THE SYNCHRONIZATION BEHAVIOR OF BASAL GANGLIA

Y. Cakir

Abstract-A network model of basal ganglia (BG) that comprises striatum, internal and external segments of globus pallidus, subthalamic nucleus, substantia nigra pars reticulate neuronal sub populations and thalamus is constructed. The dynamic behavior of network is investigated using Izhikevich neuron model. The influences of dopamine, the synaptic strength and the neuronal interconnection density on the synchronization behavior of the subpopulations are investigated. In the case of dopamine depletion, the increased striatal synchronization is observed. Considerable effect on the synchronization of other basal ganglia sub populations is not observed in the case of dopamine depletion. The highest synchronization values are observed for the lowest synaptic strength and the neuronal interconnection density. The decreased neuronal interconnection density causes a decrease in the fluctuation in synchronization in thalamic neurons depending on dopamine value.

Keywords—basal ganglia, synchronization, Izhikevich neuron model

1. INTRODUCTION

THE basal Ganglia (BG), together with thalamus and cortex comprise a complex network that is responsible for variety of functions and dysfunctions. Basal ganglia has a key role in the abnormal neural oscillations, observed in Parkinson's disease or dystonia. Parkinson's disease is a neurodegenerative disorder whose patients experience motor deficits such as slowness of movement, rigidity, a low frequency rest tremor, and difficulty with balance and also non-motor deficits such as depression, constipation, pain, genitourinary problems, and sleep disorders. The motor deficits are due to the degeneration of dopamine containing neurons in the Substantia Nigra pars compacta (SNc) and consequent loss of dopamine in the striatum [1].

Neural activity in the brain of parkinsonian patients is characterized by the intermittently synchronized oscillatory dynamics [2]. In the patients with Parkinson's disease and in animal models of this disorder, the neurons in the basal ganglia and the related regions in thalamus and cortex show changes including altered firing rates and patterns, pathologic oscillatory activity and increased inter-neuronal synchronization [3]. Subthalamic Nucleus (STN), Globus Pallidus Internal (GPi), and Globus Pallidus External (GPe) neurons in the basal ganglia show high levels of synchrony in Parkinsonian conditions [4].

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More recent studies suggest the key role of the BG in "nonmotor" diseases such as absence epilepsy which is a generalized non-convulsive epilepsy. In these diseases, the symptoms accompany various oscillatory patterns of neural activity often synchronized across the BG, cortex and other brain areas [1]. Recent studies in animals and humans have revealed the existence of several types of oscillatory activity in the various nuclei of the basal ganglia and, although still poorly understood, are believed to play an important function in both the normal physiology and pathophysiology of this system [5].

On the other hand, Alzheimer's disease (AD) is the most prominent aging dependent neurodegenerative disorder. Brain atrophy caused by neuronal loss is a prominent pathological feature of Alzheimer's disease. Cholinergic neurons are most damaged in AD brain [6]. Reduction in potential healthy synapses and the strength of connections among the neurons is observed in case of AD [7].

Since in the case of brain diseases pathologies such as neuronal lost, dopamine depletion and reduction of synaptic strength is observed, in this work, the synchronization level of neuronal activity of Basal ganglia regions such as Striatum, GPi/SNr, GPe, STN and Thalamic relay neurons is investigated according to these pathologies. For different dopamine levels, different synaptic strength values and neuronal interconnection densities, the synchronization level is obtained and analyzed. The simulation results reveal that the striatal synchronization is increased in the case of dopamine depletion. For the lowest synaptic strength and the neuronal interconnection density, the highest synchronization values are observed.

2. BASAL GANGLIA AND THALAMUS NETWORK STRUCTURE

The Basal ganglia region of network given in Fig. 1 consists of Striatum, Globus Pallidus External (GPe), Globus Pallidus Internal (GPi), Substantia Nigra pars reticulate (SNr) and Subthalamic Nucleus (STN) sub networks. GPi and SNr are different neuronal populations but in this work they are considered as a single GPi/SNr structure due to their closely related inputs and outputs and similarities in cytology and function [8].

