A network model with pyramidal cells and GABAergic non-FS cells in the cerebral cortex

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Abstract

Recent experiments revealed that there exist electrophysiologically, anatomically, and functionally distinct two classes of GABAergic interneurons in the cerebral cortex, FS cells and non-FS cells. We propose a network model of cortical local circuits including dendritic inhibition, which is the anatomical hallmark of the non-FS cells. While conventional lateral inhibition models always converge to winner-take-all states if the self-excitation is strong, our model does so only for appropriate inputs but otherwise converges to another states, in which all the neurons have little activities, even if the self-excitation is strong enough to keep winner's activity after the extinction of the inputs.

1. Introduction

There are a wide variety of GABAergic interneurons in the cerebral cortex. Recent experiments using intracellular dual recordings revealed that these GABAergic neurons are divided into mainly two classes, fast spiking (FS) cells and non-FS cells including low threshold spiking (LTS) cells, according to their electrophysiological features. Additionally, this physiological grouping largely accords with the anatomical grouping based on the difference in the locations where they make the synapses on the target pyramidal cells: while FS cells tend to make their synapses at the somata or proximal regions of the dendrites, non-FS cells are apt to target the distal sites of the dendrites [2,3,4,26]. Therefore, as for non-FS cells, localized interaction between the excitatory synaptic inputs and inhibitory ones on each dendritic branch could occur, raising the possibility of the local computation, proposed by Koch *et al.* as "dendritic gate" [11], such that inhibitory inputs on a certain dendritic branch effectively shunt excitatory inputs on the same branch but do not have effects on other branches. Increasing evidences indicate that the non-FS GABAergic system is functionally different in many situations from the FS system [2,3,30], and so it seems to be important to analyze the interaction between pyramidal cells and non-FS cells not only in the single cell level but also in the network level. In this paper, we propose a firing rate-based neural network model of cortical local circuits consisting of pyramidal cells and non-FS GABAergic cells, in which we include location-restricted effects of dendritic inhibitions mediated by non-FS cells. We examine the dynamical properties of the model, showing that this nonlinearity of single neurons changes the network behavior from those of conventional lateral inhibition networks.

2. The model

We propose a neural network model which corresponds to relatively small regions of the cerebral cortex, possibly cortical columns [27], including excitatory pyramidal cells and inhibitory GABAergic interneurons of such types as LTS or other non-FS cells that make synapses onto the distal dendrites of the target pyramidal cells (Fig. 1). We consider four types of connections: 1) excitatory feed-forward connections from input sources such as thalamus, other subcortical regions, or other parts of the cortex to the pyramidal cells, perhaps via excitatory interneurons, 2) excitatory connections from the pyramidal cells' axonal collaterals to the non-FS cell that is activated by the pooled activities of all the pyramidal cells, 3) inhibitory connections from the pooled non-FS cell to each dendritic branch of every pyramidal cell, and 4) excitatory connections from the pyramidal cells' axonal collaterals to each dendritic branch of themselves. As to 1), we assume that all the synapses from the same input source converge to single dendritic branches of individual pyramidal cells, because it has been shown that synaptic inputs from different input sources are preferentially mapped on to different specific regions of the dendritic tree [21]. Connections 2) and 3) constitute the dendritic lateral inhibitions between the pyramidal cells via the pooled non-FS cell, whereas connections 4) form self-excitations. All the strengths of these connections except for 1) are assumed to be uniform. The firing rate dynamics of n pyramidal cells and a pooled non-FS cell can be described by the following differential equations (Fig. 1):

$$\begin{aligned} \frac{dx_j}{dt} &= \frac{1}{\tau_p}\left(-x_j + \sum_{i=1}^{m} \sigma_{dend}\left(w_{ji} I_i + \frac{\alpha}{m} x_j - \frac{\beta}{m} y \right) \right) \quad \text{for} \quad j = 1, \cdots, n \\ \frac{dy}{dt} &= \frac{1}{\tau_G}\left(-y + \frac{\gamma}{n} \sum_{j=1}^{n} x_j \right). \end{aligned} \tag{1}$$

