Independent Component Analysis in Neuron Model

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Abstract

Independent Component Analysis (ICA) and related algorithms provide a model for explaining how sensory information is encoded in our brains. However, it remains unclear how neural network uses its biological plasticity to achieve this ICA-like processing: maximization of information transmission or minimization of redundancy. Here, we consider a neuron model proposed by Savin, Joshi, and Triesch (2010), which includes three forms of plasticity in real neural network: spike-timing dependent plasticity (STDP), intrinsic plasticity (IP), and synaptic scaling. We investigate both theoretical and experimental aspects of the model and found that the three types of plasticity play important but different roles in efficiency and quality of learning. Although this neuron model cannot compete with classic ICA algorithms in solving blind separation problem, it provides a biological perspective that can potentially explain how our brains learn and why our brains have such high capacity and complexity.

1 Introduction

How our brains learn and encode a huge amount of information has been a long debating question. Information theories predict that neural network learn the sensory input by maximizing information encoded and reducing redundancies. Models based on independent component analysis (ICA) provide successful explanation of various properties in sensory coding in the cortex [1]-[2]. However, it remains unclear how networks of spiking neurons using realistic plasticity rules to realize such computation.

1.1 Independent component analysis (ICA)

Independent component analysis is a computational signal processing technique for solving blind separation problem. ICA can extract statistically independent components from complex and high-dimensional signals such as EEG. Several theories and algorithms have been proposed for ICA problem, including infomax approach by Bell and Sejnowski [3], maximum likelihood estimation, negentropy maximization [4] and minimization of mutual information [5]. Te-Won Lee et al demonstrates that these approaches lead to the same iterative learning rules for solving blind separation problem of mixed independent sources [6]. However, few of them consider spiking neurons scenario and provide biological plausible mechanism for learning.

1.2 ICA-like processing in neural network

Although classic ICA algorithms are based on information and probability theories rather than biological mechanisms, they share similar concepts and learning strategies with principles of neural network. For example, Jutten and Herault [7]-[8] propose an adaptive algorithm, Jutten-Herault algorithm, for separating independent sources based on biological observations, which consider the architecture of a recursive neural network. Beck and Bell [9] adopt the idea of maximization of entropy of output spikes distribution in a feed-forward network, which lead to maximization of information flow between spiking neurons.

Different from classic ICA, the neuronal learning is based on the mechanisms of metabolic constraints and many types of neuronal plasticity. However, several properties of natural or artificial neural network, including spike-timing dependent plasticity (STDP) and Hebbian-like learning, have shown to correspond to concepts of classic ICA algorithms [2]. The optimization criteria in classic ICA such as maximization of information transmission [10], maximization of entropy, and minimization of mutual information can lead to the learning principles in neural network like maximization of information encoded and minimization of redundancies. The ICA-like processing in neural network provides neuron models for us to explain and investigate how learning is achieved in our brains.

1.3 Sparse Coding

A related principle is sparse coding, which suggests that neurons encode sensory information by using only a small number of active neurons at each time point [11]. The model gives a metabolic efficiency perspective on the properties of V1 receptive field [12]. The concept is that spikes are energy-consuming and neurons must be regulated under tight energy constraints, which lead to sparse activity. Experiments report the near exponentially distribution of firing rates is observed in visual area when natural scenes present, which support the idea of sparse coding [13].

1.4 Neuronal plasticity and learning

From the previous section, we discussed why the brain needs the ICA-like learning to encode and represent the information. The goal of this paper is to investigate the biologically plausible mechanism that brain actually achieves ICA-like learning. This paper introduces the neuron model that performs ICA-like learning using a three biological plasticity: intrinsic plasticity, spike-timing dependent plasticity, and synaptic scaling. As weights are updated based on certain learning rules in the classic mathematical ICA, parameters such as synaptic strength or the threshold voltage of a neuron are updated in a biological fashion in this neuronal ICA model.

2 Methods

2.1 Neuron Model

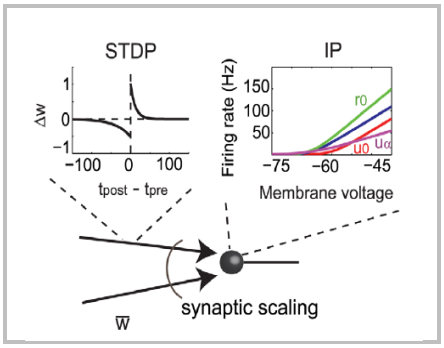

Figure 1: Neuronal model [14]

In our simple neuronal model, presynaptic neurons are connected to postsynaptic neurons, and the synaptic strength is modeled as weight vector W. Firing rate of the postsynaptic neuron is changed by the intrinsic plasticity of the postsynaptic neuron. Weights are changed by spike-timing dependent plasticity and synaptic scaling, as shown in figure 1.

