BUILDING NEUROCOMPUTATIONAL MODELS AT DIFFERENT LEVELS FOR BASAL GANGLIA CIRCUIT

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Abstract: *The target of computational neuroscience studies can be considered in two-folds: understanding the connection between the physiology and functional aspects of the brain to develop new approaches for diagnosing and treatment of neurological disorders and behavioral deficits and understanding mind and consciousness to develop new intelligent technologies. The methods and approaches used in computational neuroscience have to overcome the complexity of the system in all aspects. So, different methods and approaches are developed for different scales not only for observing the phenomena, but also for modeling. In this paper, an approach is proposed to build a connection between different levels of modeling. A simple, linear system will be shown to give an understanding of the working principle of basal ganglia circuit which is modeled with a detailed spiking neural network approach. First, spiking neural network of basal ganglia circuit will be introduced and the role of dopamine on its functioning will be shown; then a simple linear system model will be given, and the relation between two models will be explained. The aim of this work is to show that even a simple model which is not sufficient for detailed understanding of the neuronal process, could give a coarse understanding of a complex phenomenon. Such simple models could be used as a starting point in building complex models and also can be benefited for implementing intelligent technologies.*
Keywords: *Basal ganglia circuit, spiking neural network, mass model, dopamine.*

1. Introduction

In computational neuroscience literature, there are numerous models of neurons and neural structures at different levels. One reason of this diversity is the collection of data at different levels using different measurement tools and methods. While based on single neuron measurements, it is possible to obtain data to build a detailed model of a neuronal behavior based on the role of ion channels [1,2], it is also possible to pinpoint the regions of the brain that are active during a task by fMRI (functional magnetic resonance imaging) and obtain neural field model which can mimic the collective activity of neurons at a specific region during a specific task [3]. The resolution of measurements also depend on the scale. While, temporal resolution of single neuron measurements and EEG (electroencephalogram) / LFP (local field potential) are better, spatial resolution of fMRI is superior to other techniques. Thus the models corresponding to different levels have to cope with all these different scales and the dynamics of the brain is either modeled by a set of nonlinear, ordinary differential equations or partial differential equations. Besides these, there are hybrid models, where some structures are modeled at neuron level; others are modeled as mass model [4,5].

Of course the role of different aspects on neurological disorders and diseases is another reason of this diversity. While mutual activity of neurons is responsible for some processes and the malfunctioning in their collective behavior give rise to deficits, the activity of ion channels and the concentration of ions and neurotransmitters are important in other cases. So models differ as they target all these different phenomena at different levels. This variety of models is needed since all provide information necessary to understand the complexity of cognitive processes and neurological disorders and diseases. In computational neuroscience, the level of the model built has to be decided considering the experimental results to be used and the cognitive process dealt with. In some cases, models at different levels should be considered together to have a better understanding.

There is another aspect which should be noted for the models in computational neuroscience other than building the connection between the physiology and functional aspects of the brain. As pointed out in abstract, computational neuroscience also focuses on understanding mind and consciousness to develop new intelligent technologies. For this aspect, the simplicity of the models is crucial, since implementation on a hardware and real time applications is possible only if the computational burden is manageable. Even though there are some attempts to develop special hardware for neural structures as

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SpiNNaker [6], neuromorphic hardware as Neurogrid [7], the scale of model should be kept small to use the models in mobile and robotics applications [4]. Thus, while modeling the behavior of a group of spiking neurons, the number of neurons in the model is not same as the number of neurons in the neuronal structure to be modeled, but scaled to a number that is capable to display the behavior. Also, the membrane potential of a neuron is modeled by either by first order dynamical systems as integrate and fire models [8,9] or second order dynamical systems as Izhikevich model [10], even though a detailed model could be obtained by adding ion channel dynamics to Hodgkin-Huxley model [11]. Thus, in neurorobotics applications and in developing new learning rules simple models are preferred, rather than detailed models.

In this paper, the objective is whether it is possible to foresee the behavior of a complicated computational model by a simple one. If this is fulfilled than it would be possible to build a connection between different levels of modeling and a tool can be developed to ease detailed modeling. Since models at each level are versatile as they point different aspects of the neural phenomenon, the aim is not to replace a detailed model by a simple one, but to use a coarse approach to understand a complex but detailed one. To show the possibility of such an approach basal ganglia network will be considered, and it will be modeled by spiking neural models and by a simple linear system.

