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Modeling Dynamic Behavior of Striatal Medium Spiny Neurons with Point Neuron Models

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Abstract: The aim of this work is to investigate the role of network organization and the neuron model type on the collective dynamic behavior of striatal population. For that purpose, two different scale neuron models which are phenomenological Izhikevich and conductance-based Hodgkin-Huxley (HH) type are used to investigate the dynamic behavior of MS neurons. Two network architectures are proposed with inhibitory and excitatory synaptic currents. In these networks, while all MS neurons affect each other with inhibitory synaptic currents, an excitatory current is applied to all MS neurons in the first layer, to represent the cortical inputs. A mathematical model of a medium spiny neuron of striatum based on HH type neuron model is proposed using different calcium channels and its dynamical behavior is investigated. It is observed that when the original HH model is used, regular spiking type behavior is observed. Including the high threshold calcium current, after hyperpolarization calcium current and voltage gated potasium current into the model improves the modeling capabilities. With extended ion channels, in addition to regular spiking behavior, bursting with resting stage are obtained. Then, Izhikevich neuron model is used in the network structures to compare the dynamic behaviors and computational time.

Keywords: Straitum, Medium spiny neurons, Hodgkin-Huxley neuron model, Izhikevich model

1. INTRODUCTION

In computational neuroscience, using the tools of dynamical systems theory is becoming common in order to investigate the mechanisms underlying not only for the cognitive processes but also for neurological disorders and diseases, (Izhikevich 2007; Terman et al. 2002). The models developed in computational neuroscience are used in all levels: the modeling of single nerve cells, for investigating neural system infrastructure and describing the formation of cognitive processes, (Terman et al. 2002; Hodgkin and Huxley 1952; Yucelgen et al. 2012; Gurney et al. 2004).

The striatum is the crucial component of the basal ganglia which is associated with a variety of functions including control of voluntary motor movements, procedural learning, routine behaviors or habits, (Bolam et al. 2000; Samuelsson et al. 2007) . Striatum receives input from the cerebral cortex and is the primary input to the basal ganglia system. The principal neurons of striatum are medium spiny neurons (MSN) which are GABAergic cells, which means that they inhibit their targets with small cell bodies and dendrites. Thus, they are classified as inhibitory neurons. Depending on the species, MSNs comprise 90–95% of the total neuronal population in the striatum of the basal ganglia. Medium spiny neurons have dopamine receptors, where dopamine has a dual action on MSNs; it inhibits the (D2-type) MSNs in the indirect pathway and excites (D1-type) MSNs in the direct pathway, (Elibol and Sengor 2014; DeLong and Wichmann 2007). Consequently, when dopamine level is reduced in the striatum, the indirect pathway becomes overactive and the direct pathway becomes underactive. The lack of dopamine in the striatum is regarded as a major cause of motor-related Parkinson's disease symptoms, such as tremors, bradykinesia, and postural instability.

As the striatum is the main gateway to the basal ganglia, it activates a group of interconnected subcortical nuclei that are crucial for motor planning, (Kim et al. 2013; Ayling et al. 2007). Therefore, modeling of dynamic behavior of striatum is an important component of work on Parkinson's disease.

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In recent years, an increasing number of computational models have addressed various aspects of basal ganglia (Terman et al. 2002; Hodgkin and Huxley 1952; Yucelgen et al. 2012; Gurney et al. 2004; Wolf et al. 2005; Baladron and Hamke 2015). Besides, modeling basal ganglia, there are works that focus on modeling striatal medium spiny (MS) neurons. The AMPA, GABAA and NMDA receptors in the MS neurons are modeled to examine the relationship of the different glutamatergic receptors with the membrane response. The MS neuron was created in the NEURON simulation environment using a 29-compartment model, (Wolf et al. 2005). The model demonstrates that the NMDA current is capable of sustaining certain membrane states.

Batista et al. (2014) investigated the small-world network of neurons with chemical synapses using Hodgkin-Huxley model to verify the relation between stimulus and response for spiking neurons. Only the chemical excitatory synapses are taken into account to reduce the number of parameters.

McCarthy et al. (2011) modeled MSNs using single-compartment models with Hodgkin-Huxley type dynamics. All excitatory input from the cortex and thalamus is modeled using a background excitation term and Gaussian noise. It is shown that beta oscillations which are correlated with bradykinesia, can merge from inhibitory interactions between striatal MSNs.

The behaviors of individual neurons such as tonic spiking, phasic spiking, tonic bursting, mixed mode, spike latency etc. in response to simple pulses are illustrated in the work by Izhikevich (2004). The capabilities of the present models of spiking and bursting neurons such as integrate and fire, integrate and fire with adaptation, Morris-Lecar, Izhikevich and Hodgkin-Huxley are depicted for a single neuron. The network behaviors of the aforementioned models are mentioned but the details such as network architecture are not given.

