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Capita Selecta

Assessing the need for biological plausibility in cognitive modelling: a focus on basal ganglia without segregated pathways

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Overview

The present document describes the technical work done for my Capita Selecta. First, the implementation of the basal ganglia model is described. As I believe some of the code can be useful for other Nengo users, a pull request has been opened and is currently being reviewed. The second part describes some of the experiments that have been simulated. The research proposal describing the scientific context of the project, the slides of the midterm presentation and the final poster that was presented at the CJC-SC seminar are available as appendices. For more information about the project see the Github and ResearchGate pages.

1 Implementation

The spiking version of the biologically constrained model of basal ganglia (sBCBG) proposed by Liénard and Girard (2014) was originally implemented in the NEST simulator. In order to analyse its action selection capacities and for further integration in broader cognitive models, the first part of my Capita Selecta has been dedicated to the implementation of this model in Nengo.

1.1 Straightforward translation

In most cases, there is a direct correspondence between the functions of NEST and the functions of Nengo, making the translation straightforward. For example, creating a population of leaky integrate-and-fire neurons (LIF) can be done with either `nest.Create` or `nengo.Ensemble`.

1.2 Spiking threshold

The two simulators differ in some ways. NEST is more biologically grounded and includes more precise parameters. Table 1 shows the correspondence between some NEST and Nengo parameters that are used to define the neuron models in sBCBG. It is not possible to directly define the spiking threshold of the neurons of a population in Nengo. In the case of LIF, the subthreshold dynamics are linear with respect to the current. This observation tells us that scaling the threshold is mathematically equivalent to inversely scaling the input current by the exact same factor. Such tricks were used to address the differences between the two simulators.

NEST	Nengo	Description
V_min	min_voltage	Minimum voltage of the membrane
t_ref	tau_ref*1000	Refractory period (ms)
tau_m	tau_rc*1000	Membrane time constant (ms)
C_m	tau_rc / resistance	Capacity of the membrane
V_th	-	Threshold
I_e	-	Constant external input

Table 1: Selected leaky integrate-and-fire neuron parameters in NEST and Nengo

1.3 Constant external Input

The table also shows that there is no direct support for a constant external input. This feature is available in many spiking simulators but is less useful for a functional approach using Nengo. However, it is necessary to implement sBCBG. It can be done by simply connecting a Nengo Node to the population

with the desired current. Like every connection, the weight has to be scaled by the spiking threshold of the postsynaptic population for the reasons mentioned in the previous section.

1.4 Connections

In Nengo, neural populations encode a vector and the connections are usually optimized "under the hood" to compute some arbitrary function of this vector. However, this is inherent to the functional approach of this simulator, whereas other simulators focus on biological features and use a connectivity matrix or basic rules to connect neural ensembles. The NEST implementation of sBCBG makes use of two rules in particular:

- `fixed-indegree` randomly connects neurons in the source population with the neurons in the target population such that each neuron in the target population has a fixed indegree.
- `fixed_total_number` randomly connects neurons in the source population with the neurons in the target population such that the total number of connections equals a given parameter N.

Although it is not common, it is possible to do a neuron-to-neuron connection in Nengo with the `transform` argument. The following code has been written to build a connectivity matrix with either the `fixed-indegree` or the `fixed_total_number` rules.

```
def connectivity_matrix(rule, value, n_pre, n_post):
  connectivity = np.zeros((n_post, n_pre))

  if rule=='fixed_indegree':
    for post_neuron in range(n_post):
      for in_i in range(value):
        pre_neuron = rnd.randint(0, n_pre)
        connectivity[post_neuron, pre_neuron] += 1

  elif rule=='fixed_total_number':
    for connection in range(value):
      pre_neuron = rnd.randint(0, n_pre)
      post_neuron = rnd.randint(0, n_post)
      connectivity[post_neuron, pre_neuron] += 1

  else:
    raise ValueError('Nengo error: unknown rule in connectivity_matrix')

  return connectivity
```

1.5 Poisson generators

The inputs of sBCBG are cortico-striatal neurons (CSN), pyramidal tract neurons (PTN) and the centromedian complex of the intralaminar thalamus (CMPf). These populations are modelled with Poisson generators (i.e. firing with exponentially distributed interspike intervals). To implement these input populations in Nengo, a new neuron type has been implemented. A pull request with this Poisson neuron model has been submitted to the Nengo Github repository and is currently under review[1]. The

[1] https://github.com/nengo/nengo/pull/1505

following code is the main part of its implementation and conforms to the style guide of the Nengo contributor guide.

```
class Poisson(NeuronType):
    """Poisson spiking generator

    Parameters
    ----------
    amplitude : float, optional (Default: 1)
        Scaling factor on the neuron output. Corresponds to the relative
        amplitude of the output of the neuron.
    seed : int, optional (Default: 1)
        The seed used for random number generation.
    """

    probeable = ('spikes',)

    def __init__(self, amplitude=1, seed=1):
        super(Poisson, self).__init__()

        self.amplitude = amplitude
        self.rng = np.random.RandomState(seed)

    def rates(self, x, gain, bias):
        out = self.current(x, gain, bias)
        out = np.maximum(0, out) * self.amplitude
        return out

    def step_math(self, dt, J, output):
        p = J*dt # find spiking probabilities
        output[:] = (self.rng.rand(*J.shape)<p).astype(float) # sample
        output[:] *= self.amplitude / dt
```

1.6 Semantic Pointer Architecture module

Nengo SPA is an external package that implements the Semantic Pointer Architecture in Nengo. The last step of the implementation part of the project was to add sBCBG as an action selection module. The structure of the code was based on the traditional action selection module of the SPA based on Gurney et al. (2001). The thalamus model was not modified.

Structure of the code

1. Instantiate a sBCBG network with N channels if there are N competing actions
2. Connect an input node with N dimensions to an ensemble that represents the input with 100 LIF neurons
3. Connect the ensemble to the CSN population of the sBCBG network. This connection is scaled by a factor to properly activate the population of medium spiny neurons in the striatum (by default, the factor is 10). This is done with a function encoded by the connection. For this reason, we

cannot connect the input node to the CSN directly, as functions are not allowed on pass-through nodes.

4. Connect the GPi (output nucleus of sBCBG) to an output node. As sBCBG populations are connected directly (see section 1.4), it is necessary to decode the activity of the GPi to send a one dimensional vector to the thalamus (the utility of actions). For that, we use a simple solver with each neuron contributing equally to the vector. This is done by using a $M \times 1$ matrix filled with ones, with M the number of neurons in one GPi channel (each channel corresponds to one action). The connection is filtered with a lowpass synapse to compensate the all or none nature of spikes.

2 Simulations

Five notebooks have been created to highlight important experiments of the project:

1. **Runtime** compares the runtime of sBCBG and the spiking implementation of the model proposed by Gurney et al. (2001) as a function of the number of competing actions.

2. **Parkinson's disease condition** shows the oscillations emerging when the dopamine is reduced in the GPe and the STN nuclei. The method for simulating Parkinson's disease is described in the research proposal and by Lienard et al. (2017).

3. **Action selection** compares the ability of a model to select the action with highest utility. The following models are compared: sBCBG in normal condition, sBCBG in parkinsonian condition and the spiking implementation of the model proposed by Gurney et al. (2001).

4. **Activity of the nuclei** shows the activity of the nuclei during the action selection task

5. **Syllable sequencing task** tries to reproduce the syllable sequencing task proposed by Senft et al. (2018), though simplified, and compares sBCBG with the spiking implementation of the model proposed by Gurney et al. (2001).

3 Bibliography

Gurney, K., Prescott, T. J., Redgrave, P., May 2001. A computational model of action selection in the basal ganglia. i. a new functional anatomy. Biological Cybernetics 84 (6), 401–410.
URL https://doi.org/10.1007/PL00007984

Liénard, J., Girard, B., Jun 2014. A biologically constrained model of the whole basal ganglia addressing the paradoxes of connections and selection. Journal of Computational Neuroscience 36 (3), 445–468.
URL https://doi.org/10.1007/s10827-013-0476-2

Lienard, J. F., Cos, I., Girard, B., 2017. Beta-band oscillations without pathways: the opposing roles of d2 and d5 receptors. bioRxiv.
URL https://www.biorxiv.org/content/early/2017/07/17/161661

Senft, V., Stewart, T. C., Bekolay, T., Eliasmith, C., Kröger, B. J., 2018. Inhibiting basal ganglia regions reduces syllable sequencing errors in parkinson's disease: A computer simulation study. In: Front. Comput. Neurosci.

Appendices

I Research proposal

Assessing the need for biological plausibility in cognitive modelling: a focus on basal ganglia without segregated pathways

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Abstract

Both old and new anatomical findings challenge mainstream theory of the basal ganglia circuitry and of how this structure performs action selection. A new biologically plausible model of basal ganglia takes into account these findings. Here I propose to compare this model with a simpler one which is in accordance with the mainstream theory. Both models will be used within an existing cognitive architecture to perform a well-known naturalistic task, the Tower of Hanoi, and be evaluated according to behavioural data from human studies. I believe it will be a good way to assess the functional performance of both models and the need for biological plausibility in cognitive modelling. The cognitive architecture will also provide a good framework for studying the relations between basal ganglia, high level cognition and movement disorders like Parkinson's disease.

Keywords: Computational neuroscience, Cognitive modelling, Basal ganglia, Parkinson's disease

1. Project description

Drawing inspiration from neuroscience has led to great progresses in artificial intelligence (AI). However, it is not yet clear to what extent biological plausibility is needed for human-level AI. Basal ganglia (BG) have been studied extensively and can serve as a case study to assess the need for biological plausibility. These brain nuclei have been modelled at different scales and with various levels of complexity. In Section 1.1.1, the traditional theory of how BG work will be presented and criticised, with neuroscientific studies suggesting that this theory is too simplistic. A BG model in accordance with the traditional theory has previously been implemented within cognitive architectures to perform naturalistic tasks. Comparing the ability of the cognitive architectures to reproduce human behavioural data when using this model, along with a more detailed one, can shed light on the trade-off between complexity and performance. This project can also be derived as a general method for evaluating computational theories and models. Furthermore, the detailed model described in Section 1.1.2 can be simulated in Parkinsonian conditions. Hence, it could be used to study the impact of the disease on behaviour. Societal, technical and scientific relevance of this project will be discussed in more details in Section 3.

1.1. Background

1.1.1. Basal ganglia

Basal ganglia (BG) form a brain structure thought to be fundamental for learning and decision processes. These strongly interconnected subcortical nuclei are often assumed to perform action selection modulated by reinforcement learning. Their dysfunction results in a wide range of motor impairments such as Parkinson's disease.

An old but still very popular box-and-arrow model of BG, the aim of which was to explain these disorders, was proposed by Albin et al. [2]. In the motor areas of the cortex and in the BG, each motor command is represented as a discrete channel of neurons. With their model, Albin et al. [2] argue that BG are divided into a direct and an indirect pathway and that motor disorders result from an imbalance of the activity of these pathways. The direct pathway is commonly referred to as the facilitating pathway as it results in the selection of the action corresponding to the channel whose direct pathway is activated (i.e. results in the excitation of the corresponding neurons in the motor cortex). Conversely, the indirect pathway results in the inhibition of the competing motor commands in the cortex. In the striatum, medium spiny neurons (MSN) of the direct pathway target the internal globus pallidus (GPi) and express D1 dopamine receptor. MSN of the indirect pathway target the external globus pallidus (GPe) and express D2 receptor (see the solid lines in Figure 1).

