Decisions and Downward Causation in Neural Systems

Azadeh Hassanejad Nazir[1] and Hans Liljenström[1,2]

[1] Dept. of Energy and Technology, SLU, P.O. Box 7032, 750 07 Uppsala, Sweden
[2] Agora for Biosystems, P.O. Box 57, SE-19322 Sigtuna, Sweden
hans.liljenstrom@slu.se

Abstract. For any complex system, consisting of several organizational levels, the problem of causation is profound. Usually, science considers upward causation as fundamental, paying less or no attention to any downward causation. This is also true for the nervous system, where cortical neurodynamics, and higher mental functions are normally considered causally dependent on the nerve cell activity, or even the activity at the ion channel level. This study presents a computational approach to decision making and downward causation in cortical neural systems. We have developed models of paleo- and neocortical structures, in order to study their *mesoscopic* neurodynamics, as a link between the *microscopic* neuronal and *macroscopic* mental events and processes. We demonstrate how both noise and chaos may play a role for the functions of cortical structures. While microscopic random noise may trigger meso- or macroscopic states, the nonlinear dynamics at these levels may also affect the activity at the microscopic level.

Keywords: Decision making, free will, mesoscopic neurodynamics, causation.

1 Introduction

Decision making is perhaps the most important of our cognitive processes related to behavior, and is crucial for survival of all higher animals. In humans, conscious decision making is linked to a sense of free will. Normally, when we decide to act in a certain way, when we choose one of several options, we take for granted that we can do that out of free will, i.e. without any constraints or influences extrinsic to our conscious self. If our willful acts should have any evolutionary effects, our physical body must be under the influence of our consciousness, which is an example of downward (top-down) causation, where a higher level of reality has a causal power over lower levels.

In fact, most of our conscious human behavior and actions, including science, presupposes free will, which yet often is considered an "illusion" in present day science [1,2]. George Ellis [3] expresses this as: "All scientific experiments are based on purposeful activity and free will, enabling decisions based in abstract analysis that lies beyond the explanatory scope of physical science". He also emphasizes that "bottom-up, same-level, and top-down causation all occur at the same time, in concert, enabling the emergence of genuine complexity based in modular hierarchical systems", a point also made by Dennis Noble [4].

For a nervous system, as for complex systems in general, different phenomena appear at different levels of aggregation. Emergent phenomena may result from a "bottom-up" causation, based on *micro* level phenomena. Yet, higher *macro* levels may also "control" lower ones (c.f. the so-called enslaving principle of Haken [5]). This interplay between micro and macro levels is part of what frames the dynamics of neural systems. Of special interest is the *meso* level, i.e. the level in between the micro and the macro, as this is where bottom-up meets top-down [6].

Mesoscopic brain dynamics is partly a result of a dynamic balance between opposing processes such as inhibition and excitation, which often results in oscillatory and chaotic-like behaviour [6]. Yet, this dynamics is mixed with noise, generated at a microscopic level by spontaneous neural activity. It is also affected by macroscopic activity, such as slow rhythms generated by cortico-thalamic circuits or neuromodulation from different brain regions. Effects of arousal, attention, or mood, through neuromodulation or other means, could be seen as a top-down interaction from macroscopic activity to mesoscopic neurodynamics.

We address this issue, with the aim of elucidating the causal pathways in brain dynamics, where downward causation from larger to smaller scales could be regarded as evidence that multi-level 'both-way' causation occurs. We concur that downward causation is necessary but not sufficient for demonstrating the existence of free will. In order to demonstrate downward causation, one has to show that a change in some high level variable(s) reliably results in a change in lower-level variable(s).

We use computational models of different brain structures to investigate how cortical neurodynamics may depend on structural properties, such as connectivity and neuronal types, and on intrinsic and external signals and fluctuations. We also investigate to what extent the complex neurodynamics of cortical networks can influence the neural activity of single or populations of neurons. We have previously shown how neuromodulation and attention can synchronize and in other ways "control" the activity of neurons and neural populations in networks [7,8]. In this work, we develop a neural network model of the three most prominent brain structures involved in decision making, and study the chains of causation involved. Our results are suggestive for the neural basis for emotional and rational decision making, also aiming at a greater understanding of the interplay between different brain structures in neural information processing.

2 Methods

We propose a neural network model, which attempts to describe an adaptive decision making process (DM), under varying internal and external contexts. The model includes the three most crucial structures in DM: amygdala, orbitofrontal cortex (OFC) and lateral prefrontal cortex (LPFC). These neural regions are involved in both emotional/intuitive and rational/cognitive aspects of DM [9-12].