The striatum sub network structure is as that defined in [9] but include the latest findings about the network structure given in [10]. The rate of Fast Spiking Interneurons (FSI) in the striatum is roughly 10 %. The half of the left 90 % consists of D1 dopamine receptor type Medium Spiny Neurons (MSN) and the other half of D2 dopamine receptor type MSN's. FSI neurons in the striatum has inhibitory

GABAergic connections with the other FSI neurons and D1 and D2 MSN neuronal population. Also, there is gap junction type connection between FSI neurons. The D2 MSN neurons has inhibitory GABAergic connection with other D2 MSN neurons, D1 MSN neurons population and GPe neurons. The D1 MSN neurons has inhibitory connections with the other D1 MSN neurons and GPi and SNr neuronal populations.

The GPe receives excitatory input from STN neurons and other GPe neurons over local axon collaterals which have an inhibitory influence. The GPe sends an inhibitory projection to GPi and to STN. The subthalamic nucleus is the only portion of the basal ganglia that produces an excitatory neurotransmitter glutamate which excites both the GPe and GPi/ SNr output nuclei.

The external inputs of Basal ganglia are Thalamocortical signal originating from cortex region and dopamine signal originating from SNc region that modulate the striatal neurons. Thalamus region in the network consists of Thalamocortical relay cells that receive excitatory sensorimotor inputs from cortex region. This neurons function is affected by inhibitory synaptic connections from GPi/SNr basal ganglia neuronal population. The striatal activity has excitatory effect on the thalamus via the D1 MSN neurons connection from the striatum to the output nuclei called direct pathway, and an inhibitory effect via the D2 MSN neurons connection to the output nuclei over GPe and the STN called indirect pathway [8, 11, 12].

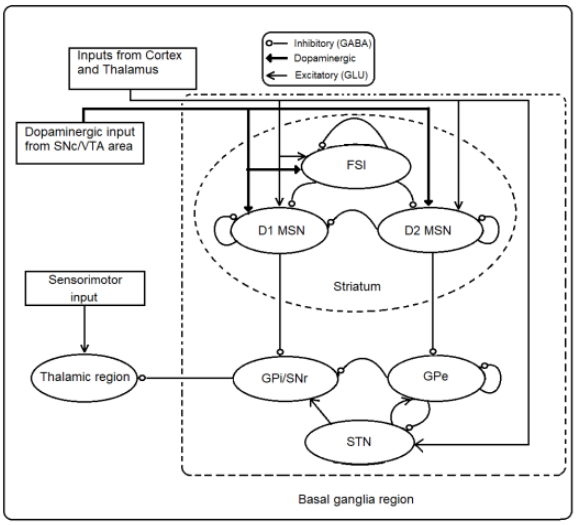


Fig. 1. Structure of Basal ganglia thalamocortical network

3. IZHIKEVICH NEURA MODEL

The neuronal populations of Basal ganglia and Thalamus are modeled by using phenomenological *Izhikevich* model given in Eq. 1 [9,13,14].

Here C is the membrane capacitance. The dimensionless variables v and u represent the membrane potential of the neuron and the membrane recovery, respectively. v_r, v_t are the resting and threshold potentials.

$$\begin{aligned} C\dot{v} &= k(v-v_r)(v-v_t)-u+I \\ \dot{u} &= a[b(v-v_r)-u] \\ v &\geq v_{peak} \;\; v \rightarrow c, u \rightarrow u+d \end{aligned} \tag{1}$$

a, *b*, *c*, and *d* are dimensionless parameters. v and u account for the activation and inactivation of ionic currents, respectively. When the membrane potential reaches vpeak, the values of v and u are assigned with *c* and u+d as given in the last expression of Eq. 1. [9, 13, 14]. I denotes the currents consisting of the externally applied and the synaptic current, ($I = I^{out} + I^{syn}$). The externally applied current represents the effects of inputs coming from outside of the network. All populations in network consist of 100 neurons connected randomly by predefined connection probability.

The striatum region neurons are affected by dopamine neuromodulator. Dopamine inhibits the D2 dopamine receptor type MSNs in the indirect pathway and excites D1 dopamine receptor type MSNs in the direct pathway [15]. The FSI and D1 and D2 MSN neuron models of Striatum region are as:

$$C\dot{v}_{fs} = k\left[v_{fs} - v_r(1-\eta\phi_1)\right](v_{fs}-v_t) - u_{fs} + I \tag{2}$$

$$\dot{u_{fs}} = \begin{cases} -au_{fs}, & if \;\; v_{fs} < v_b \\ a\left[b(v_{fs}-v_b)^3 - u_{fs}\right], & if \; v_{fs} > v_b \end{cases} \tag{3}$$

$$C\dot{v}_{D1} = k(v_{D1}-v_r)(v_{D1}-v_t) - u + I + \phi_1 g_{DA}(v_{D1}-E_{DA}) \tag{4}$$

$$C\dot{v}_{D2} = k(1-\alpha\phi_2)(v_{D2}-v_r)(v_{D2}-v_t) - u + I \tag{5}$$

where the reset condition of D1 and D2 MSN neurons is as that given in Eq. 1.

