Simulation of Ageing Using Venn-Networks

Fernando Buarque de Lima Neto
Polytechnic School of Engineering
University of Pernambuco
50.750-410 – Recife – PE, Brazil
fbln@upe.poli.br

Philippe De Wilde
Electrical and Electronic Engineering
Imperial College London
SW7 2BT – London, UK
p.dewilde@imperial.ac.uk

Abstract

This work presents simulations of ageing of a neo-cortical inspired tissue controlling virtual fingers by using Venn-network. This model is able to produce, simultaneously, (a) multi-localized activations in a 2-dimensional grid of artificial neurons and (b) flexion of virtual fingers. Another highlight of the model is the compositional manner, which it can be set up. That is, small patches of processing artificial regions can be assembled side-by-side and interconnected freely forming a 'patch-work' of competitive 2-dimensional network. The results of the aging simulations show that Venn-network, when appropriately configured, can exhibit and, more important, explain some physiologically observable decay in motor response due to neuronal death. Because of its architecture, the changes in behavior of the fingers are also accompanied by modifications in the pattern of activations of the neurons.

1. Introduction

A ubiquitous fact in biological systems is their functional decline due to ageing processes. These processes are not uncomplicated and often affect various granularities and constituent parts of the system. Ranging from sub-cellular to macroscopic changes, alterations (chiefly performance decline) are gradually more evident over time as the organism "grows old". The nervous system, unfortunately, is not immune to this very undesirable decay that results in idiosyncratic reduction of memory power, intellectual and physical abilities, as well as neuroendocrine alterations (*e.g.* affecting the mood and sleeping patterns) [1][3].

Although not aiming to model ageing processes as a whole, the objective of the present experiment is to investigate if an artificial neural network, namely Venn-network, could be able to implement brain function degradations similarly to what happens in ageing processes. The unpretentious paradigm selected here relates loss of computing power in simulated networks to (nervous) cell death, as Price [1] affirms that this fact is a common feature always present in ageing processes.

2. Venn-network – Overview

Venn-networks (short for Venn-like neural networks), is a neural inspired architecture proposed by Buarque [4,5] an used in this work for simulating aging processes. It creates a non-linear association between input and output spaces by utilizing a 2-dimensional (2D) map composed of processing elements (artificial neuron). Similar to cortical columns, each processing unit of the 2D map represents the average activity observed in the patch of the cortex being modeled. Each processing element can receive connections from all types of fibers of the model simultaneously.

Venn-network allows definition of arbitrary number of cyto-architectonical regions – that includes many processing elements; and arbitrary sets of inbound, outbound and interconnecting fibers. The processing regions are the main loci of neural signal processing. On the other hand, fibers carry these signals all around the architecture regions. Every fiber has associated to it a number of features such as *synaptic weights*, *delay and cardinality.* These features confer to each bunch of fibers very distinct behaviors. Fibers are referred according to their functionality, namely, (a) afferent as the ones specialized for incoming signal; (b) efferent as the ones specialized for outgoing signal; connections; and (c) u-fibers, the ones specialized in interconnecting regions. Figure 1 contains a schematic view of the architecture. In the figure the reader can observe all elements that make this kind of neural network so unique: different types of columns, different types of fibers, and different types and dimensions of regions.

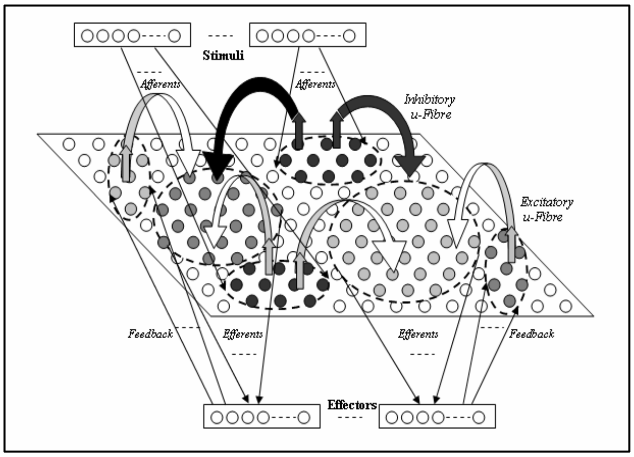


Figure 1. Schematic view of Venn-network architecture; *afferents*, *efferents (*including *feed-back)* and *u-fibers* (both inhibitory and excitatory) can be seen as well as regions, and external components: *stimuli* sources and *effectors*. Note that arrows indicate bunch of fibers that connect origin and target in a massively parallel manner.