The model of Hodgkin-Huxley (HH) is biologically realistic for the nerve cells, and the most widely used mathematical model of neuron behavior. This model is capable of defining the effect of especially potassium and the sodium channels. In general, even though the behavior of nerve cell is described by this model, it is far away to define all the behaviors observed in nerve cells. However, by adding new ion channels to the structure, it is possible to exhibit the behaviors such as bursting and tonic bursting.

Modeling striatal MS neurons and their collective behavior is gaining more importance as, these models reveal the possibilities of understanding cognitive processes as decision making, reward based learning (Bechara et al. 2000; Schultz et al. 1997) and treatment procedures as deep brain stimulation.

In this work, striatum which is an important input structure of basal ganglia is considered. As seen from literature, either HH or other type models is used in the works related to dynamic behavior of neurons. Apart from many work in the literature, here two different scale models which are phenomenological and conductance-based are used to investigate the dynamic behavior of MS neurons. In addition, two different network architectures are considered. First, a mathematical model of Hodgkin-Huxley type is proposed for MSNs, the dynamic behavior of neuron cell is investigated with different calcum channels. Then, Izhikevich neuron model is used in the network structures to give a comparison and to discuss the advantages and disadvantages of simple and detailed neuron models. In the two proposed network architectures, while all MS neurons affect each other with inhibitory synaptic currents, an excitatory current is applied to all MS neurons in the first layer, to represent the cortical inputs. The excitatory currents are modeled as a constant current with additive Poisson distributed noise. It is shown that even though both models give similar responses, the computational time is too high in HH model.

2. STRIATAL MEDIUM SPINY NEURON MODEL

MS neurons constitute 90% of striatum, which is the most effective input structure of Basal Ganglia circuits and they suppress the other structures which are affected by the striatum. The membrane potential of these neurons alternate between a resting level called the down state and a more depolarized level called the up state (Wilson 1993; Plenz and Kitai 1998).

2.1. Hodgkin-Huxley Based Striatal Medium Spiny Neuron Model

One of the most important features of striatum is that it is not an easy to stimulate it. But, when it is stimulated, it shows the bursting behavior. Therefore, the computational model which defines the behavior of the striatum should be capable of producing this bursting behavior. The bursting behavior is characterized by a silent phase of near steady state resting behavior, then, an active phase of rapid, spike-like oscillations. The bursting behavior is carried out by the dynamics of calcium channels in neuroscience literature (Gerstner and Kistler 2006; Guthrie 2009).

Electrical activity in neurons is propagated via ionic currents through neuron membranes. Most of these transmembrane currents involve one of four ionic species: sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), or chloride

(Cl^-). The electrochemical gradients which are the major driving forces of neural activity is created by the concentration differences of these ions on the inside and outside of the cell. While high concentration of Na^+ and Cl^- and a relatively high concentration of Ca^{2+} are present on the extracellular medium, the intracellular medium has high concentrations of K^+ and negatively charged molecules denoted by A^- . Considering the works by Terman et al. (2002), Shen et al. (2004), Song et al. (1996), the neuronal dynamics of stratium is described by the Hodgkin–Huxley type model. The dynamical behavior of the stratium cell potential is modelled by the differential equation given in Eq. 1.

$$C_m \dot{v}_{Str} = I - I_L - I_K - I_{Na} - I_{L_{Ca}} - I_{AHP} - I_{Ca} - I_{Kv1} \tag{1}$$

Where, C_m is the membrane capacitance per unit area and v_{str} denotes the membrane potential of the striatum. I_L is the leak current, I_K, I_{Na} which are spike producing currents are ionic currents related to the ion channels embedded in the neuron membrane. I_{LCa}, I_{AHP} and I_{Ca} are high threshold calcium current, after hyperpolarization calcium current and calcium current, respectively. Apart from many work in the literature about Hodgkin–Huxley model, I_{AHP} and I_{Kv1} currents are included to Eq.1. The high threshold calcium current (I_{LCa}) has been shown to be present in MS neurons (Song and Surmeier 1996) and therefore is included in this model. A Ca^{2+}-activated, voltage-independent after hyperpolarization K^+ current (I_{AHP}) is defined according to Terman et al. (2002), I_{Kv1} current is defined according to Shen et al. (2004) Ohmic leak current, I_L, which is carried mostly by Cl^- ions, is defined as in Eq. 2.

$$I_L = g_L (v_{Str} - V_L) \tag{2}$$

where, g_L is the leak conductance which is constant in the model. V_{str} and V_L are stratium membrane voltage and equilibrium voltage, respectively.