Modeling basal ganglia network has been considered in computational neuroscience literature extensively [9,11-16] due to its role in voluntarily action selection, reward related learning and in neurological deficits and diseases as Parkinson's disease, Huntington's disease, and in behavioral deficits as addiction. Especially, models of basal ganglia network are developed to understand deep brain stimulation [2]. In recent years more attention is paid to the role of basal ganglia circuits in high level cognitive processes as decision making [17], substance dependence [18,19].

In the following section, a brief introduction to the basal ganglia circuits will be given, especially focusing on the role of dopamine in action selection. Then, the proposed spiking neuron model will be introduced and a simple linear mass model will be given. The simulation results obtained using BRIAN simulator and XPPAUT will be given and these results will be discussed. It will be shown that a connection between the the firing rate of the spiking neuron model and dynamic behavior of simple linear model can be drawn.

2. Basal Ganglia Circuit

Basal ganglia circuits proposed to have important role in motor activation and cognitive processes [20] especially their role in reward based learning and decision making pointed in various works [17,21-23]. Impairment of basal ganglia circuits manifest deficits in motor actions observed in neurodegenerative diseases such as Parkinson's and Huntington's disease, and also cause behavioral deficits observed in attention deficit hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD) and addiction [19,24-27]. These behavioral disorders and motor movement disorders are treated by deep brain stimulation (DBS), a well-known treatment of Parkinson's disease [28,29]. The role of basal ganglia in psychiatric disorders is considered more recently [30,31], and their treatment by DBS makes basal ganglia a target for functional and restorative neurosurgery [32,33].

In computational neuroscience, modeling basal ganglia circuits is an important subject and many models are proposed. While mostly focus more on the role of basal ganglia circuits in voluntary movement and action selection [9,11-16], there are also models for reinforcement learning [34-42]. Some models deal with the malfunctioning of basal ganglia circuits and how treatments can be developed [2,43-46]. Most of these work except [13,42,45] focus on simple spiking neuron networks. Here, both mass model simpler than the ones in [13,42,45] and spiking neuron networks will be considered.

Striatum is considered as the input structure of basal ganglia and it is together with Subthalamic nucleus (STN), Globus pallidus (internal(GPi), external(GPe) and ventral pallidum) and substantia nigra (pars compacta and pars reticulata) [47,48] form the direct, indirect and hyper-direct pathways of cortico- striatal circuit [20,24]. Normally, direct and indirect pathways are at an equilibrium state. Little perturbations on the output of basal ganglia circuit which correspond to the GPi/SNr (substantia nigra pars reticulata), result in the selection of an action. The role of the hyper-direct pathway is to perform the fine tuning between several possible output choices which are conducted by direct or indirect pathway [49]. Dopamine from substantia nigra pars compacta and ventral tegmental area modify the activation in basal ganglia circuit by acting on striatum.

The afferents of striatum are mainly cortical pyramidal neurons located in layer V and occasionally in layer III [50]. During motor activation, posterior putamen and the dorsolateral anterior putamen receive inputs from motor and motor association cortex [21,51].

Striatum is mostly composed of medium spiny neurons (MSNs) which comprise 80-95% [47] (90 - 95 % [48]) of striatum, remainder is mostly interneurons. Even though MSNs are structurally homogeneous they have different chemical properties and are classified according to their response to neurotransmitter dopamine (DA) [48]. The two most effective groups are MSN with D1 type and D2 type receptors. D2 type receptors are more abundant and they are considered to be promoting the selection of the latent reinforcers [52].

The stimulation of the D1 type DA receptors cause neuronal excitation while the stimulation of the D2 type receptors cause neuronal inhibition. Both D1 and D2 type receptors exist together on the membrane of a neuron and their net effect drives the neuronal output. While D1 type MSNs inhibit GPi neurons and form direct pathway, D2 type MSNs inhibit GPe neurons and form indirect pathway. The direct pathway promotes the action initiation and selection whereas indirect pathway prevents actions [21,53]. The role of DA on striatum behaviour is vital,

many neurological diseases and disorders are due to malfunctioning of dopamine neurons in striatum [2,16,46].