3. Experimental set-up

3.1. Network structure

Investigations of ageing in this work are divided into two sets Sim2905A and Sim2905B. The first set considers that cell death happens uniformly distributed over the cortical map, and the second set assumes a more localized loss of nerve cells. Both sets use the same network structure; that is four regions and one pair of: stimuli sources, effectors, bundles of afferent, efferent, and efferent-feedback (see Figure 2). Synaptic values used here are unique and obtained from an *ad-hoc* training of the topology. Hence, there is no training involved in the current set of simulations. The rationale for this decision is justifiable by the fact that we want to measure output performances of networks submitted to ageing processes on top of well-trained and "known" topologies. Further experiments, not the objective of this work, could be carried out on re-training the aged Venn-networks.

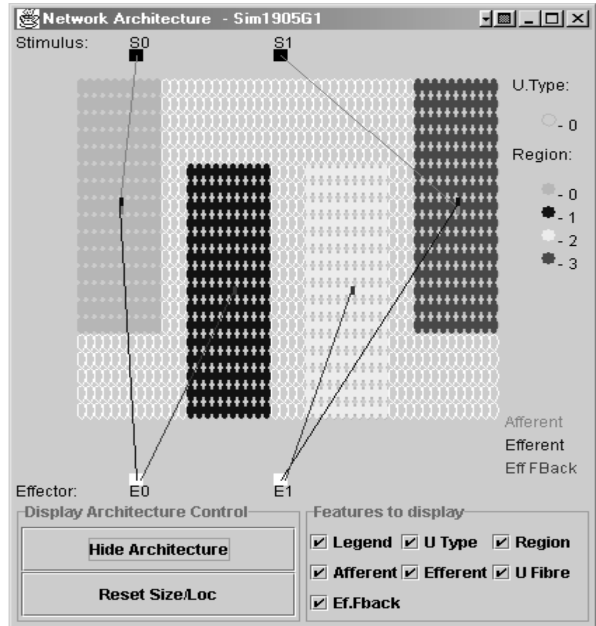


Figure 2. Illustrative view of a Venn-network structure utilized in all simulations of this work. Top rectangles (on each corner) represent motor regions controlling two pairs of virtual hands E1 and E2 (effectors); each hand has five fingers. Bottom rectangles represent sensory regions receiving feedback from effectors. The connecting lines are, in top- bottom order, *afferent*, *efferent* and *efferent-feedback* pairs of fibers. No *u-fibers* are used in this work.

3.2. Simulation configuration

In both sets of simulations carried out here, four processing phases were used for evaluating output performances. Throughout all simulations the only parameter modified per processing phase was *cell death rate*. The values considered for this parameter in each processing phase were: 0.0, 0.079, 0.15, and 0.20. These values correspond to the normalized-to-one decimal logarithmic function *log(n+1)* applied on the *ad hoc* selected series representing the neuronal decay: 0, 20%, 40% and 60%.

To increase accuracy of results and minimize effects of race condition the number of repetitions carried out per simulation was fixed at five. The selection of this high value is partially due to the randomness of the generation process that governs neuronal death in all simulations.

3.3. Input data

The input data for all processing phases of every simulation (*i.e.* all repetitions of Sim2905A and Sim2905B) are the same 111 patterns, corresponding to digitalized finger position of a piano player, *left and right hands,* performing an arbitrarily selected portion of the Mozart's *Sonata Facile* [6]. Each pattern contains distinct numeric information about flexion of all ten fingers of the piano player in a given time (regardless of their position on the keyboard). In other words, the encoding mechanism relates keystrokes within timestamp (**t**) to normalized numerical values. The convention used was 0.0; 0.5; and 1.0 to represent respectively: (a) no finger flexion, (b) the same finger flexed after a brief release of a keyboard key and (c) sustained finger flexion on a key. So far only three discrete values are used; in future experiments normalized values between [0, 1] could be used to encode strength of finger flexion.

