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Computational Neuro-Genetic Modelling: Methods, Systems, Applications

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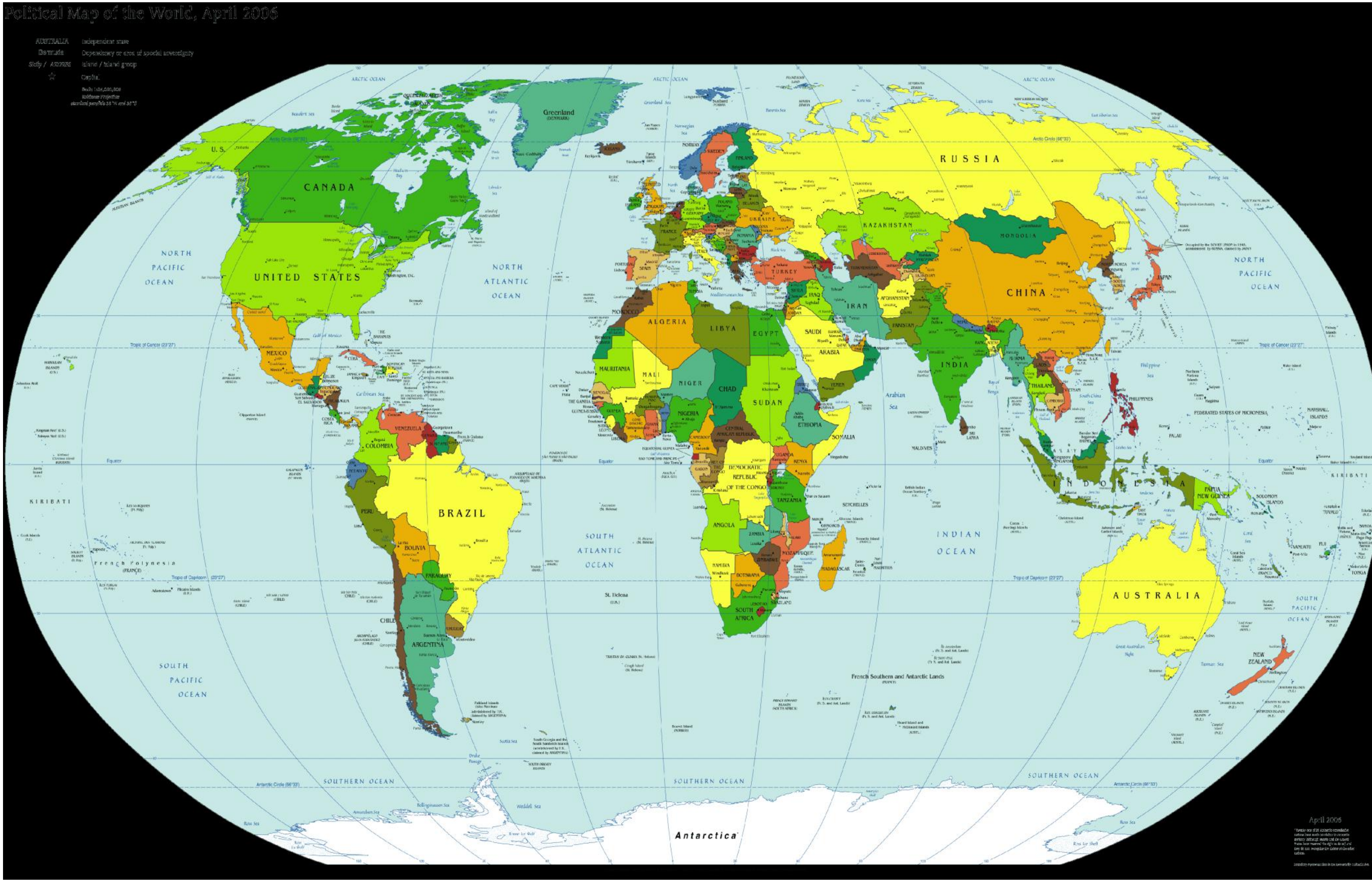
(http://www.inns.org)

The Scope of the Contemporary NN Research

Understanding, modelling and curing the brain
Cognition and cognitive modelling
Memory
Learning, development
Neurogenetic modelling
Neuro-informatics
Mathematics of the brain
Brain ontologies
Bioinformatics
Molecular computing
Quantum information processing; **Q**uantum inspired neural networks
Novel methods of soft computing for adaptive modelling and knowledge discovery; **H**ybrid NN-, fuzzy-, evolutionary- algorithms;
Methods of evolving intelligence (EI);
Evolving molecular processes and their modelling
Evolving processes in the brain and their modelling
Evolving language and cognition
Adaptive integrated/embedded systems

The Scope of the Contemporary NN Applications:

- Adaptive speech, image and multimodal processing;
- Biosecurity
- Adaptive decision support systems;
- Dynamic time-series modelling; Adaptive control;
- Adaptive intelligent systems on the WWW;
- Medicine,
- Health,
- Information Technologies,
- Horticulture,
- Agriculture,
- Business and finance,
- Process and robot control,
- Arts and Design;
- Space research
- Earth and oceanic research


Political Map of the World, April 2006
CANADA
UNITED STATES
NORTH PACIFIC OCEAN
NORTH ATLANTIC OCEAN
Greenland
MEXICO
BRAZIL
ARGENTINA
SOUTH ATLANTIC OCEAN
SOUTH PACIFIC OCEAN
ALGERIA
LIBYA
EGYPT
SUDAN
RUSSIA
KAZAKHSTAN
MONGOLIA
CHINA
IRAN
INDIA
INDIAN OCEAN
AUSTRALIA
INDONESIA
SOUTHERN OCEAN
Antarctica
KIRIBATI

Main references:

N.Kasabov (2007) Evolving Connectionist Systems:
The Knowledge Engineering Approach, Springer, London

Benuskova, L. and N.Kasabov, Computational neuro-genetic modelling, Springer, New York, 2007, 290 pages

N.Kasabov, Evolving Intelligence in Humans and Machines: Integrative Connectionist Systems Approach, Feature article, IEEE CIS Magazine, August, 2008, vol.3, No.3, www.ieee.cis.org, pp. 23-37

N.Kasabov, Integrative Connectionist Learning Systems Inspired by Nature: Current Models, Future Trends and Challenges, Natural Computing, Int. Journal, Springer, Vol. 8, 2009, Issue 2, pp. 199-210.

N.Kasabov, To spike or not to spike: A probabilistic spiking neural model, Neural Networks, Volume 23, Issue 1, January 2010, 16-19

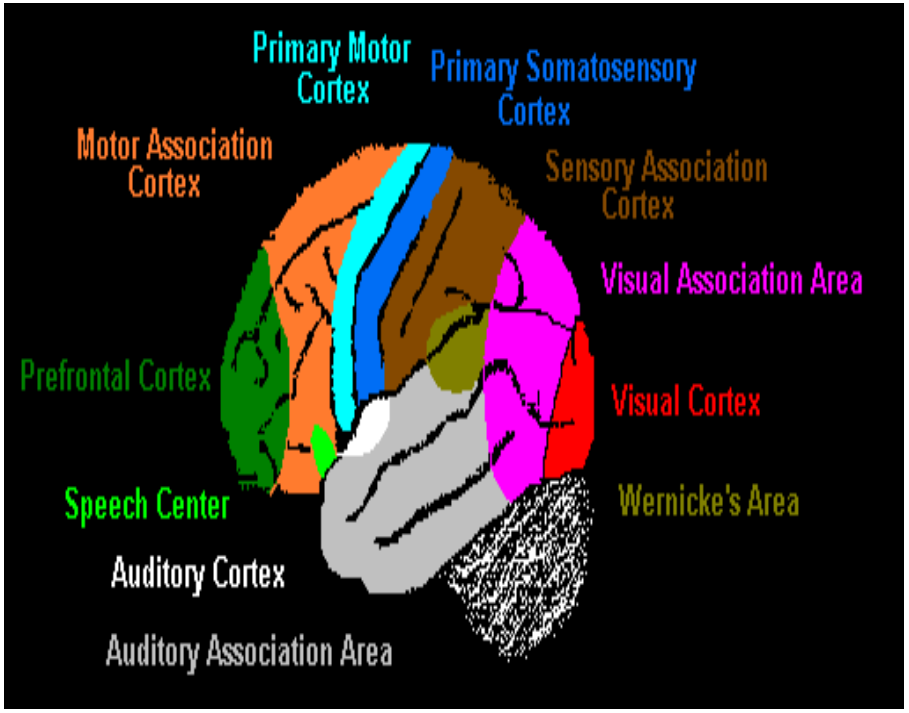
N.Kasabov, R.Schliebs, H.Kojima (2011) Probabilistic Computational Neurogenetic Framework: From Modelling Cognitive Systems to Alzheimer's Disease. *IEEE Tr Autonomous Mental Development*, in print

Content of the talk

Moto: Based on biological evidence, new CNGM can be developed to solve complex generic and specific tasks of computational intelligence (CI).