Dopamine has a dual action on MSNs; it inhibits the (D2-type) MSNs and excites (D1-type) [15]. $\phi_1 g_{DA}(v_{D1} - E_{DA})$ in D1 MSN neuron model is for simulating the hyperpolarizing effect of D1 activation and $k(1 - \alpha\phi_2)$ in D2 MSN neurons is for reflecting increased sensitivity to the injected current where ϕ1 and ϕ2 are the parameters for specifying the level of dopamine, taking value between 0 and 1. The external current is modeled as

$$I^{out} = I_o + \rho\alpha_i(t) + \sigma I_{cortical} \tag{6}$$

where Io , ρ and σ are the constants, α(t) is white noise with zero mean value and $I_{cortical}$ is a pulse shaped signal with unit amplitude and frequency of 2 spike/s, simulating cortical input [15]. The synaptic input to all MSNs is $I^{syn} = I_{gaba-fs} + I_{gaba-ms}$.

The internal synaptic current of any neuron is the sum of exponentially decaying presynaptic currents of connected neurons, which reach to considered neuron with a delay. The synaptic current of any i'th MSN neuron of type Ii=Igaba_fs + Igaba-ms is given in Eq. 7.

$$I_i(t) = g_{gaba\_fs}\sum_l r_{il}(t-\tau_{il})\left[E_{gaba\_fs} - V_i\right] - g_{gaba\_ms}\sum_j r_{ij}(t-\tau_{ij})\left[E_{gaba\_ms} - V_i\right] \quad (7)$$

where ggaba_fs and ggaba_ms are the chemical conductivity strengths, and τil and τij represents time delays from l'th and j'th presynaptic neurons to i'th postsynaptic neuron. Time delay between striatal neurons was assumed zero during analysis in this work. Egaba_fs and Egaba_ms are the reversal potentials for the inhibitory synapses and was taken as Egaba_fs = Egaba_ms =- 60 mV. For each spiked connected neuron, (jth connected to ith neuron), the connection strength between these neurons is updated as $r_{ij} \leftarrow r_{ij} + \omega_{ij}$, here ωij is the inhibitory synaptic strength from neuron j to neuron i. It is taken as constant, ωij =ω [9, 14, 16].

rij is the dynamic variable that decreases exponentially depending on decay time (synaptic time constant) τs=τgaba_*fs*=τgaba_ms as given in Eq. 8.

$$\tau_s \dot{r}_{ij} = -r_{ij} \quad (8)$$

The gap junction type connection between FSI neurons provide an extra current of electrical type and is included in the model as I_{gap}. The synaptic input for FSI neurons is as:

$$I^{syn} = I_{gaba-fs}(1 - \varepsilon\phi_2) + I_{gap} \quad (9)$$

where $(1 - \varepsilon\phi_2)$ represents dopamine effect and junction current for any i'th FSI neuron is defined as is in Eq. 10.

$$I_i(t) = g_{gap}\sum_k \left[V_k - V_i\right] \quad (10)$$

Here ggap is the constant electrical synaptic strength from neuron *k* to *i*. Vk and Vi are pre and postsynaptic neuron's membrane potential.

By using the simplified form of Izikhevich neuron model given in Eq. 11, the rest of basal ganglia network is constructed [8].

$$\dot{v} = 0.04v^2 + 5v + 140 - u + I$$
$$\dot{u} = a(bv - u)$$
$$if\ v \geq 30\ mV, then\ v \rightarrow c, u \rightarrow u + c \quad (11)$$