The time-dependent variation in conductances allows a neuron to generate an action potential, or spike. The electrical conductance of individual channels may be controlled by gates, which open and close the channels. The gates may be sensitive to membran potential (voltage-gated Na+ or K+ channels), intracellular agents (Ca^{2+}-gated K^+ channels), extracellular agents (AMPA, NMDA, or GABA receptors). The voltage-gated persistent K^+ current with four activation gates is one of the four major currents. It is defined as Eq.3.

$$I_K = g_K n^4 (v_{Str} - V_K) \tag{3}$$

where n is the activation variable for K^+, the parameter g_K (mS/cm^2) is the K^+ conductance and ($Vstr$ -VK) is the K^+ driving force. n^4 is the probability that a potassium channel is open. The voltage-gated transient Na^+ current with three activation gates and one inactivation gate is defined as Eq. 4.

$$I_{Na} = g_{Na} m^3 h (v_{Str} - V_{Na}) \tag{4}$$

where $m(h)$ is the probability of an activation (inactivation) gate being the open state. The high threshold calcium current, L-type calcium current, is represented by Goldman-Hodgkin-Katz equation, Eq. 5.

$$I_{L_{Ca}} = g_{L_{Ca}} m_{L_{Ca}}^2 \frac{v_{Str} z^2 F^2}{RT} \frac{\left[Ca^{2+}\right]_e e^{-\frac{v_{Str} zF}{RT}} - \left[Ca^{2+}\right]_i}{1 - e^{-\frac{v_{Str} zF}{RT}}} \tag{5}$$

where g_{LCa} is channel permeability to calcium ions, m_{LCa} is calcium valency, F is Faraday's constant, R is Boltzmann's constant, T is temperature as celcius. V_{str} is the voltage of the stratium cell. $[Ca^{2+}]$ is intracellular or extracellular calcium concentration, z is calcium valency. Calcium concentration inside the cellular membrane was used to regulate both a large-conductance and a small-conductance (I_{AHP}) calcium-dependent potassium current. After hyperpolarization K^+ current (I_{AHP}) and Ca current are defined as followings:

$$I_{AHP} = \frac{g_{AHP} (v_{Str} - V_K) \left[Ca^{2+}\right]_i}{\left[Ca^{2+}\right]_i + k_L} \tag{6}$$

where $[Ca^{2+}]_i$ is intracellular calcium concentration, k_L is the dissociation constant of Calcium dependent AHP current, Terman et al. (2002).

$$I_{Ca} = g_{Ca} s_\infty^2 (v_{Str} - V_{Ca}) \tag{7}$$

It is shown by Shen et al. (2004) that, $K_{V1.2}$ -containing K^+ channels regulate subthreshold excitability of striatal medium spiny neurons. $K_{V1.2}$ channels regulate first spike latency and repetitive discharge in MSNs. Voltage gated potasium current is assumed to conform to a HH-like formalism considering the work by Shen et al.(2004).

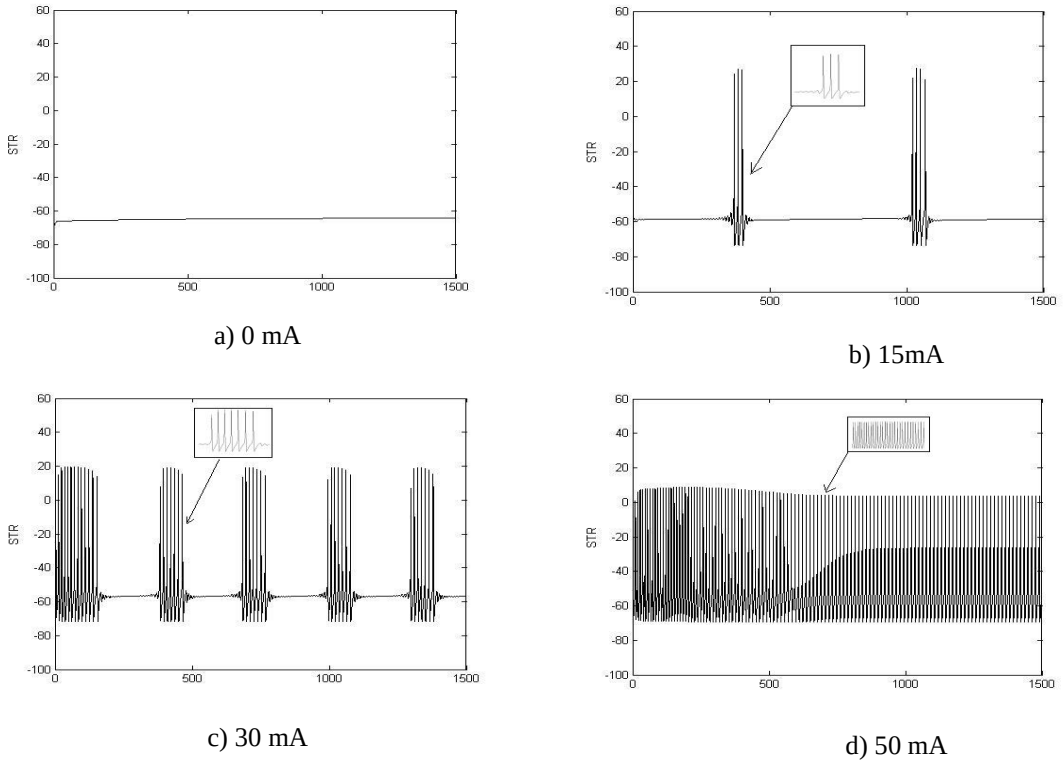
$$I_{Kv1} = g_K m^2 h(v_{Str} - V_K) \tag{8}$$

where g_K is the maximum conductance, *Vstr* is the striatum voltage, V_K is the K^+ equilibrium potential. The gating variables *m, n* and *h* are functions of time and striatum membrane potential that satisfied the partial differential equations given in Izhikevich (2007). The parameters of the HH based model are biophysically meaningful and measurable. The model parameters used in the equations, 1-8 are depicted in Table 1.