2.2 Intrinsic Plasticity

2.2.1 Biological meaning of intrinsic plasticity

Modern theories of memory has focused on the experience-dependent changes in synaptic function, but certain learning takes are related to the changes in the intrinsic excitability of neurons. The excitability of neurons can be altered by changing the function of voltage-gated ion channels, threshold voltage, neurotransmitter, and etc. Intrinsic plasticity (IP) refers to such change in intrinsic neuronal excitability [15]. One evidence of intrinsic plasticity in biology is shown in figure 2.

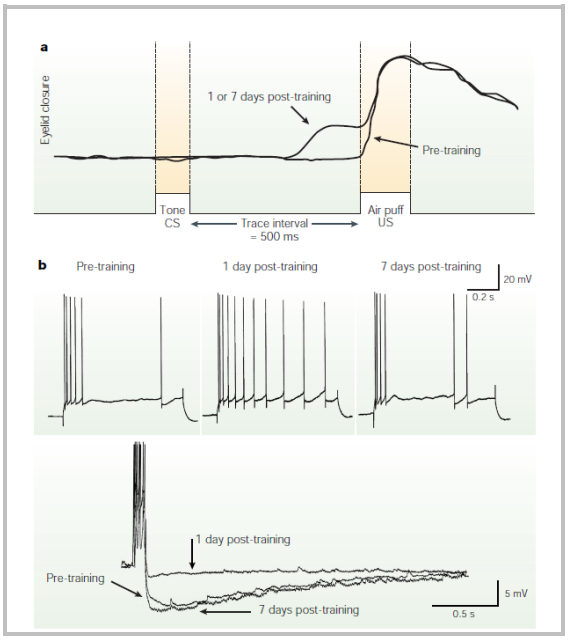

Figure 2: Intrinsic plasticity in biology. [15]

In this experiment, trace eyelid conditioning in rabbits was evoked. The neutral tone (conditioned stimulus, CS) was paired with an air puff (unconditioned stimulus, US). Before the training, the reflexive blink was the only response to the air puff (figure 2a). After one to seven days of training, carefully timed eye blink was evoked before the arrival of air puff (figure 2a). When the activity of CA3 and CA1 neurons was measured with the microelectrode recording, the activity of neurons increased after one day of the training. However, after 7 days of training, the intrinsic excitability returned to the baseline values while the memory for the association remains strong (figure 2b, 2c). This transient, about 1 to 3 days, increase in excitability suggests that the intrinsic plasticity mechanism adjusted the ion channel properties and induced the persistent changes in neuronal excitability.

Intrinsic plasticity has been reported for various systems to play an important role in maintain the system homeostasis [16]. Furthermore, previous studies have shown that IP is closely related to learning in behaving animals [15]. In a computational point of view, it has been studied that IP maximizes the information transmission of a neuron under the metabolic constrains [17]. Therefore, IP plays an important role in achieving ICA-like learning in brain.

2.2.2 Mathematical model of intrinsic plasticity

As discussed in the previous section, intrinsic plasticity changes the firing rate of the neuron. The mathematical model of the firing rate of the neuron is defined as:

$$g(u_t) = \boldsymbol{r_0} \log(1 + \exp\left(\frac{u_t - \boldsymbol{u_0}}{\boldsymbol{u_a}}\right))$$

In this model, u_t is total postsynaptic potential, and $g(u_t)$ is the firing rate of the postsynaptic neuron. r_0, u_0, and u_a are three parameters determining the intrinsic excitability of a neuron. u_0 represents the threshold voltage; when the membrane potential is below u_0, the firing rate goes to 0. Above u_0, the firing rate increase linearly with the membrane potential with the slope r_0/u_a.

Intrinsic plasticity changes the neuron's intrinsic excitability by adjusting three parameters r_0, u_0, and u_a. Figure 3 shows the change in the firing rate when three parameters are tweaked. In figure 3, default case is depicted in blue. Green is the case where r_0 is increased by a factor of 1.5, red is the case where u_0 is increased by 5 mV, and purple is the case where u_a is increased by a factor of 1.2. This figure shows that r_0 gives the slope of the curve, u_0 shifts the entire curve left or right, and u_a rescales the membrane potential axis.