1) Biological motivations.
2) Spiking neural networks (SNN) and evolving SNN (eSNN).
3) Computational Neuro-Genetic Models (CNGM).
4) Applications
5) Future development

1. Biological motivations



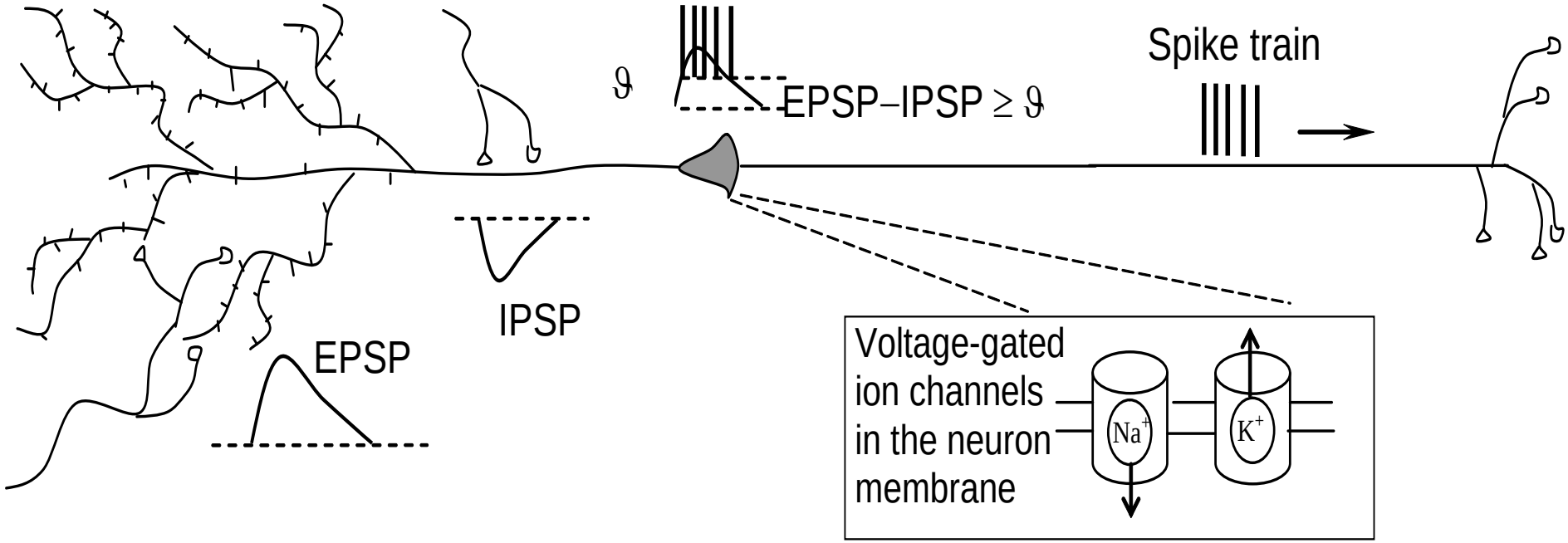
Inspiration from the brain

- The brain evolves through genetic "pre-wiring" and life-long learning
- Evolving structures and functions
- Evolving features
- Evolving knowledge
- Local (e.g. cluster-based) learning and global optimisation
- Memory (prototype)-based learning, "traceable"
- Multimodal, incremental learning
- Spiking activity
- Genes/proteins involved
- Quantum effects in ion channels

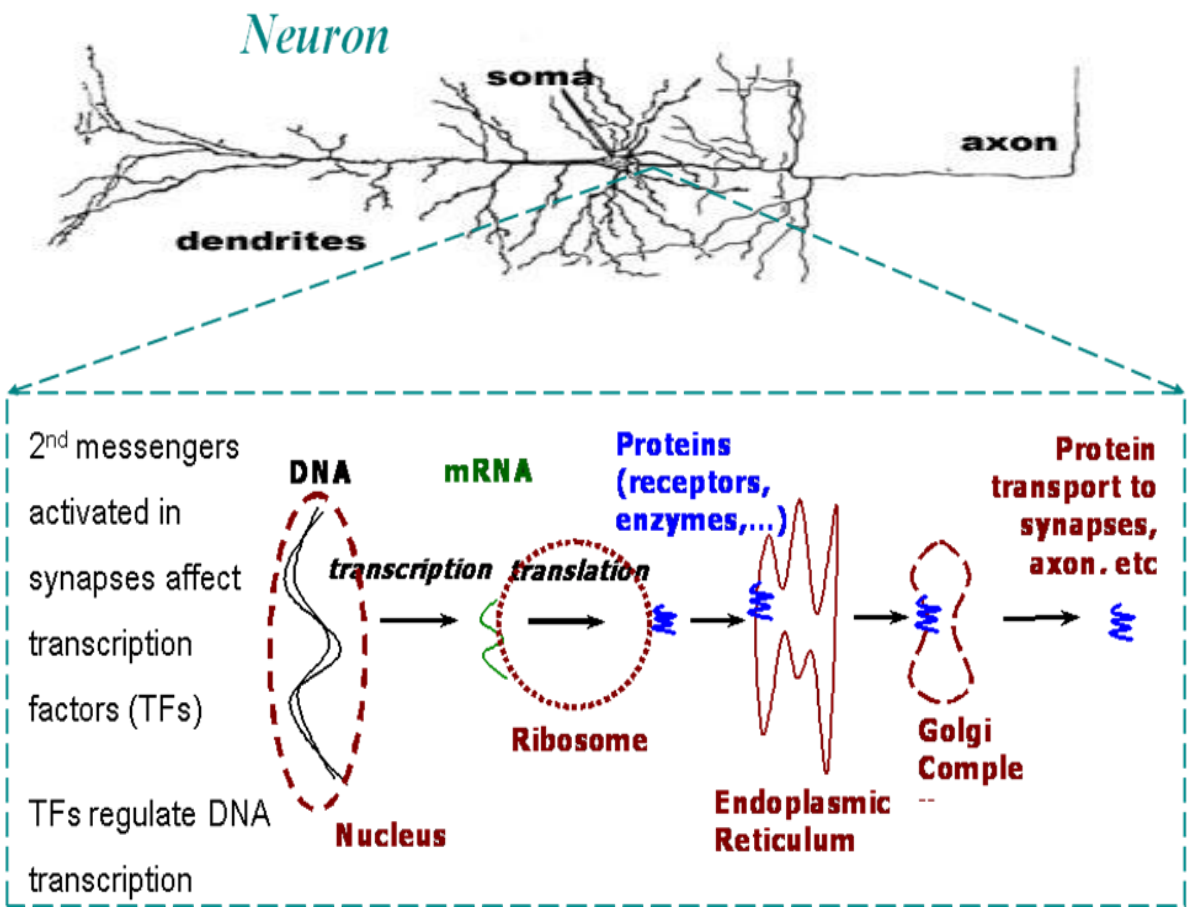
Rich neurophysiological information about the spiking activities in the brain is already available

(Singer, Abeles, Freeman, Villa, Grossberg, Kojima, Yamaguchi, ...)

Electric synaptic potentials and axonal ion channels responsible for spike generation and propagation: EPSP = excitatory postsynaptic potential, IPSP = inhibitory postsynaptic potential, ϑ = excitatory threshold for an output spike generation.