Abbreviations used in the text

AI: Artificial intelligence
BG: Basal ganglia
MSN: Medium spiny neurons
GPi: Internal globus pallidus
GPe: External globus pallidus

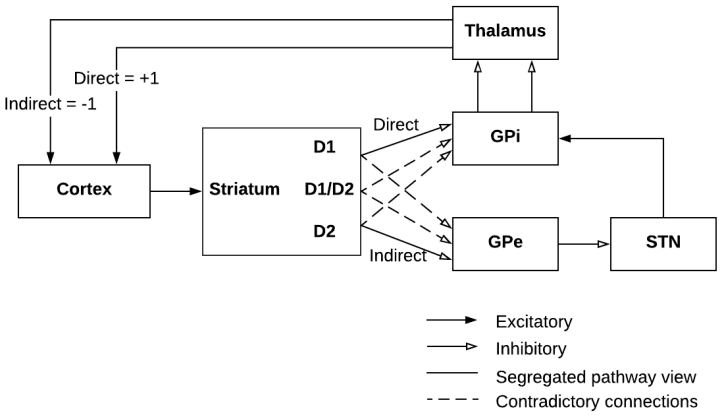


Figure 1: Direct and indirect pathways (solid lines) and contradictory connections (dashed lines). D1, D2 and D1/D2 refer to MSN populations of the striatum that express D1, D2 or both D1 and D2 dopamine receptor respectively. In the traditional view (solid lines), channels with D1 as most active MSN subpopulation receive a positive feedback via the direct pathway (+1). Channels with D2 as most active MSN receive a negative feedback via the indirect pathway which includes the STN (-1).

However, several findings suggest that the simple view in which the direct and indirect pathways are perfectly segregated is an over-simplification of the primates' and rats' BG connectivity. Almost every MSN projects to both GPi and GPe in primates [16, 12] and rats [11, 24]. Furthermore, up to 60% of MSN express both D1 and D2 dopamine receptors [3, 15, 1]. Figure 1 (dashed lines), summarises these findings that contradict the segregation view.

1.1.2. Biologically constrained model of basal ganglia

Liénard and Girard [13] have recently proposed a biologically constrained model of the primates' BG which takes into account the data that contradict the segregation view, as well as other anatomical and electrophysiological data. The parameters that are pretty well known, such as the number of neurons, the maximal discharge rates, or the axonal delays, were considered as fixed parameters. The other parameters such as the mean axonal varicosity counts, or the difference between the resting and threshold potential for each population, were optimized according to the fit of the models with available anatomical data, and with in vivo electrophysiological experiments. After optimisation, the authors found 15 solutions (i.e. configurations of the model) that matched their constraints. The overall architecture of the model is shown in Figure 2 (right).

The model can exhibit emergent action selection properties [13] and plausible Parkinson-like dynamics [14] although it was not optimized for these purposes. Even more recently, the model has been implemented with spiking neurons by Girard et al. [8]. When it is simulated in Parkinsonian conditions, this implementation exhibits dynamics that are less stereotypical than those exhibited with the non-spiking model [5, 9].

1.1.3. Nengo

Nengo is a software tool that can be used to simulate large-scale neural models. Its aim is to bridge the gap between low-level biological details and high-level modelling of cognitive functions such as sensory processing, memory formation, reasoning, and motor control [4]. Nengo has been used to implement Spaun, a model of multiple brain areas capable of performing eight different tasks[1] [19]. Spaun, among other projects developed with Nengo, make extensive use of BG as a program sequencer.

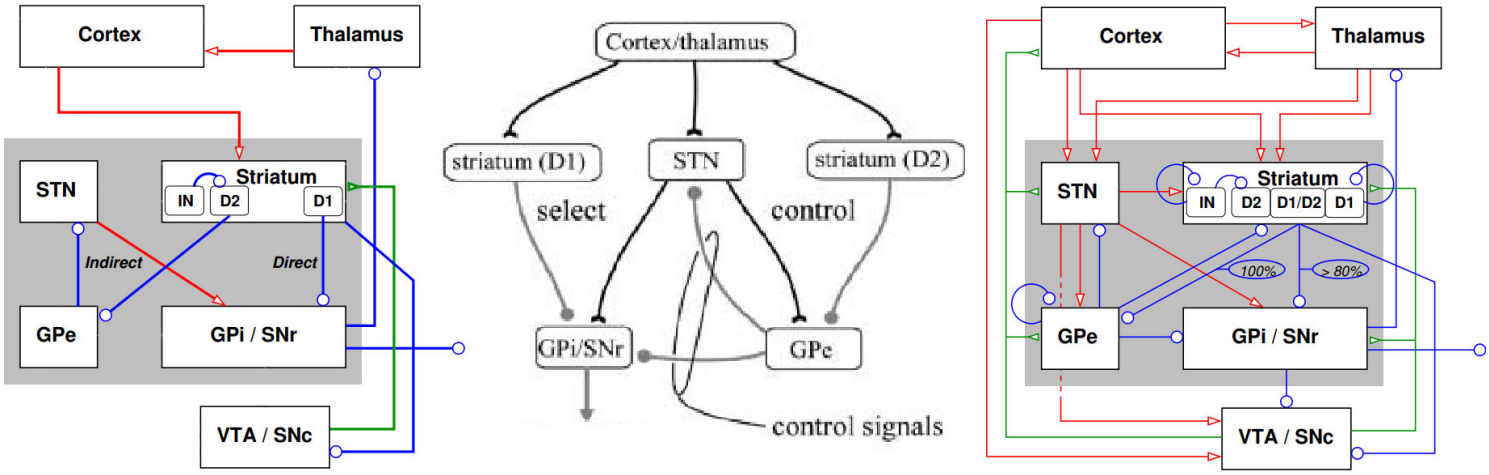


Figure 2: Comparison of the architecture of three models: the canonical model proposed by Albin et al. [2] (left), the model of Gurney et al. [10] on which Nengo's default action selection module is based upon (center), and the most biologically accurate model proposed by Liénard and Girard [13] which does not include segregated pathways (right). Adapted from [9] and [10].

[1]Description and examples for each task are available on `https://xchoo.github.io/spaun2.0/videos.html`

All of the projects that use a BG [6, 20, 19, 21, 18] used a spiking implementation of the model described by Gurney et al. [10] which includes segregated pathways. An explicit reference to the canonical model of Albin et al. [2] is made in the supplementary material of the report of Spaun [7]. As detailed above, these models inaccurately describe primates' and rats' BG. In his thesis, Tripp [22] does mention the results of Aizman et al. [1], Lévesque and Parent [12] while discussing the limitations of Albin et al. [2] but does not take them into account later in his work with Nengo. Aside from the segregation of projections of the striatum, the models of Albin et al. [2] and Gurney et al. [10] omit a lot of connections captured by the model of Liénard and Girard [13] (see Figure 2 for a comparison).

1.1.4. Cognitive architecture of the Tower of Hanoi task

Stewart and Eliasmith [21] have developed a neural cognitive architecture capable of solving arbitrary Tower of Hanoi problems in a human-like way in Nengo. This task involves three pegs and a fixed number of disks of different sizes. A disk can be moved from the top of a peg to the top of another peg if there is not a smaller disk in the new peg. Given an initial configuration, the goal is to move the disks to a goal configuration (typically, to move all disks from one peg to another). Figure 3 shows the optimal series of steps needed to solve the four-disk Tower of Hanoi when all disks start on one peg and have to be moved onto a different peg. Non-neural symbolic cognitive models of this task already exist and match empirical data extremely well. However, the neural cognitive architecture developed by Stewart and Eliasmith [21] also provides insights on how this task can be solved with spiking neurons and an architecture inspired by the human brain.

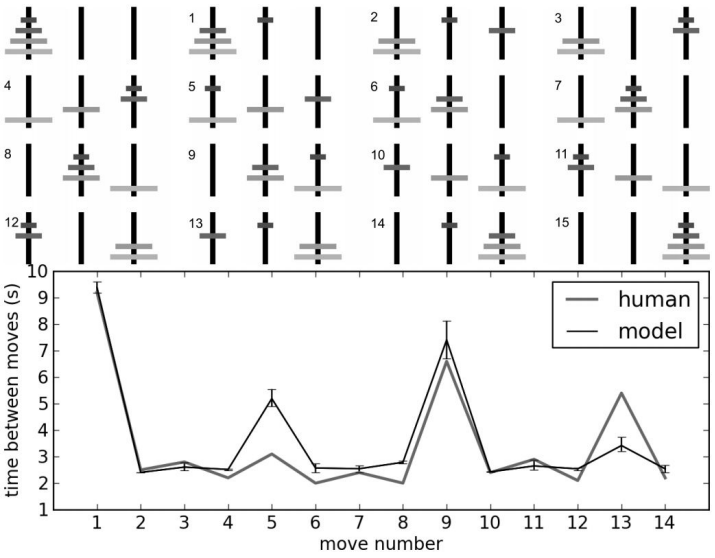


Figure 3: The sequence of moves to ideally solve the fourdisk Tower of Hanoi (top). Time delay for expert human performance and the neural model (bottom). Taken from [21].

The cognitive architecture is composed of cortical states, an action selection module based on the BG model proposed by Gurney et al. [10], a thalamus, a motor output and a visual input (see Figure 4). There is only one free parameter, namely the synaptic connection weights of the input to the short-term memory which controls how quickly the memory will store new inputs and forget old information. Nevertheless, the model is able to successfully solve the Tower of Hanoi, given any initial/goal configuration. The time delay at each move were compared with expert human players (see Figure 3).