The well-known and extensively studied basal ganglia action selection circuit [24] has two main pathways: direct and indirect. Both pathways start with the stimulus from cortex to the neostriatum (caudate and putamen - Str) and unite again at the output nucleus of the basal ganglia, GPi/SNr.

The direct pathway is responsible for action selection while the indirect pathway is responsible for the inhibition of the unwanted actions. Direct pathway starts with the glutamatergic projections from the cortex to the MSNs of striatum. Some of the striatal neurons have direct GABAergic (gamma aminobutyric acid) projections to the GPi. The connection between GPi and thalamus (THL) is inhibitory while the connection from thalamus to cortex is excitatory. The net result of the direct pathway is the inhibition of the inhibition on thalamus so the motor cortical areas are stimulated which is called disinhibition in the neuroscience literature [47,48]. In the indirect pathway different type of striatal neurons have inhibitory GABAergic connections to GPe. GPe inhibits STN while STN stimulates GPi via glutamatergic connection. The net result of the indirect pathway is the disinhibition on the GPi so thalamus is inhibited and motor cortical areas are less stimulated.

In addition to the direct and indirect pathways, there is another excitatory connection between the cortex and the STN and this is called the hyper-direct pathway [49]. Via hyper-direct pathway cortical activity is transferred to GPi over STN and the striatal pathways are short-circuited. By the help of the excitation from the STN, already tonically active GPi inhibits thalamus stronger. For action selection, ventral oralis nuclei of thalamus is active [54] and its efferent is layer IV of supplementary motor area of cortex [21,48,54,55].

3. Computational Models of Basal Ganglia Circuit at Different Levels

In this section, two different levels of modeling basal ganglia circuit will be considered and a spiking neuron model based on Izhikevich type neurons will be introduced first. Then, a simple continuous time differential equation set will be used to define a mass model of basal ganglia circuit. Such mass models for basal ganglia circuit have been given previously [13,42,56,57], but all these were composed of discrete time, nonlinear dynamical systems. These two model focus on the effect of dopamine on action selection and the role of dopamine is modeled by a parameter.

3.1. Spiking neural network model

In this work, a computational model of basal ganglia-thalamocortical circuit for action selection which is shown in **Figure 1** is first built using point neurons. The point neuron model used in forming these groups is Izhikevich neuron [58] and the equations governing the neuron model with the reset condition are given in **Equation 1** and **Equation 2**, respectively. While forming the model the neural activation pattern of each

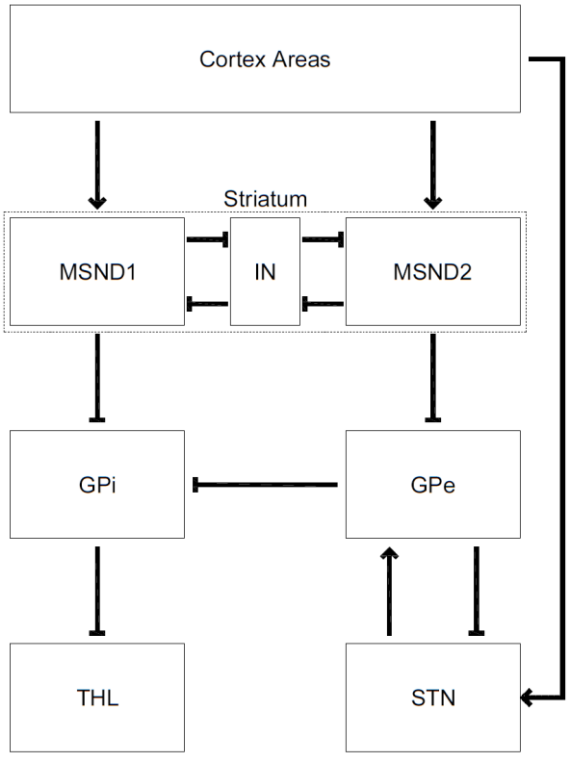

Figure 1. Cortex - Basal Ganglia and Thalamus circuit.

