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# Selective amplification using a contracting model of the basal ganglia

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## ABSTRACT

Nous proposons un nouveau modèle des boucles cortico-baso-thalamo-corticales, intégrant des projections des ganglions de la base (GB) habituellement négligées, et dont la dynamique est maîtrisée par l'usage de la théorie de la contraction. L'interconnexion du système amplificateur cortex-thalamus avec celui, sélectif, des GB, permet une sélection nette des informations destinées aux cibles sous-corticales et une conservation des données d'entrée avec amplification sélective des signaux gagnants dans le cortex frontal.

## KEY WORDS

Basal ganglia, loops, action selection, contraction analysis

## 1 Introduction

The basal ganglia (BG) are a set of interconnected sub-cortical nuclei, common to all vertebrates, and thought to constitute a generic evolution solution to the problem of selection. They interact with various cortical and subcor-

tical regions involved in sensorimotor, cognitive or limbic processes. The interaction of the BG with the cortex takes place in parallel cortico-baso-thalamo-cortical (CBGTC, see Fig. 1) loops [1].

The precise selection role of the BG depends on the involved circuits. It can be, for example, to select the most appropriate behaviour in a given context, the target of a saccade among the multiple points of interest present in the visual field, the piece of information to be stored in working memory, etc. Each element competing for selection is represented by a channel inside the BG, and is considered selected when it is disinhibited [2]. At rest, the BG tonically inhibits all the neural circuits corresponding to the competing elements. When one of them wins the competition, the inhibition of the corresponding channel is removed and the target circuit is thus activated.

Numerous computational models of the basal ganglia have been proposed in the last ten years [3, for a review]. However most of them rely on the "direct/indirect pathways" scheme proposed by [4], which is quite outdated and doesn't take into account numerous connections. Even the latest models [5, 6] neglect some of these interesting projections. Finally, despite the fact that

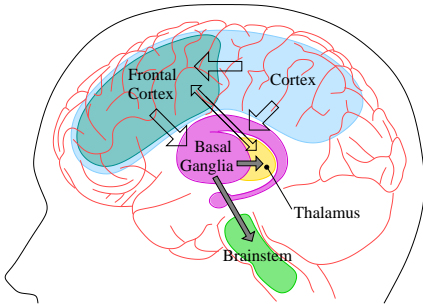


Figure 1: Cortico-baso-thalamo-cortical loops. The basal ganglia receive inputs from the whole cortex, but establish loops with the frontal areas only. Shaded arrows: inhibitory projections.

the CBGTC contains numerous internal loops susceptible to generate various dynamic behaviors, the dynamics of these models was not analyzed.

Consequently, we propose a new model of the CBGTC loops based on our BG model [7] including usually neglected connections, and prove the stability of its operation using contraction analysis [8]. Contraction analysis is an extension to nonlinear systems of the stability analysis for linear systems. It is well adapted to study the dynamics of artificial neural networks made of nonlinear components. Moreover, contraction has the advantage of being preserved through basic system combinations (hierarchies, feedback, etc.), it is thus possible to study the contraction of isolated modules and then to obtain the contraction of a large system made of these modules. A preliminary version of this work has been presented in [9]

## 2 Nonlinear Contraction Analysis

A nonlinear time-varying dynamic system is called *contracting* if initial conditions or temporary disturbances are forgotten exponentially fast. Thus, finding a particular trajectory of a contracting system is sufficient to be sure that it will eventually tend to this trajectory.

Consider a  $n$ -dimensional time-varying system of the form:

$$\dot{\mathbf{x}}(t) = \mathbf{f}(\mathbf{x}(t), t)$$

where  $\mathbf{x} \in \mathbb{R}^n$  and  $t \in \mathbb{R}_+$  and  $\mathbf{f}$  is  $n \times 1$  non-linear vector

function which is assumed to be real and smooth.