As in the striatal neurons, the input current $I_i = I_i^{app} + I_i^{syn}$ of any i'th neuron in the rest of basal ganglia consists of the external applied current I^{app} and the summation of synaptic currents coming from other neurons having synaptic connections with it expressed as I_i^{syn}. The external currents for GPi/SNr, GPe and STN populations are constant valued, whereas the externally applied current to Thalamus region is puls shaped signal with frequency of 14Hz comprising sensorimotor input (ISM) to this region. The synaptic current is in the form of

$$I_i^{syn} = \sum_j I_{ij}^{syn} = \sum_j g_{ij}\, r_{ij}(t-\tau_{ij})[E_S - V_i] \quad (12)$$

where the synaptic current from j'th neuron to i'th neuron has coupling strength denoted as g_{ij} and τ_{ij} denotes synaptic delay time between pre and postsynaptic neurons and E_S is reversal potential that is $E_S = 0\ mV$ for excitatory and $E_S = -80mV$ for inhibitory connection. The update mechanism of connection strength r_{ij} between pre and postsynaptic neurons is the same as update process in striatum neurons. Beside striatal neurons, the synaptic delay time between pre and postsynaptic neurons in the basal ganglia are taken as described in [8]. The striatum region neuron parameters used in analysis are chosen as that described in [9] and are given in Table 1.

TABLE 1
PARAMETERS FOR FSIS AND MSNs

Parameters for FSI neurons		Parameters for MSN neurons	
a	0.2	C	50 pF
b	0.025	b	-20
d	0	c	-55 mV
k	1	v_r	-80 mV
v_{peak}	25 mV	v_{peak}	40 mV
v_b	-55 mV	k	1.14
C	80 pF	v_t	-33.8 mV
c	-60 mV	a	0.05
v_r	-70 mV	d	377
v_t	-50 mV	α	0.03
η	0.1	g_{DA}	22.7 nS
ε	0.625	E_{DA}	-68.4 mV
E_{gaba_fs}, E_{gaba_ms}	-60 mV	E_{gaba_fs}, E_{gaba_ms}	-60 mV
τ_{gaba_fs}	4 mS	τ_{gaba_fs}, τ_{gaba_ms}	4 mS
g_{gaba_fs}	20 nS	g_{gaba_ms}	4.36 nS

The network parameters of the Thalamus and other sub population of Basal ganglia, including GPi/SNr, GPe and STN neuronal populations was given in Table 2 and 3.

TABLE 2
GPi/SNr, GPe STN AND THALAMUS NEURONAL POPULATION'S NEURON PARAMETER VALUES

Population	a	b	c	d	τ_s ms	E_s mV	I^{app}
GPe	0.01	0.58	-65	4	5	-80	10
GPi/SNr	0.01	0.58	-65	4	100	-80	15
STN	0.01	0.26	-65	2	10	0	1
Thalamus	0.01	0.23	-65	0.45	10	0	I_{SM}

The parameters values are determined by considering the recent studies on the basal ganglia neurons dynamics and [8, 9, 12] to simulate the neuronal firing rates and patterns. Different from [8], the present network includes the detailed striatum sub network whereas in [8], the striatum is depicted as an external input only. The basal ganglia thalamic network in [12] is mass modeled whereas the present network

structure includes Izhikevich neuron-based network model of basal ganglia region.

TABLE 3
THE CONNECTIVITY RELATIONS BETWEEN SUB NETWORKS

Connectivity, α→β	Synaptic conductance, g
MSN D1 → GPi/SNr	0.75
MSN D2 → GPe	0.75
STN → GPi/SNr	0.075
STN → GPe	0.075
GPe → STN	0.025
GPe → GPe	0.025
GPe → GPi	0.015
GPi →Thl	0.01

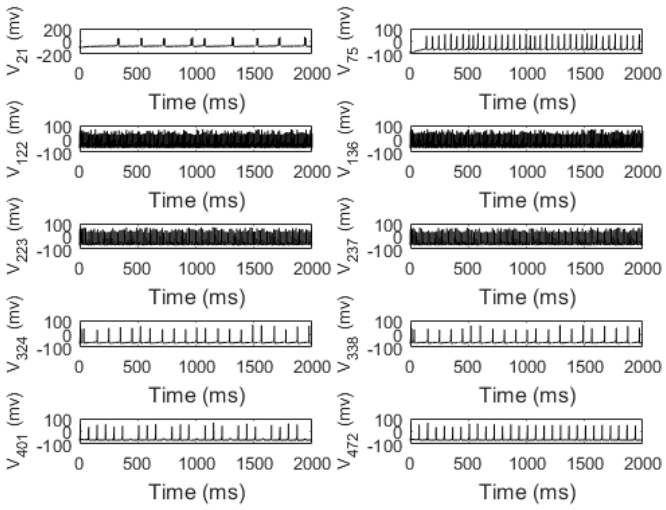
4. SIMULATION RESULTS

Since in the case of diseases such as Alzheimer's and Parkinson's disease, the neuron death, the synaptic connectivity degenerations and decrease in dopamine level is observed, the synchronization in the neuronal populations is analyzed by changing the synaptic connectivity number and the synaptic strength and by reducing the amount of dopamine. The degree of synchrony in the network is calculated according to the method developed by Hansel and Sompolinsky [17]. The synchronization measure is computed as:

