Joining distributed pattern processing and homeostatic plasticity in recurrent on-center off-surround shunting networks: Noise, saturation, short-term memory, synaptic scaling, and BDNF

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A B S T R A C T

The activities of neurons vary within small intervals that are bounded both above and below, yet the inputs to these neurons may vary many-fold. How do networks of neurons process distributed input patterns effectively under these conditions? If a large number of input sources intermittently converge on a cell through time, then a serious design problem arises: if cell activities are sensitive to large inputs, then why do they not all saturate at their maximum values in response to large inputs and thereby become incapable of processing analog differences in inputs across an entire network? Grossberg (1973) solved this noise-saturation dilemma using neurons that obey the membrane, or shunting, equations of neurophysiology interacting in recurrent and non-recurrent on-center off-surround networks, and showed how different signal functions can influence the activity patterns that the network stores in short-term memory. These results demonstrated that maintaining a balance between excitation and inhibition in a neural network is essential to process distributed patterns of inputs and signals without experiencing the catastrophes of noise or saturation. However, shunting on-center off-surround networks only guarantee that cell activities remain sensitive to the relative sizes of inputs and recurrent signals, but not that they will use the full dynamic range that each cell can support. Additional homeostatic plasticity mechanisms are needed to anchor the activities of networks to exploit their full dynamic range. This article shows how mechanisms of synaptic scaling can be incorporated within recurrent on-center off-surround networks in such a way that their pattern processing capabilities, including the ability to make winner-take-all decisions, is preserved. This model generalizes the synaptic scaling model of van Rossum, Bi, & Turrigiano (2000) for a single cell to a pattern-processing network of shunting cells that is capable of short-term memory storage, including a representation of how BDNF may homeostatically scale the strengths of excitatory and inhibitory synapses in opposite directions.

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1. Introduction: Balancing excitation and inhibition to process patterns in neural networks

The activities of neurons vary within small intervals that are bounded both above and below, yet the inputs to these neurons may vary many-fold. How do networks of neurons process distributed input patterns effectively under these conditions? A classical example of this situation occurs during the visual perception of brightness. The retina receives luminance signals, which are a product of reflectances and illumination levels (Hurlbert, 1986; Lambert, 1760; Wyszecki & Stiles, 1982), from objects in the world. Surface reflectances, or the percentages of light reflected by a surface in each wavelength, provide information about the material properties of objects. The spatiotemporal patterning of these reflectances across a network of neurons represents objects in a scene. From these patterns of luminance signals, the visual system is able to estimate object reflectances across a scene by compensating for an immense dynamic range of mean illuminations throughout each day and night, and for a wide dynamic range of luminances across each scene.

This process of “discounting the illuminant” is not sufficient, however, to efficiently see the world, because illuminant-discounted signals may represent only the relative amounts of light that each object surface reflects to the eyes. For effective perception, the brain also needs to compute an absolute lightness scale, by a process called “anchoring”, that can represent the full-range of experience from dim moonlight to dazzling sunlight (Gilchrist, 1977, 1980; Gilchrist & Bonato, 1995; Wallach, 1948, 1976). Grossberg and Hong (2006) and Hong and Grossberg (2004) have developed a neural model of anchoring to explain and quantitatively
simulate a variety of perceptual and brain data about perceived
tightness.

The lightness anchoring problem is a special case of a much more
general problem that all neurons must face throughout life. Indeed,
at every stage of neural processing, neuronal networks receive
patterned inputs that represent many types of information.
Moreover, many different input pathways may converge on a
single target cell. Suppose that activities, or short-term memory
(STM) traces, of cells in a network are defined by \( x_1, x_2, \ldots, x_n \).
Each of the activities \( x_i \) may fluctuate within fixed finite
limits. Such a bounded operating range for each \( x_i \) has the advantage that
fixed decision criteria, such as output thresholds, can be defined.
On the other hand, if a large number of input sources intermittently
converge on a cell through time, or if an individual input can vary
greatly in its intensity through time, then a serious design problem
arises: If the activities \( x_i \) are sensitive to large inputs, then why do
not small inputs get lost in internal cellular noise? If the activities
\( x_i \) are sensitive to small inputs, then why do they not all saturate
at their maximum values in response to large inputs and thereby
become incapable of processing analog differences in inputs across
an entire network?

