticity**

One of the central features of the model is the simple relationship between the output patterns and the connections, i.e., the formalized Hebb learning rules (Eqs. 1 and 2). These rules allow new patterns to be embedded in the network by modifying the synapses both incrementally in time and locally in space; the change to each synapse depends only on the activities of the postsynaptic and presynaptic neurons during the learning of the new pattern. Local updating of the synapses makes the present model particularly suitable for large, complex systems that are continuously updated as patterns are modified or added. This feature also pertains to some other network models of sequence generation (41, 72–75).

We introduced the relation between the sequential form of the $T^S_q$ synapses (Eq. 2) and their slow dynamic response (Eqs. 5 and 6) as an ad-hoc assumption. These two features may, in fact, be closely related to each other. If one considers the evolution of the synaptic strengths in terms of a dynamic learning mechanism, the different final forms of the $T^S_q$ and the $T^I_q$ synaptic components may be the result of the different time-scale of their dynamic response. For example, the $T^I_q$ components can relate two experiences that are separated by the characteristic response time of the slow components, while the $T^S_q$ components can only aid in recalling the presence of either experience. It would be interesting to test this idea in a biologically plausible model of learning. Finally, we note two other potential applications of the model. One involves the relation between learning rules that depend on the history of neuronal activity and the temporal associations inherent in classical conditioning (76–78). A second involves the recognition of sequences of sensory input (15, 42, 79, 80).

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