Here m: the number of the input sources, I_i: the intensity of the i-th component of the input sources (defining the m-dimensional input vector as $\mathbf{I} \equiv (I_1 \cdots I_m)$), x_j: the firing rate of the j-th pyramidal cell, y: the firing rate of the pooled non-FS cell, $w_{ji} (\geq 0)$: the weight of excitation from the i-th input to the j-th pyramidal cell, $\frac{\alpha}{m} (\geq 0)$: the weight of excitation from every pyramidal cell to each dendritic branch of itself (self-excitation), $\frac{\beta}{m} (\geq 0)$: the weight of inhibition from the pooled non-FS cell to each dendritic branch of every pyramidal cell, $\frac{\gamma}{n} (\geq 0)$: the weight of excitation from every pyramidal cell to the pooled non-FS cell, and τ_p and τ_G: the time constants of the dynamics of the pyramidal cells and the non-FS cell, respectively. $\sigma_{dend}(z)$ denotes the transfer function consisting of the thresholding and the upperbound:

$$\sigma_{dend}(z) = \begin{cases} 0 & (z \leq 0) \\ z & (0 < z \leq \frac{\eta}{m}) \\ \frac{\eta}{m} & (\frac{\eta}{m} < z) \end{cases}.$$

This transfer function represents the nonlinear input summation on each dendritic branch: i) rectification (thresholding) represents the effect of non-FS cell-mediated shunting inhibition such that $GABA_A$ receptor-channel opening on a certain branch effectively shunts EPSPs or prevents dendritic spike generation and propagation on the same branch but does not

affect other branches [13,16,24], ii) the upperbound ($\frac{\eta}{m}$) represents saturation on each branch. In this way, in this model, the nonlinear transfer function is imposed on each dendritic branch before summing all the branches. On the other hand, in the conventional models consisting of self-excitation and conventional lateral inhibition, only one nonlinear function is imposed after the linear summation of all the inputs [1,5,8,31]. In order to compare dynamical properties of our model with those of the conventional models, here we define a kind of prototypical conventional model as following:

$$\begin{aligned} \frac{dx_j}{dt} &= \frac{1}{\tau_p}\left(-x_j + \sigma_{soma}\left(\sum_{i=1}^{m} w_{ji} I_i + \alpha x_j - \beta y\right)\right) \quad \text{for} \quad j = 1,\cdots,n \\ \frac{dy}{dt} &= \frac{1}{\tau_G}\left(-y + \frac{\gamma}{n}\sum_{j=1}^{n} x_j\right), \end{aligned} \tag{2}$$

in which $\sigma_{soma}(z)$ denotes the transfer function consisting of the thresholding and the upperbound:

$$\sigma_{soma}(z) = \begin{cases} 0 & (z \le 0) \\ z & (0 < z \le \eta) \\ \eta & (\eta < z) \end{cases}.$$

Only the difference between this conventional model (Eq. 2) and our new model (Eq. 1) is the order of the transfer function (σ) and the summation ($\sum$).

3. Simulation results

In both the conventional model (Eq. 2) and our new model (Eq. 1), there are two regimes about the strength of the self-excitation (α). If $\alpha < 1$, whenever the input is absent, all the neurons are quiet because the decay term ($-x$) exceeds the self-excitation term ($+\alpha x$). If $\alpha > 1$, on the other hand, the self-excitation term exceeds the decay term so that activities of some neurons persist even after the input is turned off. In this paper, we concentrate on the latter case, namely $\alpha > 1$, in which the recurrent excitation is strong enough to keep the persistent activities after the extinction of the external inputs.