Biological neurogenetic processes



Gene information processing principles:

- Nature via Nurture
- Complex interactions between thousands of genes (appr. 6000 expressed in the brain) and proteins (more than 100,000)
- Different time-scales
- Stochastic processes

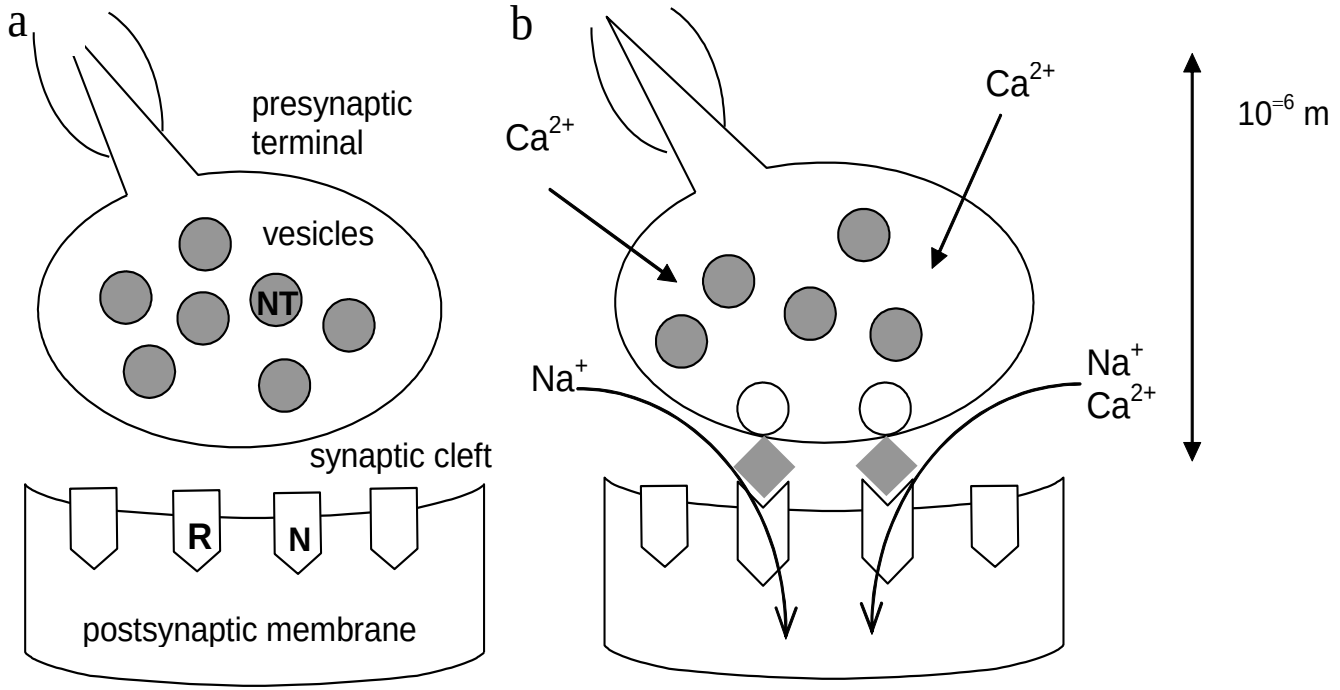
Offer the potential for:

- Integrating molecular and neuronal information processing (possibly with particle level as well)

The challenge:

How do we integrate molecular and spiking neuronal processes in a SNN?

Molecular (protein) level of spiking activities



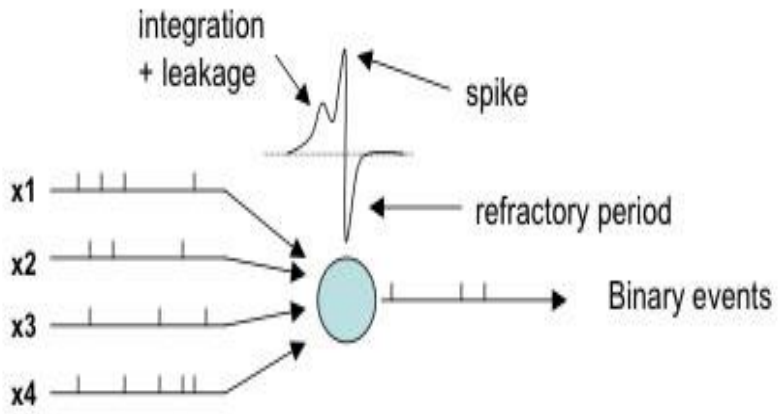
Scheme of synaptic transmission:

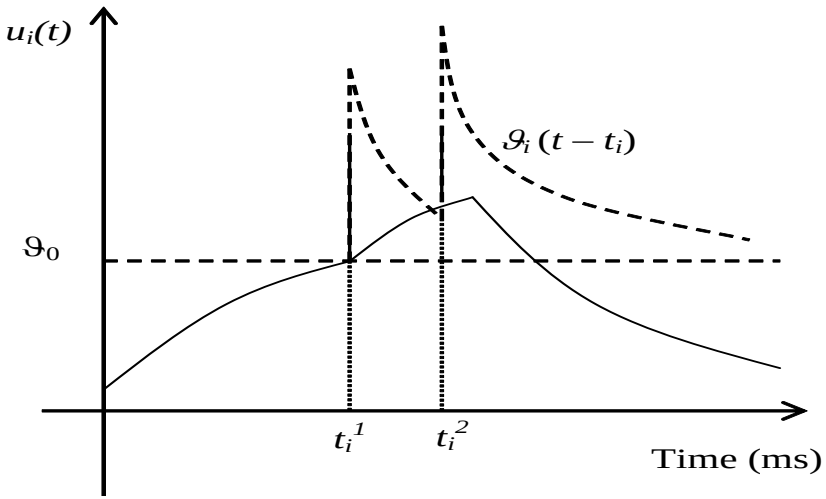
a) A synapse is ready to transmit a signal.

b) Transmission of electric signal in a chemical synapse upon arrival of action potential into the terminal.

Abbreviation: NT = neurotransmitter, R = AMPA-receptor-gated ion channel for sodium, N = NMDA-receptor-gated ion channel for sodium and calcium.

2. SNN and evolving SNN (eSNN)





Information processing principles in neurons and neural networks:

- LTP and LTD;
- Trains of spikes;
- Time, frequency and space;
- Synchronisation and stochasticity;
- Evolvability…

They offer the potential for:

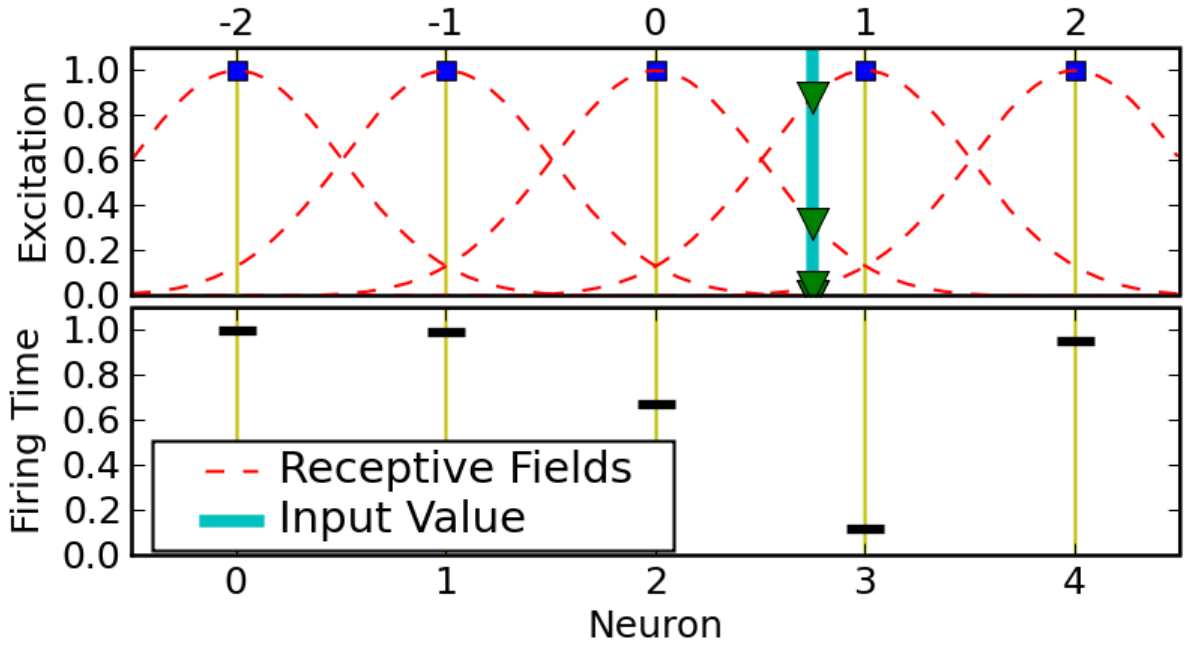
- Modelling cognitive functions through patterns of neuronal spiking activity;
- Modelling neuronal activities based on genes and proteins;
- Integration of different 'levels 'of information processing.