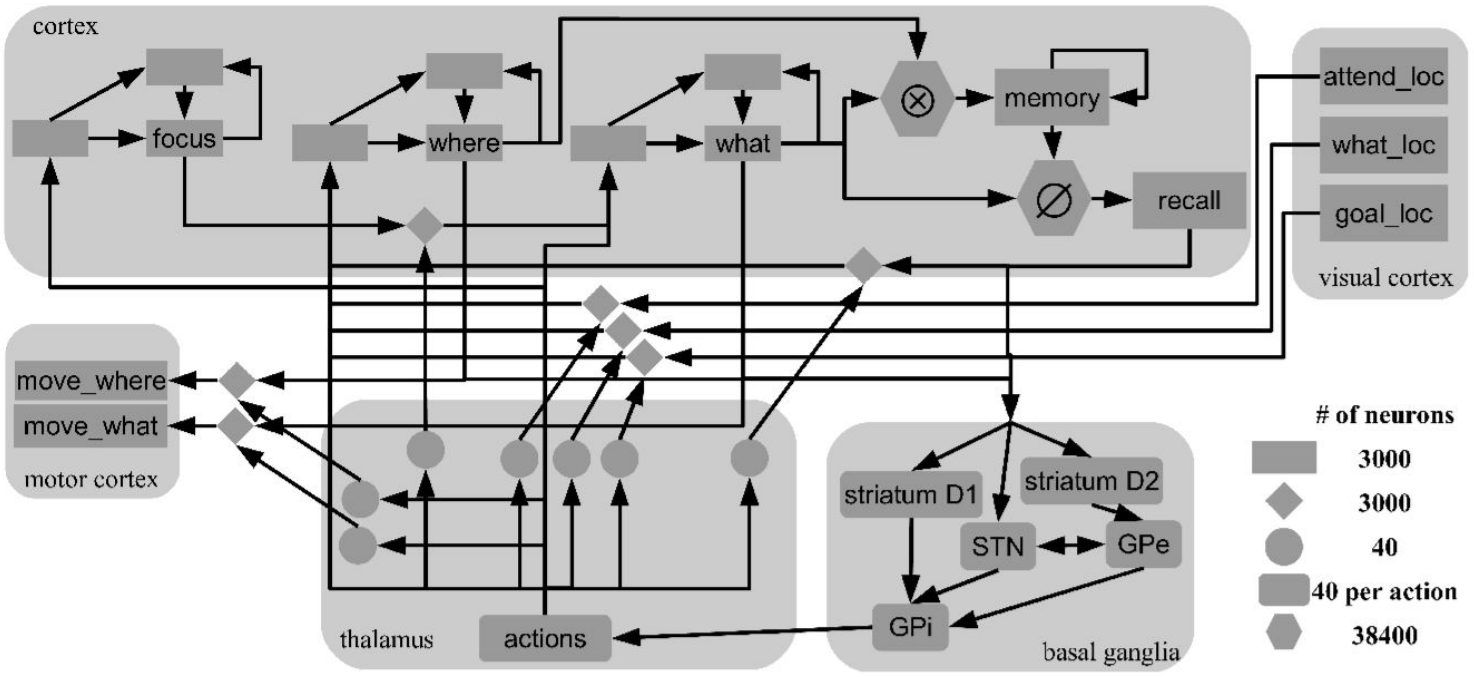


Figure 4: The Tower of Hanoi model. Total # neurons: 150,640. Taken from [21].

1.2. Aim of the project

During this project, the new model of BG described in section 1.1.2 will be used as a replacement of the action selection module of the Tower of Hanoi cognitive architecture. Action selection properties of BG models are usually tested with dummy inputs that do not explicitly represent a command, making the results hard to interpret. The Tower of Hanoi architecture provides the opportunity to assess the functional performance of models in a naturalistic context with a meaningful task. It is possible to directly compare the motor output of the architecture with the results of behavioural studies implying the Tower of Hanoi task. Stewart and Eliasmith [21] used the delay between moves to evaluate the plausibility of their model's behaviour (see Figure 3). One can hypothesise that a more biologically detailed action selection module like the biologically constrained model of Liénard and Girard [13] will improve the human-likeliness of the architecture, and better fit these behavioural data.

Furthermore, the Tower of Hanoi cognitive architecture will serve as a framework to study the relations between high-level cognition and Parkinson's disease, characterised by dopamine depletion leading to beta-oscillations in BG. The Tower of Hanoi is widely used as a test of planning and problem solving abilities. It is therefore used for studying the impact of Parkinson's disease on planning and executive function (e.g. [17, 23]). It will be interesting to compare the ability of the architecture to reproduce known pathological behaviours with both models of BG. A detailed model of the functioning brain would permit to better understand the relations between biological processes and symptoms. Hopefully, this study will form a significant building block for this long-term objective.

Last but not least, it can turn out that the new model is more efficient from the algorithmic point of view. Stewart and Eliasmith [21] noticed that their Tower of Hanoi architecture sometimes makes mistakes they could not explain. As the human BG do not seem to include perfectly segregated pathways, the current BG model available by default in Nengo may not be an optimal action selection device. If this more biologically accurate bottom-up model performs better, it could be useful for possibly commercial applications in Artificial Intelligence. Incidentally, ABR, a company whose aim is to make commercially available neuromorphic applications using Nengo, was recently founded and probably makes extensive use of Nengo's model of BG as an action selection device.

1.3. Research plan

The new model was originally implemented using NEST, another simulator for spiking neural networks. By contrast with Nengo, NEST is not aimed to implement cognitive processes, but rather to study the dynamics of biological neural systems. The first thing to do will be to implement it in Nengo. With the latter, connections are usually automatically chosen in accordance with the computation that needs to be performed. However, Liénard and Girard [13] did not define their model on the functional level, but rather with specific connectivity and details of neural populations. Although Nengo was not originally designed to implement such bottom-up models, it is possible to specify direct connections and neuron characteristics. Implementing the model in Nengo will probably be the main difficulty of the project.

To check the validity of the implementation, action selection tests will be performed in both Nengo and NEST. Common approaches for evaluating action selection include using more or less noisy and similar inputs for commands, checking if the most active command is selected, and measuring the time necessary to perform this task. Both implementations should exhibit the same dynamics (in terms of timing and activation levels) with the same inputs. Comparisons between action selection performances of both the new model and Nengo's implementation of Gurney et al. [10] will also be made. Nengo's implementation of the new model will also need to exhibit the same oscillations as NEST's implementation when the effects of Parkinsonian dopamine depletion are simulated. The oscillation index has been used to quantify the oscillations in the range 15-30 Hz from the activity of the neural populations of the model in pathological conditions. It is defined as follows:

$$OI = \frac{\int_a^b S(f)\, df}{\int_0^{F_{max}} S(f)\, df} \tag{1}$$

with a and b the limits of the frequency range of interest, F_{max} the maximal frequency of the spectrum, and $S(f)$ the frequency power function. Oscillation index has been shown to increase when the dopamine depletion is simulated with NEST and will need to be reproduced in Nengo (see Figure 5).

If the implementation is made properly, it should not be hard to switch between action selection modules in the cognitive architecture for the Tower of Hanoi task. A qualitative examination of errors may provide preliminary indications on the human-likeness of the new architecture. Comparisons of the delays between moves with both the new and old models (e.g. with mean square error or plots like Figure 3) will then be carried. Lastly, the behaviour of the architecture in Parkinsonian conditions will be compared with behavioural results from Schneider, J. S. [17] and Vakil et al. [23].

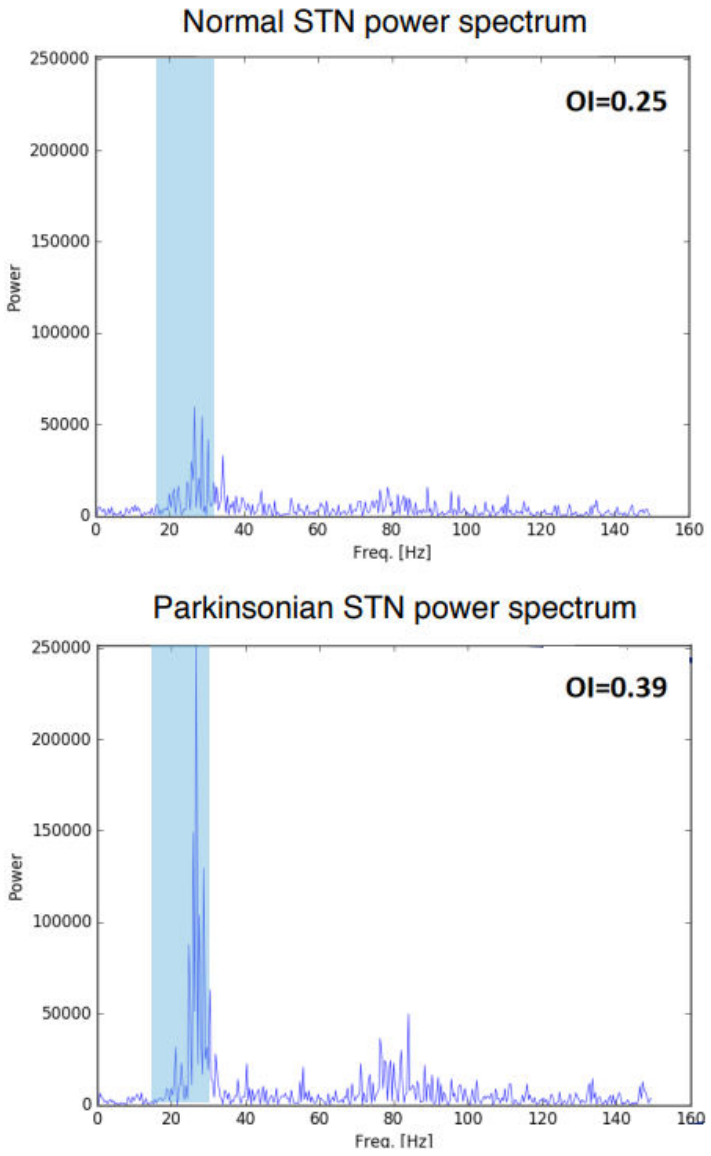


Figure 5: Parkinson-like oscillations (15-30 Hz) emerge in the STN when the dopamine depletion is simulated in the model. Adapted from [9].

Results of this study may provide insights to reduce the number of solutions of the model (see Section 1.1.2 for details about the 15 solutions resulting from the optimisation process).

2. Schedule

This research project will last approximately 21 weeks and will require a workload of 168 hours. It will be divided into three main phases, namely the setup, the implementation of the model in Nengo, and the use of the model within the architecture for solving the Tower of Hanoi (see Table 1). Project deliverables will be a report describing the study and the results, and the source code of the implementation of the biologically constrained model of the BG in Nengo. My supervisors, Serge Thill and Marcel van Gerven, will use these deliverables to evaluate my Capita Selecta of 6 EC.

#	Task	Start	Finish	Workload (h)
1	**Total**	**01/10/2018**	**25/02/2019**	**168**
2	**Setup**	**01/10/2018**	**21/10/2018**	
3	Install Nengo	01/10/2018	02/10/2018	1
4	Familiarise with Nengo	03/10/2018	21/10/2018	24
5	**sBCBG**	**22/10/2018**	**20/01/2019**	
6	Implement sBCBG in Nengo	22/10/2018	23/12/2018	71
7	Test action selection	24/12/2019	20/01/2019	16
8	Test PD oscillations	24/12/2019	20/01/2019	16
9	**sBCBG in TOH model**	**21/01/2019**	**25/02/2019**	
10	Get familiar with TOH model	21/01/2019	03/02/2019	16
11	Replace BG with sBCBG	04/02/2019	10/02/2019	8
12	Analyse results	11/02/2019	25/02/2019	16

Table 1: Dates of main tasks

3. Societal, technical, and scientific relevance

3.1. Treatments for Parkinson's disease

Parkinson's disease is the second most common neurodegenerative disease after Alzheimer's disease. Approximately 6 million men and women worldwide have it. The prevalence of the disease is expected to double in a lot of countries by the year of 2040, as a consequence of the increase of life expectancy. As it reduces movement control, Parkinson's disease impairs the individuals ability to function in daily life situations. Consequently, it affects the quality of life, social interactions, and the financial situation of these patients. Hence there is an urgent need to understand the biological underpinnings of this disease to better treat them.