component of basal ganglia circuit is considered and to model different patterns different parameters of Izhikevich neuron model are used. These parameter values are given in the upper part of **Table 1**.

$$v' = 0.04v^2 + 5v + 140 - u + ge - gi \qquad (1)$$
$$u' = a(bv - u)$$

$$\text{If } v > 30 \text{ mV, then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d \end{cases} \qquad (2)$$

In **Equation 1**, g_e and g_i represents the excitatory and inhibitory connections to the neuron. As a neuron is connected to numerous neurons sum of excitatory and inhibitory neurons effects the behavior of the neuron [59]. The dynamics of synapses and the parameter values are given in **Equation 3**, **Equation 4** and **Table 2**, respectively.

$$g'_x = -\frac{g_x}{\tau_{syn}}, x \in \{e, i\} \qquad (3)$$

Here index e corresponds to excitatory, and i corresponds to inhibitory connections.

$$v^{(j)} > V_{thr} \text{ then } g_x^{(k)} \leftarrow g_x^{(k)} + w_{j-k,x} \quad (4)$$

As the connection increases when a presynaptic neuron fires, $w_{j-k,x}$ denotes this increase in the weight from neuron j to neuron k either excitatory or inhibitory.
While cortex in the model has 200 excitatory pyramidal neurons which have regular spiking activity. Striatum model consists of three groups of neuron populations: D1 type medium spiny neurons (MSND1), D2 type medium spiny neurons (MSND2) and interneurons (IN).

Table 1. Izhikevich model parameters and connection weights. ⊣ is used for inhibitory and → for excitatory connections.

Parameters	RS	FS
a	0.02/ms	0.1/ms
b	0.25/ms	0.2/ms
c	-65mV	-65mV
d	8mV/ms	2mV/ms
Connections	$w_{j-k,x}$	Probabilities
CRTX → MSND1	0.75V/s	0.5
IN ⊣ MSND1	1V/s	0.5
CRTX → MSND2	0.75V/s	0.5
IN ⊣ MSND2	1V/s	0.5
MSND1 ⊣ IN	1V/s	0.25
MSND2 ⊣ IN	1V/s	0.25
MSND2 ⊣ GPe	1V/s	0.25
STN → GPe	1V/s	0.25
GPe ⊣ STN	1V/s	0.25
CRTX → STN	1V/s	0.25
MSND1 ⊣ GPi	0.75V/s	0.25
GPe ⊣ GPi	0.75V/s	0.25
GPi ⊣ THL	1V/s	0.25

MSND1 and MSND2 neuron populations are composed of 100 neurons and IN group has 25 neurons. MSND1 and MSND2 are modeled as regular spiking neurons like cortex excitatory neurons. The neurons in GPi, GPe, IN and STN are modeled as fast spiking neurons with initial values v_i=-75, u_i =-16, while cortex inhibitory neurons have as initial condition v_i=-65, u_i=-15. The connection weights and probabilities for the model in **Figure 1** are given in the lower part of **Table 1**. The number of point neurons considered for all structures are given in **Table 3**.
In order to model the role of dopamine on action selection, the synaptic connections have to be changed with dopamine and here, this is accomplished as in [60,61]. Thus, the Equations given in **Equation 3**, **Equation 4** will be modulated with dopamine as in **Equation 5**.

$$g'_x = -\frac{g_x}{\alpha.\tau_{syn}}, x \in \{e,i\} \quad (5)$$

Here, with these equations implemented to the model, the effect of Dopamine can be investigated by changing DA parameter. In order to show different effect of dopamine on MSND1 and MSND2, the parameter α =DA is used for MSND1 group and α =1/DA is used for MSND2 group.

Table 2. Synaptic time constant and frequencies of Poisson groups.

τ_{syn}	CRTX	GPe, THL	STN	GPi
10ms	100Hz	150Hz	200Hz	250Hz

Table 3. Number of neurons in each neural population given in Figure 1.