The DM is modeled at a level of mesoscopic neurodynamics, using attractor neural networks [13]. Oscillatory rhythms encode information related to perception, cognition and emotional associations in this model. The emerged oscillatory activities are the result of interactions between neural populations. The network activity can be envisioned as local field potentials (LFP) or electroencephalogram (EEG) readouts. Each unit in our model represents a group of neurons firing in synchrony. The oscillatory network behavior is a result of the interaction between excitatory and inhibitory neural groups, as described in more detail in [13]. The general structures of these three systems and their interconnections are shown in Fig 1, whereas the more detailed network structure is shown in Fig. 2.

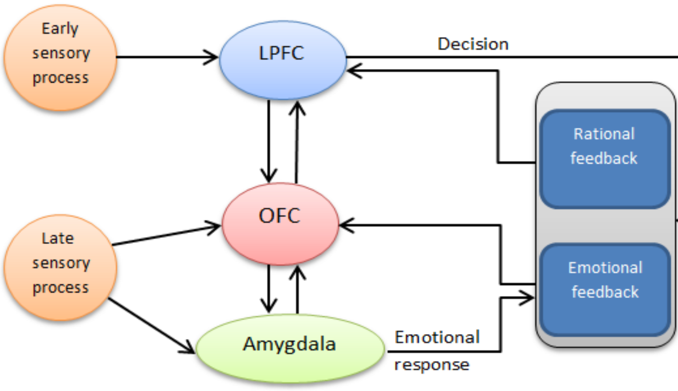

Fig. 1 A representation of the interactions of the three main neural structures in the decision making process. Amygdala and orbitofrontal cortex (OFC) are considered as the main organizations in emotional decision making and lateral prefrontal cortex (LPFC) plays a crucial role in rational analysis. The inputs come from all sensory modalities.

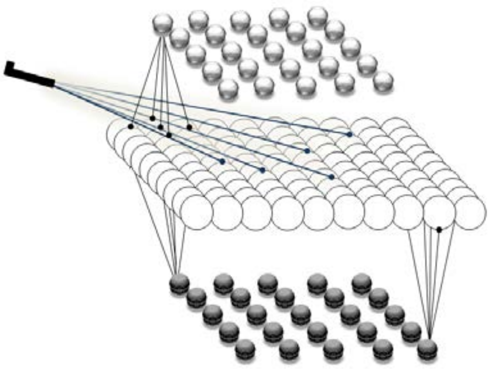

Fig. 2. This figure represents three neural structures. The upper and lower layers are composed of 25 inhibitory neurons and the network in the middle is composed of 100 excitatory neurons. The external inputs stimulate (subset of) excitatory neural network. The stimulation of excitatory neurons is the start of activity of the system. Stimulated excitatory neurons excite inhibitory neurons which result in the excitation-inhibition balance.

The excitatory sub-layer of each model structure consists of a network of 100 neural units (populations), while each of the two inhibitory networks is composed of 25 inhibitory units. The excitatory network nodes are connected recurrently, while there is no internal connection among inhibitory network nodes. An excitatory-inhibitory balance results from the bidirectional connectivity of excitatory units with the two inhibitory networks on either side (see Fig. 2). The network structure allows for a complex neurodynamics, in particular oscillations with varying amplitudes and frequencies. The oscillatory properties are characteristic for each structure, where the activity of each neural unit can be regarded as the mean membrane potential of the population resulting in a graded, rather than spiking, neuronal output.

External stimuli are driven by afferent neurons (not explicitly modeled) to a subset of the network of excitatory units. The difference between inputs to the emotional systems and rational one is modeled by different magnitudes. Excited units transmit signals to their excitatory neighbors, as well as to neighboring inhibitory neurons, which provide controlling inhibition to all excitatory neurons.

The function of the three neural structures requires the formation and update of cell assemblies. The network oscillations of each cell assembly, *s*, is the representation of stored experiences, attitudes and associated feelings towards the consequence of choosing any of the present options as the ultimate decision. Any decision taken can be followed by different fallouts. The size of a cell assembly with *s* excitatory units constitutes one of the parameters that represent the significance of a given option. Internal and external stimuli (context), as well as magnitude and frequency of the oscillatory network activity are properties of the cell assembly activity important for option selection.

The intrinsic motivation, Q (from the input-output equation in [13], which can originate from either emotion or cognition, is another effectual variable. A larger value of Q indicates a higher motivation for choosing a particular option. Motivation is a function of an individual's experiences and environmental factors, but is for the sake of simplicity, currently modeled with a constant value.