In order to examine the general dynamical properties of the models described by Eq. 1 or Eq. 2, we assume a random feed-forward weight matrix $W = (w_{ji})$, where $j = 1,2,\cdots,n$ and $i = 1,2,\cdots,m$, and each component of W is a random value taken from the uniform distribution on $[0 \quad \frac{1}{m}]$. In this case, if the input vector $\mathbf{I} = (I_1 \cdots I_m)$ is parallel to one of the feed-forward connections of a certain pyramidal cell, that is, one (e.g. the k-th) of the row vectors of $W = (w_{ji})$, $\mathbf{w}_k = (w_{k1} \cdots w_{km})$, then the total feed-forward synaptic excitation of the very k-th pyramidal cell is expected to be the largest among all the pyramidal cells, because W is a random matrix and then $\mathbf{I}$ should not have any correlation with all the afferents (row vectors of W) except for $\mathbf{w}_k$. In this sense, we refer this input vector $\mathbf{I}$ parallel to $\mathbf{w}_k$ as the input pattern *corresponding to* the k-th pyramidal cell. Each pyramidal cell has its own corresponding input pattern in the m-dimensional input space, and the whole network *stores* n input patterns in the feed-forward connectivity. Here, we will

regard the model (Eq. 1 or Eq. 2) as a kind of pattern discriminator: we examine the responses of the pyramidal cells to the input corresponding to a certain pyramidal cell (the k th cell) with various amount of random noise on it:

$$\mathbf{I} = s\mathbf{w}_k + (1-s)\boldsymbol{\xi}, \tag{3}$$

where $\boldsymbol{\xi} = (\xi_1 \cdots \xi_m)$ represents a random noise whose component is a random value taken from the uniform distribution on $[0 \quad \frac{1}{m}]$, and s represents a signal ratio ($0 \le s \le 1$). We will compare the ability of the conventional model (Eq. 2) and our new model (Eq. 1) as a pattern discriminator in the following.

It has been known that the conventional models with the strong self-excitation ($\alpha > 1$) always converge to the winner-take-all states, in which only a single or several neurons have positive activities while the other neurons' activities are 0, regardless of the input. Here we confirm these features using Eq. 2 for comparison with our new model. As shown in Fig. 2b, when the input corresponding to the 50 th pyramidal cell without noise (Fig. 2b - top: $s = 1$) or with noise up to a certain degree (Fig. 2b: $s = \frac{1}{2}, \frac{1}{4}$) is presented, the activity of the 50 th cell reaches the upperbound (η) while the other cells are inactive. This can be interpreted as that the network achieved a correct pattern discrimination. On the other hand, when the input is distorted by a large noise (Fig. 2b: $s = \frac{1}{8}$) or the input is completely random (having no correlation with the afferents of the 50 th cell, Fig. 2b - bottom: $s = 0$), the 50 th cell usually no longer becomes active but instead the other cell reaches the activity upperbound. Since which cell becomes the winner depends on the input and the initial conditions, this situation can be interpreted as that the network failed a pattern discrimination: it made a misjudgment by choosing a wrong cell that did not correspond to the input. If the inhibition is weak (for example, one-fifth of the value used in Fig. 2b), multiple cells would reach the activity upperbound at the same time for heavily noise-distorted or random inputs (results not shown). In any cases, however, the system described by Eq. 2 (with $\alpha > 1$) finally reaches a certain winner(s)-take-all state, in which one cell or a few cells reach the activity upperbound while all the other cells are inactive, whether it means correct pattern discrimination or misjudgment.

Next, we examine our new dendritic inhibition model described by Eq. 1 (with $\alpha > 1$) in the same situation as above. Fig. 2c shows the steady-state activities of the pyramidal cells, and Fig. 2d shows the time-courses of the 50 th pyramidal cell (solid line) or the other cells (dotted line with error-bars indicating the standard deviations) in our dendritic inhibition model. When the input corresponding to the 50 th pyramidal cell without noise (Figs. 2c & 2d - top: $s = 1$) or with noise up to a certain amount (Figs. 2c & 2d: $s = \frac{1}{2}, \frac{1}{4}$) is presented, the activity of the 50 th cell reaches the upperbound while the other pyramidal cells' firing rates remain to be quite low values (usually lower than one percent of the activity upperbound under the parameters in Fig. 2). This can be interpreted as that the network achieved a correct pattern discrimination. Since this state is not a winner-take-all state in a usual sense because the losers have very small but nonzero activities, we refer it as a