3.4. Simulation execution

Once parameters and input data were set, five repetitions of Sim2905A were carried out for all four processing phases. For that, a especially programmed simulator for Venn-networks was utilized, *i.e.* the GVNS-Generalized Venn-network Simulator [4]. Next, a special parameterization was made in the GVNS to make it possible that repetitions of Sim2905B had death of nerve cells to be restricted to specific parts of the cortical map.

4. Results

Table 1 and table 4 shows average output error of all five repetitions of Sim2905A. Table 1 shows additive error per left and right finger – L(n) and R(n) across five repetitions. Table 4 displays output error per unit of time T(n), also average for all testing patterns.

In all graphics, there is great variability of impact of cell death across repetitions and fingers. Even for the same time span, i.e. same neuronal decay among cell populations (artificial neurons), this variability is observed. Even so, within each repetition and time span the cumulative cell death leads to consistent increase in the output error. This means, reduction of output performance per finger because of neuronal decay. Table 2 shows snapshots of cortical activations, with corresponding cell death and evoked finger flexions in table 3. All results for one (same) pattern presented to the network at each one of the four time spans. Finally, table 5 shows different possible layouts available in GVNS to set-up cell death.

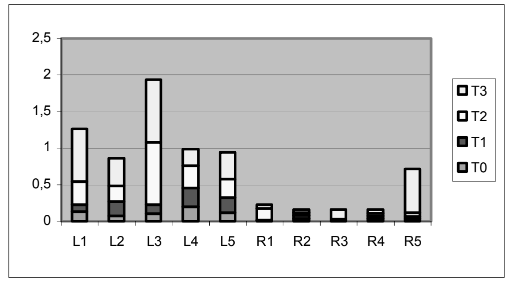
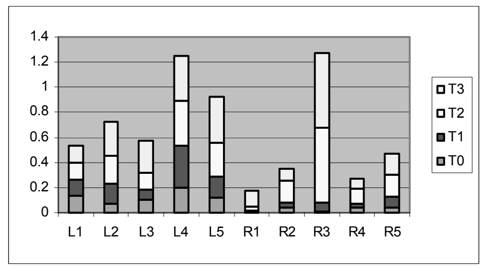
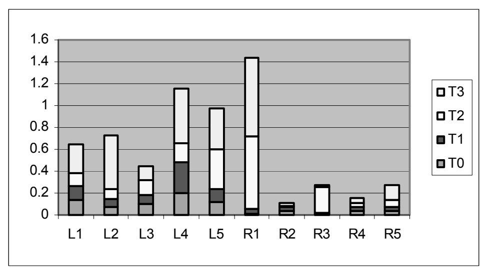
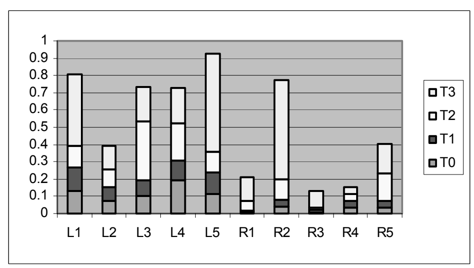
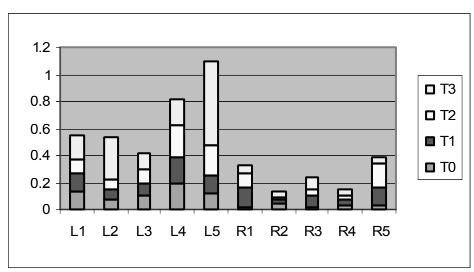
	Additive error - per finger L(n) or R(n)
Repetition 1	
Repetition 2	
Repetition 3	
Repetition 4	
Repetition 5	

Table 1. Additive output error per finger of (L)eft and (R)ight effectors, i.e. virtual hands, across five repetitions. *T(n)* represents ageing though time T0 .. T3.