$$S = \frac{\frac{1}{T}\sum_{t=1}^{T}\left[\left(\frac{1}{N}\sum_{i=1}^{N}V_i(t)\right)^2\right]-\left[\frac{1}{T}\sum_{t=1}^{T}\left(\frac{1}{N}\sum_{i=1}^{N}V_i(t)\right)\right]^2}{\frac{1}{N}\sum_{i=1}^{N}\left[\frac{1}{T}\sum_{t=1}^{T}V_i(t)^2-\left(\frac{1}{T}\sum_{t=1}^{T}V_i(t)\right)^2\right]} \quad (13)$$

The neuronal activity of some neurons obtained for ϕ1 = ϕ2=1 comprising normal level of dopamine are given in Fig. 2a as an example of neuronal activity of network.

The network populations connectivity is obtained for the probability value of 0.1, i.e. any neuron has a connection probability of 0.1 to other and synaptic strength as wij=0.01. The firing pattern of all neurons in the network is given in Fig. 2b. The first 10 neurons are striatal FSI neurons, the following 45 neurons are D1 MSN (neurons number 11-55) and the next 45 neurons are D2 MSN neurons (neurons number 56-100). The neurons from 101 to 200 are GPi/SNr, neurons from 201 to 300 are GPe, neurons from 301 to 400 are STN and the last 100 neurons are Thalamic relay neuronal populations. It is seen that dopamine has different effect on D1 and D2 MSN neurons firing, and because of the applied 14 Hz external sensorimotor signal, the thalamic relay neurons fires approximately at this frequency.



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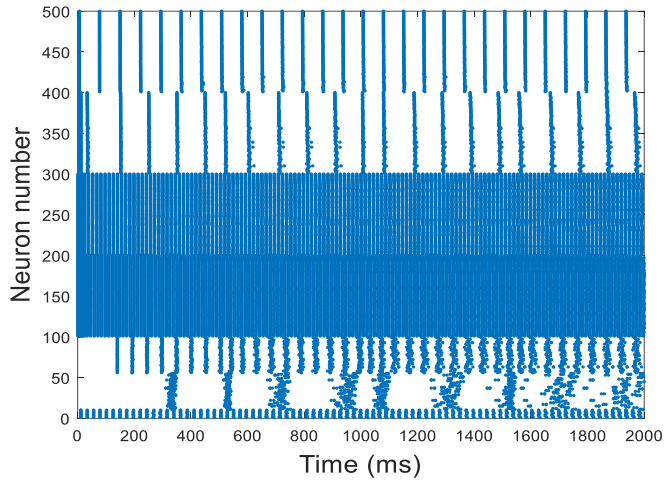


b)

Fig. 2

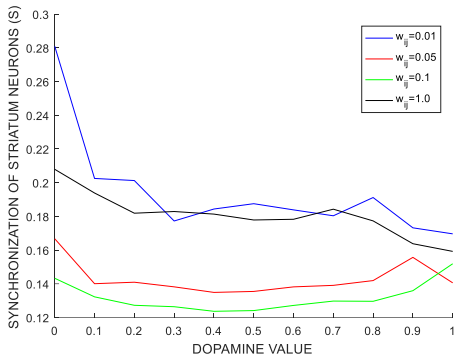
a) The neuronal activity of randomly selected neurons,

b) the raster plot of network obtained for connection probability of 0.1, dopamine level

ϕ1 =ϕ2=1 and wij=0.01

By decreasing the connection probability from 0.5 to 0.1 for 2000 ms, the network activity was obtained for striatal neurons synaptic strength wij values of 1.0, 0.1, 0.05 and 0.01 and dopamine values between ϕ1 =ϕ2=0 (comprising total dopamine depletion) and ϕ1 =ϕ2=1 (comprising no lack of dopamine). The synchronization measure, S for these parameter values are calculated.