Neural Population	# of neurons	Behaviour
CRTX	200	RS
MSND1	100	RS
MSND2	100	RS
IN	25	FS
GPi	100	FS
GPe	100	FS
STN	100	FS
THL	100	FS

3.2. Mass model

A mass model equations for basal ganglia circuits are formed by linear differential equations given in **Equation 6**. This model is inspired by the firing rate results of spiking neuron model given in previous section and knowledge of state space behavior of linear dynamical systems.
The behavior of cortex areas $crtx$ are modeled by a Heaviside function. Striatum is represented by two state variables msn_{D1} and msn_{D2}, which have afferent exitatory connection from $crtx$ weighted by dopamine level (DA) denoted by w_{DA1} and w_{DA2}, for msn_{D1} and msn_{D2}, respectively. Each of neural structures other than striatum is modeled by a single state variable, thus gp_e, gp_i, stn and thl are represented by a single dynamical variable and they all have afferent and efferent connections, corresponding to the circuit given in **Figure 1**. The role of dopamine on action selection is investigated by changing dopamine level DA from low to high levels where the values of DA are taken as 0.25, 0.5 and 0.75 for low, normal, and high level respectively [62,63].

$$msn_{D1}' = -msn_{D1} + w_{DA1}.crtx$$
$$msn_{D2}' = -msn_{D2} + w_{DA2}.crtx$$

$$gp_e' = -(gp_e - 0.5) - 0{,}5.msn_{D2} + 0{,}5.stn$$
$$stn' = -(stn - 1) - 0{,}5.gp_e + 0{,}5.crtx$$
$$gp_i' = -(gp_i - 1) - 0{,}5.msn_{D1} - 0{,}5.gp_e \quad (6)$$
$$thl' = -(thl - 1) - gp_i$$
$$w_{DA1} = DA$$
$$w_{DA2} = 1 - DA$$

4. Results and Conclusion

The simulations are done for spiking neural network model and mass model proposed in Section 3. In both cases the role of dopamine on action selection is investigated similarly. The levels of dopamine are changed through time and its effect on the results are shown with the activity of THL population. This activity which will be called THL activity is depicted with raster plot and firing rate for spiking neuron model and with curves obtained by the dynamical system for mass model.

4.1. Spiking neural network results

The simulations for spiking neural network model are carried in Python based simulation environment BRIAN [59]. During the simulations the level of dopamine is modified by changing parameter α in **Equation 5**. As the effect of dopamine on D1 and D2 type receptors is different, and α =DA for MSND1 group and α =1/DA for MSND2 group, to change the value of α , DA is taken as 1 for normal level, 0.9 for low level and 1.1 for high level. This modification is done through time and the level of dopamine is taken to be at normal level for the first 500ms and then switched to high level till 1500ms and then switched to normal level till 2500ms, to low level till 3500ms and again to normal level till the simulation ended at 4000ms. This switching is done to investigate the effect of dopamine level change on the action selection through time. The simulation results are given for a randomly chosen single neuron from MSND1 and MSND2 population and synaptic activity in **Figure 2**, where the effect of dopamine on single neuron activity can be followed. As it can be followed from **Figure 2**, the neuron chosen from MSND1 population is more active when the level of dopamine is high, and the neuron chosen from MSND2 population is more active when the level of dopamine is low. This change in activity is also projected to synaptic dynamics and synaptic activity in both populations show difference as the membrane potentials.

The outcome of the activity of neuronal populations in basal ganglia circuit determines the activity of THL which can be interpreted as the result of action selection. Thus eventhough the single neuron activity of striatum MSN population reflect the effect of dopamine, the overall result of all this neuronal activity is revealed in THL, so the population activity of MSND1, MSND2 and THL are given in **Figure 3**, with raster plots and firing rates of neurons in MSND1, MSND2 and THL populations.

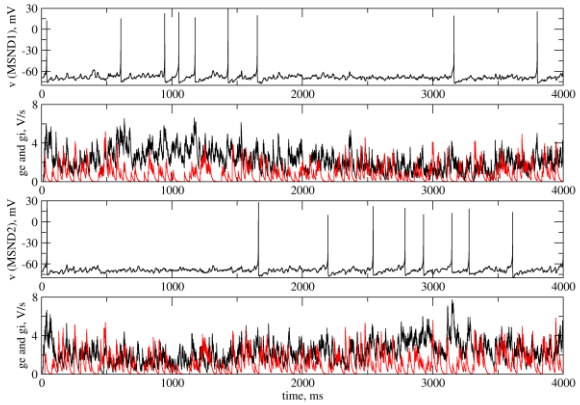

Figure 2. Membrane potential $v(t)$ and synaptic dynamics $g_e(t)$ and $g_i(t)$ for neurons which are randomly selected in MSND1 and MSND2 groups. g_e and g_i is black and red, respectively. DA level is 1 (normal), 1.1 (high), 1 (normal), 0.9 (low) and 1 (normal) for 0-500ms, 500ms-1500ms, 1500ms-2500ms, 2500ms - 3500ms and 3500ms-4000ms time intervals.