winner-take-all-*like* state. On the other hand, when the input is distorted by a large noise (Figs. 2c & 2d: $s = \frac{1}{8}$) or the input is completely random (Figs. 2c & 2d - bottom: $s = 0$), the 50 th cell no longer becomes a winner. In addition, contrary to the conventional model, there are usually no cells that reach the activity upperbound. Instead, all the cells have very small activities (usually lower than one percent of the activity upperbound under the parameters in Fig. 2). We refer to this state as an "I don't know" state. In terms of pattern discrimination, this response can be regarded as telling that the input is not similar to any of the stored patterns. Thereby our dendritic inhibition model described by Eq. 1 (with $\alpha > 1$) hardly misjudge the random or heavily distorted input as a wrong pattern.

Simulation results under various noise levels are summarized in Fig. 3. With the same parameters as Fig. 2, both models converge to the "correct" winner-take-all state (*i.e.* the 50 th cell becomes the winner) when a signal ratio (s) is larger than about 0.3 (Figs. 3a & 3b). As a signal ratio decreases, the conventional model tends to converge to the "misjudge" winner-take-all states (*i.e.* some cell other than the 50 th cell becomes the winner) while our model tends to converge to "I don't know" states. Actually, our model sometimes converges to "misjudge" winner-take-all states (as shown in Fig. 3b, up to about 15 % in this parameter). When the inhibition is strengthened half time (Fig. 3c), our model becomes even more careful, that is making almost no misjudgments, although it becomes less sensitive.

Here is an intuitive explanation about the difference between the conventional model and our model. In the conventional model, the transfer function is imposed on the linear summation of all the inputs which include the feed-forward excitations to the dendritic trees, the self-excitation, and the lateral inhibition. Therefore, the neurons compete in their inputs' linear summation. Thereby a slight difference in the input linear summation would be amplified by the strong ($\alpha > 1$) self-excitation via positive feedback, resulting in the convergence to the winner(s)-take-all states. On the other hand, in our model, the self-excitation and the lateral inhibition are added on each dendritic tree, and the transfer function is also imposed on each dendritic tree locally. Therefore, the neurons do not compete in their inputs' linear summation but compete at a single dendritic branch level. Specifically, assume that there is a neuron whose input's linear summation is slightly weak compared with the other neurons. Although this neuron is "weak" in terms of its input' linear summation, such a dendritic branch of this neuron that receives a strong feed-forward excitation can still receives some positive total input after the subtraction of the inhibition from the non-FS cell, resulting in that this "weak" neuron is hardly inactivated completely. The small but nonzero activity of this neuron contributes to raising the activity of the non-FS cell, preventing other neurons from becoming the complete winner. In this way, there is no complete loser, and also no complete winner as well, leading to the convergence to the "I don't know" state. Although this is not a proof but merely an intuitive explanation, the existence and the stability of the "I don't know" states can be proven in the limit of $m \to \infty$ [17].

4. Discussion

We discuss here the possibility that our simplified network model is related to the real neocortical neuronal networks. At first, consider the problem that though our model includes only non-FS type of GABAergic cells, actually there coexist both types, non-FS and FS, of GABAergic cells in the cerebral cortex. In general, it goes without saying that models including both types of GABAergic cells should be constructed. Nevertheless, the network model with only the non-FS cells, as those proposed above, may have direct biological meanings in some situations because of several reasons: 1) FS cells and non-FS cells may be included in different circuitries at least in some parts of the neocortex. For example, in the rat visual cortex, axons of non-FS cells have been found to be mainly distributed vertically to upper layers while those of FS cells primarily confined to layer V, suggesting that non-FS cells mainly mediate intracolumnar inhibition, whereas FS cells primarily mediate intralaminar inhibition [29] (see also refs. of [29]). If so, our model could be thought as a feasible model of the cortical column, 2) FS cells and non-FS cells may be differentially regulated by some neural modulators. It has been found that both acetylcholine [29] and dopamine [6] modulate the FS-mediated somatic inhibition and the non-FS-mediated dendritic one to the opposite directions, depressing the former but promoting the latter, suggesting that those neurotransmitters switch the two modes of inhibition and that under certain conditions either FS or non-FS plays a dominant role.