Models of Spiking Neurons

- Spiking neuron models incorporate the concept of time and phase in relation to the neuronal and synaptic states
- **Microscopic Level:** Modeling of ion channels, that depend on presence/absence of various chemical messenger molecules
  - Hodgkin-Huxley's
  - Izhikevich's
- **Macroscopic Level:** Neuron is a homogenous unit, receiving and emitting spikes according to defined internal dynamics
  - Spike response model (SRM) (Maass)
  - Integrate-and-Fire models (IF, LIF) (Maass, Gerstner)
  - Thorpe's model
  - A probabilistic spiking neuron model (pSNM)
- **Integrative**
  - A quantum inspired optimisation of evolving SNN
  - A neuro-genetic evolving SNN (ngeSNN)

Rank Order Population Encoding

- Distributes a single real input value to multiple neurons and may cause the excitation and firing of several responding neurons
- Implementation based on Gaussian receptive fields introduced by Bothe *et al* . 2002



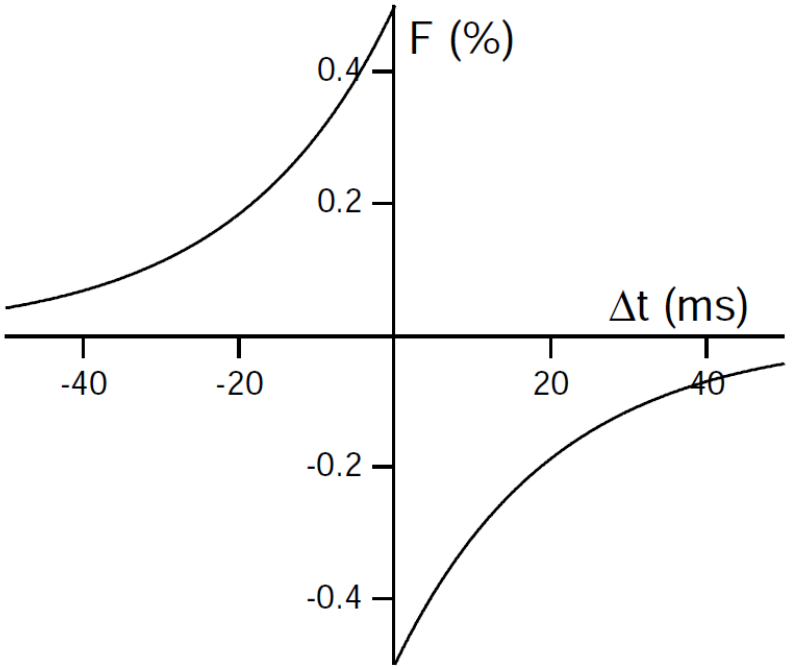
Learning in SNN

- Due to time dependence, learning methods are rather complex in SNN
- Recurrent SNN introduce additional difficulties and complexity
- Unsupervised Hebbian learning
- **Spike-timing dependent plasticity (STDP)**
- Reinforcement learning (e.g. for robotics applications)
- SpikeProp – supervised error back-propagation, similar to learning in classical MLP
- (Linear) readout functions for the Liquid State Machines (Maas et al)
- ReSuMe – Remote Supervised Learning, capable of learning the mapping from input to output spike trains
- Weight optimization based on evolutionary algorithms (EA)
- Combined EA and STDP

Spike-Time Dependent Plasticity (STDP)

- Hebbian form of plasticity in the form of long-term potentiation (LTP) and depression (LTD)
- Effect of synapses are strengthened or weakened based on the timing of post-synaptic action potentials

Pre-synaptic activity that precedes post-synaptic firing can induce LTP, reversing this temporal order causes LTD



Thorpe's Model

- Simple but computationally efficient neural model, in which early spikes are stronger weighted – time to first spike learning
- Model was inspired by the neural processing of the human eye and introduced by S. Thorpe et. al. 1997
- PSP $u_i(t)$ of a neuron i:

$$u_i(t) = \begin{cases} 0 & \text{if fired} \\ \sum_{j \mid f(j) < t} w_{ji} m_i^{order(j)} & \text{else} \end{cases}$$

- w_{ji} is the weight of the connection between neuron j and i, $f(j)$ is the firing time of j, m_i a parameter of the model (***modulation factor***)
- Function *order* (j) represents the rank of the spike emitted by neuron j and receive at neuron i

eSNN

(Kasabov, 2007; Wysoski, Benuskova and Kasabov, 2006-2010)

- Creating and merging neurons based on localised information
- Uses the first spike principle (Thorpe et al.) for fast on-line training
- For each input vector

a) Create (evolve) a new output spiking neuron and its connections

b) Propagate the input vector into the network and train the newly created neuron

$$u_i(t) = \begin{cases} 0 & \text{if fired} \\ \sum_{j \mid f(j) < t} w_{ji} m_i^{\text{order}(j)} & \text{else} \end{cases}$$

$$\Delta w_{ji} = m^{\text{order}(j)}$$

Weights change based on the spike time arrival

c) Calculate the similarity between weight vectors of newly created neuron and existing neurons:

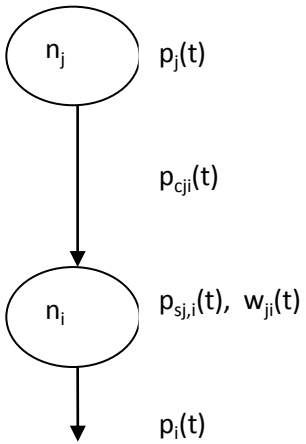
IF similarity > *SIMthreshold* THEN Merge newly created neuron with the most similar neuron, where N is the number of samples previously used to update the respective neuron.

d) Update the corresponding PSP threshold ϑ:

$$W \Leftarrow \frac{W_{new} + NW}{1+N} \qquad \vartheta \Leftarrow \frac{\vartheta_{new} + N\vartheta}{1+N}$$

- Three main parameters of the eSNN: Modulation factor *m*; Spiking threshold ϑ; *SIM*threshold

Probabilistic spiking neuron model, pSNM (*Kasabov, Neural Networks, Jan. 2010)*



The information in pSNM is represented as both connection weights and probabilistic parameters for spikes to occur and propagate. The neuron (n_i) receives input spikes from pre-synaptic neuron n_j (j=1,2,…,m). The state of neuron n_i is described by the sum of the inputs received from all *m* synapses – the postsynaptic potential, PSPi(t). When PSPi(t) reaches a firing threshold $\vartheta i(t)$, neuron ni fires, i.e. emits a spike.

The PSPi(t) is now calculated using a new formula:

$$PSP_i(t) = \sum_{p=t_0,..,t} \sum_{j=1,..,m} e_j \, g(p_{cj,i}(t-p)) \, f(p_{sj,i}(t-p)) \, w_{j,i}(t) + \eta(t-t_0)$$

where: e_j is 1, if a spike has been emitted from neuron n_j, and 0 otherwise; $g(p_{cj,i}(t))$ is 1 with a probability $p_{cji}(t)$, and 0 otherwise; $f(p_{sj,i}(t))$ is 1 with a probability $p_{sj,i}(t)$, and 0 otherwise; t_0 is the time of the last spike emitted by n_i; $\eta(t-t_0)$ is an additional term representing decay in the PSP. As a special case, when all or some of the probability parameters are fixed to "1", the ipSNM will be simplified and will resemble some already known spiking neuron models, such as SRM.

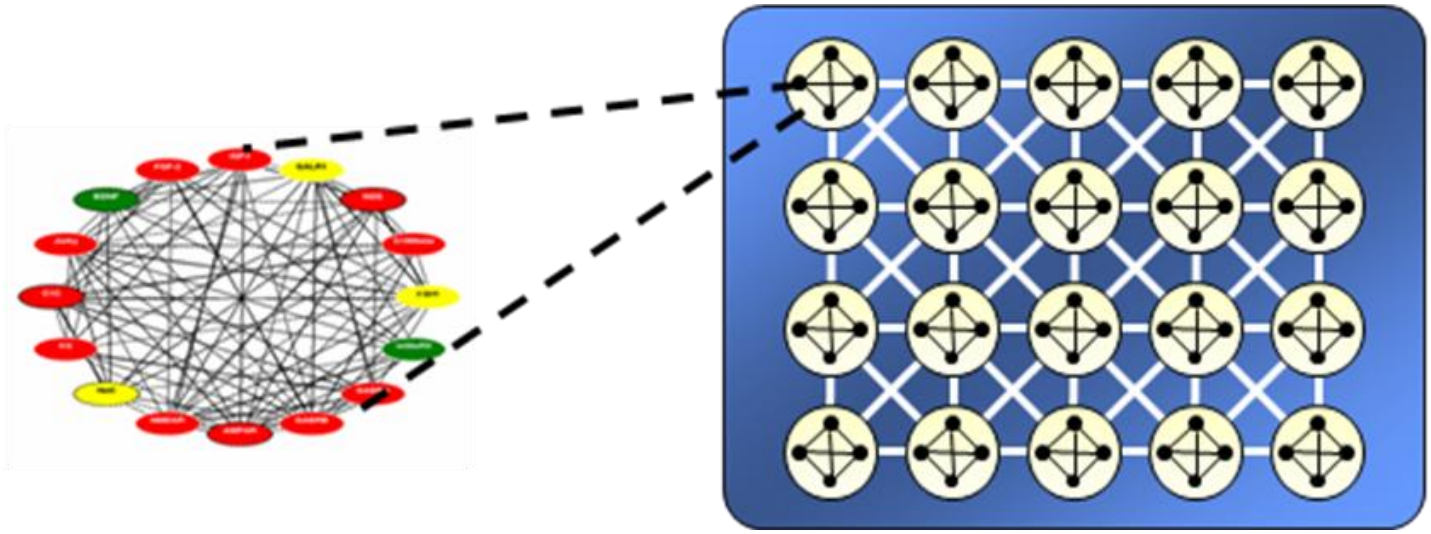
3. Computational Neurogenetic Models

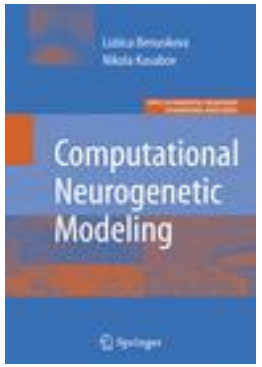
- Functions of neurons and neural networks are influenced by internal networks of interacting genes forming an abstract GRN model.