3.2. Biological plausibility vs. explainable AI

Neuroscience-inspired artificial intelligence approaches such as deep learning currently have tremendous success. However, they face a big problem of interpretability. Evolution is obviously not constrained by explainability. As a matter of fact, the human brain contains approximately 6×10^{14} messy synaptic connections. The boundaries between what is needed for human-level intelligence, and what is just specific to the biological substrate are still far from being fully understood. Although the view that human action selection is performed with direct and indirect pathways can be appealing in its simplicity, it has been challenged by anatomical data (see Section 1.1.1). This study would provide an interesting framework to compare a simple and easily interpretable model, and a more accurate biologically constrained model of BG. I believe it could shed light on the necessity of biological plausibility for producing efficient artificial intelligent behaviour.

3.3. Interpretable and multidisciplinary evaluation of computational models

Researchers often regret the lack of bridges between disciplines. Although it is not always a trivial task, I believe that implementing computational models in cognitive architectures can provide useful and easily interpretable results. This method could permit the comparison of behavioural data from psychological studies and the dynamics of low-level models from

computational neuroscience. It can also be argued (for ethical, legal, and economic reasons) that simulations with cognitive architectures can be used as replacements for animal experiments.

4. Context and personal relevance

The present document is a research proposal for both AAPS and Capita Selecta courses (6 EC). The project is a follow-up to the internship I did at the Institute for Intelligent Systems and Robotics (ISIR) under the supervision of Benoit Girard in Paris. During this internship, I quantified the effect of dopamine depletion on the spiking version of the BG model proposed by Liénard and Girard [13]. I think this project will be a good approach for me to expand my knowledge on cognitive modelling with Nengo while using a model I am already familiar with.

References

[1] Aizman, O., Brismar, H., Uhlén, P., Zettergren, E., Levey, A. I., Forssberg, H., Greengard, P., Aperia, A., Mar 2000. Anatomical and physiological evidence for d1 and d2 dopamine receptor colocalization in neostriatal neurons. Nature Neuroscience 3, 226 EP –, article.
URL http://dx.doi.org/10.1038/72929

[2] Albin, R. L., Young, A. B., Penney, J. B., 1989. The functional anatomy of basal ganglia disorders. Trends in Neurosciences 12 (10), 366 – 375.
URL http://www.sciencedirect.com/science/article/pii/016622368990074X

[3] Aubert, I., Ghorayeb, I., Normand, E., Bloch, B., 2000. Phenotypical characterization of the neurons expressing the d1 and d2 dopamine receptors in the monkey striatum. Journal of Comparative Neurology 418 (1), 22–32.
URL https://onlinelibrary.wiley.com/doi/abs/10.1002/%28SICI%291096-9861%2820000228%29418%3A1%3C22%3A%3AAID-CNE2%3E3.0.CO%3B2-Q

[4] Bekolay, T., Bergstra, J., Hunsberger, E., DeWolf, T., Stewart, T., Rasmussen, D., Choo, X., Voelker, A., Eliasmith, C., 2014. Nengo: a python tool for building large-scale functional brain models. Frontiers in Neuroinformatics 7, 48.
URL https://www.frontiersin.org/article/10.3389/fninf.2013.00048

[5] Chateau-Laurent, H., 2018. Comportement dynamique d'un modele a potentiels d'action des ganglions de la base, en conditions normale et pathologique (parkinson).
URL https://www.researchgate.net/publication/328857664_Comportement_dynamique_d'un_modele_a_potentiels_d'action_des_ganglions_de_la_base_en_conditions_normale_et_pathologique_Parkinson

[6] DeWolf, T., Eliasmith, C., 2013. A neural model of the development of expertise. In: 12th international conference on cognitive modelling.

[7] Eliasmith, C., Stewart, T. C., Choo, X., Bekolay, T., DeWolf, T., Tang, Y., Rasmussen, D., 2012. Supplementary material for a large-scale model of the functioning brain. Science 338 (6111), 1202–1205.
URL http://science.sciencemag.org/content/sci/suppl/2012/11/28/338.6111.1202.DC1/1225266.Eliasmith.SM_revised.pdf

[8] Girard, B., Heraiz-Bekkis, D., Doya, K., 2017. A spiking model of the monkey basal ganglia without segregated pathways, but with emerging selection capabilities and parkinson-like oscillations. In: Seventh International Symposium on Biology of Decision Making. Bordeaux, France, poster.

[9] Girard, B., Linard, J., Chateau Laurent, H., Doya, K., 2018. Selection and oscillations in a spiking model of the basal ganglia. In: Neuralnet2018 - Understanding neural networks : from dynamics to functions. Paris, poster.

[10] Gurney, K., Prescott, T. J., Redgrave, P., May 2001. A computational model of action selection in the basal ganglia. i. a new functional anatomy. Biological Cybernetics 84 (6), 401–410.
URL https://doi.org/10.1007/PL00007984

[11] Kawaguchi, Y., Wilson, C., Emson, P., 1990. Projection subtypes of rat neostriatal matrix cells revealed by intracellular injection of biocytin. Journal of Neuroscience 10 (10), 3421–3438.
URL http://www.jneurosci.org/content/10/10/3421

[12] Lévesque, M., Parent, A., 2005. The striatofugal fiber system in primates: A reevaluation of its organization based on single-axon tracing studies. Proceedings of the National Academy of Sciences 102 (33), 11888–11893.
URL http://www.pnas.org/content/102/33/11888

[13] Liénard, J., Girard, B., Jun 2014. A biologically constrained model of the whole basal ganglia addressing the paradoxes of connections and selection. Journal of Computational Neuroscience 36 (3), 445–468.
URL https://doi.org/10.1007/s10827-013-0476-2

[14] Lienard, J. F., Cos, I., Girard, B., 2017. Beta-band oscillations without pathways: the opposing roles of d2 and d5 receptors. bioRxiv.
URL https://www.biorxiv.org/content/early/2017/07/17/161661

[15] Nadjar, A., Brotchie, J. M., Guigoni, C., Li, Q., Zhou, S.-B., Wang, G.-J., Ravenscroft, P., Georges, F., Crossman, A. R., Bezard, E., 2006. Phenotype of striatofugal medium spiny neurons in parkinsonian and dyskinetic nonhuman primates: A call for a reappraisal of the functional organization of the basal ganglia. Journal of Neuroscience 26 (34), 8653–8661.
URL http://www.jneurosci.org/content/26/34/8653

[16] Parent, A., Hazrati, L.-N., 1995. Functional anatomy of the basal ganglia. ii. the place of subthalamic nucleus and external pallidium in basal ganglia circuitry. Brain Research Reviews 20 (1), 128 – 154.
URL http://www.sciencedirect.com/science/article/pii/016501739400008D

[17] Schneider, J. S., 2007. Behavioral persistence deficit in parkinson's disease patients. European Journal of Neurology 14 (3), 300–304.

[18] Senft, V., Stewart, T. C., Bekolay, T., Eliasmith, C., Kröger, B. J., 2018. Inhibiting basal ganglia regions reduces syllable sequencing errors in parkinson's disease: A computer simulation study. In: Front. Comput. Neurosci.

[19] Stewart, T. C., Choo, F.-X., Eliasmith, C., 2012. Spaun: A perception-cognition-action model using spiking neurons. In: CogSci.

[20] Stewart, T. C., Choo, X., Eliasmith, C., 2010. Dynamic behaviour of a spiking model of action selection in the basal ganglia neural structure. In: 10th International Conference on Cognitive Modeling.

[21] Stewart, T. C., Eliasmith, C., 2011. Neural cognitive modelling: A biologically constrained spiking neuron model of the tower of hanoi task. In: CogSci.

[22] Tripp, B., 2009. A search for principles of basal ganglia function. Ph.D. thesis.
URL http://hdl.handle.net/10012/4179

[23] Vakil, E., Hassin-Baer, S., Karni, A., 2014. A deficit in optimizing task solution but robust and well-retained speed and accuracy gains in complex skill acquisition in parkinsons disease: Multi-session training on the tower of hanoi puzzle. Neuropsychologia 57, 12 – 19.
URL http://www.sciencedirect.com/science/article/pii/S0028393214000463

[24] Wu, Y., Richard, S., Parent, A., 2000. The organization of the striatal output system: a single-cell juxtacellular labeling study in the rat. Neuroscience Research 38 (1), 49 – 62.
URL http://www.sciencedirect.com/science/article/pii/S0168010200001401

II Midterm presentation

Assessing the need for biological plausibility in cognitive modelling

a focus on basal ganglia without segregated pathways

What is at stake?

- Is biological plausibility needed for AI and cognitive modelling?

  → compare the results of a simple vs more detailed model

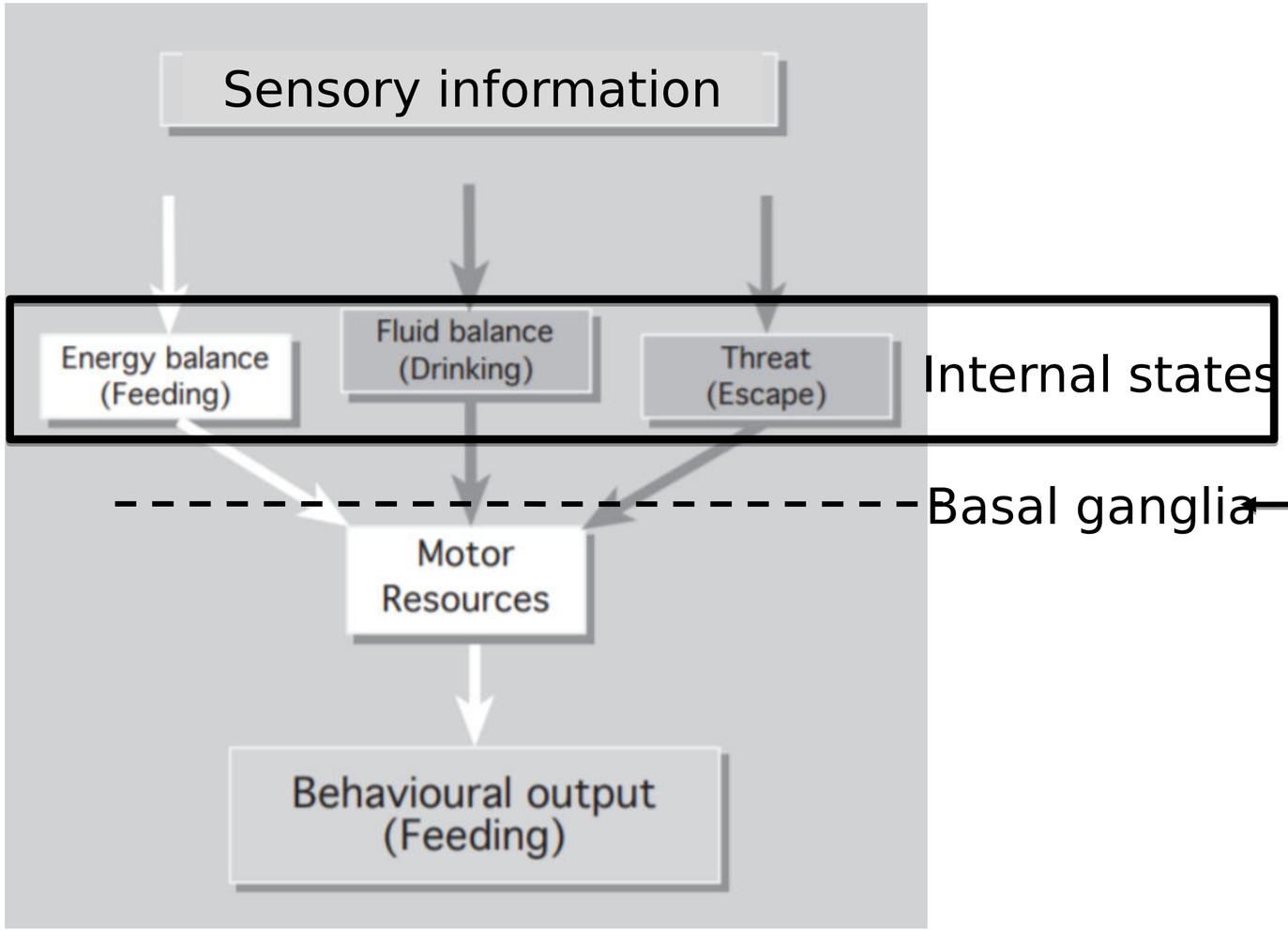
- How to interpret the evaluation of computational models?