The next problem is that although our model includes only GABAergic interneurons, actually there exist more excitatory glutamatergic interneurons than GABAergic ones in the cerebral cortex [10]. In fact, our model does include some types of excitatory interneurons *implicitly* in the feed-forward excitatory connections and in the description of self-excitation, because those connections can be regarded as reduced representation of the relaying excitatory interneurons. However, it seems to be an important next step to analyze the model including excitatory interneurons explicitly.

There is one more problem that there exist wealth of direct interactions between GABAergic interneurons, which are not included in our model that assume the pooled non-FS cells representing the population of those cells. In addition to chemical synapses, it was found that FS cells and non-FS cells densely make electrical couplings with only the same cell-type as themselves [7]. Although the biological meaning of these couplings is still elusive, it was proposed that they would be one of the underlying mechanisms of spike synchronization [18]. These cooperative effects of couplings on neuronal spiking activities are, though not necessarily inconsistent with, but beyond the scope of our firing rate-based model, and so more detailed models using spiking neuron models should be constructed.

In this paper, we analyzed both the conventional model and our new model in the regime that the self-excitation is strong

enough for keeping the activities persistently after the extinction of the inputs. When the self-excitation is weak so that the activities retain only during the existence of the inputs, the conventional models also support "I don't know" states, and then the situation is different from those described in the above. The comprehensive analysis of our model in both regimes, including the mathematical proof of the existence and the stability of the "I don't know" states in the limit of $m \to \infty$, will appear elsewhere [17]. Although there are few data about the actual strengths of the recurrent excitation in the real brain, it could vary from region to region. Strong recurrent excitation such that we assumed may be used for keeping some kinds of working memories.

Dendrite inhibition was originally implemented by Spratling and Johnson as a discrete-time neural network model [22, 23], in which they have shown that appropriate strengths, of both feed-forward excitatory and dendritic inhibitory connections, for a certain type of pattern recognition could be self-organized by assuming a kind of Hebbian learning rule. Although we do not concern learning and plasticity in this paper, it might be expected that the weights of the feed-forward connections of our model could also be self-organized by similar Hebbian rules. On the other hand, there are no clear evidences about Hebbian learning at GABAergic synapses, while rather anti-Hebbian plasticity has been reported in some cases [28]. Since our model assumes uniform strengths on GABAergic synapses, it does not violate biological plausibility.

As we have shown, the neural network model consisting of self-excitation and non-FS cells-mediated dendritic lateral inhibitions has a different dynamical property from the conventional model in the regime that the self-excitation is strong enough to keep the activities after the extinction of the inputs. While the conventional model always converges to a certain winner(s)-take-all state and thus would make a misjudgment for heavily noise-distorted or random patterns, our model supports "I don't know" states and therefore hardly makes a discrimination error. This careful pattern discrimination might be used in the human or the animal's brain to represent things that have critical meanings for them, such as human faces or animals' body odors. Convergence to "I don't know" states in spite of the strong recurrent excitation may act as a network-level mechanism to keep the total firing rate at a low level. From computational neuroscience's point of view, it has been proposed that single cortical pyramidal cells with branched dendrites implement two stages of the nonlinear transfer functions, one on each dendritic branch and the other on somata, and thus themselves could be regarded as two-layer artificial "neural networks" [19]. It seems likely that various types of specialized computations emerge from the networks consisting of such a component having a rich nonlinearity in itself, and our model may be regarded as a simple example of them.