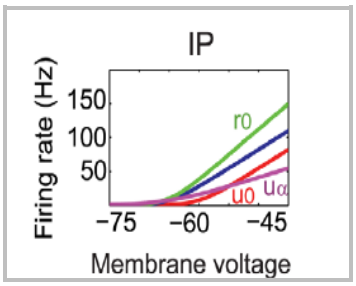

Figure 3: Intrinsic plasticity [14]

How does the intrinsic plasticity change the intrinsic excitability? What are the updating rules for the three parameters? Joshi and Triesch studied the mathematical update rule of the intrinsic plasticity [18]. As in classic ICA algorithm, brain attempts to maximize the mutual information between input and output distribution of a neuron, because the sensory information should be encoded by a limited number of responses for a fixed energy budget. Information theory argues that exponential distribution of firing rate can achieve the maximum mutual information because the exponential distribution has the maximum entropy for a fixed mean. In fact, biological evidence has been reported that the neurons in the visual cortex of cats and macaques respond with an approximately exponential distribution of firing rates in response to stimulation with natural videos. From the mathematical argument of information theory and biological evidence, it has been proposed that the one of the goals of intrinsic plasticity (IP) might be to obtain such an exponential distribution of firing-rate [18]. Based on this argument, learning rules for the three parameters r_0, u_0, and $u_a$were derived by minimizing the Kullback-Liebler divergence (KLD) between the output distribution and a desired exponential distribution (figure 4).

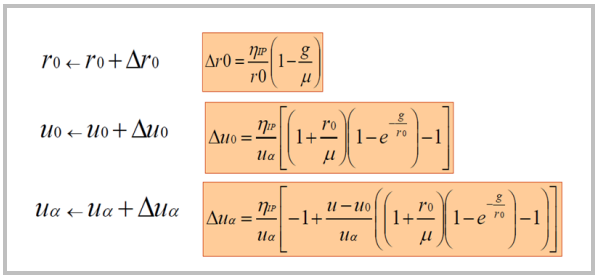

Figure 4: Learning rules for the IP

2.3 Spike-timing dependent plasticity (STDP)

2.3.1 Biological meaning of STDP

According to the Hebbian rules, synaptic efficiency is increased when the synapses persistently causes the postsynaptic neurons to generate action potentials. A simpler phrase can explain Hebbian rule as following: "those who fire together, wire together". With the technical development, spike timing of neurons were studied more precisely, and it turned out that synaptic strength was strengthened when the presynaptic neuron fires shortly before the postsynaptic neuron. Spike-time dependent plasticity refers to such biological process that alters the synaptic strength between neurons in response to the relative timing of presynaptic and postsynaptic action potentials. Since both Hebbian rule and STDP increases the efficiency of synapses in transferring the information, they are also closely linked to ICA-like learning in brain.

2.3.2 Mathematical model of STDP

STDP rule can be implemented when the input and output are spike-based. Yet, for simplicity, classic Hebbian learning rule was used: $\Delta w_i = \eta u_i' g(u_t)$. Here, w_i is the synaptic weight between ith presynaptic neuron and a postsynaptic neuron, η is synaptic learning rate, u_i' is the membrane potential of ith presynaptic neuron, and $g(u_t)$ is the firing rate of the postsynaptic neuron for the postsynaptic potential u_t.

2.4 Synaptic scaling

Biologically, synaptic scaling mechanism multiplicatively scales the synaptic weights to preserve the average input drive received by the neuron [19]. This mechanism allows single neurons to regulate the overall action potential firing rate by normalizing the synaptic strengths of all neurons in a network. Without the homeostatic feedback loop, persisting correlated neural activity continues up regulating the synaptic strengths. Then at some point, insignificant perturbation can trigger chaotic synchronous network-side firing which leads to the unstable neural activity. Therefore, synaptic scaling mechanism is required to homeostatically regulate firing rate. Synaptic weights were normalized by dividing each weight by $\frac{\sum w_i}{w_{total}}$, so the total weight was maintained.

2.5 Experiment setup: Simple demixing problem

For our project, a simple neuronal model based on the biologically plausible fashion was used to solve a classic ICA problem: simple demixing problem to separate two mixed signals in independent source signals. To achieve this problem, we used a simple neuronal model with two presynaptic neurons connected to one postsynaptic neuron (figure 5).