  → use the models in a cognitive architecture that performs a naturalistic task

- How to model the relations between Parkinson’s disease and high-level cognition?

  → simulate it within the cognitive architecture

Focus on basal ganglia



Internal states

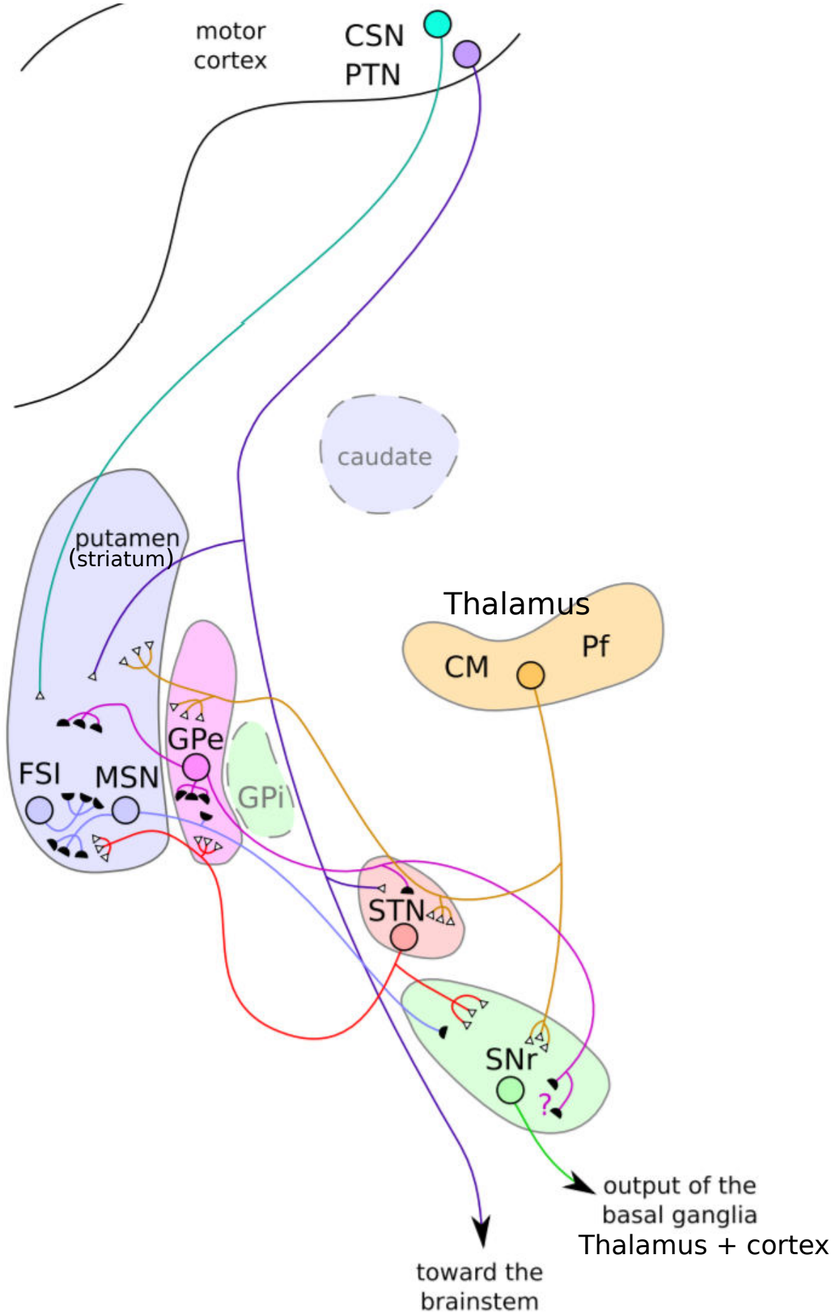
Basal ganglia ← Dopamine modulation (RL)

Focus on basal ganglia

- Inputs: cortex → Striatum
- Dopaminergic input: SNc Striatum (Parkinson’s disease)
- GPe
- STN
- Outputs: GPi/SNr → Thalamus → Cortex

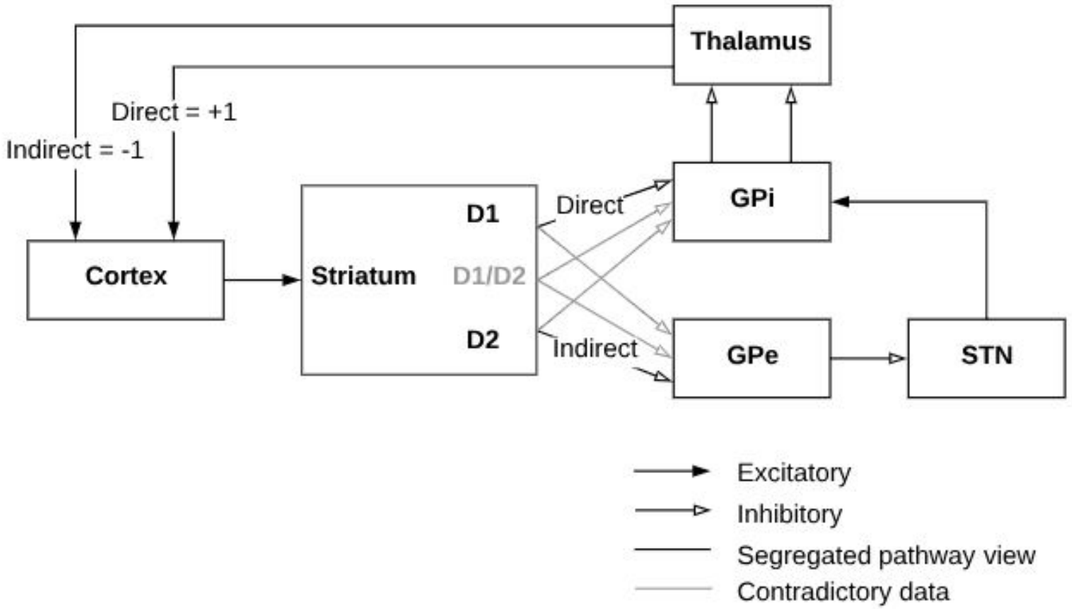
Anatomy of basal ganglia

- Inputs: cortex -> Striatum
- Computation in intrinsic nuclei:
  - GPe
  - STN
- Outputs: GPi/SNr -> Thalamus -> Cortex



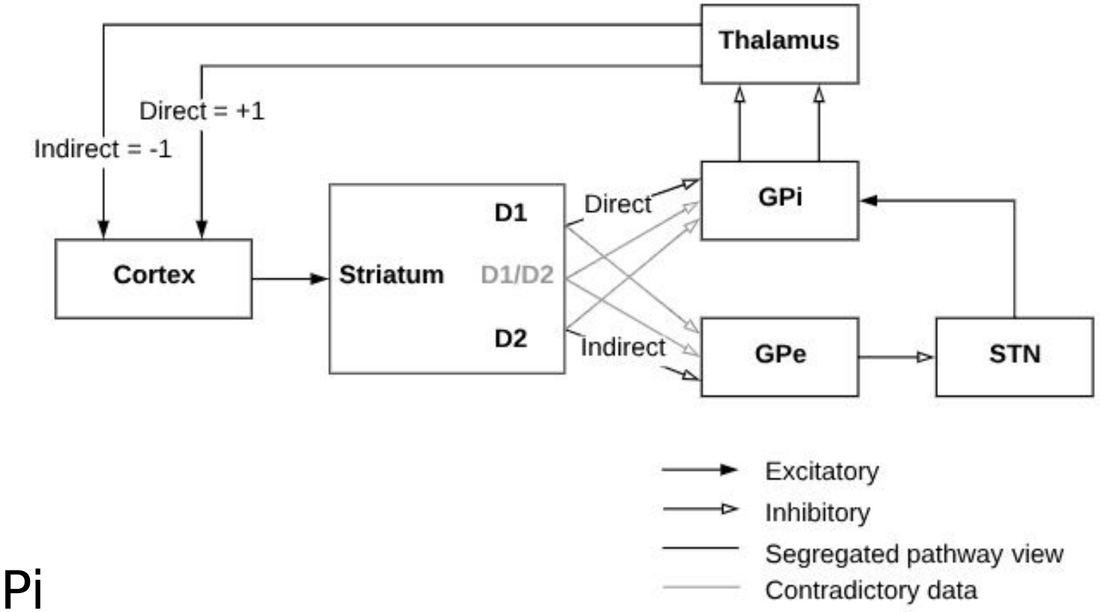
The myth of segregated pathways

- Canonical model: Albin et al. (1989) contains segregated pathways
  - Direct pathway: selects best action
  - Indirect pathway: inhibit concurrent actions
- Contradicted by anatomical data