- The GRN and the SNN function at different time scales

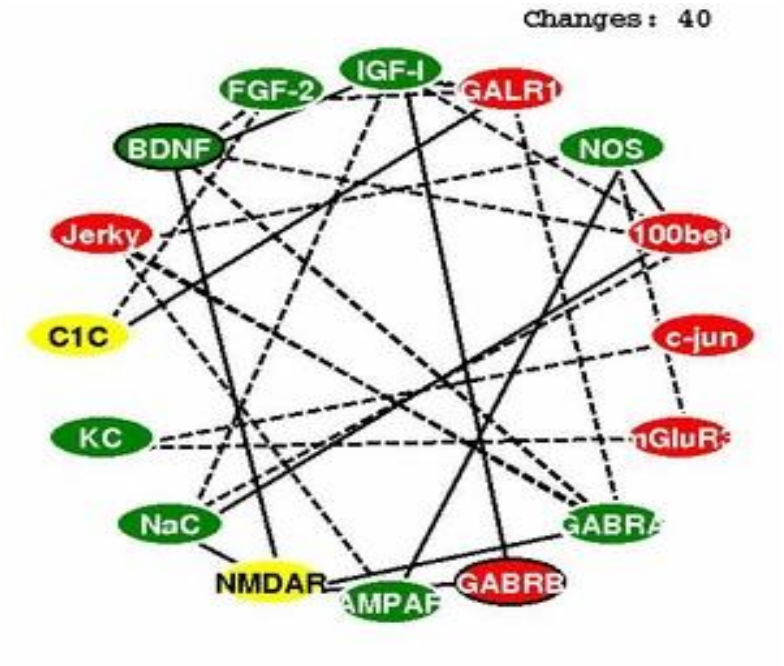
- The challenge is how to integrate a GRN model into a SNN model.

A CNGM is a SNN that incorporates a gene regulatory network (GRN) to capture the interaction of genes related to neuronal activities of the SNN.





GRN as a dynamical system



$$P_j(t) = P_j(0) g_j(t)$$

$$g_j(t+1) = \sigma\left(\sum_{k=1}^{n} w_{jk}(t) g_k(t) \right)$$

A spike response CNGM of a neuron (integrating gene activation with neuronal spiking activity)

(Kasabov, Benuskova, Wysoski, 2005)

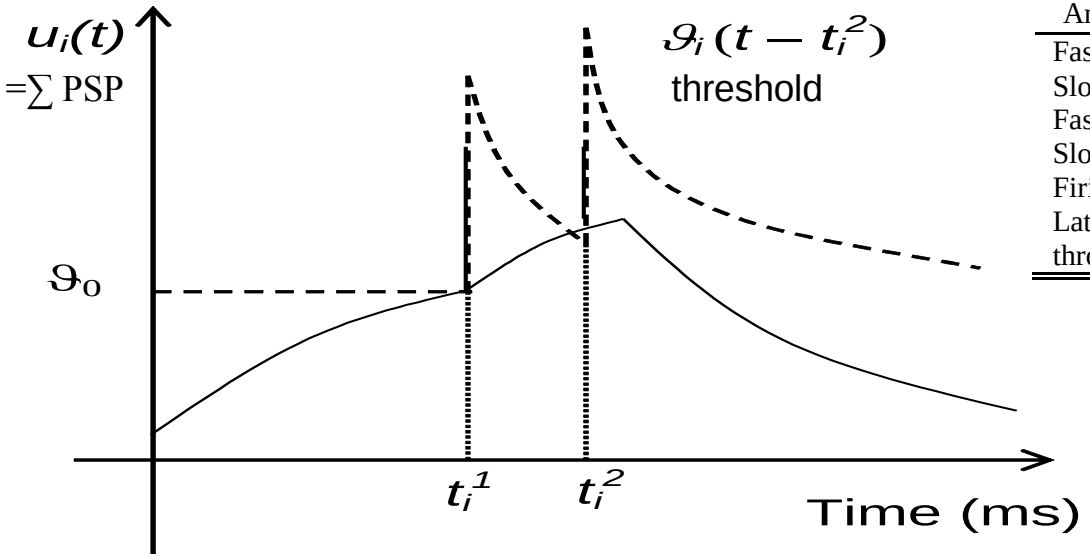


Table. Neuronal Parameters and Related Proteins

Neuronal parameter Amplitude and time constants of	Protein
Fast excitation PSP	AMPAR
Slow excitation PSP	NMDAR
Fast inhibition PSP	GABRA
Slow inhibition PSP	GABRB
Firing threshold	SCN, KCN, CLC
Late excitatory PSP through GABRA	PV

$$PSP_{ij}^{type}(t - t_j - \Delta_{ij}^{ax}) = A^{type}\left(\exp\left(-\frac{t - t_j - \Delta_{ij}^{ax}}{\tau_{decay}^{type}}\right) - \exp\left(-\frac{t - t_j - \Delta_{ij}^{ax}}{\tau_{rise}^{type}}\right)\right)$$