Grossberg (1973) has called this problem the noise-saturation
dilemma. Grossberg (1973) proved mathematically that neurons
which obey the membrane, or shunting, equations of neurophysiology
(Hodgkin, 1964) can solve the noise-saturation dilemma if their
bottom-up inputs and recurrent interactions are organized in
on-center off-surround networks. Such a network is also called a recurrent
competitive field, or RCF. In other words, RCFs keep their stored activities large enough to avoid being distorted by
internal cellular noise, yet not so large as to activate cells maximally,
saturate their responses, and destroy a record of analog input
differences. A shunting on-center off-surround network can thus contrast-normalize and preserve the analog sensitivity of its
cell activities in response to an input pattern, no matter how large the
inputs to the network may be chosen.

After this initial discovery, theorems and computer simulations
were provided for increasingly complicated non-recurrent and
recurrent shunting on-center off-surround networks to demonstrate how they respond when their interaction strengths, feedback sig-
nal functions, and other network parameters are varied. The earliest analyses of this kind include those of Elias and Grossberg
(1975) and Grossberg and Levine (1975, 1976). Specialized shunt-
ing networks have hereby been classified in terms of their spec-
cific pattern processing and memory storage properties, thereby
providing a storehouse of networks to serve as a resource for explaining and predicting a wide range of behavioral and brain
data. Such networks have also helped to solve technological prob-
lems wherein stable content-addressable memories are needed.
Increasingly general theorems have been proved, using both Lyapunov functions and functional, about how recurrent coopera-
tive–competitive networks can be designed so that they always converge to stable limiting patterns (e.g., Cohen & Grossberg, 1983
(Cohen–Grossberg theorem and Lyapunov function); Gross-
berg, 1978a, 1978b, 1980). These theorems clarify what design features are essential for effective pattern processing among many
specialized networks. The results include the Lyapunov function that was popularized by Hopfield (1984).

All of these results demonstrated that maintaining a balance
between excitation and inhibition in a neural network is essential
for the network to be able to process distributed patterns of
inputs and signals without experiencing the catastrophes of
noise or saturation. The results also show that the simplest
properties of shunting on-center off-surround networks can only
ensure that cell activities remain sensitive to the relative
sizes of inputs and recurrent signals, but not that they will use the
full dynamic range that each cell can support. In other words,
additional mechanisms are needed to “anchor” the activities of
networks to exploit their full dynamic range. This article shows how
such anchoring can be achieved by incorporating synaptic scaling mechanisms of homeostatic plasticity within recurrent on-
center off-surround networks in such a way that their pattern
processing capabilities, including the ability to make winner-take-
all decisions, is preserved. These results suggest how BDNF may
homeostatically scale the strengths of excitatory and inhibitory
synapses in opposite directions (Rutherford, Nelson, & Turrigiano,
1998).

2. How stored patterns depend on feedback signal functions in
a recurrent competitive field

The theorems of Grossberg (1973) analyzed how the feedback
signal functions in recurrent on-center off-surround networks
whose cells obey membrane, or shunting, equations (Fig. 1(a))
transform input patterns before they are stored in short term
memory as equilibrium activity patterns. In these simplest
networks, the on-center of self-excitatory feedback is narrow, and
the off-surround of recurrent lateral inhibition reaches all other
cells. Such a network is defined by

\[
\dot{x}_i = -A x_i + (B - x_i) (I_i + f(x_i)) - x_i \left[ J_i + \sum_{k \neq i} f(x_k) \right].
\]

In (1), \( x_i \) is the activity of the \( i \)th cell, or cell population; \( A \) is
the passive decay rate; \( B \) is the excitatory saturation point of cell
activity; \( I_i \) is the excitatory input to the \( i \)th cell; \( f(x_i) \) is the
on-center positive feedback signal; \( J_i \) is the inhibitory input to the \( i \)th
cell; and \( \sum_{k \neq i} f(x_k) \) is the negative feedback from the off-surround.
Eq. (1) may be derived from the following equation for cell activity, or
voltage \( V(t) \):

\[
C \frac{d}{dt} V = (V^p - V) g^p + (V^+ - V) g^+ + (V^- - V) g^-,
\]

where \( C \) is capacitance; the constants \( V^+, V^-, \) and \( V^p \) are excit-
tory, inhibitory, and passive saturation points of \( V \), respectively, 
and \( g^+ \), \( g^- \), and \( g^p \) are conductances that can be changed by inputs
(Hodgkin, 1964). In (1), \( x_i = V_i \). \( A = g^p \), \( V^+ = V^p = 0 \), \( B = V^+ \),
and \( g^+ \) and \( g^- \) are the total on-center and off-surround inputs, re-
spectively. The choice of the feedback signal function \( f \) determines how
an input pattern is transformed before it is stored in short-
term memory (i.e., before the network reverberates the stored pat-
tern for all time), and indeed whether it will be stored in short-term
memory at all.