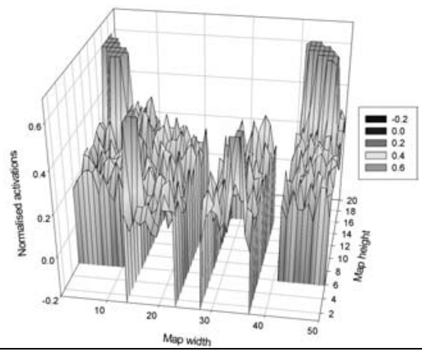
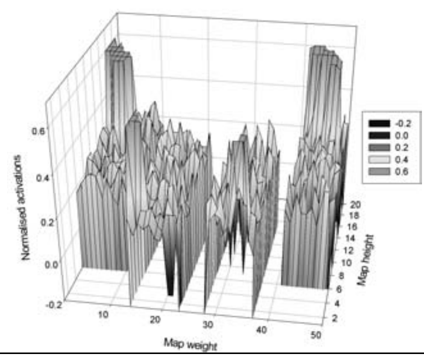
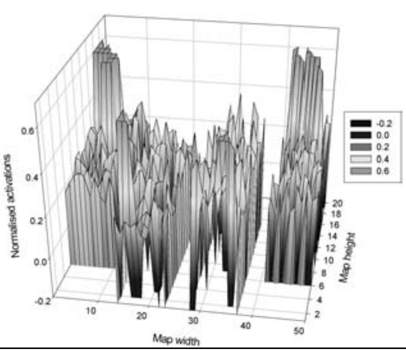
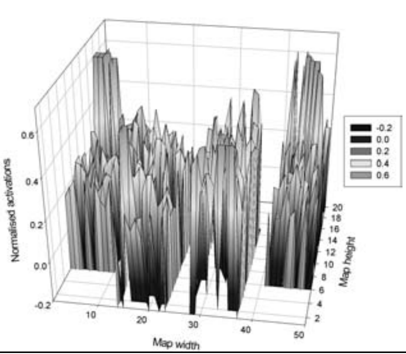
	Presentation of the same typical pattern
T(0)	
T(1)	
T(2)	
T(3)	

Table 2. Snapshots of cortical activations thru time spans.

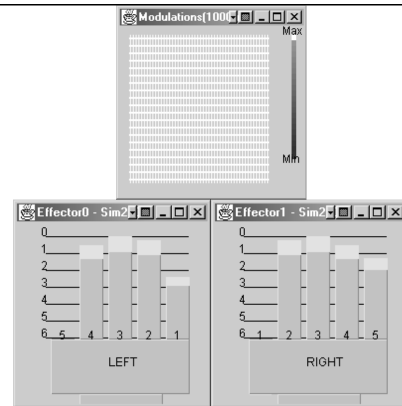
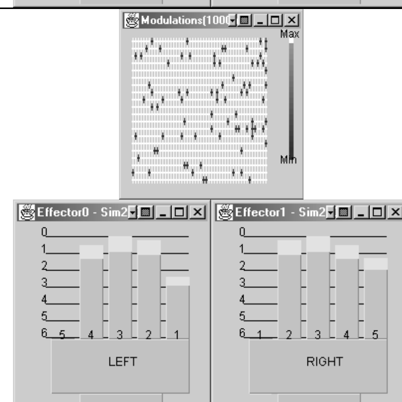
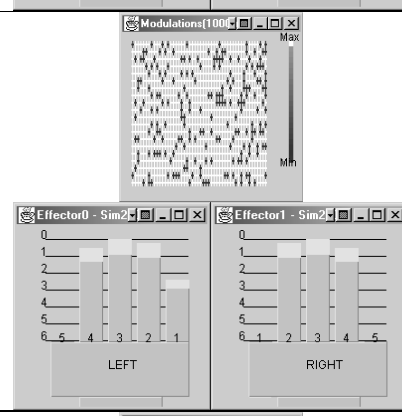
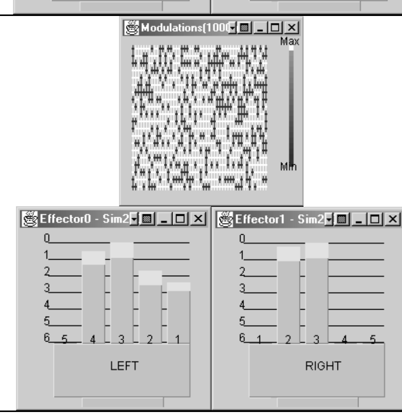
	Simulator screens: dead cells/both hands
T(0)	
T(1)	
T(2)	
T(3)	