For the classic ICA problem, we generated two source signals u_1 and u_2. Then we mixed two signals by taking the linear superposition using the rotation matrix A.

$$A = \begin{pmatrix} \cos(\alpha) & \sin(\alpha) \\ -\sin(\alpha) & \cos(\alpha) \end{pmatrix}, \qquad u' = Au$$

Mixed signals u_1' and u_2' are the inputs to the neuronal system. Membrane potential of the presynaptic neurons are u_1' and u_2', respectively. Membrane potential of the postsynaptic neuron is $u_t = w_1 u_1' + w_2 u_2'$, and the firing rate of the postsynaptic neuron is $g(u_t) = r_0 \log(1 + \exp\left(\frac{u_t - u_0}{u_a}\right))$. w_1, w_2, r_0, u_0, and u_a were updated using the update rules discusses in the previous section. Following parameters were used: $\eta_{IP} = 4 \cdot 10^{-5}$, $\eta_{syn} = 2 \cdot 10^{-5}$, $\mu =$ 2Hz, $w_{tot} = 1$, $\mu = 2$Hz. Weights and parameters for the intrinsic plasticity were initialized as following: $w_1 = 0.8$, $w_2 = 0.2$, $r_0 = 5$Hz, $u_0 = 0$mV, $u_\alpha = 2.5$mV.

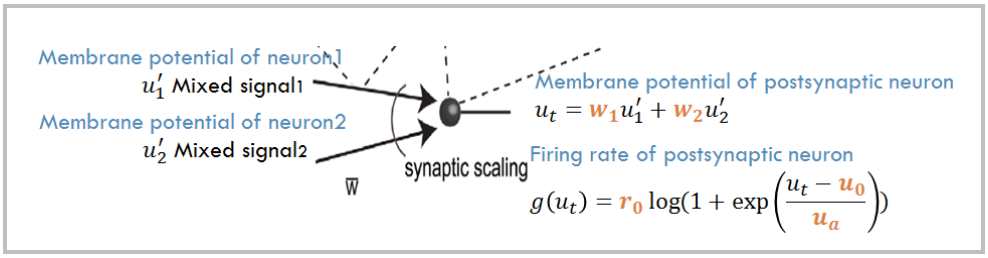

Figure 5 Experiment setup for simple demixing problem

3 Results

3.1 Classic demixing problem

3.1.1 Independent Laplacian distributed input and sinusoidal wave input

For the source signal, independent Laplacian distributed input and sinusoidal wave input were used. u_1 was randomly generated from the Laplacian distribution with unit variance: $p_{u_1}(u_1) = \frac{1}{\sqrt{2}} e^{-\sqrt{2}|u_1|}$, and u_2 was generated as sinusoidal wave. Two signals were mixed with the rotation matrix with $\alpha = \frac{\pi}{3}$. Source signal and mixed signal are shown in figure 6.

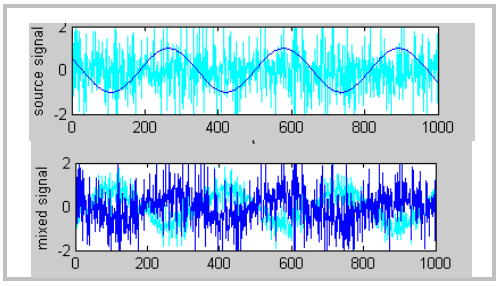

Figure 6: Source signal and mixed signal

Three parameters r_0, u_a, u_0 were updated by intrinsic plasticity and weight vector w_1, w_2 were updated by STDP and synaptic scaling (figure 7). After training for the $5 \cdot 10^5$ samples, parameters were stabilized. The distribution of the firing rate of the postsynaptic neuron became close to the exponential distribution.

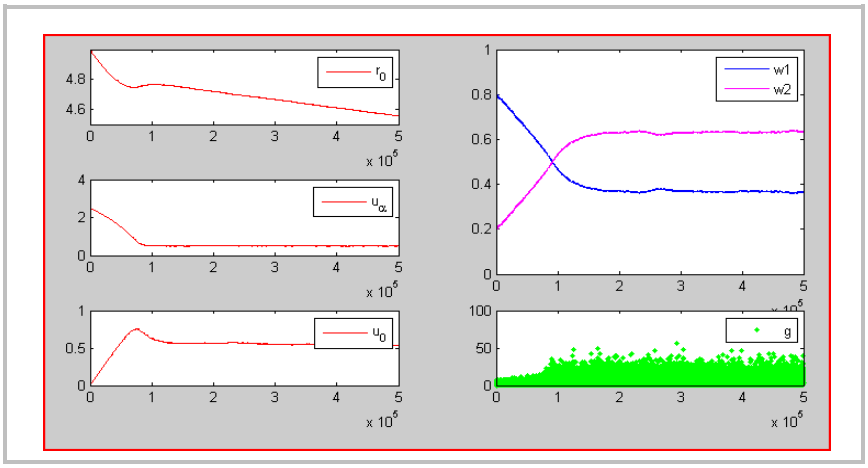

Figure 7: The evolution of three intrinsic parameters (r_0, u_a, u_0), weight vector $[w_1, w_2]$ and firing rate of the postsynaptic neuron, g.