type = fast excitation, slow_excitation, fast_inhibition, slow_inhibition

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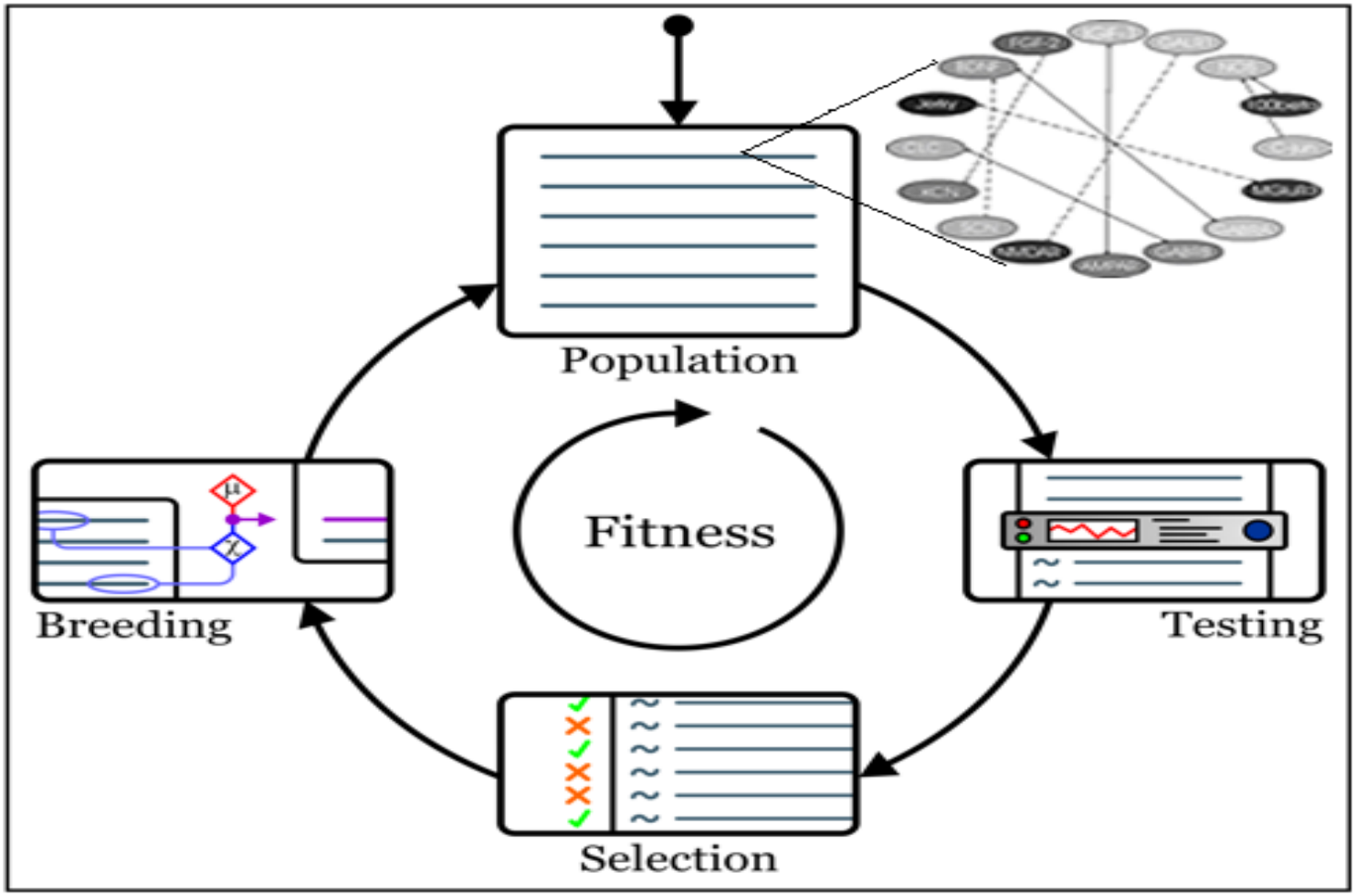
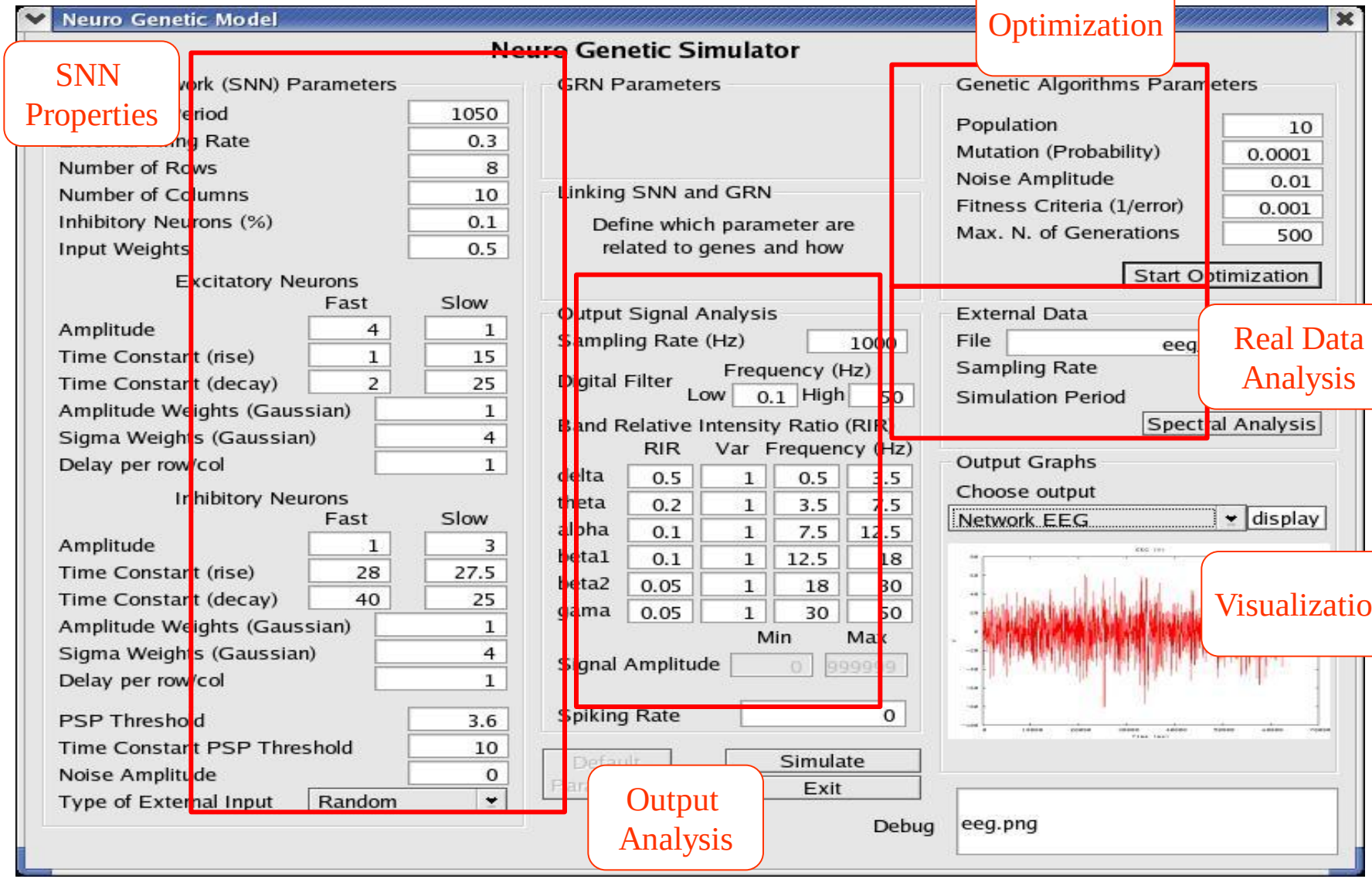
GA optimization of a GRN model



CNG Simulator (Available from KEDRI, www.kedri.info)



qi *E*volutionary Algorithms

- QiEA use a q-bit representation of a chromosome of n "genes" at a time t:

$$Q(t) = \{q_1^t, q_2^t, ..., q_n^t\}$$

- Each q-bit is defined as a pair of numbers (α, β) – probability density amplitudes.

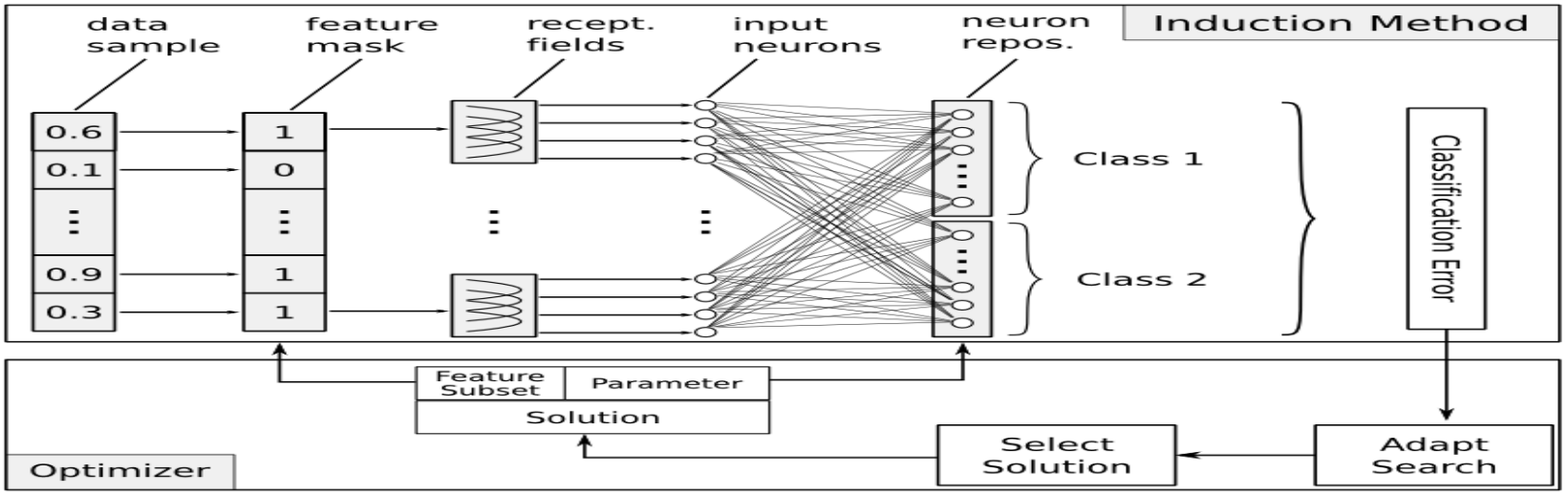
$$|\alpha_i|^2 + |\beta_i|^2 = 1$$

- A *n* element q-bit vector can represent probabilistically 2^n states at any time
- The output is obtained after the q-bit vector is collapsed into a single state
- Changing probability density with quantum gates, e.g. rotation gate:

$$\begin{bmatrix} \alpha \\ \beta \end{bmatrix} = \begin{bmatrix} \cos(\Delta\theta) & -\sin(\Delta\theta) \\ \sin(\Delta\theta) & \cos(\Delta\theta) \end{bmatrix} \begin{bmatrix} \alpha_{t-1} \\ \beta_{t-1} \end{bmatrix}$$

- M. Defoin-Platel, S.Schliebs, N.Kasabov, Quantum-inspired Evolutionary Algorithm: A multi-model EDA, IEEE Trans. Evolutionary Computation, Dec., 2009.