Accordingly, size of cell assemblies, properties of stimuli and motivation are functions affecting the predicted value of each option. As a result, the total value of each option is the product of the mentioned parameters as below. $\boldsymbol{f}$ and $\boldsymbol{A}$ are the matrices of oscillatory frequencies and oscillation amplitude, respectively. Rows of these matrices show the population of the cell assembly and columns represents the duration of oscillatory activity. Averaged values of neural activities of all units over a time interval ΔT, $\overline{\boldsymbol{f}}$ and $\overline{\boldsymbol{A}}$, are computed. The dot product of two vectors, times the number of cell populations active in the cell assembly, $|s|$,results in the value of any option.

$$V(option) = \left|s_{opt}\right| \cdot \langle \overline{\boldsymbol{f}_{opt}}, \overline{\boldsymbol{A}_{opt}} \rangle = \left|s_{opt}\right| \cdot \langle \overrightarrow{f_{opt}}\,,\ \overrightarrow{A_{opt}} \rangle, \quad \forall\, opt = 1, \ldots, n$$

The value for each option should be computed in all of the three neural structures. V_{OFC} , V_{Amy} and V_{LPFC} are the vectors of alternatives' values which are the basis of emotional and cognitive values. It is important to remember that OFC and amygdala are structures involved in emotional valuation and LPFC is involved in cognitive valuation [11,14].

The emotional and rational decisions are based on the "winning" response activity. The competition between the stored patterns toward the cue can be determined with the help of cosine similarity of the frequency vectors $\vec{f}$. The highest value of V will win the competition, and result in a decision to choose that option. The ultimate decision is the resultant of the emotional and rational decisions.

3 Results

The choice of an action, which one of several options we will eventually decide upon, depends on various external and internal factors. We may have different preferences, depending on our general attitudes with regard to the options. Accordingly, each option has a total value that is a weighted sum of different types of values.

In our model, an individual's preferences/priorities are determined by the neural activity of cell assemblies in the three brain structures considered, amygdala, OFC and LPFC, which represent the attitude, expectancy value, and rules towards the outcome of a decision. Amygdala is considered to form emotional memories, and give an emotional response, depending on varying external and internal stimuli. Each option is represented by an active cell assembly. Positive and negative emotion is modeled as a level of "satisfaction", with five degrees of satisfaction used here, -2, -1, 0, +1, and +2. The emotion towards different options depends on external and internal contexts. Given the number of options, various contexts can be defined.

Based on objective and personal preference, different rational attitudes and rules govern cognition. A combination of declarative and procedural memories forms the basis of the rational analysis. The ultimate decision might be affected by the contingency of having the predicted outcome and the intensity/severity of different internal and external context. In the absence of external/internal context, the priority should be followed and the option with the highest priority would be chosen in each system.

In Fig. 3, the neural activity of cell assemblies in the emotional and cognitive system is illustrated. U_{ex}, U_{ff} and U_{fb} represent the mean neural activity of excitatory, feedforward inhibitory and feedback inhibitory networks. The top frames show the activities of stimulated (red oscillations) and non-stimulated(blue oscillations) neural units. The bottom frames illustrate the excitation-inhibition balance between the inhibitory networks (feedforward and feedback networks) and the excitatory units.

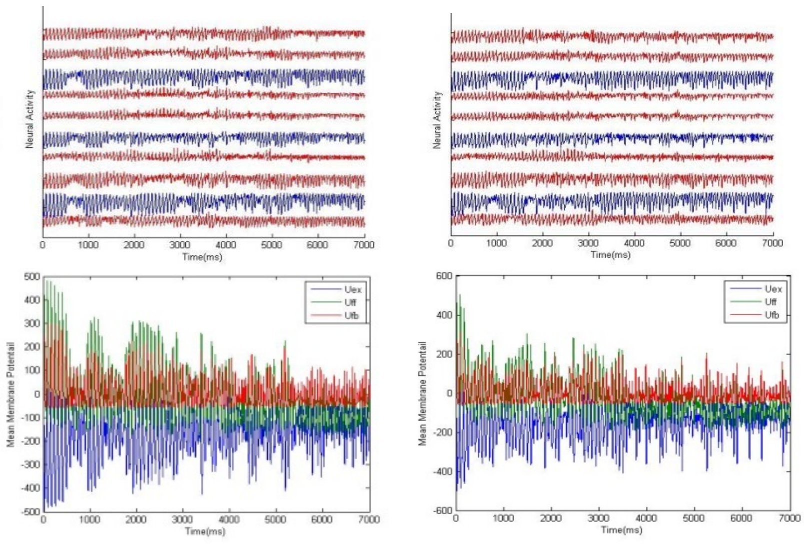

Fig. 3. The LPFC cell assembly activity changes as a result of different contexts (left, right). These activities represent two different options, as an outcome of the decision making process.