The theorems in Grossberg (1973) assumed that inputs were on
until time \( t = 0 \), when they were shut off to allow the network
to transform and store the input pattern in short-term memory
using its recurrent interactions. The theorems included all possible
initial values \( x_i(0) \) of the activities, corresponding to the effect
of all possible input patterns. As shown in Fig. 1(b), if the signal function
is linear (e.g., \( f(w) = Aw \)), then the initial input pattern is
preserved. If the signal function is slower-than-linear (e.g., \( f(w) = Aw^2 \)),
then noise is suppressed. In fact, noise is suppressed so vigorously that only the cell, or cell population, with the largest input survives the competition, and its activity
is stored in short term memory. This is thus a winner-take-all (WTA)
network.

In order to enable cells with activities less than the maximum
to be stored in short-term memory, a sigmoid cell function suffices
(e.g., \( f(w) = Aw^2/(B^2 + w^2) \)), because it is a hybrid of the
other signal functions. Any signal function needs to be faster-than-linear at low activity levels in order to suppress noise. And any biologically plausible signal function needs to be bounded at high activity values. A sigmoid signal function is the simplest one that combines both constraints: at low activity levels, it is faster-than-linear. At high activity values, it is slower-than-linear. Because it smoothly interpolates these extremes, it is approximately linear at intermediate activities. Thus, a sigmoid signal function can begin to contrast-enhance an input pattern as the shunting on-center off-surround network interactions begin to normalize cell activities and drive them into the approximately linear range, where they can be stored as a partially contrast-enhanced pattern.

The net effect of a sigmoid signal function on network dynamics is to define a quenching threshold, or initial activity level below which activities are suppressed, and above which they are contrast-enhanced and stored in short-term memory. The quenching threshold can be tuned, thus leading to a tunable filter: in the limit of a high quenching threshold, it can perform like a WTA network. If in response to an unexpected event the quenching threshold goes down, then the network can store a distributed pattern of input features until hypothesis testing can select the features that can better predict future outcomes.

The question that the current article asks is: How can these transformations be preserved in a network that anchors the equilibrium activities using homeostatic plasticity?

3. Homeostatic plasticity

Turrigiano and her colleagues (e.g., Desai, Cudmore, Nelson, & Turrigiano, 2002; Rutherford et al., 1998; Turrigiano, 1999; Turrigiano, Keslie, Desai, Rutherford, & Nelson, 1998; Turrigiano & Nelson, 2004, 2005 and Wierenga, Ibata, & Turrigiano, 2005) have made and developed the seminal discovery that the strengths of excitatory and inhibitory connections in many neural tissues can adjust themselves to stabilize network dynamics through a homeostatic plasticity process called synaptic scaling. In particular, among other factors, it has been shown that the neurotrophin brain-derived neurotrophic factor (BDNF) has opposite effects on pyramidal neuron and interneuron quantal amplitudes, and thereby modifies the ratio of pyramidal neuron to interneuron firing rates (Rutherford et al., 1998). Such a mechanism can stabilize pyramidal neuron firing rates when synapse number and strength change, by allowing neurons to adjust the total amount of excitatory current that they receive while preserving the relative differences between inputs (Turrigiano et al., 1998).

van Rossum, Bi, and Turrigiano (2000) described a model for synaptic scaling in an individual neuron. The dynamics of the synaptic scaling process in this model are defined as follows:

\[
\frac{dw}{dt} = \beta w[a_{\text{goal}} - a].
\]

where

\[
\tau \frac{da}{dt} = -a + \sum_i \delta(t - t_i).
\]

In Eq. (3), \(\frac{dw}{dt}\) is the rate of change of the weight \(w\) due to synaptic scaling, and \(a\) is an activity level that time-averages the effects of postsynaptic spikes on the target neuron. The synaptic scaling process up-regulates the weight when the average activity falls below a desired target level, \(a_{\text{goal}}\), via the term \([a_{\text{goal}} - a]\) and down-regulates it when the average activity grows too large. The average activity level in (4) increases every time the neuron
spikes, as defined by the sum of delta functions, \( \delta(t - t_i) \), and decays exponentially between spikes. In (3), the non-negative parameter \( \beta \) determines the rate of the synaptic scaling process, and parameter \( \alpha_{\text{goal}} \) determines the target activity level. Parameter \( r \) in (4) specifies the rate with which the average activity level changes.