Table 3. Snapshots of dead cells and both hands positions

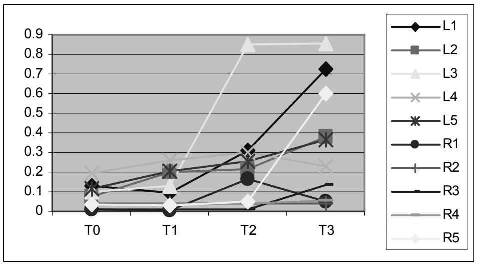
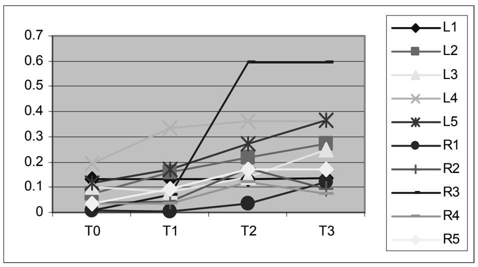
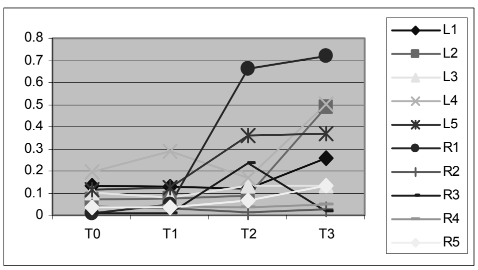
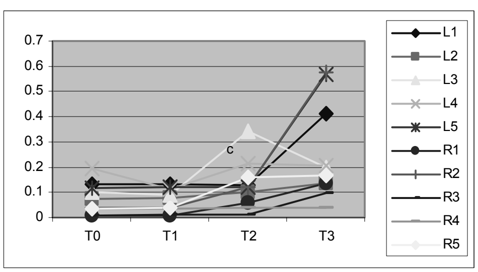
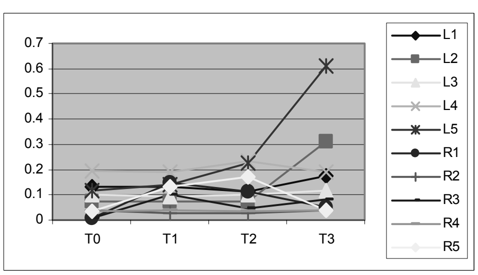
	Output error - per unit of time T(n)
Repetition 1	
Repetition 2	
Repetition 3	
Repetition 4	
Repetition 5	

Table 4. Average output error per unit of time for T(n), i.e. progression of the ageing process, across all fingers *L(n) and R(n),* which represent all ten finger number.

5. Discussion

Randomness of cell death generation prevented further comparisons across repetitions. The slightly uneven variability of impact on performance among fingers is due to use of mono-region for controlling each hand. Further experiments, using this time, one region per finger, instead of one region for the whole hand, proved to be a more robust set-up (as the biological system is indeed).

The overall discrepancy of performances between hands found here is coherent with other additional experiments carried out later. They may well indicate unequal difficulties of tasks being performed by the two virtual hands. Probably originated from the data-set (i.e. directly from Mozart's sonata). Nonetheless, all discrepancies observed in all experiments, reveal a marked reduction in the ability of the network to perform the proposed tasks. This single observation provides a corroboration that Venn-networks present graceful degradation of performance due to network decay; which is also an observable fact in the brain [7].

6. Future works

The wide range of selection for neuronal demise, that can be made for defining which regions or even areas are going to be affected by ageing processes, are a very instigating tool for confronting the model with actual damages of cortical regions. The possibilities currently available in the **GVNS**, see table 6, are: (1) selective of areas, (2) exclusive of areas (all map), and (3) partially exclusive of areas. The latter only differs to the second by considering areas (regions) that are currently being utilized (defined) for processing, whereas in the second modality the whole map (except selected areas) is "candidate" for ageing. Note the additional possibility to mix diverse regions, *e.g.* left-motor. In this example some maps resemble the area selected for ageing, e.g. sensor-selective and motor-partially exclusive. However, this only occurred because the number of groups of regions is limited to two, which would not be the case in a more complex topology. All modalities shown in the example used the same cell death parameter of 20%.