4 Discussion

The neural activity at the microscopic level of single neurons is the basis for the neurodynamics at a mesoscopic network level, and fluctuations may sometimes trigger coherent spatio-temporal patterns of activity at this higher level [15,16]. Oscillations and irregular chaotic-like behaviour can be generated by the interplay of neural excitatory and inhibitory activity at the network level [6,15]. This complex network dynamics, in turn, may influence the activity of single neurons, causing them to fire coherently or synchronously. Upward and downward causation are interdependent.

We have modelled a major part of the neural system involved in decision making, including the amygdala, the orbitofrontal cortex and the lateral prefrontal cortex. These systems represent emotional, as well as cognitive aspects of the decision making. With our model we have demonstrated how different cell assemblies, representing different optional choices available to the individual may compete with respect to level of activity. The winning assembly was simply the one with the largest neural activity, measured as the product of the three assembly characteristics (number, frequency, amplitude). The different options get different values, depending on internal (e.g. motivation, attitudes, values) as well as external (e.g. environment, social interaction) factors. For any particular (sensory) input signal, the final decision made by an individual may shift depending on internal and external context.

Apparently, our decisions are based on biological (genetic, neural, physiological), but also social and environmental factors, constituting a complex web of causation, making it hard or even impossible to predict a decision or action for any given individual. The unpredictable outcome could be seen as a result of random neural activity, or as a result of free will. In the first interpretation, upward causation could be an explanation, otherwise, downward causation. The question is if it is possible to experimentally settle the case, or if it will remain a matter of philosophical debate.

Acknowledgments
Grants from the EC-FP7 for COMPLEX, (Grant 308601) and from the John Templeton Foundation (Grant 39987) are gratefully acknowledged.

References

1. Wegner, D.M. (2003) The mind's best trick: how we experience conscious will. *Trends in Cognitive Sciences* **7**:65-69.
2. Soon, C. S., Brass, M., Heinze, H-J., & Haynes, J-D. (2008). Unconscious determinants of free decisions in the human brain. *Nature Neuroscience* **11** :543.
3. Ellis, G. (2009) Top-down causation and the human brain. In : *Downward Causation and the Neurobiology of Free Will* (Eds. Murphy, N., Ellis, G. & O'Connor, T.). Berlin: Springer, pp. 63-81.
4. Noble, D. (2012) A theory of biological relativity: no privileged level of causation, *Interface Focus* 2:55–64.
5. Haken, H. (1983) *Synergetics: An Introduction*. Berlin: Springer-Verlag.
6. Freeman, W.J. (2000) *Neurodynamics: An Exploration in Mesoscopic Brain Dynamics*, Berlin: Springer.

7. Liljenström, H. & Hasselmo, M.E. (1995) Cholinergic modulation of cortical oscillatory dynamics, *J. Neurophysiol*. **74**:288-297.
8. Gu, Y. & Liljenström, H. (2007) A neural network model of attention-modulated Neurodynamics, *Cogn. Neurodyn* **1**:275-285..
9. Whalen, P.J. & Phelps, E.A. (Eds.) (2009) The Human Amygdala. *The Guilford Press.*
10. Zald, D. (Ed.) Rauch, S. (Ed). (2006) The Orbitofrontal Cortex. *Oxford University Press*
11. Dixon, M.L. & Christoff, K.E. (2014) The lateral prefrontal cortex and complex value-based learning and decision making. *Neurosci Biobehav Rev*. 45C:9-18.
12. Roesch,M. & Schoenbaum,G. (2006) From associations to expectancies: orbitofrontal cortex as gateway between the limbic system and representational memory. In: Zald,D. (Ed.) Rauch,S.(Ed.) The Orbitofrontal Cortex. *Oxford University Press*
13. Liljenström, H. (1991) Modeling the dynamics of olfactory cortex using simplified network units and realistic architecture, *Int. J. Neur. Syst.* **2**:1-15.
14. Doya, K., (2008). Modulators of decision making, *Nature Neuroscience* **11**:410-416.
15. Århem, P. and H. Liljenström, *Fluctuations in Neural Systems: From Subcellular to Network Levels*, in *Handbook of Biological Physics*, F. Moss and S. Gielen, Editors. 2001, Elsevier: Amsterdam. pp. 83-129.
16. Basu, S. and Liljenström, H. (2001) Spontaneously Active Cells Induce State Transitions in a Model of Olfactory Cortex, *BioSystems* **63**:57-69.