The full model of van Rossum et al. (2000) embeds synaptic scaling in a spiking neuron. However, the model does not include the membrane equation of neuronal activities, either individually or interacting in a network. Nor does it describe the opposite effects of neurotrophic factors such as BDNF in scaling the strengths of excitatory and inhibitory synapses in opposite directions. The current homeostatic recurrent competitive field (hRCF) model provides perhaps the simplest realization of such factors that preserves critical properties of distributed pattern processing and short-term memory storage in response to a wide range of feedback signal functions.

4. A model of pattern processing in a recurrent competitive field with synaptic scaling

Accordingly, we generalize the van Rossum et al. (2000) synaptic scaling model to a hRCF with adaptive weights \( w_i \) and \( W \) that scale the strengths of recurrent excitation and inhibition in opposite directions. We assume that these weights are sensitive to an average activity \( \alpha \), akin to that in Eq. (4), which is sensed by all the weights. Assuming that all the weights \( w_i \) start out equal, they will remain equal for all time, so that we can assume all \( w_i = w \). A similar assumption allows us to assume that all \( W_i = W \). With this simplification, we extend Eq. (1) for an RCF to the synaptically scaled recurrent competitive field:

\[
\dot{x}_i = -Ax_i + (B - x_i)(I_i + f(x_i)w) - x_i \sum_{k \neq i} (I_k + f(x_k)W),
\]

where

\[
\dot{a} = \frac{1}{\tau} \left( -a + \sum_{i=1}^{N} x_i \right),
\]

\[
\dot{w} = \beta w(G - a),
\]

and

\[
\dot{W} = \beta W(-G + a).
\]

Eq. (5) is the same as (1) with the addition that the excitatory weight \( w \) modulates the efficacy of the on-center positive feedback signal \( f(x_i) \) and the inhibitory weight \( W \) modulates the efficacy of the off-surround negative feedback signals \( f(x_k) \) in the opposite direction. It is also assumed in (5) that the bottom-up off-surround inhibitory input \( I_k \) that is defined in (1) shares the same off-surround connections as the recurrent off-surround inputs, leading to a total bottom-up input term \( \sum_{k \neq i} I_k \). Such an assumption is often made in an RCF, since it assures that bottom-up and recurrent connections are balanced by the same excitatory and inhibitory anatomical connections. In the current study, since all inputs shut off before the period of network reverberation and storage, this assumption does not influence the results, but does decrease the convergence time in our simulations.

The scaling weights \( w \) and \( W \) in (5), (7) and (8) tune the balance of excitation and inhibition in the network. By (5), weight \( w \) determines the intensity of excitation. By (7), it increases when the average activity level is too small and decreases when the average activity level is too large. By (5), weight \( W \) determines the intensity of inhibition. By (8), it decreases when the average activity level is too small and increases when the average activity level is too large. These two weighting processes embody synaptic scaling in the hRCF, and are identical save for a sign change. In (7) and (8), \( \beta \) is the learning rate parameter and \( G \) is the target level of the scaling function level, replacing \( \alpha_{\text{goal}} \) in (3). In (6), the scaling function \( a \) is sensitive to the total network activity \( \sum_{i=1}^{N} x_i \). It uses the same constant time parameter, \( \tau \), as in van Rossum et al. (2000). Because this hRCF is a continuous network instead of a single spiking neuron, the instantaneous activity the average activity level tracks the sum across \( x_i \), it plays an integrative role in the hRCF much like BDNF seems to play in the brain. In response to this mediating factor, the excitatory and inhibitory adaptive weights, \( w \) and \( W \), respectively, multiplicatively adjust in opposite directions in Eqs. (7) and (8), as they have been reported to do in vivo (Rutherford et al., 1998). In particular, it follows from (7) and (8) that the product \( w(t)W(t) \) of the excitatory and inhibitory weights is constant for all time.