If there exists a uniformly positive definite metric  $\mathbf{M}(\mathbf{x}, t) = \Theta(\mathbf{x}, t)^T \Theta(\mathbf{x}, t)$  such that the generalized Jacobian:  $\mathbf{F} = (\Theta + \Theta \mathbf{J}) \Theta^{-1}$  is uniformly negative definite, then all the all system trajectories converge exponentially to a single trajectory with convergence rate  $|\tau_{max}|$ , where  $\tau_{max}$  is the largest eigenvalue of the symmetric part of  $\mathbf{F}$ . The system is said to be *contracting*.

## 3 Model description

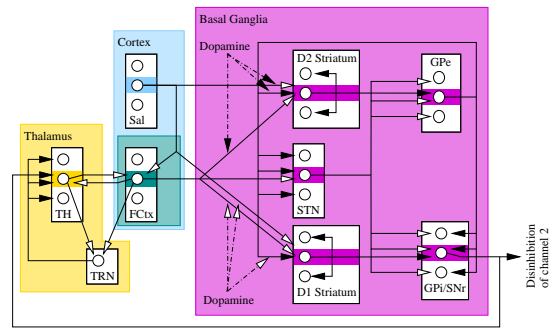


Figure 2: CBGTC loop model. Three channels competing for selection are represented; for clarity, the projections of the second channel only (the shaded one) are represented; white arrowheads: excitatory projections; black arrowheads: inhibitory projections; TRN: thalamic reticular nucleus; other abbreviations: see text. Dopamine level has a modulatory effect on the striatal input.

The model is built out of rate-coding artificial neurons (leaky integrators), defined as follows:

$$\begin{cases} \tau \dot{a}(t) = -a(t) + I(t) \\ y = [a - \epsilon]_+ \end{cases}$$

$\tau$ : time constant of the neuron,  $a(t)$ : activation,  $y(t)$ : output,  $I(t)$ : input,  $\epsilon$ : firing threshold. Note that leaky integrator neurons are trivially contracting (the Jacobian is  $-\frac{1}{\tau}$  and the transfer function defines a convex region).

In accordance to neurobiological data [10], the BG part of the model includes projections from the external globus pallidus (GPe) to the striatum, which are usually neglected, moreover the projections from the GPe to the subthalamic nucleus (STN), the internal globus pallidus

(GPi) and the substantia nigra pars reticulata (SNr) are considered diffuse (see Fig. 2, basal ganglia box). The cortical inputs to the BG, or saliences (Sal), are represented by a vector of values associated to each channel, for simplicity. These values of course represent the result of the convergence of information from various cortical areas. The BG circuitry selects the channel with the highest input (or salience) using various mechanisms: recurrent lateral inhibitions in the input nuclei (D1 and D2 striatum), off-center on-surround architectures in the D1-STN-GPi and D2-STN-GPe complexes, reinforced by negative feedback loops between GPi and D1, and GPe and D2. It was proved [7] to be contracting and to perform efficient selection.

Given known thalamus anatomy and connectivity [11], a simple thalamo-cortical module is added to the existing BG model (see Fig. 2, thalamus and cortex boxes). Depending on the CBGTC loop considered, the thalamic nuclei involved (ventro-lateral, medio-dorsal, etc.), as well as the sensory and frontal cortical areas, may vary. They are represented by the TH nucleus and the Sal and FCtx areas in Fig. 2. As we model one CBGTC loop, we consider the reciprocal channel-specific thalamo-cortical connections but exclude the cross-loop projections [12].

The Th-FCtx excitatory loop is proposed to have a role of amplification of the sensory signal, its net gain is however kept under 1, so that it amplifies the cortical signal without saturating (and thus avoid infinitely self-sustained activity). The inhibitory projections of the BG onto the thalamus limits the amplification of the unselected channels and thus favors a selective amplification of the winning channels. The interest of such an architecture is that in the frontal cortex, the selected channel has its activity amplified, but the information about the other channels isn't lost. On the contrary, the subcortical target circuits of the BG are under very selective inhibition, so that the circuit corresponding to the winning channel only is disinhibited and able to affect the animal's behavior. The equation system defining the model is given in the appendix.