Table 1. Parameters used in Eq. 1-8

parameter	C_m	g_L	g_K	g_{Na}	$g_{L_{Ca}}$	g_{AHP}	g_{Ca}	R	z
value	1	0.4	36	120	0.001	11	0.24	8314	2
parameter	V_L	V_K	V_{Na}	V_{Ca}	$[Ca^{2+}]_i$	$[Ca^{2+}]_e$	k_L	T	F
value	-54.6	-77	55	140	variable	2.5	5	310 K	96485

The simulation result of HH based for striatal medium spiny neuron model which is obtained using the in-house built MATLAB codes is depicted in Fig. 1 for different input currents (0, 15, 30 and 50 mA).



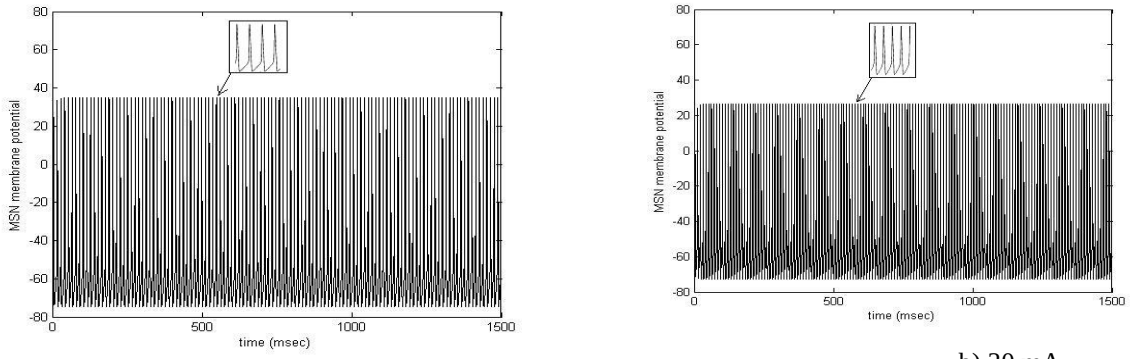
a) 0 mA b) 15mA

c) 30 mA d) 50 mA

Figure 1. The simulation results of HH- based for striatal medium spiny neuron given in Eq. 1 for constant input currents, I a) 0mA, b)15mA, c) 30mA and d) 50 mA.

It is observed that the behaviors of membran potential for 15 mA and 30 mA constant input currents is in the elliptic bursting form. In the elliptic bursting, small amplitude oscillations occur during the resting phase and the amplitude of spikes gradually increases and decreases. The model exhibit resonator type behavior in which neurons have damped and sustained subthreshold oscillations, as seen in Fig. 1. There is a resting and repetitive spiking states. Increasing the initial current leads an increase in the number of spikes between resting stages.

To investigate the influence of currents included in Eq. 1 i.e., I_{LCa}, I_{AHP} and I_{Kv1}, they are excluded and the simulation results are depicted in Fig. 2 for 15 and 30 mA inital currents. Having only the currents related to voltage-gated potassium, sodium, calcium and leak channels leads to the results different from Fig. 1.



a) 15 mA b) 30 mA

Figure 2. The simulation results of Hodgkin–Huxley model with potassium, sodium, calcium and leak currents are considered and input currents are a)15 mA and b) 30 mA, respectively.

When the original Hodgkin–Huxley model is used, regular spiking type behavior is observed. Including the I_{LCa}, I_{AHP} and I_{Kv1} currents into the model improves the modeling capabilities, as it can be followed from Fig.s 1 and 2. With extended ion channels, in addition to regular spiking behavior, bursting with resting stage can be obtained as in Fig. 1 b) and c). The behavior in Fig. 2 a), corresponds to fast spiking neuron behavior, which is observed especially in inhibitory neurons.

Based on these simulation results, in order to get bursting type behavior of MS neurons HH equations have to be extended including I_{LCa}, I_{AHP} and I_{Kv1} currents, giving rise to a single neuron model of order six. So especially in forming large scale neuronal network models, this model will not be versatile, even though it is a biologically realistic model presenting the role of ion channels on the dynamic behavior of the MS neurons.