Quantum inspired optimisation of features and parameters of eSNN



1) The principle of quantum probability feature representation:

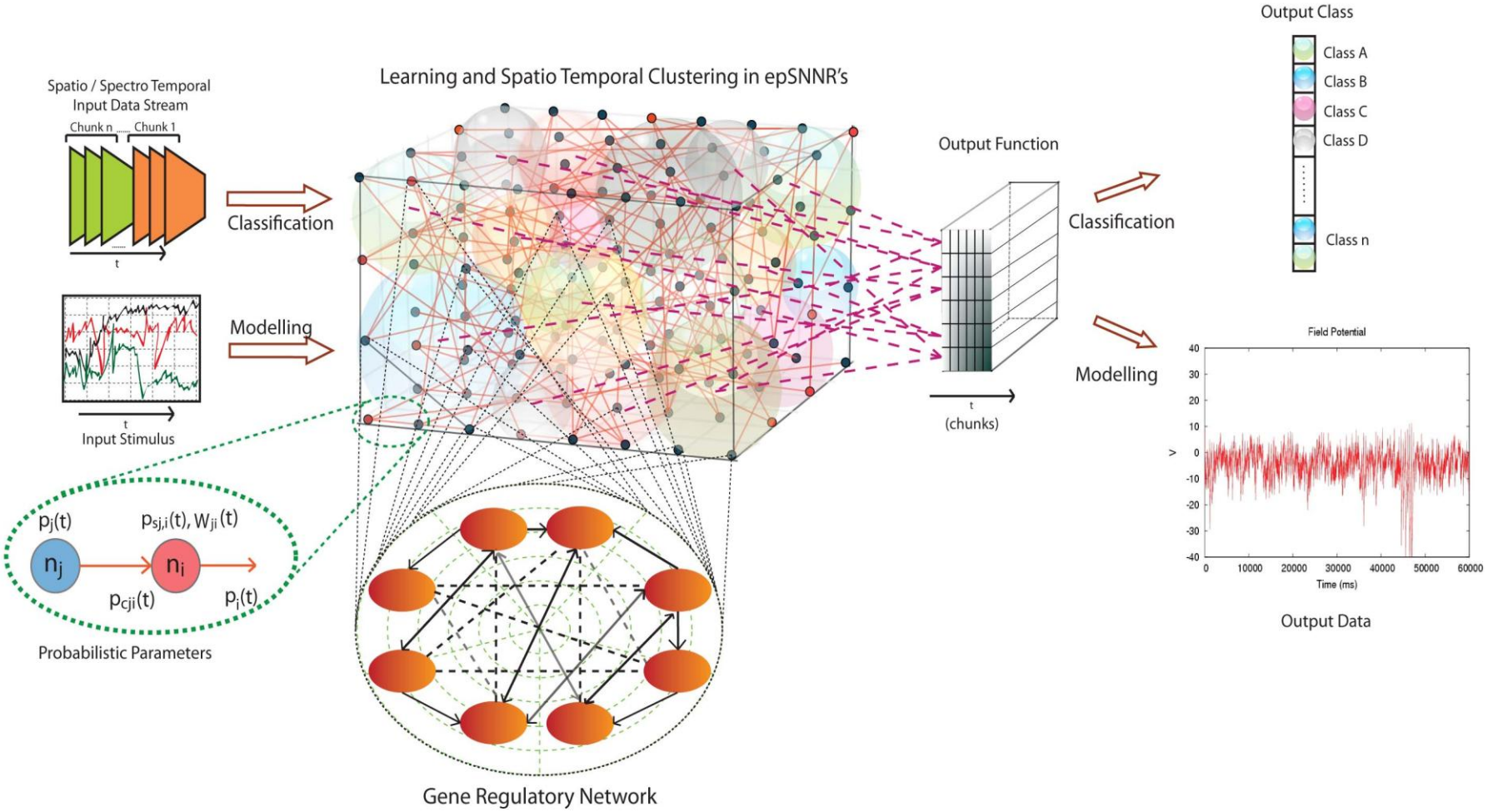
At any time a feature is both present and not present in a computational model, which is defined by the probability density amplitudes. When the model computes, the feature state is 'collapsed' in either 0 (not used) or 1 (used).

2) Quantum probability representation of the connections in eSNN.

3) Quantum probability representation of the eSNN parameters.

N.Kasabov, Integrative connectionist learning systems inspired by Nature: Current models, future trends and challenges, Natural Computation, Springer, 2009, 8:199-218.

Genes regulate the probability parameters of the probabilistic neuronal models in a Evolving Spiking Neuro-Genetic Reservoir (eSNGR)



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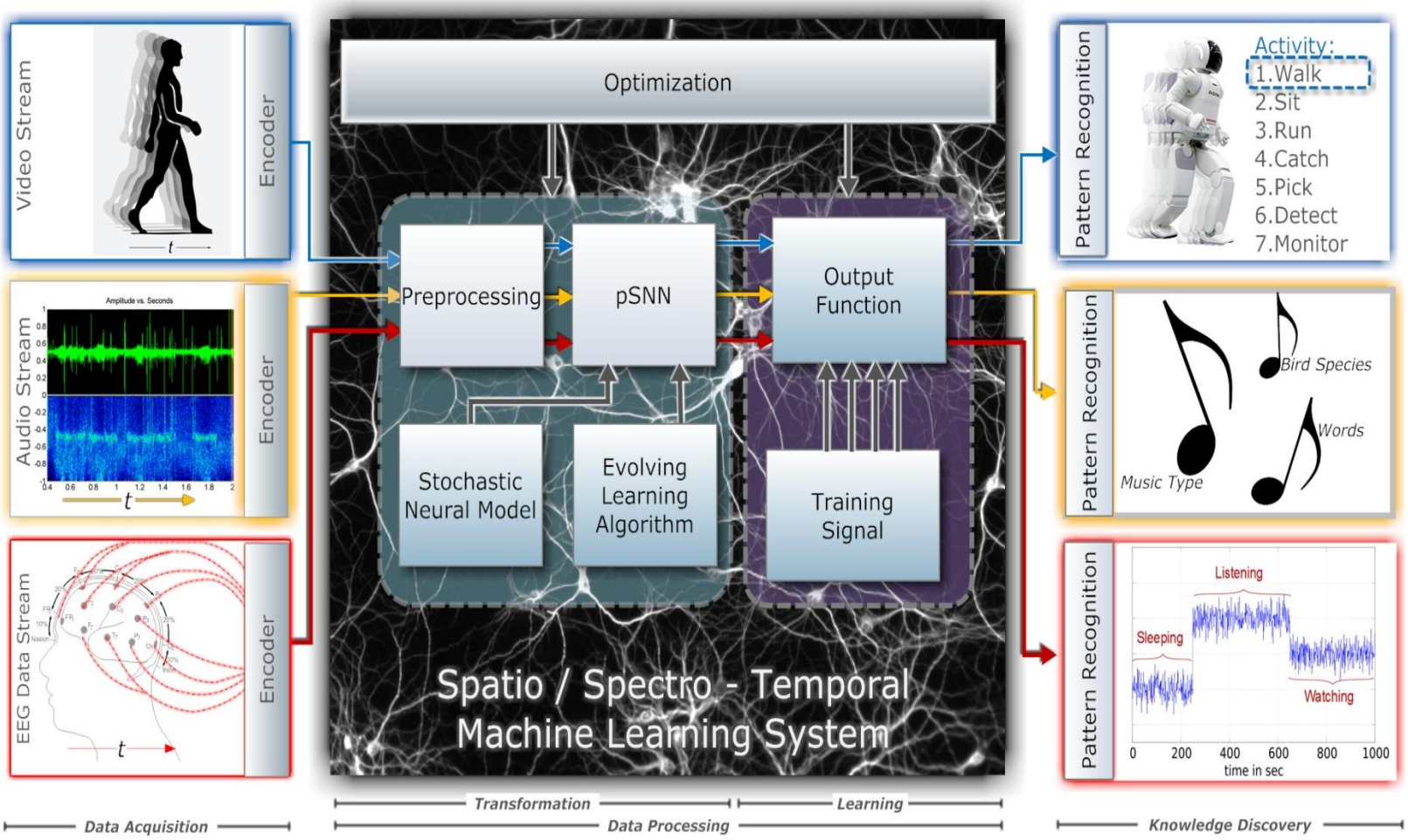
4. Applications of eSNN and CNGM

- Spatio- and spectro- temporal data analysis, modeling and pattern recognition:
  - Audio-visual,
  - EEG,
  - fMRI
  - Ecological
  - Environmental
- Modelling brain functions, e.g. epilepsy
- Modelling neurodegenerative diseases, e.g. AD
- Modelling and creation of cognitive and emotional systems (e.g. Robots)
- Integrating CNGM with brain-gene ontology systems