5. Results

5.1. Parameters and initial conditions

Unless noted otherwise, the model parameters assume the values given in Table 1. As noted in Table 1, the network size of five cells is set just large enough that the pattern processing behavior is readily apparent. Network connectivity is all-to-all, so larger networks are dramatically slower to simulate. The decay rate \( A \) is set to a reasonable value given the activation bound, but the network is not particularly sensitive to this value. The activation bound, or excitatory saturation level, \( B \) is similarly unconstrained. The chosen value is numerically stable and consistent with prior work. The sigmoid inflection point (see Table 2) is set given the activation bound. Networks with a sigmoid signal function tend to quench cells whose activity falls below this value and uniformize the activities of cells whose activities lie above it. The synaptic scaling parameters \( \tau \) and \( \beta \) are set such that synaptic scaling operates on a much slower time scale than the fast network dynamics. The target activity level \( G \) is set such that synaptic scaling process will try to maintain an average aggregate network activity equivalent to that of one fully-activated cell. This is a safe choice for every signal function. The signal functions are chosen from Table 2.

This set of signal functions includes at least one example of each major class of pattern processing behaviors. The faster-than-linear and sigmoid signal functions include two degrees of nonlinearity. The activities \( x_i \) remain non-negative due to shunting inhibition, so there is no need to put an explicit threshold on the signal functions to ensure that they are always non-negative.

### Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N )</td>
<td>Number of nodes</td>
<td>5</td>
</tr>
<tr>
<td>( A )</td>
<td>Decay rate</td>
<td>1</td>
</tr>
<tr>
<td>( B )</td>
<td>Upper activation bound</td>
<td>3</td>
</tr>
<tr>
<td>( \alpha )</td>
<td>Inflection point of sigmoid signal function</td>
<td>0.5</td>
</tr>
<tr>
<td>( \tau )</td>
<td>Time scale for slow averaging process</td>
<td>400</td>
</tr>
<tr>
<td>( \beta )</td>
<td>Homeostasis rate constant</td>
<td>0.005</td>
</tr>
<tr>
<td>( G )</td>
<td>Target activity level</td>
<td>3</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>Name</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Linear</td>
<td>( f(x) = x )</td>
</tr>
<tr>
<td>Slower than linear</td>
<td>( f(x) = \frac{x^2}{2} )</td>
</tr>
<tr>
<td>Faster than linear: ( n = 2 )</td>
<td>( f(x) = x^2 )</td>
</tr>
<tr>
<td>Faster than linear: ( n = 4 )</td>
<td>( f(x) = x^4 )</td>
</tr>
<tr>
<td>Sigmoid: ( n = 2 )</td>
<td>( f(x) = \frac{x^2}{2} - \frac{x^4}{4} )</td>
</tr>
<tr>
<td>Sigmoid: ( n = 4 )</td>
<td>( f(x) = \frac{x^2}{2} - \frac{x^4}{4} + \frac{x^6}{6} )</td>
</tr>
</tbody>
</table>
Fig. 2. (a)–(d) Network dynamics; (e)–(h) pattern processing at different time steps (1, 170, 340, 500) with a linear signal function. See text for details.

Unless noted otherwise, the initial conditions are set as follows: \( x_i(0) = 0 \), \( a(0) = G \), \( w(0) = 1 \), and \( W(0) = 1 \). The network is not, however, sensitive to its initial conditions, so long as the initial values \( w(0) \) and \( W(0) \) are positive. Negative values are outside the normal range, and values of zero would disable tuning. The average activity \( a(0) \) is set as if the network were already at an equilibrium average activity level of \( G \) to keep down the simulation time, but non-equilibrium initial values produce identical steady-state behavior.

5.2. Simulation protocol

Each simulation consists of 500 presentation intervals, each running for ten time units. In the first five time units of each interval, a random input pattern is presented to the network. Each element in the input pattern is independently chosen from a uniform random distribution in the range \( [0, 1] \). For the remaining five time units, input is switched off and the network is allowed to reverberate. In both the input and no-input cases, five time units are sufficient for the fast network dynamics to equilibrate. At the end of each presentation interval, each \( x_i \) is set back to the initial value of zero. The values of \( a \), \( w \), and \( W \) carry over from interval to interval.