2.2. Izhikevich based striatal medium spiny neuron model

HH based medium spiny neuron model given in section 2.1 is physiologically realistic one. But, the examining the behavior of a group of cells with this neuron structure is not so easy. Therefore, Izhikevich cell model, which is simple compared to HH model and gives similar results, is considered also. This phenomenological neuronal model differs from conductance-based HH-type models. The model was designed to reproduce firing responses instead of introducing all of the ionic currents. Biophysically accurate HH-type neuronal models can be reduced to a two-dimensional (2-D) system of ordinary differential equations by bifurcation methodologies. The equations of Izhikevich neuron model are given in Eq. 9.

$$\begin{aligned} \dot{v} &= 0.04v^2 + 5v + 140 - u + I \\ \dot{u} &= a(bv - u) \\ v \geq 30 \;\; & v \to c, u \to u + d \end{aligned} \tag{9}$$

where v and u are dimensionless variables, represents membrane potential of the neuron and membran recovery respectively. *a*, *b*, *c*, and *d* are dimensionless parameters. *v* and *u* account for the activation of K^+ ionic currents and inactivation of Na^+ ionic currents, respectively. When membrane potential threshold reaches 30 mV, v and u are assigned to the values given in the last expression of Eq. 9. with reset condition. *I* is current input including synaptic current and external applied current. The parameters used here for different neuron behavoirs in Izhikevich neuron model is taken from Izhikevich (2004). Izhikevich neuron model can be used to model the dynamic behavior of MS neurons, with a much simplier representation than conductance-based HH neurons.

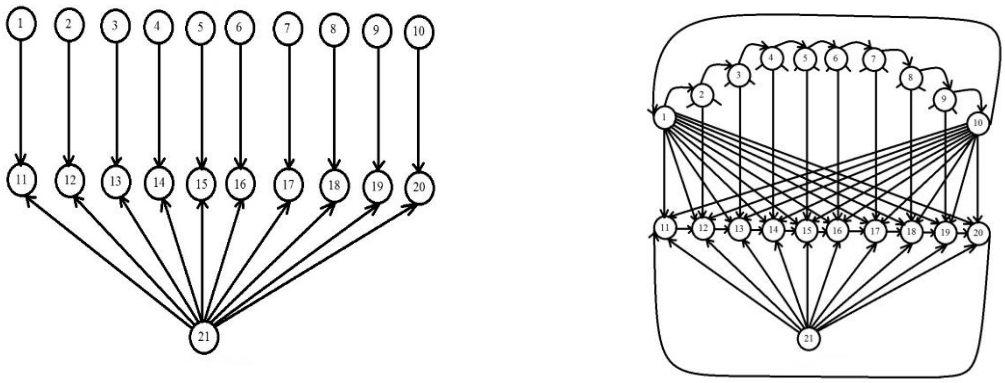
3. NETWORK STRUCTURE OF STRIATAL MEDIUM SPINY NEURONS

MS neurons inhibit each other through a local network of collaterals and receive excitatory projections from the cerebral cortex. Forming inhibitory synapses between striatal microcircuits are interpreted striatum behaving as a winner-take-all (WTA) network. However, according to experimental observations, interactions among nearby MSNs show sparse connectivity (Tunstall et al., 2002; Koos et al., 2004). Since individual connections involve one or only a few synapses, weak interactions predominate and reciprocal interactions are rare, since the majority of MS neuron pairs involved in only one-way connections. Another experimental observation by

Wilson (1993) is that highly irregular firing predominates, (Ponzi and Wickens, 2013). Though in the work of Ayling et al. (2007), it is argued that GABAergic post-synaptic potentials can exert excitatory effects on projection neurons, here we considered the conventional approach of inhibitory behavior in striatum.

Here, two different aspects that could effect the results obtained in modeling the dynamic behavior of a population of neurons in striatum is tested: the role of network organization and the role of neuron models. For network organization, two different architectures is considered. As in a striatal microcircuit, both MS neurons and interneurons is considered (Denizdurduran and Sengor, 2013; Elibol and Şengör, 2014) in all architectures one interneuron is considered along with 20 MS neurons.

In each topology given in Fig. 3(a-b), there are three layers, the first two layers comprise of ten MS neurons each, while the last layer is composed of a single interneuron. The interneuron in the third layer is connected to every neuron in the second layer and inhibits their activities, in two architectures given in Fig. 3(a-b). In the architecture, given in Fig. 3(a) every neuron in the first layer is connected to one neuron in the second layer unidirectionaly, so a one-to-one feedforward connection is considered. In the second architecture given in Fig. 3(b), every neuron in the first layer is connected to every neurons in the second layers unidirectionally (all-to-all connections). Every neuron in the first and second layer has inhibitory unidirectional connection with its neighboring neuron, so forming a feedback loop giving rise to a network architecture with feedback.



a) one-to-one feedforward b) all-to-all feedback connections.