More interestingly, final weight vector aligns itself along the direction of the mixed signals. Therefore, rotation angle α was identified by the updated weights, and we can demix our signals using the demixing matrix, A^{-1}.

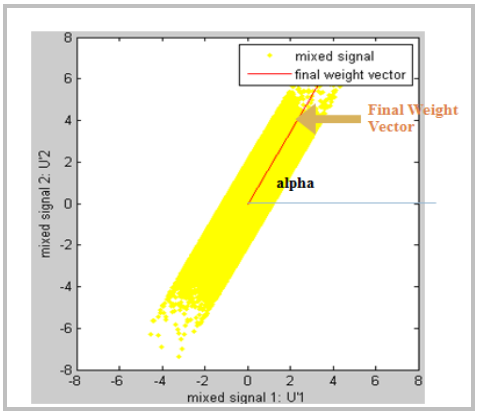

Figure 8: Distribution of mixed signals and final vector aligned

Figure 9 shows the source, mixed, and demixed signals for various rotational angles $\frac{\pi}{9}, \frac{\pi}{6}, \frac{pi}{4}$, and $\frac{pi}{3}$. As shown in the results, our neuronal model based on the biological plasticity successfully demixed the signal, and thus achieved the ICA-like learning.

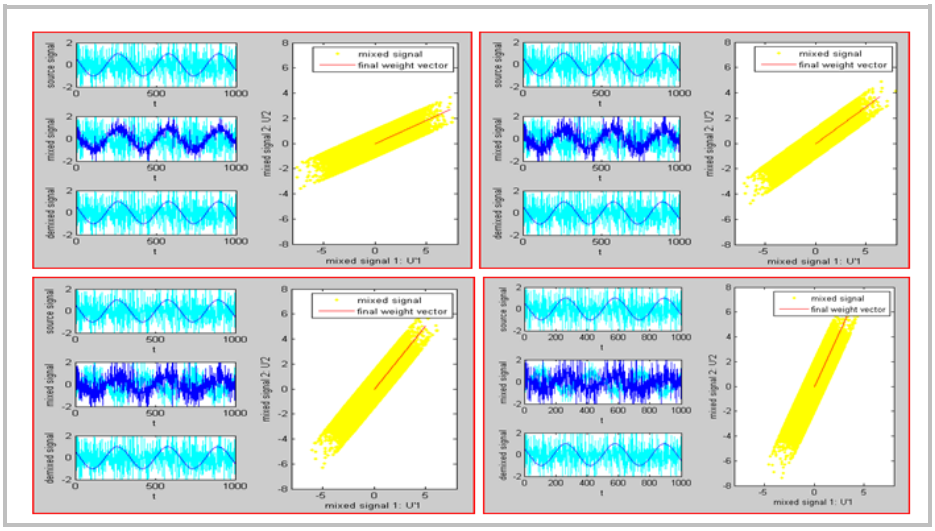

Figure 9: Independent Laplacian distributed input and sinusoidal wave input. Source, mixed, and demixed signals for various rotational angles $\frac{\pi}{9}, \frac{\pi}{6}, \frac{pi}{4}$, and $\frac{pi}{3}$.

3.1.2 Independent Laplacian distributed input and square wave input

We tried different types of inputs using Laplacian distributed input and square wave input. Though the performance of demixing was not as great as the sinusoidal wave input, the demixing was still good enough (figure 10). Later in the next section, we modified our learning rules to improve the accuracy.