The obtained synchronization measure is the mean value of the synchronization values of 5 different networks randomly generated with above mentioned connection probabilities. The obtained results are given in Fig. 3, 4 and 5.



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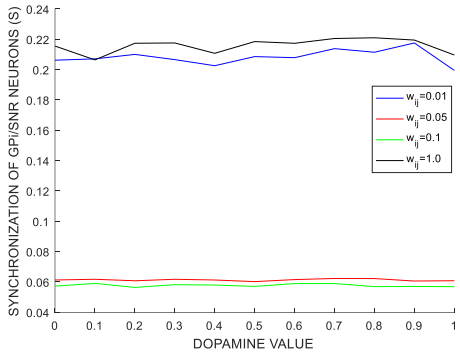

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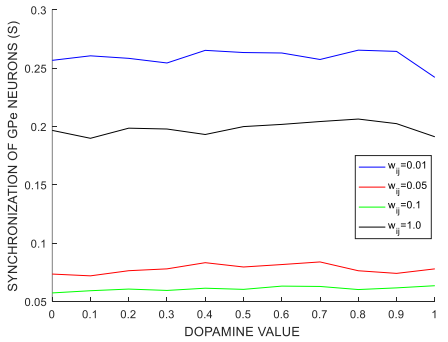

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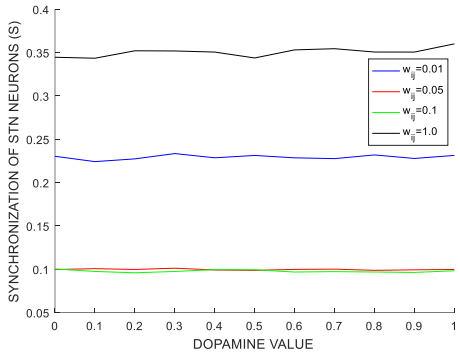

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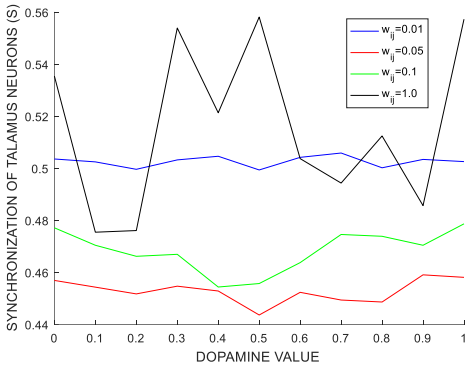

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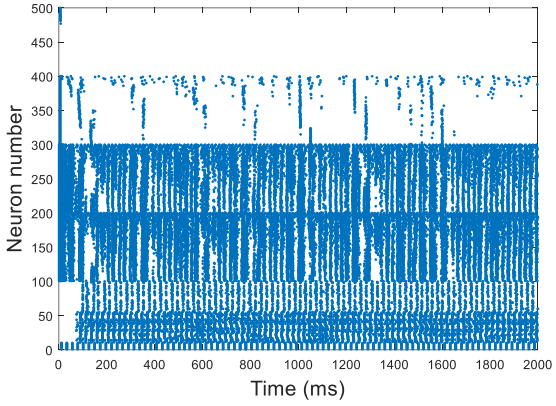
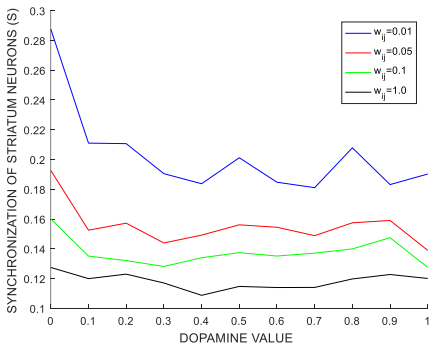


f)

Fig. 3. Synchronization measure (S) versus dopamine value obtained for connection probability of 0.5.
a) Synchronization of striatal neurons population,
b) Synchronization of GPi/SNr neurons population
c) Synchronization of GPe neurons population
d) Synchronization of STN neurons population
e) Synchronization of Thalamic neurons population
f) Rasterplot of network.