Figure 3. The network architectures for striatal neuron populations

3.1 Networks of striatal ms neurons with HH-based neuron model

The two architectures given in Fig. 3 will be first simulated with HH-based MS neuron models. In order to model the network, the connection between neurons should be defined. Thus, to the model, given in Eq. 1 synaptic current $I_{stri\rightarrow strj}$ is included as in Eq. (10)

$$C_m \dot{v}_{Strj} = I - I_L - I_K - I_{Na} - I_{L_{Ca}} - I_{AHP} - I_{Ca} - I_{Kv1} - I_{stri\rightarrow strj} \tag{10}$$

The current $I_{stri\rightarrow strj}$ which represents the synaptic input from neuron to neuron is modeled as

$$I_{stri\rightarrow strj} = \alpha_j \left(v_{strj} - E \right) \tag{11}$$

Where, α_j the synaptic conductance is modelled as a constant depending on a parameter β as $\alpha_j = 1.1 \cdot 10^{-4} \cdot \beta$. In the model, equilibrium voltage for the synapses are taken as E=0 for excitary connections and E=-70 mV for the inhibitory connections, (Terman et al., 2002).

In all architectures, while all MS neurons affect each other with inhibitory synaptic currents, an excitatory current is applied to all MS neurons in the first layer, to represent the cortical inputs. For the second layer MS neurons, such an excitatory current is applied to model the effect of background activity. These excitatory currents are modeled as a constant current I=15 mA with additive Poisson distributed noise with mean value of 10 mA. Thus all the MS neurons, has instinctively the behavior given in Fig. 1b as a single neuron.

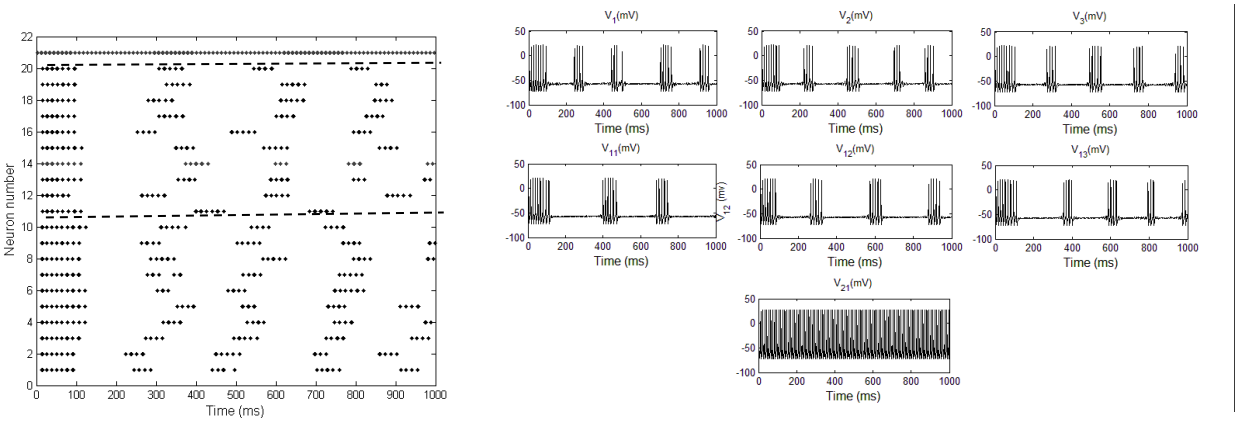
The model of interneuron is different than the one given in Eq. 10, as its behavior is fast spiking rather than bursting. So interneuron in all structures are modeled following the behavior shown in Fig. 2a , where, only

potassium, sodium, calcium and leak currents are considered and the excitatory input current representing the effect of neuronal background activity is modeled again as a constant current I=15 mA with additive Poisson distribution with mean value 10 mA. Thus, the equation for the interneuron is as given in Eq. 12

$$C_m \dot{v}_{Str-\text{int}} = I - I_L - I_K - I_{Na} - I_{Ca} \tag{12}$$

In all architectures considered, the parameter β affects the inhibition. During simulations, results are obtained with different values of this parameter where $\beta \in \{125 \quad 250 \quad 375 \quad 500\}$. Here the results obtained with $\beta = 250$ will be given and the role of β will be discussed, for each architecture.

For the first architecture given in Fig. 3a, the raster plot obtained with the above explained connections and membrane voltage versus time curves are given in Fig. 4. The behavior in the first ten rows belongs to the neurons in the first layer, the second ten belongs to the neurons in the second layer, the last one is related to single inhibitory neuron (dashed line indicates the layers).