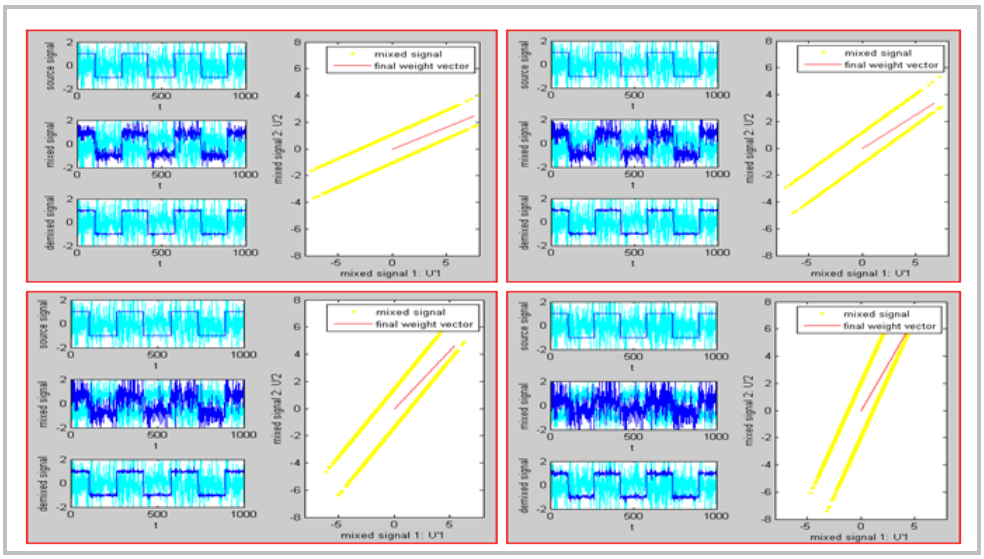

Figure 10: Independent Laplacian distributed input and square wave input. Source, mixed, and demixed signals for various rotational angles $\frac{\pi}{9}, \frac{\pi}{6}, \frac{pi}{4}$, and $\frac{pi}{3}$.

3.1.3 Square wave and sinusoidal wave inputs

Similarly, square wave and sinusoidal wave inputs were used, and our model successfully demixed the signals again (figure 11).

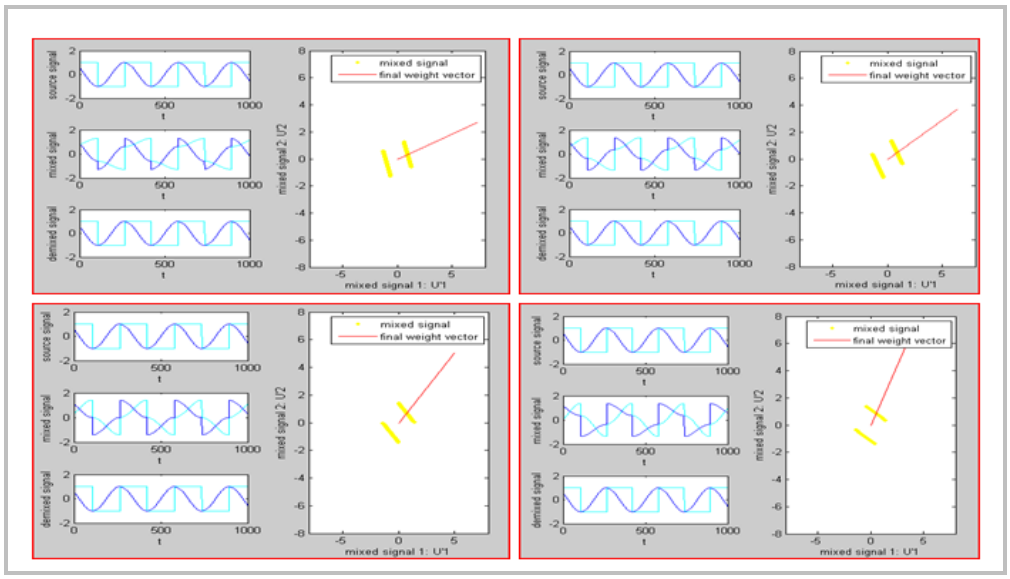

Figure 11: Square wave and sinusoidal wave input. Source, mixed, and demixed signals for various rotational angles $\frac{\pi}{9}, \frac{\pi}{6}, \frac{pi}{4}$, and $\frac{pi}{3}$.

3.2 Effect of plasticity in neuron model

Here, we further investigate the effects of three types of plasticity in the neuron model, i.e. IP, synaptic scaling and IP, on learning. The same experiment of solving classic demixing problem is used. We measure learning quality as the final weights the model learned.

3.2.1 Intrinsic plasticity

First of all, we evaluate the effects of intrinsic plasticity (IP) in the neuron model, which includes three parameters, r_0, u_0, and u_α, and corresponding update rules as shown in figure 4. We compare five different cases: full neuron model (as in 3.1), constant r_0 model, constant u_0 model, constant u_α model, and all constant model. For example, constant r_0 model means the r_0 update rule is ignored and r_0 is only a constant during learning. Also, in the experiments we use square wave and a Laplacian distributed input as two independent inputs. Same rotation matrix and mixing process is performed. The demixing outputs are shown in figure 12.