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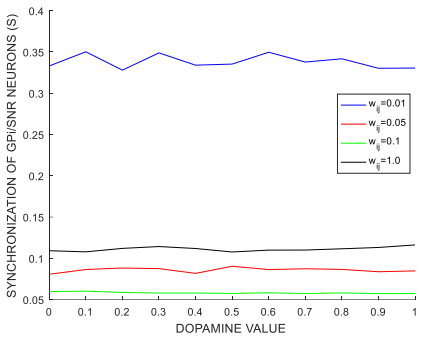

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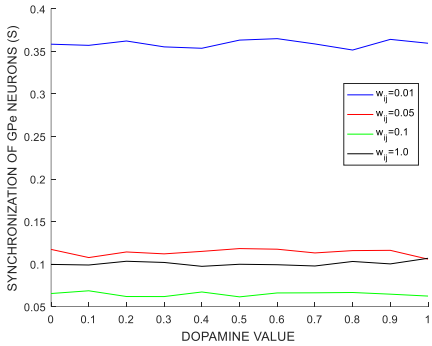

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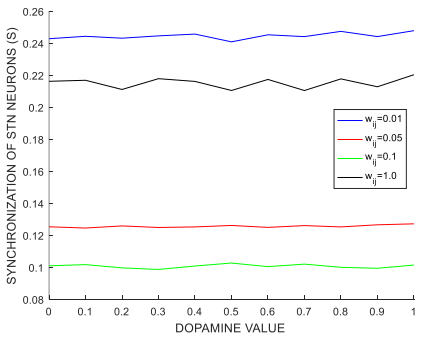

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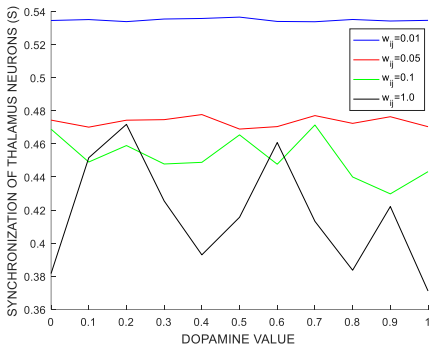

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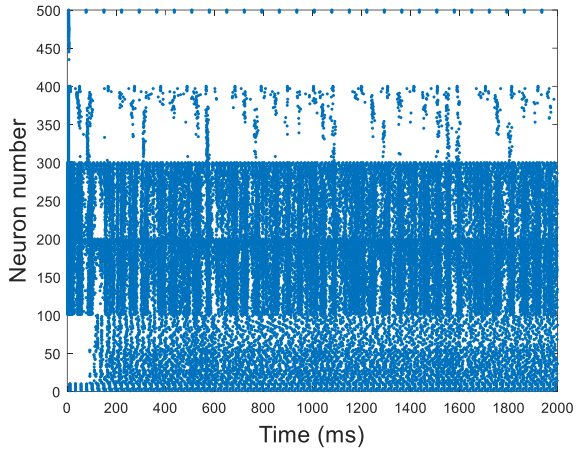
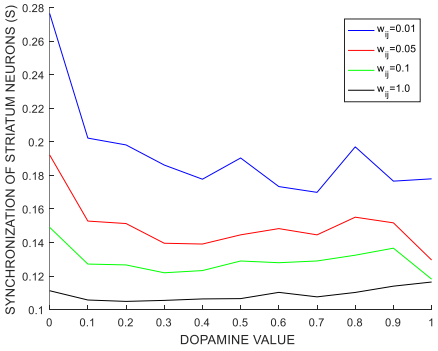


f)

Fig. 4. Synchronization measure (S) obtained for connection probability 0.25.
a) Synchronization of striatal neurons population,
b) Synchronization of GPi/SNr neurons population,
c) Synchronization of GPe neurons population,
d) Synchronization of STN neurons population,
e) Synchronization of Thalamic neurons population,
f) Rasterplot of network.



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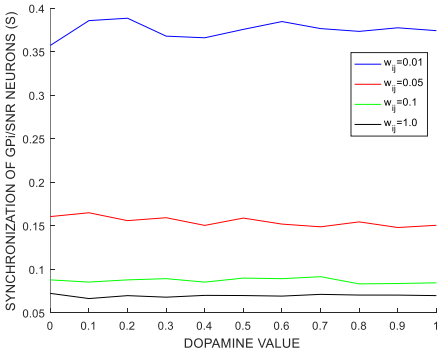