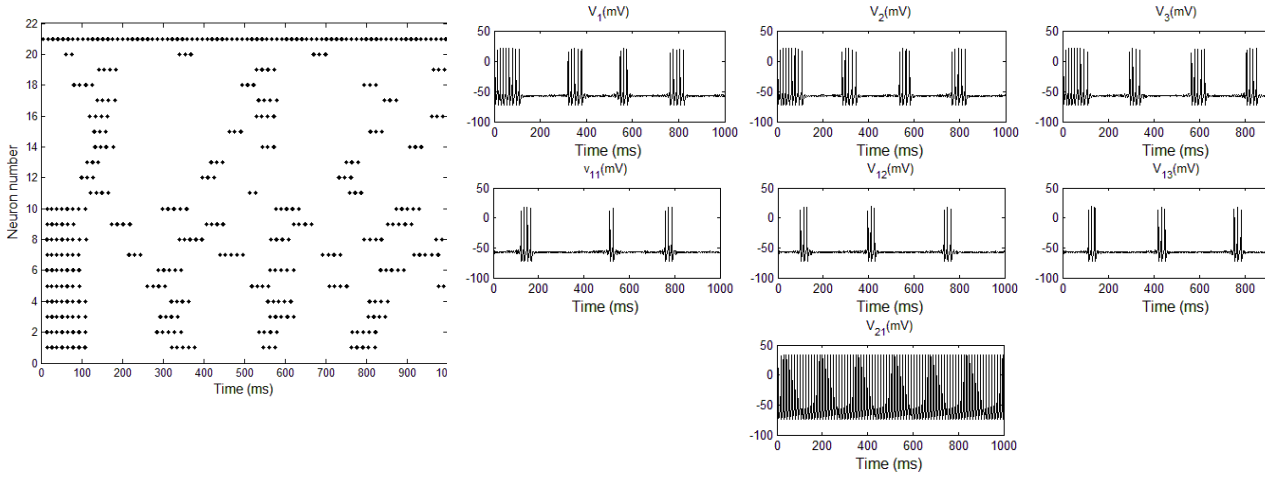


a) b)

Figure 4. a) The raster plot of 21 neurons, the first 20 being MS neurons and the 21st being inhibitory interneuron. b) The dynamic behavior of neurons in the network in Fig. 3a.

The dynamic behavior of the first three neurons in the first layer, the first three neuron in the second layer and the 21st neuron which is inhibitory are depicted in Fig. 4b. As it can be followed from Fig. 4, while the neurons in the first layer seem to spike more, there is not much difference between two layers. Even though, there is a difference in phase, the neurons in the first and second layer behave almost synchronously. In this architecture, changing the synaptic conductance, does not have much effect. Thus, for different values of β , there is not much difference in the spiking activity of first layer and second layer neurons.

The rasterplot and membrane potentials for the second architecture are depicted in Fig. 5. This architecture has feedback connections and these are inhibitory connections. The connections from the layer 1 to 2 are dense and changing the synaptic conductance affect the spiking activity. As β increases, the spiking activity in the second layer decreases. Since the connections from first layer to second layer are inhibitory, the activity in the second layer is less than the activity in the first layer.



a) The raster plot of 21 neurons b) The dynamic behavior of neurons

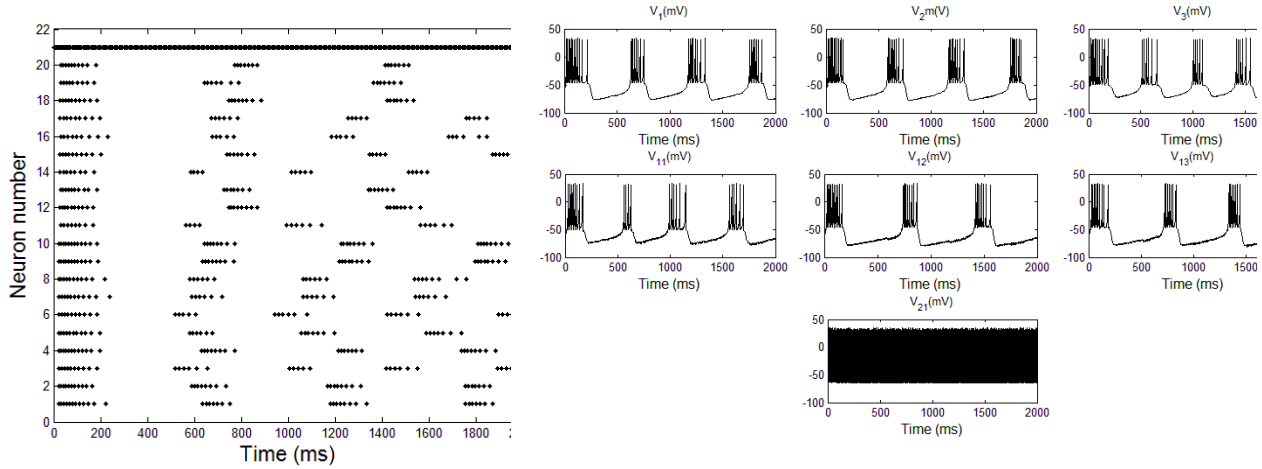
Figure 5. Simulation results for the network given in Fig. 3(b).

3.2 Network structure of striatal medium spiny neurons with Izhikevich model

The network structures given in Fig. 3 will now be considered with Izhikevich neurons. The neurons in the first two layers are modeled using the parameters giving the chattering behavior, and the inhibitory neuron is modeled as fast spiking neuron. The connection between neurons are defined as in Izhikevich (2003). The connections are constant and depending on being excitatory or inhibitory connections, input current is either has a positive or negative random value. These connections are effective, whenever the neuron fires. If neurons do not fire, the connection currents are zero.