Finally, this work did not investigate the case in which the network is allowed to re-learn in between time spans of neuronal decay. That is, re-learning a previously learned task, just after death of some neuronal populations in selected regions. This may attenuate the slope of the error curve and could be used to evaluate consequences of some neurological aspects of dementia [1,2].

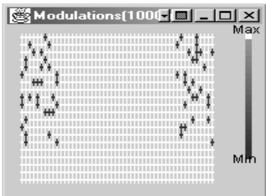
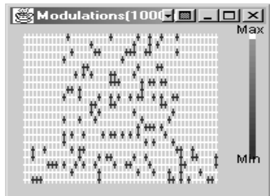
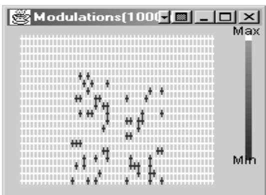
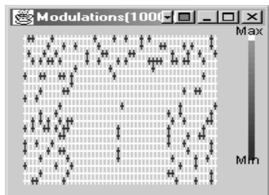
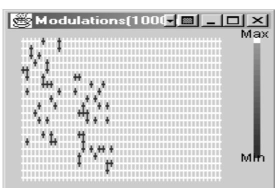
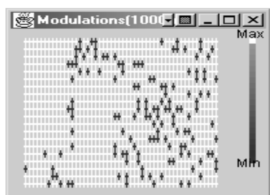
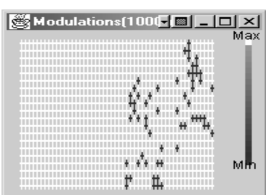
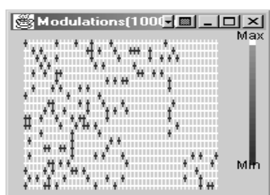
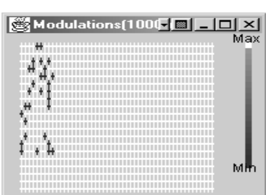
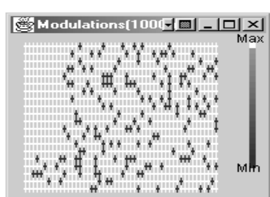
	Selective of areas	Exclusive of areas
Motor		
Sensor		
Left regions		
Right regions		
Mixed: left-motor		

Table 5. Examples of localized cell deaths caused by induced ageing of 20% of total processing elements affected (simulated here by single artificial neurons acting as cortical columns). Black dots are dead neurons, as opposed to white, meaning, functional ones.

References

[1] D. Price, "Aging of the Brain and Dementia of the Alzheimer Type", chapter 58 in [2].

[2] E. Kandel, J. Schwartz and T Jessell (Editors), *Principles of Neural Sciences,* Fourth Edition, International Edition, New York: McGraw-Hill, 2000.

[3] A. Rechtschaffen and J. Siegel, "Sleep and Dreaming", chapter 47 in [2].

[4] F. Buarque de Lima Neto, *Modelling Neural Processing Using Venn-Networks in Physiological and Pathological Scenarios*, Ph.D. dissertation, Dept. Electrical and Electronic Engineering, Imperial College, London/UK, 2002.

[5] F. Buarque de Lima Neto, "Flexion of Virtual Fingers Controlled by Artificial Neural Networks", in *Mathematics and Simulation with Biological, Economical and Musicoacoustical Applications*, edited by C. D'Attellis, V. Kluev, and N. Mastorakis, WSES Press, 2001.

[6] W. A. Mozart, "Sonata in C major: 'Sonata facile' ", in *Sonaten fuer Klavier zu zwei Haenden nach den Quellen*, edited by C.A. Martiensen newly reviewed edition by L. Koehler and A. Ruthardt. C. F. Peters Corporation, Frankfurt, 1951.

[7] P. McLeod, K. Plunkett, and E. Rolls, *Introduction to Connectionist Modelling of Cognitive Processes,* Oxford: Oxford University Press, 1998.