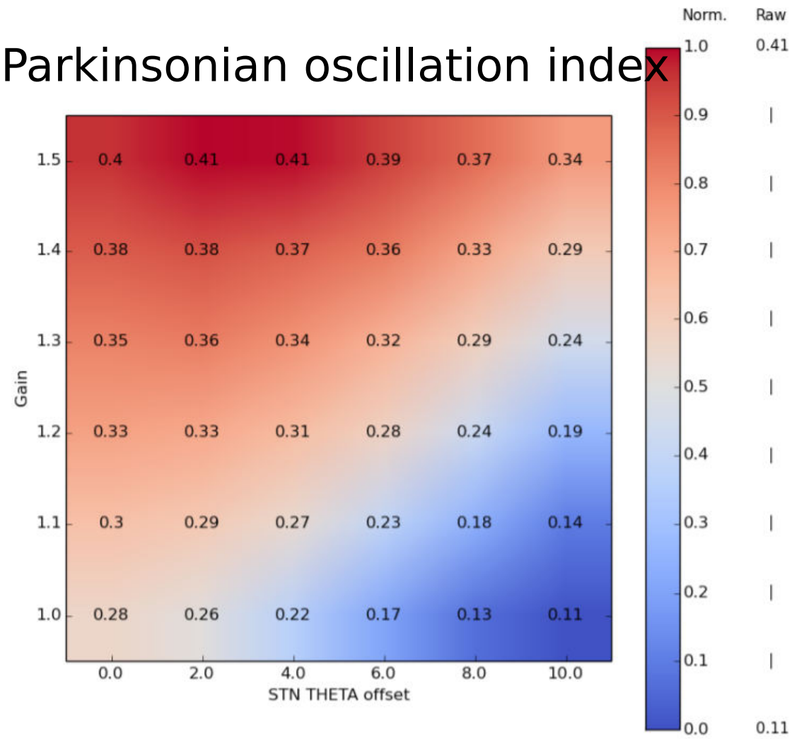
The myth of segregated pathways

- Canonical model: Albin et al. (1989) contains segregated pathways
  - Direct pathway: selects best action
  - Indirect pathway: inhibit concurrent actions
- Contradicted by anatomical data:
  - Almost every Striatum's MSN → both GPi and Gpe in both primates and rats
  - Up to 60% of MSN express both D1 and D2



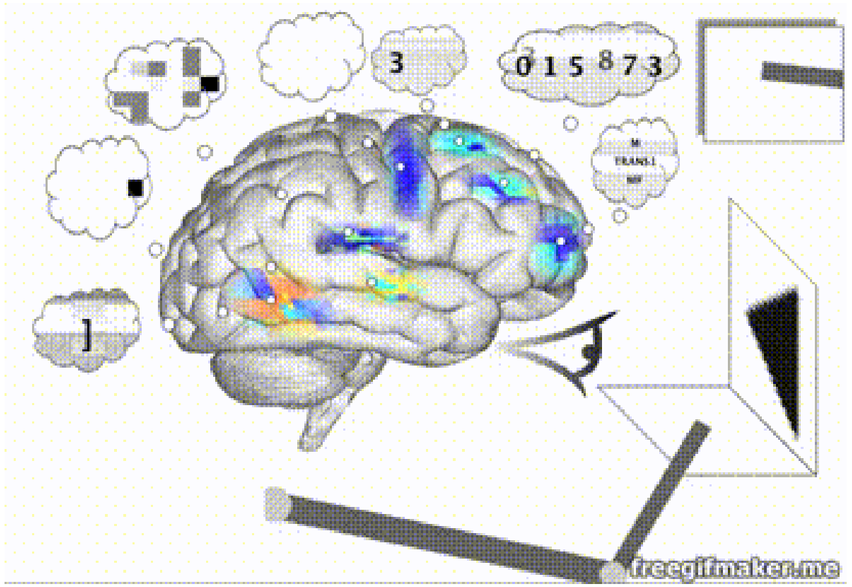
Biologically constrained model of Basal Ganglia (BioBG)

- Takes into account contradictory data
- Still capable of action selection
- Can exhibit parkinsonian behavior



Project outline

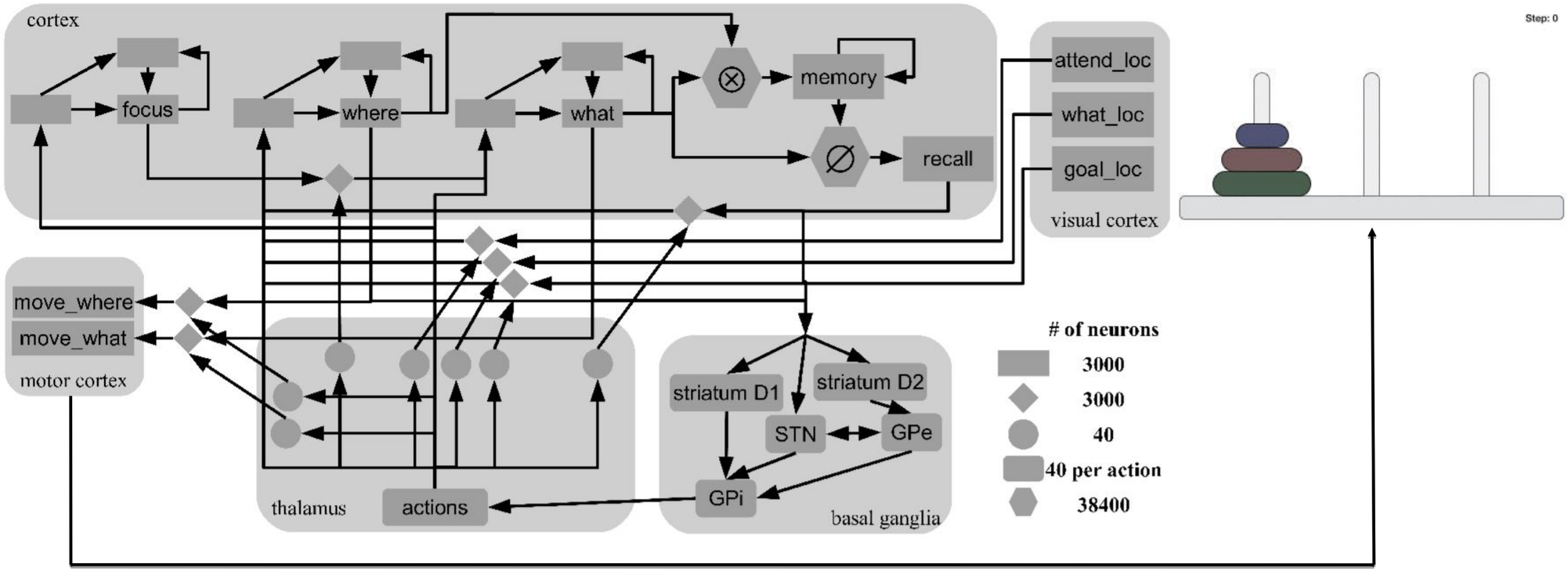
1. Implementation BioBG in Nengo, a simulator of spiking neurons for cognitive modelling
2. Find a Nengo project which uses a BG model to solve a task, and use BioBG instead



Taken from Stewart et al. (2012)

A neural model of the Tower of Hanoi Task

- Visual input/Motor output
- Cortical states
- Basal ganglia to choose next cortical states
- Thal

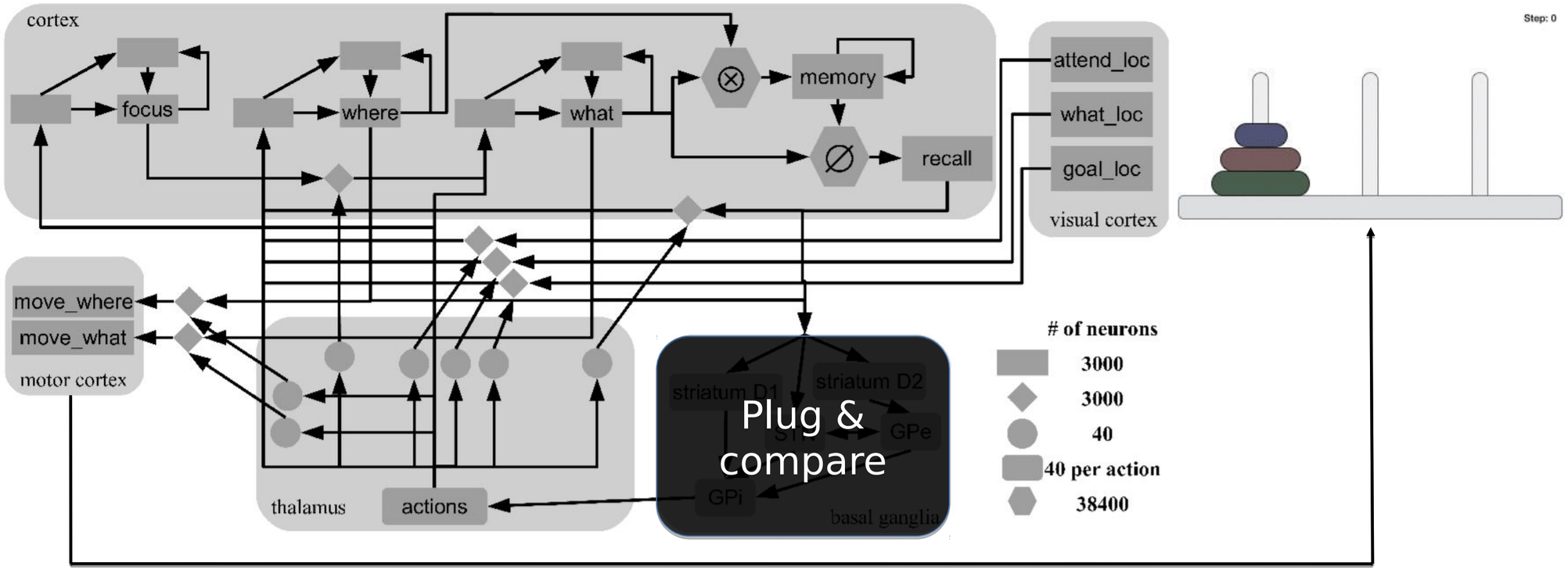


Adapted from Stewart and Eliasmith (2011)

Simple and general implementation → plug & compare

- Arbitrary number of actions
- No external dependency
- No modification to Nengo's source code

→ can l



Hypothesis 1: BioBG is more efficient

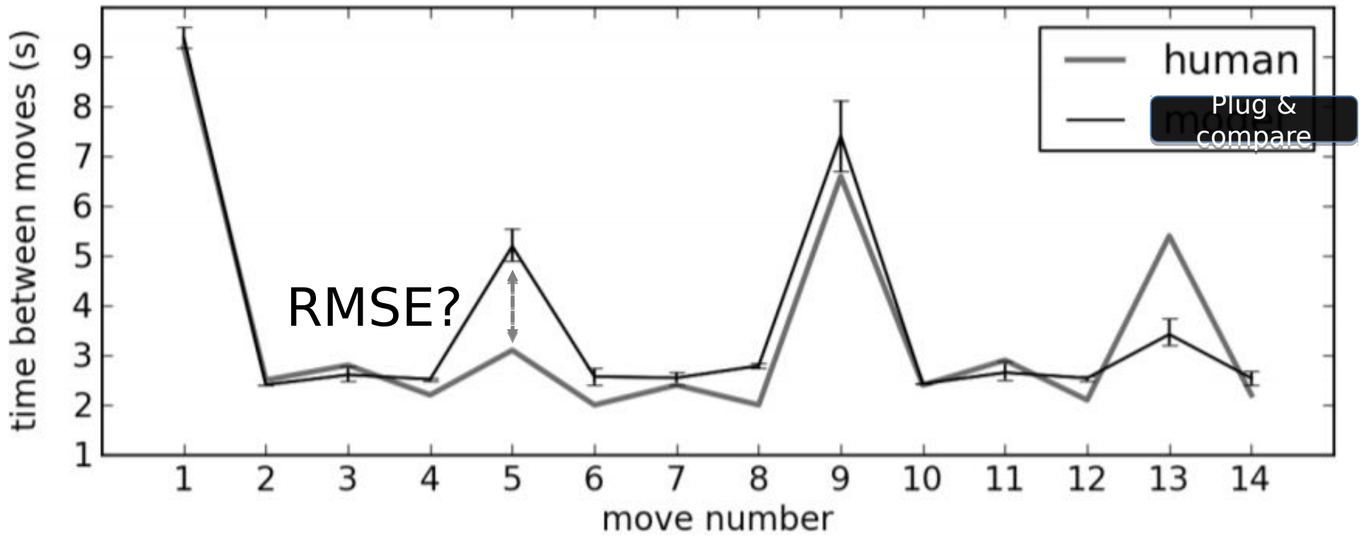
Action selection:

- Accuracy
- Speed

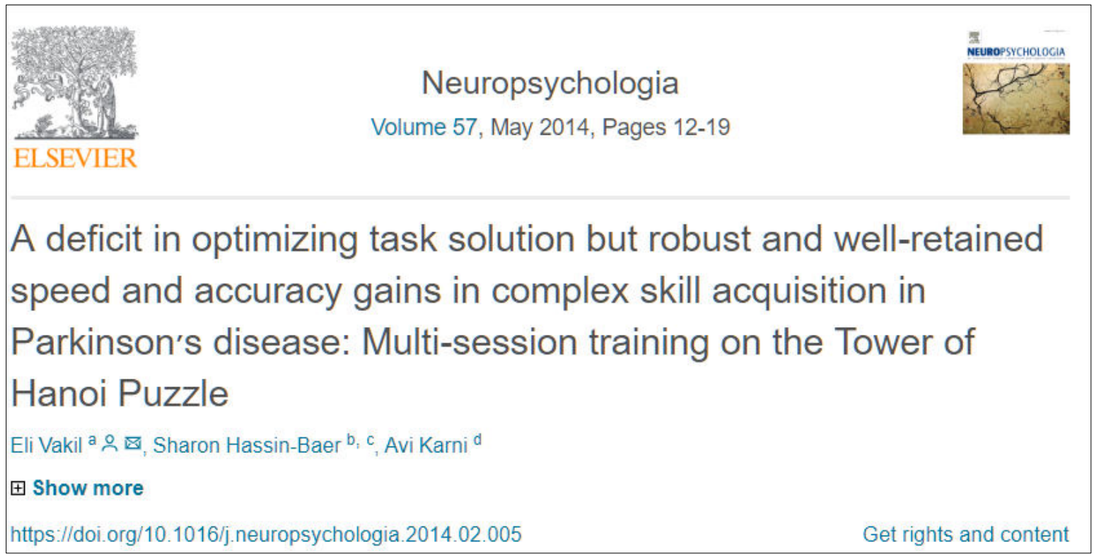
Hypothesis 2: behavioral data fitting

BioBG better fits

human data?



Hypothesis 3 (longer-term): BioBG+cognitive model can fit pathological behavioral data



ELSEVIER

Neuropsychologia

Volume 57, May 2014, Pages 12-19

A deficit in optimizing task solution but robust and well-retained speed and accuracy gains in complex skill acquisition in Parkinson's disease: Multi-session training on the Tower of Hanoi Puzzle

Eli Vakil [a], Sharon Hassin-Baer [b, c], Avi Karni [d]

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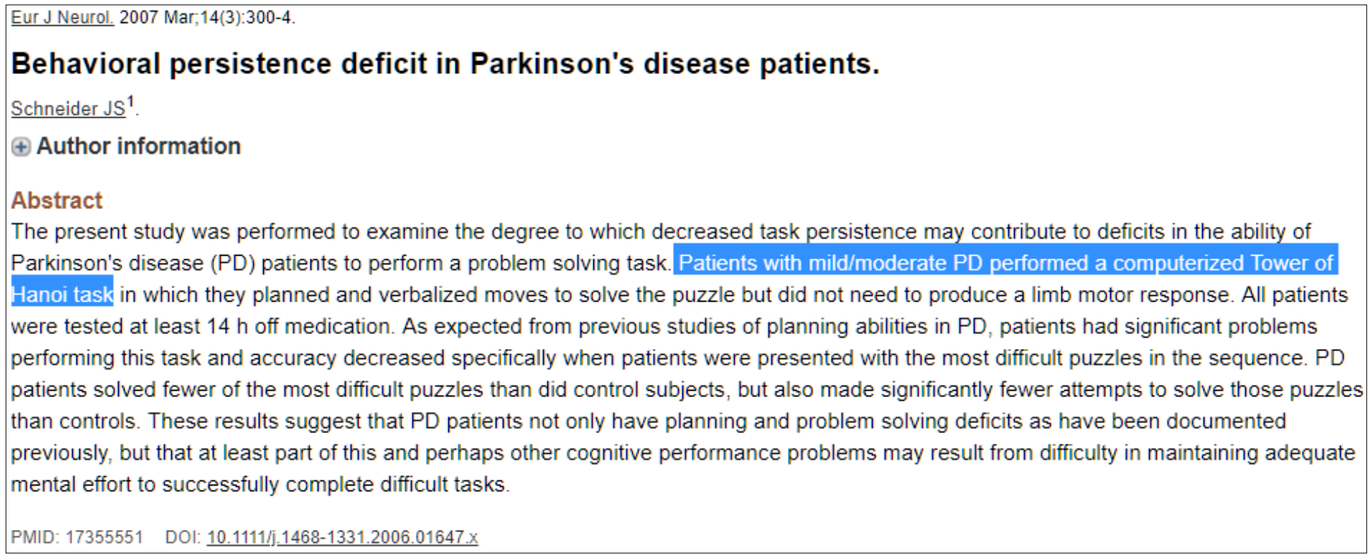


Eur J Neurol. 2007 Mar;14(3):300-4.

Behavioral persistence deficit in Parkinson's disease patients.

Schneider JS[1].

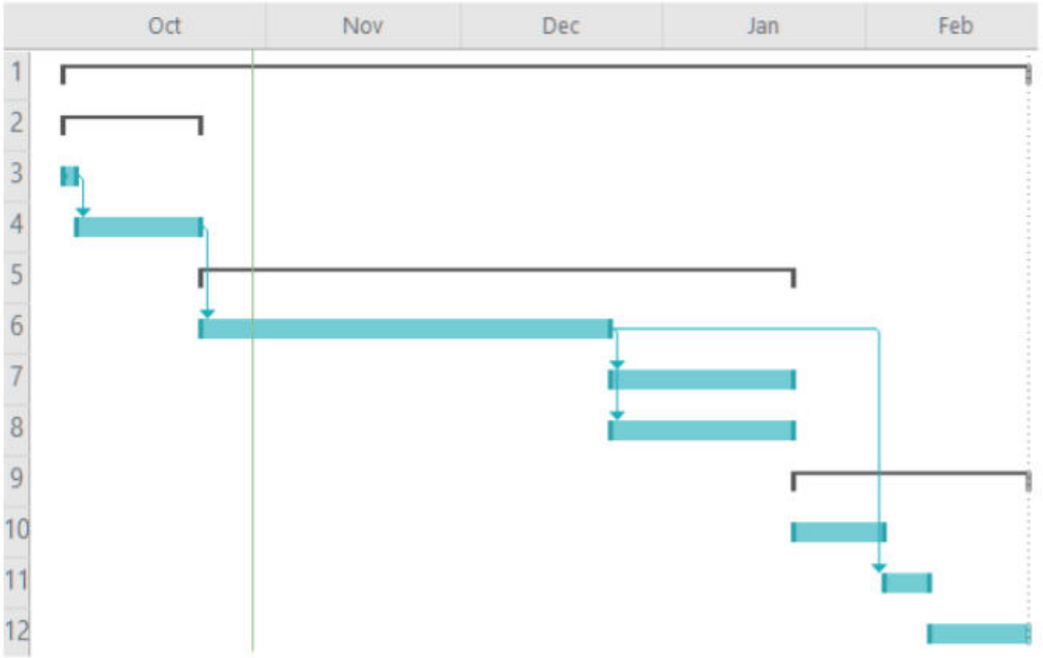
Author information

Abstract

The present study was performed to examine the degree to which decreased task persistence may contribute to deficits in the ability of Parkinson's disease (PD) patients to perform a problem solving task. Patients with mild/moderate PD performed a computerized Tower of Hanoi task in which they planned and verbalized moves to solve the puzzle but did not need to produce a limb motor response. All patients were tested at least 14 h off medication. As expected from previous studies of planning abilities in PD, patients had significant problems performing this task and accuracy decreased specifically when patients were presented with the most difficult puzzles in the sequence. PD patients solved fewer of the most difficult puzzles than did control subjects, but also made significantly fewer attempts to solve those puzzles than controls. These results suggest that PD patients not only have planning and problem solving deficits as have been documented previously, but that at least part of this and perhaps other cognitive performance problems may result from difficulty in maintaining adequate mental effort to successfully complete difficult tasks.

PMID: 17355551 DOI: 10.1111/j.1468-1331.2006.01647.x

Schedule



#	Task	Start	Finish	Workload (h)
1	**Total**	**01/10/2018**	**25/02/2019**	**168**
2	**Setup**	**01/10/2018**	**21/10/2018**	
3	Install Nengo	01/10/2018	02/10/2018	1
4	Familiarize with Nengo	03/10/2018	21/10/2018	24
5	**sBCBG**	**22/10/2018**	**20/01/2019**	
6	Implement sBCBG in Nengo	22/10/2018	23/12/2018	71
7	Test action selection	24/12/2019	20/01/2019	16
8	Test PD oscillations	24/12/2019	20/01/2019	16
9	**sBCBG in TOH model**	**21/01/2019**	**25/02/2019**	
10	Get familiar with TOH model	21/01/2019	03/02/2019	16
11	Replace BG with sBCBG	04/02/2019	10/02/2019	8
12	Analyze results	11/02/2019	25/02/2019	16

NB: “sBCBG”=“BioBG”

Take home message

I believe my project is a good framework for:

- comparing a **biologically plausible** model with a “**simpler**” one (Bottom-up vs Top-down)
- **Interpreting** the performance of **computational models**
- Studying the relations between **Parkinson’s** disease, **basal ganglia**, and high level **cognition**

III Poster

Do you need biological plausibility? A focus on basal ganglia

Hugo Chateau-Laurent
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Donders Institute
for brain, cognition and behaviour