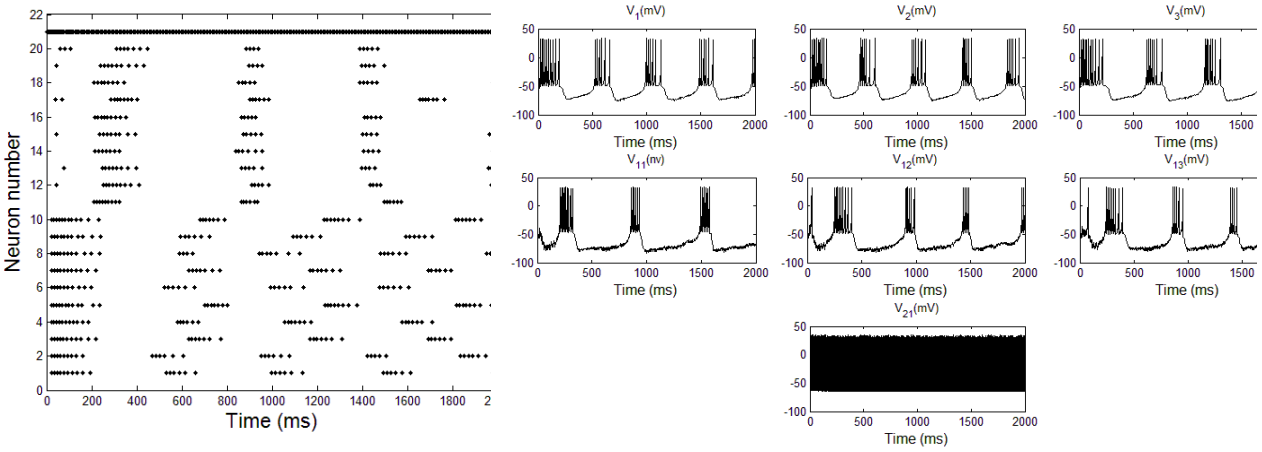
In Fig. 6, the architecture in Fig. 3a is considered and the result obtained are compared to the raster plot in Fig. 4a. Thus, using Izhikevich model, similar collective behavior is obtained. The only difference between two is the time axis, to show the similarity in behavior, a longer time span is considered. The dynamic behaviors of the single neurons in the population, first 3 neurons of the first layer, the first 3 neuron of the second layer and the neuron in the third layer which is inhibitory are depicted in Fig. 6b. These also are similar to neuron behaviors given in Fig. 4b.

The raster plot and the dynamic behavior of neurons for the network in Fig. 3b are given in Fig. 7. They show resemblence with the results obtained using HH neurons. As, it can be followed from Table 2, computational time spent is reduced dramatically, when Izhikevich neuron model is used. Run time for each architecture and model is given in Table 2.



a) The raster plot of 21 neurons b) The dynamic behavior of neurons

Figure 6. The simulation of the network in Fig. 3a with Izhikevich neuron model.



a) The raster plot of 21 neurons b) The dynamic behavior of neurons

Figure 7. The simulation of the network in Fig. 3b with Izhikevich neuron model.

The conductance-based neuron models with ion channels of the Hodgkin-Huxley type can capture the electrophysiological behavior of neurons in great detail. However, not only the computational burden but also computational time consumed is a lot more compared to the networks where simple model like Izhikevich neuron model is used.

Table 2. Execution time for two models and networks

	Arch.3a	Arch.3b
Hodgkin-Huxley model	5516.6 s.	4611.4 s.
Izhikevich model	19.2609 s.	19.0099 s.

4. CONCLUSIONS

In this work, to investigate the role of network organization and the neuron model type on the collective dynamic behavior of striatal population, two different scale neuron models, phenomenological Izhikevich and conductance-based Hodgkin-Huxley (HH) type are used. Two network architectures are proposed with inhibitory and excitatory synaptic currents. Original HH model and modified one which consist of different ion channels such as the high threshold calcium, after hyperpolarization calcium and voltage gated potasium currents, after hyperpolarization K^+ current are used for modeling a medium spiny neuron of striatum. It is observed that when the original HH model is used, regular spiking type behavior is observed. Including aforementioned currents into the model improves the modeling capabilities. With extended ion channels, in addition to regular spiking behavior, other types of behaviors are obtained.

Then, Izhikevich neuron model is used in the network structures to compare the dynamic behaviors and computational time. The collective behavior of medium spiny neurons with different topologies are investigated. The collective behavior for different network architectures are given with raster plots and examples of single neuron behaviors in different layers composed.

It is shown that, using simple neuron model gives almost the same results as the complicated neuron model. Thus, to form larger networks, it is convenient to use simple neuron model. Also, the organization of neurons do affect the collective behavior. Since it is known that the beta band synchronization arises in striatum during Parkinson's disease, maybe to look at the reorganization of neurons could give an idea about the role of dopamine.

The number of neurons used for the collective behavior is too small, in this work, but using HH model for neurons constrained the number. The role of architecture could be better investigated with larger number of neurons and with this study, it is clear that using Izhikevich neurons will help using large scale networks, with realistic results.

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