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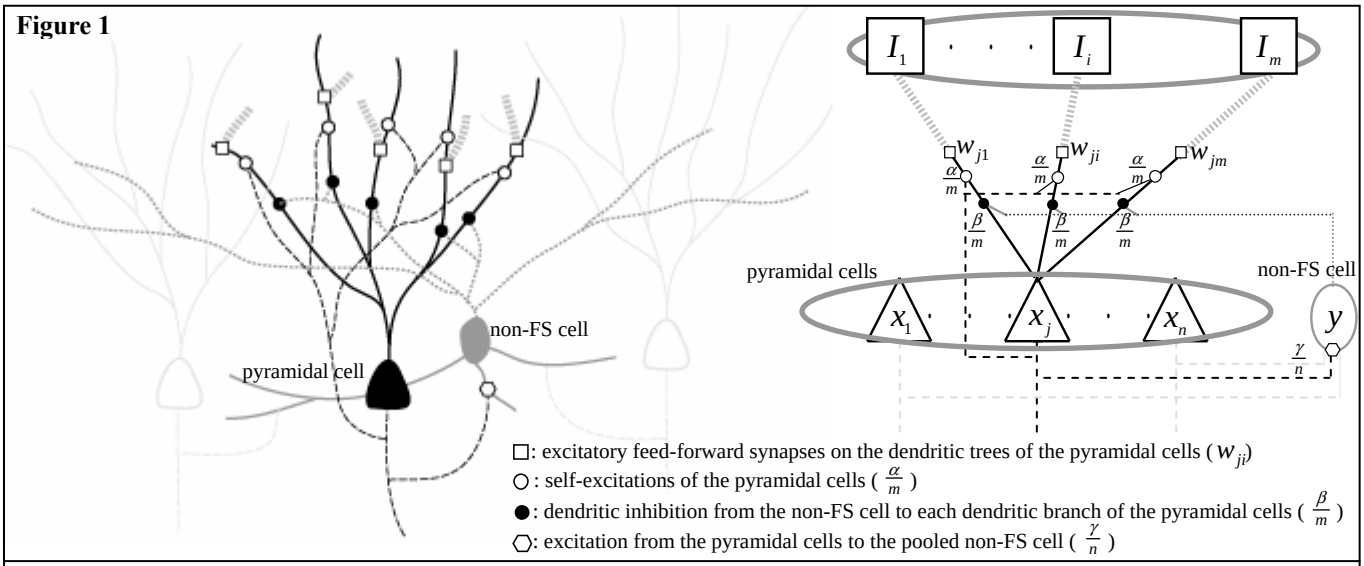
Figure legends

Fig. 1 A schematic diagram of a part of the cortical local circuit including pyramidal cells and GABAergic non-FS cells.

Fig. 2 (a) Input patterns with various noise levels. The top 30×30 image indicates the pattern corresponding to the 50 th pyramidal cell (permutated in ascending order), and the following images indicate the inputs with various noise levels (fraction of a signal: $s=1,\frac{1}{2},\frac{1}{4},\frac{1}{8}$) or a random input (the bottom $s=0$). (b, c) Steady-state activities of the pyramidal cells in the conventional model (b), or our dendritic inhibition model (c). Horizontal axis indicates the indexes ($1\sim100$) of the pyramidal cells. (d) Time-courses of the activities of the 50 th cell (solid line) and the other cells (dotted line with error-bars indicating the standard deviations) in our dendritic inhibition model. Horizontal axis indicates the time. Single trials are illustrated in (b), (c), and (d). Parameter values and conditions: $m=900(=30\times30)$, $n=100$, $\alpha=1.5$, $\beta=1$ (b: conventional model) or $\beta=0.2$ (c, d: our dendritic inhibition model), $\gamma=0.2$, and $\tau_p=\tau_{nFS}=1$, $\eta=10$; W and $\mathbf{I}$ were reset for each trial; the initial values of x_j were distributed uniformly on [0 0.1] and $y(t=0)=0$.