## 4 Results

### 4.1 Contraction Conditions

The contraction rates of the BG and thalamo-cortical (TC) modules can be determined using the combination properties described in [8]. The result concerning convex regions allows us to study the linear part of the model only. We already showed that the BG part of the model is contracting if  $w_{D1} < 1$ ,  $w_{D2} < 1$  and  $w_{D2}^{GPe} w_{GPe}^{D2} < -1 + w_{D2}$  [7]. We then concentrate on the loops of the rest of the model. Concerning the thalamo-cortical one, the analysis is simplified if  $w_{TH}^{TRN} = w_{TRN}^{TH}$ , in which case we obtain the following condition:

$$w_{FC}^{TRN^2} < 4(1 - w_{FC}^{TH} w_{TH}^{FC})$$

The remaining loops are instances of feedback combination for which we can use the algebraic condition on weights that gives rise to:

$$\tau_{BG} = 1 - (0.5w_{D2} + \sqrt{0.25w_{D2}^2 + w_{D2}^{GPe} w_{GPe}^{D2} (1 - \lambda)})$$

$$\tau_{TC} = 1 - 0.5(w_{FC}^{TH} w_{TH}^{FC} + \sqrt{w_{FC}^{TH^2} w_{TH}^{FC^2} + w_{FC}^{TRN^2}})$$

Finally, for the whole system to be contracting, a last condition on is required:

$$w_{GPe}^{TH} \max(w_{FC}^{D1}, w_{FC}^{D2}, w_{FC}^{STN}) < 4\tau_{BG}\tau_{TC}$$

### 4.2 1000 random vector test

The ability of the system to (1) perform clear selection of the channel with the largest *saliency*, by a disinhibition in the GPi/SNr, and (2) significantly amplify the frontal cortex signal of this channel only, without any influence of the initial state (a property implied by contraction), is tested by feeding a 6-channels system with a sequence of 1000 randomly drawn salience vectors. These saliences are drawn uniformly in  $[[1, 200]]$ . Each vector is presented during 0.5s, then the disinhibition in the GPi/SNr of the channel with maximum *saliency* is checked and the amplifications in FCtx are computed, before the next vector of the sequence is presented without resetting the system.

Concerning selection, the channel with maximum salience  $i_0$  is always selected (ie.  $\forall i \in [[1, 6]], i \neq i_0, y_{i_0}^{GPe} < y_i^{GPe}$ ). Two types of exceptions are expected

and observed: (1) for 0.3% of the input vectors, all the *saliences* are too small to induce any selection, because of the *D1* and *D2* input thresholds filtering the too low *saliences*; (2) for 16.7% of the input vectors, the closest competitor channel is also selected, because the difference between the two *saliences* is relatively small, so that the BG module cannot discriminate between them (see fig. 3).

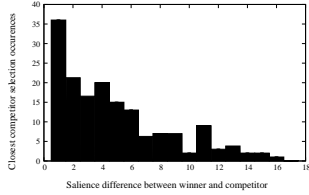


Figure 3: Selection of both the maximum salience channel and its closest competitor: histogram of salience difference between the two channels wrt. the number of occurrences of double selection.

The signal amplification in the frontal cortex is, in average, for the selected channels, 44.4%, while for the others it is 13% only (see fig. 4). By comparison, in the case of an isolated FCtx-TH-TRN circuit (ie.  $w_{GPe}^{TH} = 0$ ), the average amplification for all channels is 25.3%.

## 5 Discussion

The model proposed omits two BG nuclei projections, the STN to striatum one [10] and the D1 to GPe one [13]. The STN neurons projecting to the striatum constitute a population distinct from those projecting to the GPe, GPi and SNr, their role in the BG selection process has to be further investigated. The D1-GPe projection could improve the quality of the selection, as in [14], nevertheless, this adds a new loop whose contraction must be assessed. Finally, the inhibitory interneurons of the striatum were not modelled and might also add some selectivity.