Radboud University Nijmegen

Abstract

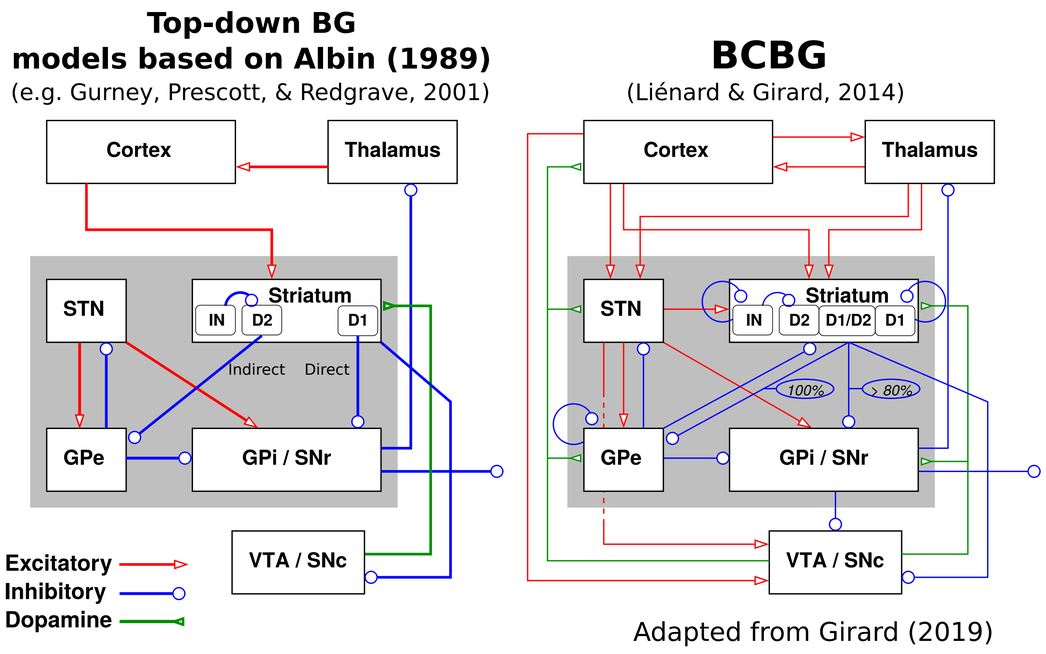
Two models of basal ganglia are used to study the relationship between biological plausibility, performance and interpretability in cognitive modelling. Some anatomical findings suggest a need to reevaluate the canonical theory of how basal ganglia may perform action selection. However, this theory is appealing in its simplicity and has given rise to efficient computational models. A more detailed model is compared to shed light on the need for biological plausibility.

Basal ganglia: function and circuitry

Basal ganglia are commonly thought to perform action selection. Albin (1989) has proposed that BG are divided in:

- **a direct pathway** that favors the action with the highest utility
- **an indirect pathway** that inhibits the execution of the competing actions

However, several findings suggest that the ouputs of the striatum are actually not segregated in primates. Liénard & Girard (2014) summarize the contradictory evidence and propose a new biologically constrained model of basal ganglia (BCBG) that has emergent selection properties.

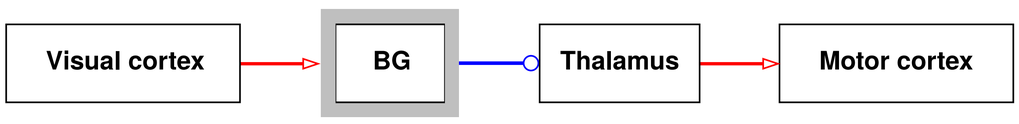


Adapted from Girard (2019)

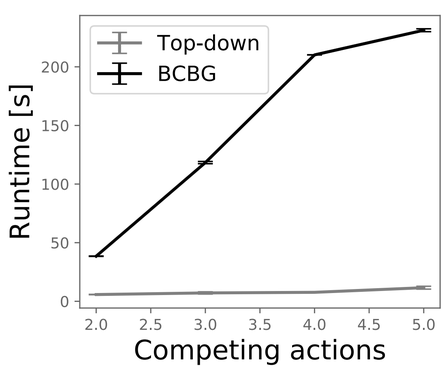
Motivation

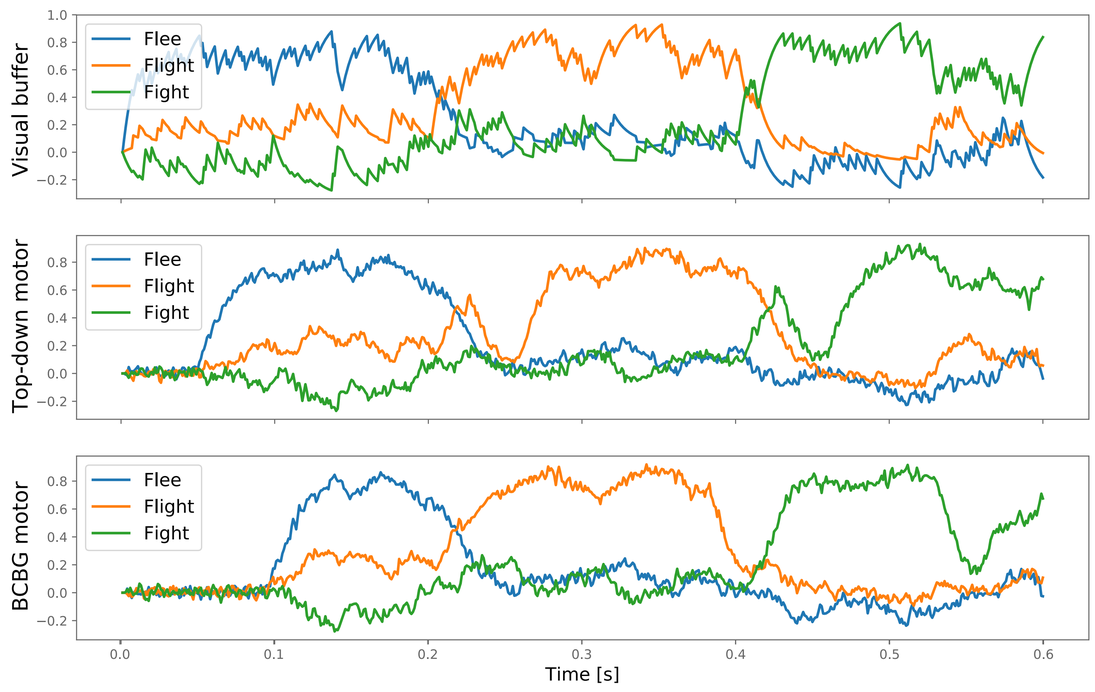
Biological plausibility is an important criterion for evaluating models in neuroscience, while artificial intelligence is more focused on efficiency for enginering ends. However, simplicity is a common goal. In neuroscience, simple and general principles are more elegant than the "bag of tricks" hypothesis and can potentially unify multiple subfields. Simplicity can also be the key to solve the interpretability concern in artificial intelligence. Neural networks are black boxes and their application in high risks domains such as autonomous driving, robotics or law is therefore limited. In these domains, agents must be able to give full account of their actions through the interpretability of their decision making process. A simple theory of how humans make decisions would be beneficial to both fields.

Action selection task

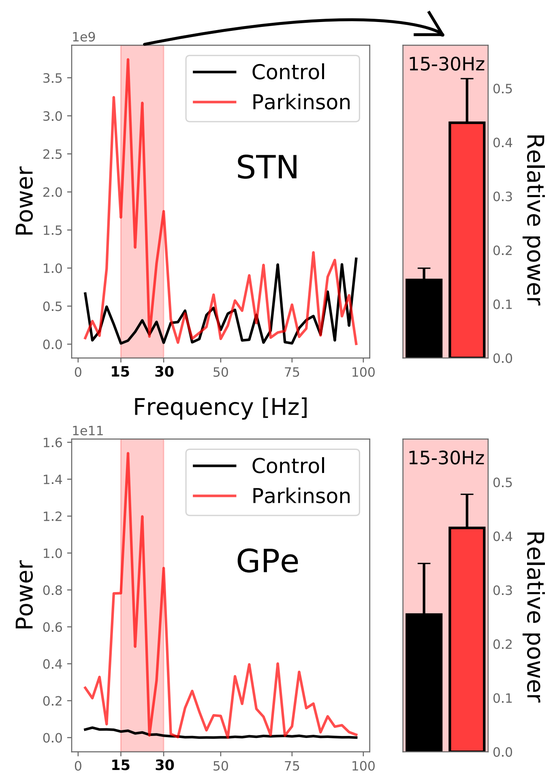


The simulations have been performed in Nengo. A visual buffer is fed a stimulus that should either drive a flee, fight or flight motor response. The visual buffer is connected to the striatum through corticostriatal neurons. The GPi/SNr nuclei serve as output of basal ganglia and project to the thalamus which itself projects to a motor buffer. BCBG is compared to the model of Gurney et al. (2001).





Parkinson's disease



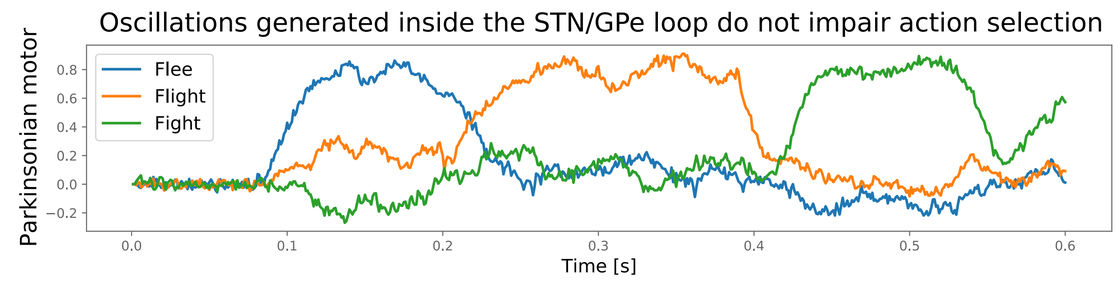
Parkinson's disease is characterized by:

- the death of dopaminergic neurons in the VTA and the SNc
- beta oscillations in the BG due to the lack of dopamine

Liénard et al. (2017) have shown that beta oscillations emerge when the dopamine is reduced in the STN-GPe loop only, while most studies focus on the depletion in the striatum.

However, it is not yet clear how the disease impairs motor control.

Oscillations generated inside the STN/GPe loop do not impair action selection



For engineering purposes, it seems more reasonable to use the model proposed by Gurney et al. (2001) or an even simpler Winner-Takes-All system. It is worth noting that BCBG is totally agnostic concerning the function of basal ganglia. Liénard & Girard (2014) have constrained the model on anatomical data only, and the selection capacities are ermergent. Therefore, BCBG will not be obsolete in case of paradigm shift. On the other hand, Gurney et al. (2001) followed a top-down approach to constrain their model with the assumption that basal ganglia perform action selection with segregated pathways.

References

Albin, R. L., Young, A. B., Penney, J. B., 1989. The functional anatomy of basal ganglia disorders. Trends in Neurosciences 12 (10), 366 – 375.

Benoît Girard.. 2019. YABG_ERC.pdf. DOI:https://doi.org/10.6084/m9.figshare.5418766.v1

Gurney, K., Prescott, T., & Redgrave, P. (2001). A computational model of action selection in the basal ganglia. Biological Cybernetics 84, 401-423.

Lienard, J., Girard, B., Jun 2014. A biologically constrained model of the whole basal ganglia addressing the paradoxes of connections and selection. Journal of Computational Neuroscience 36 (3), 445–468.

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