Fig. 3 Classification of the steady-states (vertical axis, % : fraction of 1000 trials) of the conventional model (a) or our dendritic inhibition model (b, c) for the inputs with various noise levels (horizontal axis: signal ratio (s)). Black: "correct" winner-take-all state ($x_{50}=\eta$ and $^{\forall}j(\neq50)\ x_j<\frac{\eta}{20}$), Gray: "misjudge" winner-take-all states ($^{\exists}k(\neq50)\ x_k=\eta$ and $^{\forall}j(\neq k)\ x_j<\frac{\eta}{20}$), Dotted: "I don't know" ($^{\forall}j\ x_j<\frac{\eta}{20}$), White: other cases (rare). Parameter values in (a, b) are the same as Fig. 2, while β is increased half time ($\beta=0.3$) in (c).

Figure 1



□: excitatory feed-forward synapses on the dendritic trees of the pyramidal cells (w_{ji})
○: self-excitations of the pyramidal cells ($\frac{\alpha}{m}$)
●: dendritic inhibition from the non-FS cell to each dendritic branch of the pyramidal cells ($\frac{\beta}{m}$)
⬡: excitation from the pyramidal cells to the pooled non-FS cell ($\frac{\gamma}{n}$)

Figure 2

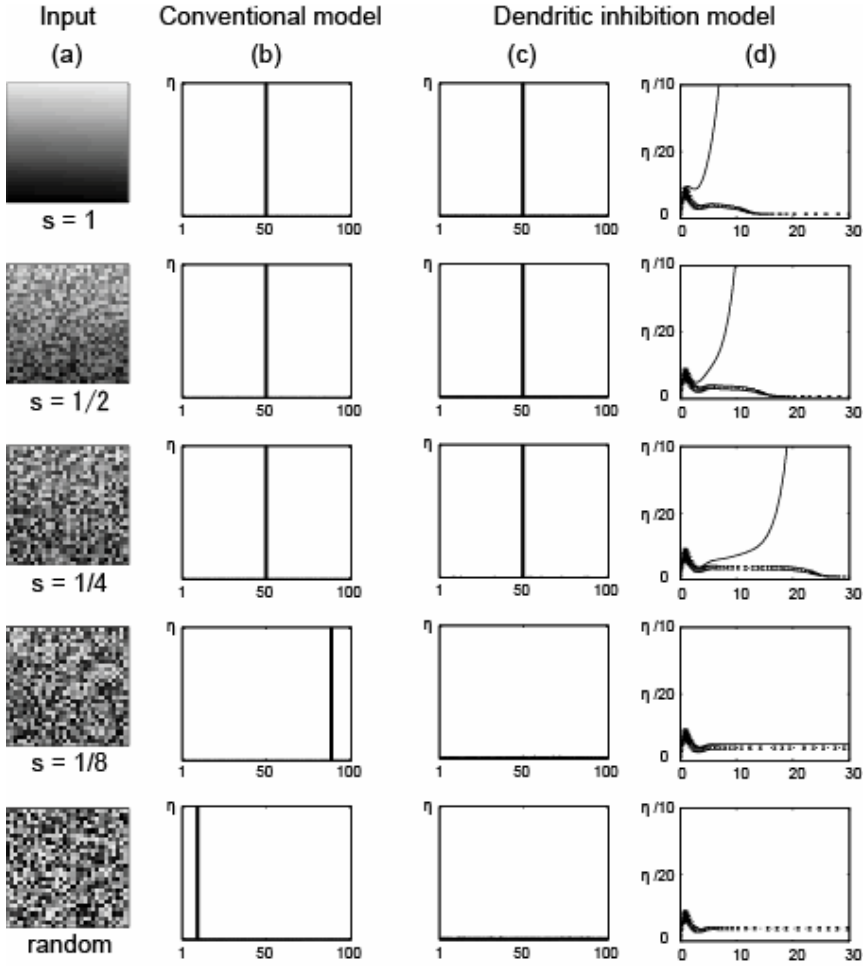


Figure 3