In time course of the change of dopamine level can be followed from firing rates easily while raster plot gives the information of neuronal population at neuron level. The firing rates inspired the idea of proposing a simple model for the interaction of neuronal population, where each population activity is denoted by a single dynamical variable as proposed in mass model.

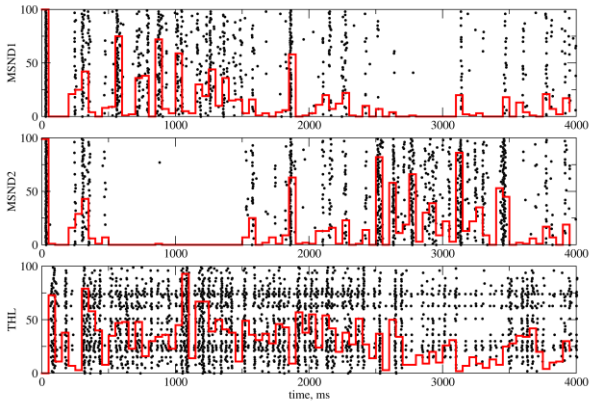

Figure 3. Raster plot and firing rates of MSND1, MSND2 and THL. When DA level is high, THL activity is higher than normal rate and DA level is low, THL activity is lower than normal rate.

4.2. Mass model results

The simulation for the mass model are done using XPPAUT, a tool developed for dynamical system analysis. In order to demonstrate the analogy between the firing rate results of spiking neuronal network and mass model, the time course of dynamical behavior of the variables

corresponding to MSND1, MSND2 and THL populations are given in **Figure 4**. Here, again the dopamine level is switched similarly from normal to high, then to normal level followed by low level and finally to normal level by

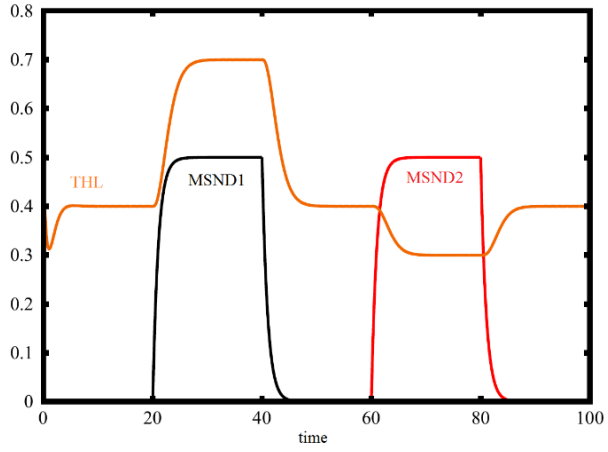

Figure 1. MSND1, MSND2 and THL activity. Mass model results show similar results spiking neural network. THL activity is effected by DA level similar to spiking neural network firing rate.

changing the parameter DA in **Equation 6**. The simulation interval is different as the it is scaled for the mass model, but the results shown in **Figure 4**, resembles the change in firing rates given in **Figure 3**.

4.3.Discussion of the results

As the simulation results given in Subsections 4.1 and 4.2 reveal, the overall activity of neuronal population can be followed from the mass model proposed as a simple linear system. Of course, with this coarse linear system approach, it is not possible to investigate the synaptic activity, or the activity in a neuronal population, but a general idea about the effect of dopamine can be followed easily. So building a simple model which consolidate the afferent and efferent connections between neuronal populations and the effect of neurotransmitter, would be informative to grasp the dynamics behind the neuronal activity. Furthermore, such a simple model can be versatile for the implementation of biologically inspired approaches and developing new learning rules as in [4,5].

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