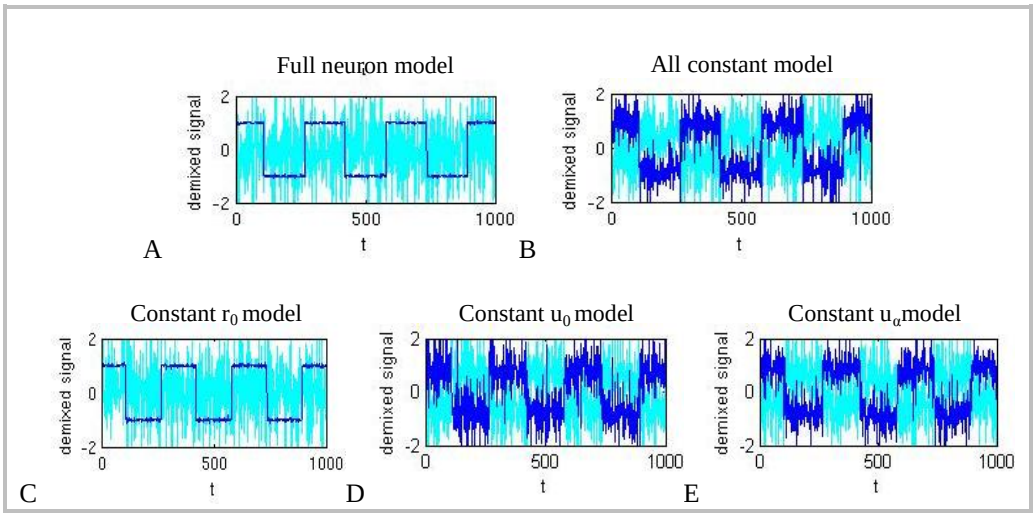

Figure 12. Full neuron model vs. simplified IP model

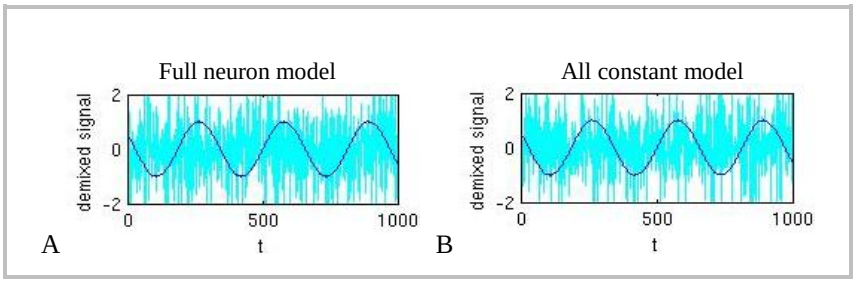

Figure 13. Full neuron model and all constant IP model with sinusoidal input

In figure 12, simplified IP models shown in B, D and E are unable to separate the two sources, which means u_0 and u_α update rules are essential for the learning. It's not surprising because u_0 and u_α are related to spiking threshold properties. It is to be noted that constant u_0 model is even worse than all constant model, which contradicts to our knowledge. By performing the experiment several times with different initial conditions, we found that the effect changes each time. Hence it's probably due to initial conditions

Interestingly, in figure 12C, we found that constant r_0 model perform well in separating square wave and Laplacian distributed input. By giving different initial conditions of r_0, the results change accordingly. Therefore by choosing a proper value for r_0, the r_0 update rule can be ignored without losing much learning ability.

However, if we take a look at how simplified IP models work for demixing sinusoidal and Laplace distributed random number, as in figure 13, we found that, surprisingly, even the all constant model is able to separate the two sources. Other simplified IP models have the similar results which are not shown here. The results suggest that the neuron model can learn to demix simple sources like sinusoidal wave without IP properties. In other words, by STDP and synaptic scaling characteristics, the neuron model is able to perform basic learning.

3.2.2 Synaptic Scaling

Next, we move on to the effects of synaptic scaling in the neuron model on learning. The non-scaled model does not take the synaptic scaling process into account. Similar to the experiment setup described in 3.2.1, we test the full neuron model and non-scaled model with three different combinations of sources. The results are shown in figure 14.