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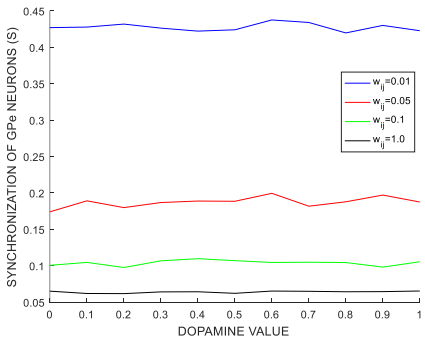

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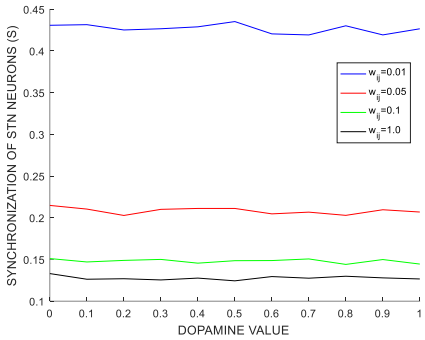

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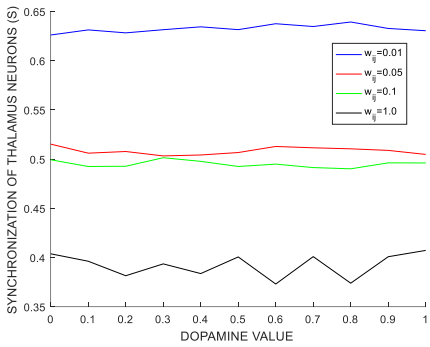

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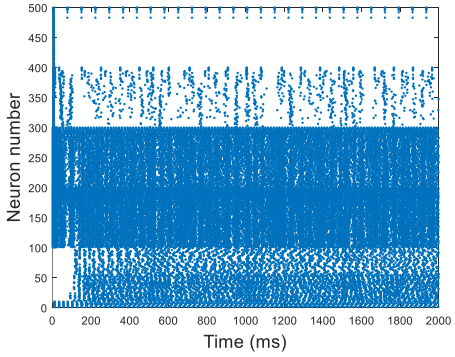

f)

Fig. 5. Synchronization measure (S) obtained for connection probability 0.1.
a) Synchronization of Striatal neurons population,
b) Synchronization of GPi/SNr neurons population,
c) Synchronization of GPe neurons population,
d) Synchronization of STN neurons population,
e) Synchronization of Thalamic neurons population,
f) Rasterplot of network.

As seen from synchronization values obtained for different connectivity values, different synaptic strength and dopamine values,

a) Decreasing the value of dopamine from its normal value of $\phi_1 = \phi_2 = 1$ to $\phi_1 = \phi_2 = 0$ increases the synchronization in striatal population which is the phenomena observed in the disease such as Parkinson in which neurophysiological hallmark is excessive synchronous activity in the basal ganglia (BG) network [8].

b) Decreasing the connection probability causes a decrease in the fluctuation in synchronization measure in thalamic neurons depending on the dopamine value. For higher synaptic strength as $w_{ij}=1$, depending on the dopamine value, the synchronization is high for connection probability of 0.5 (Fig. 3e). Decreasing connection probability to 0.1 causes a decreased fluctuation in the synchronization measure of thalamic neuron population depending on the dopamine value (Fig. 5e).

c) The synchronization measure in GPi/SNr, GPe and STN populations do not change so much depending on the dopamine decrease. For low connection probability value such as 0.1, the higher synchronization measure values are obtained for lower synaptic strength values.

d) From the rasterplots obtained from the activation for different connection probability values, it is seen that the firing in thalamic neurons decreases for higher connectivity values that means the increased inhibition is the result of higher connectivity value.

5. Conclusion

The synchronization behavior of basal ganglia is investigated by using Izhikevich neuron model. Basal ganglia comprises striatum, internal and external segments of globus pallidus, subthalamic nucleus, substantia nigra pars reticulate neuronal sub populations and thalamus. The influences of dopamine, synaptic strength and neuronal interconnection density on the synchronization behavior of the subpopulations are investigated. It is known that the neurophysiological hallmark of PD is excessive synchronous activity in the basal ganglia network. The simulation results reveal that the striatal synchronization is increased in the case of dopamine depletion which is consistent with experimental observations [8]. The decrease in the dopamine level has not shown considerable influence on the synchronization of other basal ganglia sub populations. The highest synchronization values are observed for the lowest synaptic strength and neuronal interconnection density. Decreased neuronal interconnection density causes a decrease in the fluctuation in synchronization in thalamic neurons depending on dopamine value.

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