The input pattern \( [0.2, 1, 0.4, 0.8, 0.2] \) is used to diagnose pattern processing behavior. This pattern is consistent with that used to illustrate pattern processing in Grossberg (1973). To visualize pattern processing behavior as of a given presentation interval, the network is copied and re-simulated using the diagnostic input pattern instead of a random pattern.

5.3. Signal functions determine synaptically-scaled stored activities

Simulation results with a linear signal function are shown in Fig. 2. Fig. 2(a) shows the network state at the end of each of the 500 presentation intervals for each of the model equations. The plot for \( x \) includes traces for each of the five \( x_i \) equations. The rapid mixing in \( x \) indicates that the network is successfully reverberating with each new random input pattern while the synaptic scaling process tunes the network. In this case, the tuning process up-regulates excitation and down-regulates inhibition to reach the goal activity level. Fig. 2(b)–(d) show how the variables \( a \), \( w \), and \( W \) vary through time, leading asymptotically to converging values as the network equilibrates.

Fig. 2(e)–(h) shows the pattern processing behavior sampled at four intervals (1, 170, 340, 500) over the simulation. These results were computed using the diagnostic input pattern. As the simulation proceeds, the tuning process shifts the network balance toward greater excitation. While the stored pattern remains roughly constant, the magnitude increases accordingly.

Figs. 3 through 7 use a format identical to Fig. 3. In Fig. 3, tuning with a slower-than-linear signal function tends to uniformize the input pattern as total network activity shifts to bring the network to the goal activity level.

Fig. 4 shows a faster-than-linear signal function with \( n = 2 \). The fast state dynamics show that the network can pick a new winner on each presentation interval.

Fig. 5 shows a faster-than-linear signal function with \( n = 4 \). Again, a network winner is consistently chosen as the network scales its total activity.

Figs. 6 and 7 show sigmoid signal functions with \( n = 2 \) and \( n = 4 \), respectively. The \( n = 4 \) case illustrates how the tuning process shifts the quenching threshold. Before tuning, an input of 0.4 falls below this threshold. This is why the output of cell three is quiet on the first interval. After tuning, the quenching threshold shifts lower, such that 0.4 is above the threshold.

The simulation in Fig. 2 illustrates that, when a shunting on-center off-surround network with a linear signal function incorporates the synaptic scaling weights \( w \) and \( W \), then there is a tendency for the stored pattern to become more uniform than the initial input pattern. This trend can be traced to the fact that the excitatory gain \( w \) becomes greater than 1, whereas the
inhibitory gain $W$ becomes less than 1, as the network equilibrates. In the absence of any inhibition, the network could easily saturate its activities as time goes on. The partial flattening of the stored pattern reflects the relative weakening of inhibition. A linear signal function is not biologically plausible because it tends to amplify noise. A faster-than-linear signal function at low activities, and thus a sigmoid signal function as well, suppresses noise and thereby initiates contrast enhancement of the input pattern. The simulations in Figs. 4 and 5 demonstrate that this contrast-enhancing action can overcome the uniformizing tendency of increasing the excitatory gain at the expense of the inhibitory gain in order to more fully use the dynamic range of the neurons.

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6. Conclusions

The homeostatic recurrent competitive field, or hRCF, model that is introduced in this article illustrates how a network of interacting neurons can simultaneously satisfy two types of constraints. First, it can solve the noise-saturation dilemma by protecting cell activities from getting lost in noise or from saturating at their maximal values in response to small or large inputs, respectively. Second, it can homeostatically scale the strengths of excitatory and inhibitory signals in opposite directions, mediated by a network-sensitive factor such as BDNF, to ensure that neurons respond using their full dynamic range.
Model simulations show that, when these two types of constraints are simultaneously realized, homeostatic increases in excitatory gain can tend to uniformize stored patterns, but sigmoid signal functions can overcome this tendency to contrast enhance input patterns before they are stored in short-term memory.

The current model does not describe how such synaptic scaling properties may interact with learning processes, such as long-term potentiation and depression (LTP and LTD), as they are known to do in vivo (e.g., van Rossum et al., 2000). By showing how synaptic scaling can be integrated into network processing and short-term memory storage of distributed input patterns, the present model sets the stage for incorporating known models of learning into a synthetically-scaled framework.

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