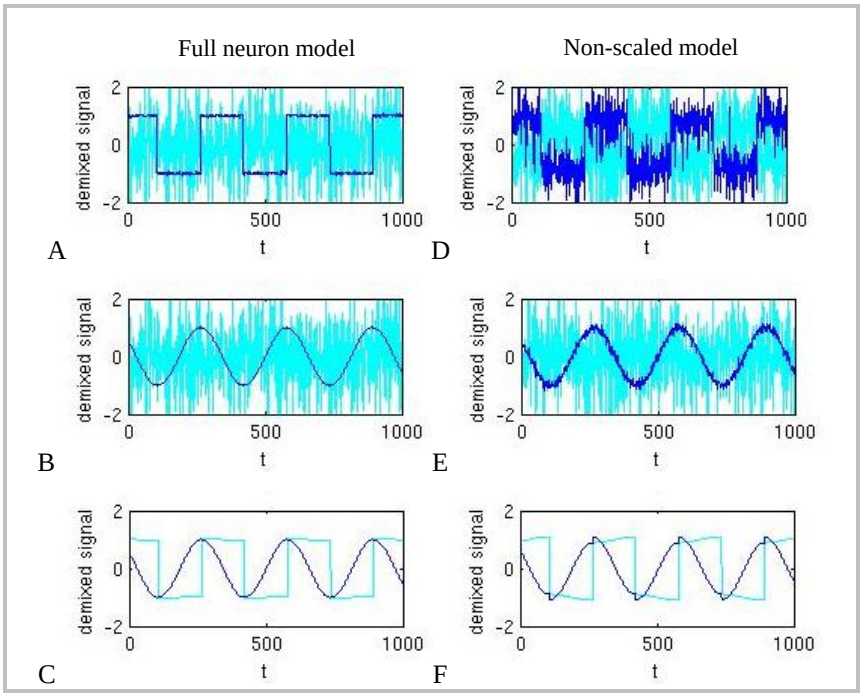

Figure 14: Full neuron model vs. non-scaled model

In figure 14 D-F, we observe that all the three combinations of two sources have worse results in non-scaled model. Unlike the all constant model described in section 3.2.1, the neuron model lack of synaptic scaling property loses the learning ability even to demix simple sinusoidal signals. Biologically, synaptic scaling represents neurons have to preserve the average input drive received. Hence synaptic scaling plasticity is essential for learning in the neuron model.

3.2.3 Spike-timing dependent plasticity (STDP)

Finally, we evaluate the effects of STDP in the neuron model. Original neuron model considers STDP weight updating rule $\Delta W_i = \eta u_i' \cdot g(u_t)$, which corresponds to one of Hebbian learning rules, $\frac{dW_{ij}}{dt} = \eta V_i \cdot V_j$. Hence we test STDP characteristic by choosing different Hebbian learning rules. Figure 15 shows the Hebbian learning rules and results.

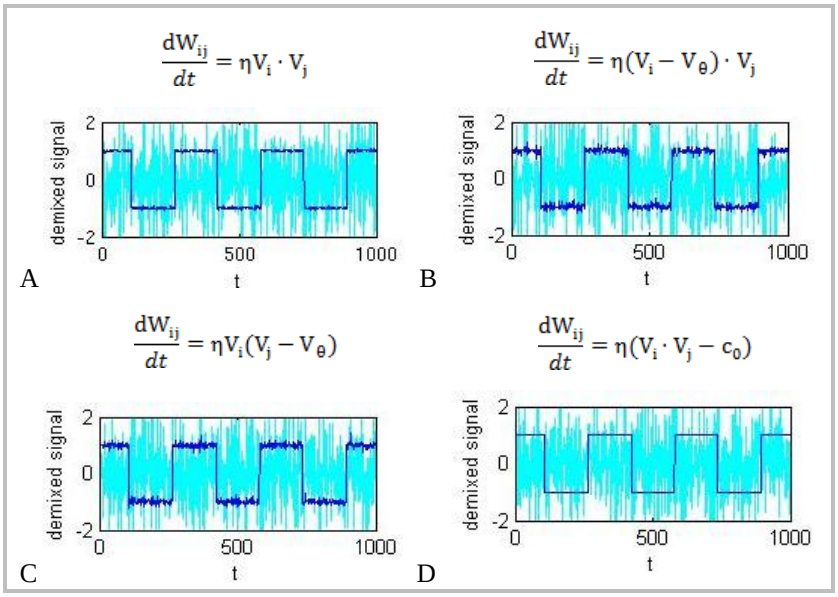

Figure 15: STDP models for different Hebbian learning rules

Compared to the result of original neuron model shown in figure 15 A, the results of Hebbian learning rules in B and C are slightly worse. On the other hand, the learning rule in D performs even a better job than the original STDP rule in the task of separating square wave and random inputs. However, if we use different initial conditions or inputs, the results would change dramatically. That is, four Hebbian learning rules have different performances under various conditions. Hence, one can choose the best STDP learning rule which fits in the experiment setup.

4 Conclusion

In the report, we investigate how the plasticity in the neuron model implements ICA-like learning in both theoretical and experimental perspectives. The model considers three types of plasticity: STDP, IP, and synaptic scaling. The corresponding update rules are derived based on the concept of maximizing information transmission and achieving exponentially distributed firing rate. The neuron model is tested by solving classic demixing problem. The results show that all three plasticity play important but different roles in ICA-like learning. IP can be simplified under specific initial conditions, while synaptic scaling property is essential for learning. STDP alters the performance of learning and should be carefully chosen to optimize the learning result. The neuron model provides a biological perspective that can potentially explain how our brains learn and why our brains have such high capacity and complexity.

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