## 6 Conclusion

We proposed a new computational model of the cortico-baso-thalamo-cortical loops, based on the idea that the

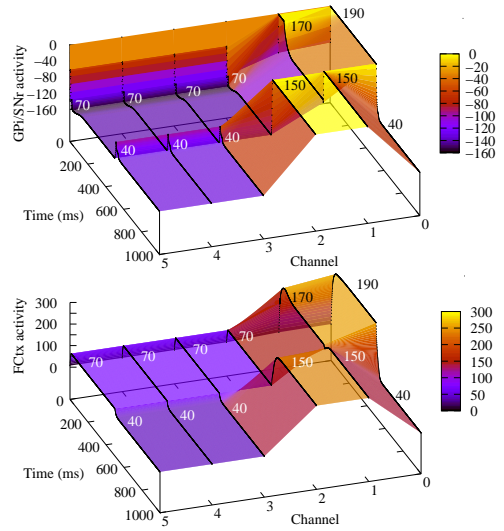


Figure 4: Example of GPi/SNr (top) and FCtx (bottom) activity with two successive salience vectors (70, 70, 70, 70, 170, 190) and (40, 40, 40, 150, 150, 40) presented during 500ms each. Top: values show the exact level of inhibition, bottom: percentage values represent the amplification bonus with regards to the input signal.

thalamo-cortical circuitry is dedicated to signal amplification and the basal ganglia to selection of the signals to be amplified. We theoretically studied the dynamic of the network and proved its stability using contraction analysis.

Beyond these dynamics considerations, basal ganglia loops models have previously been used to control autonomous robots [15, 16] or to simulate psychological tests [17]. The relevance of the present model should be tested in such contexts.

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Table 1: Parameters of the simulations.

$w_{S_{al}}^{D1}$	3.6	$w_{GPe}^{STN}$	0.35	$\tau$	0.003s
$w_{S_{al}}^{D2}$	3.6	$w_{GPe}^{GPe}$	0.08	$\lambda$	0.2
$w_{D1}$	0.4	$w_{D2}^{GPe}$	0.7	$w_{TH}^{FC}$	0.6
$w_{D2}$	0.4	$w_{D1}^{GPe}$	1	$w_{FC}^{TH}$	0.6
$w_{GPe}^{D1}$	1	$w_{STN}^{GPe}$	0.35	$w_{GPe}^{TH}$	0.5
$w_{GPe}^{D2}$	1	$w_{STN}^{GPe}$	0.35	$w_{TRN}^{TH}$	0.35
$w_{FC}^{TRN}$	0.35	$w_{TH}^{TRN}$	0.35	$w_{FC}^{D1}$	0.4
$w_{FC}^{D2}$	0.4	$w_{FC}^{STN}$	2.32		

## Appendix

The model is described by the inputs of the different neurons, with  $\sum_{j=1}^N y_j^{STN} = \Sigma_{STN}$  (parameter settings given in table 1):

$$I_i^{FC} = S_i + w_{TH}^{FC} y_i^{TH}$$

$$I_i^{TH} = -w_{GPe}^{TH} y_i^{GPe} - w_{TRN}^{TH} y_i^{TRN} + w_{FC}^{TH} y_i^{FC}$$

$$I_i^{TRN} = \sum_{j=1}^N (w_{FC}^{TRN} y_j^{FC} + w_{TH}^{TRN} y_j^{TH})$$

$$I_i^{D1} = (1 + \lambda)(w_{S_{al}}^{D1} S_i + w_{FC}^{D1} y_i^{FC} - w_{GPe}^{D1} y_i^{GPe}) - w_{D1} \sum_{\substack{j=1 \\ j \neq i}}^N y_j^{D1}$$

$$I_i^{D2} = (1 - \lambda)(w_{S_{al}}^{D2} S_i + w_{FC}^{D2} y_i^{FC} - w_{GPe}^{D2} y_i^{GPe}) - w_{D2} \sum_{\substack{j=1 \\ j \neq i}}^N y_j^{D2}$$

$$I_i^{STN} = w_{FC}^{STN} y_i^{FC} - w_{GPe}^{STN} \sum_{j=1}^N y_j^{GPe}$$

$$I_i^{GPe} = -w_{D2}^{GPe} y_i^{D2} + w_{STN}^{GPe} \Sigma_{STN}$$

$$I_i^{GPe} = -w_{D1}^{GPe} y_i^{D1} + w_{STN}^{GPe} \Sigma_{STN} - w_{GPe}^{GPe} \sum_{j=1}^N y_j^{GPe}$$

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