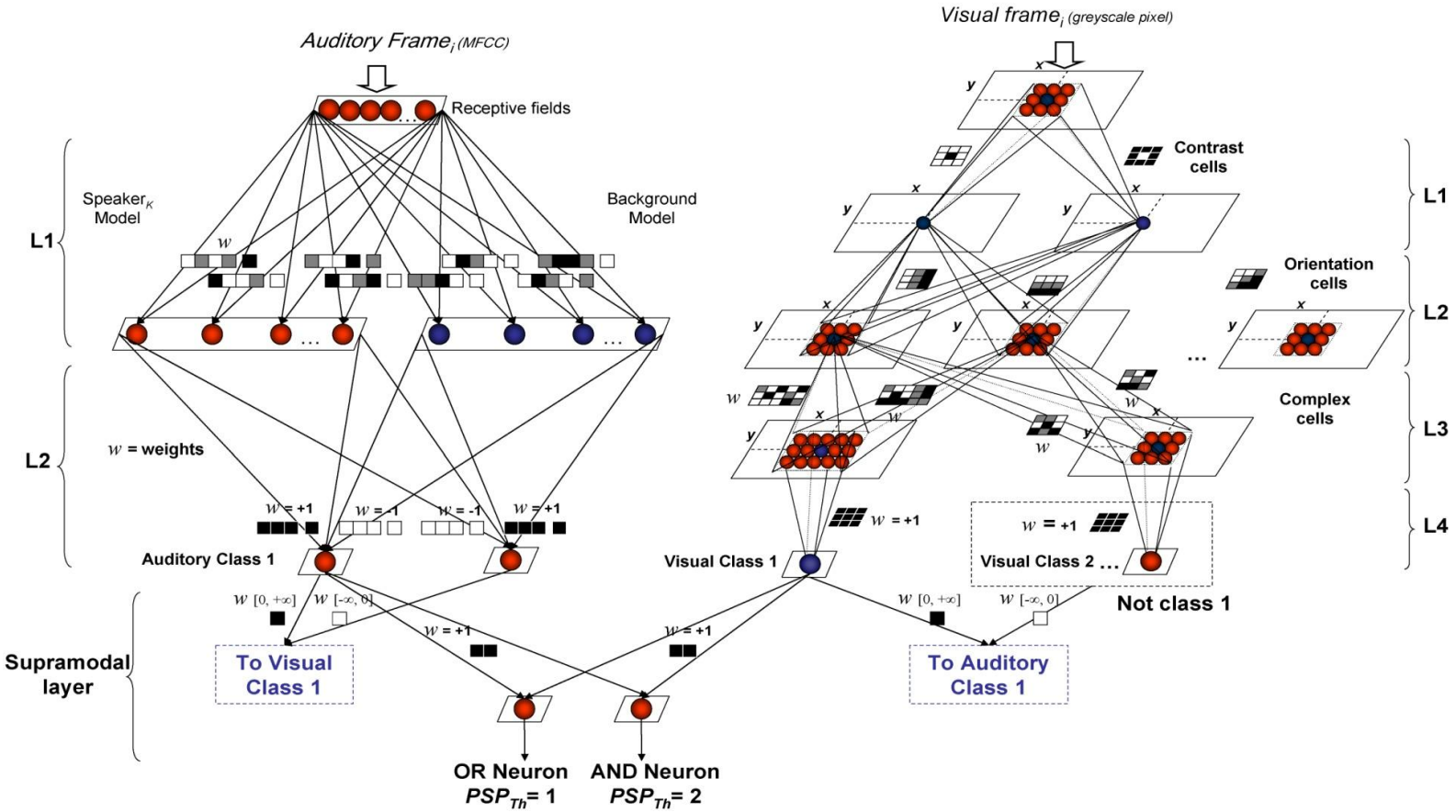
Applications of eSNN and CNGM for spatio- and spectro-temporal data analysis, modelling and pattern recognition



eSNN for integrated audio-visual information processing

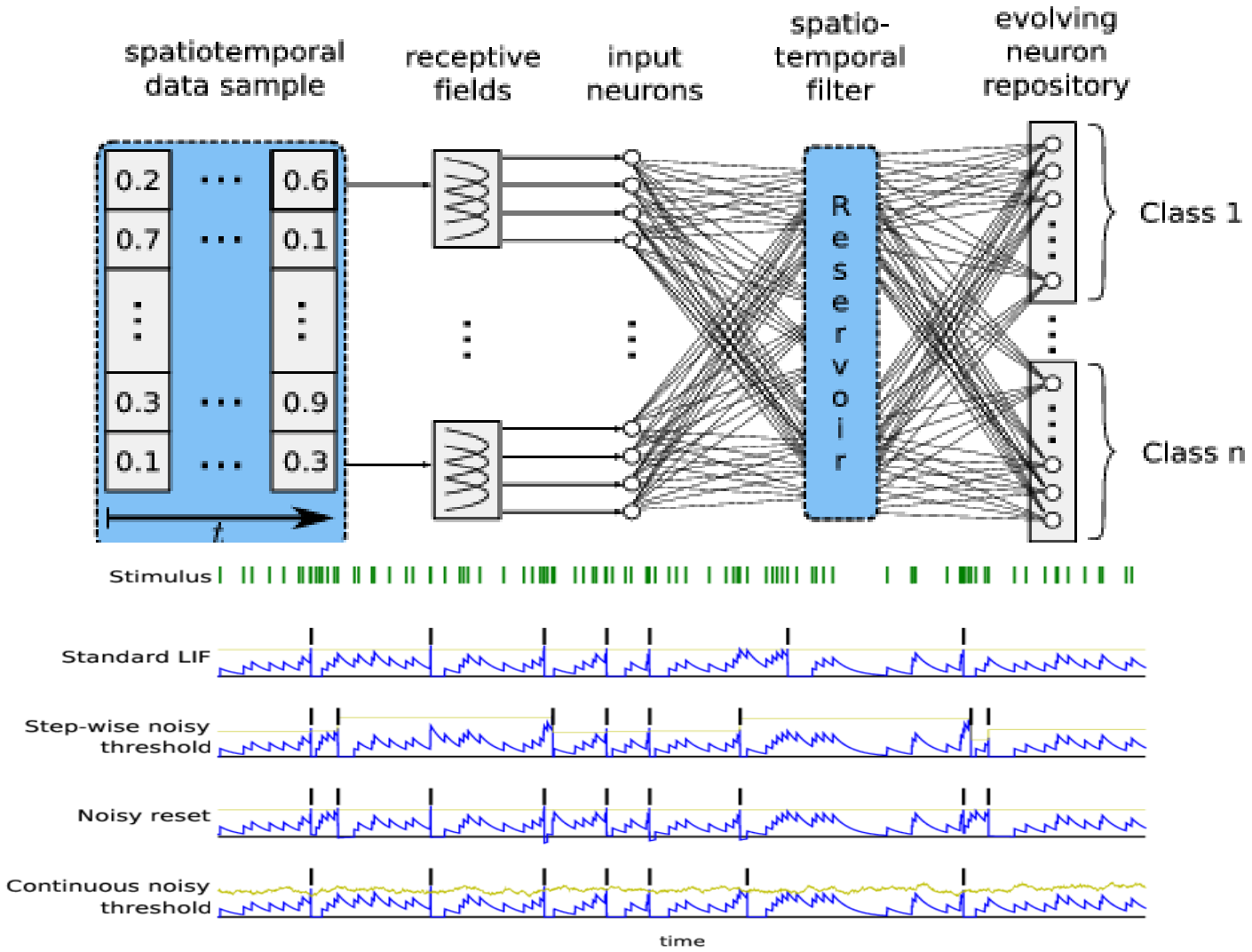
Person authentication based on speech and face data

(Wysoski, Benuskova and Kasabov, Proc. ICONIP, 2007; *Neural Networks*, 2010)



peSNN reservoir for spatio-temporal and spectro-temporal data modelling

S. Schliebs, N. Nuntalid, and N. Kasabov,Towards spatio-temporal pattern recognition using evolving spiking neural networks, Proc. ICONIP 2010, LNCS



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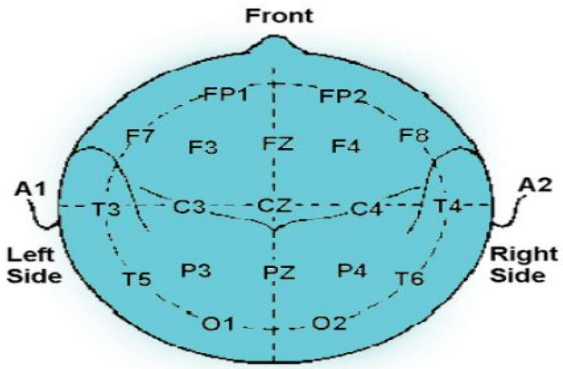
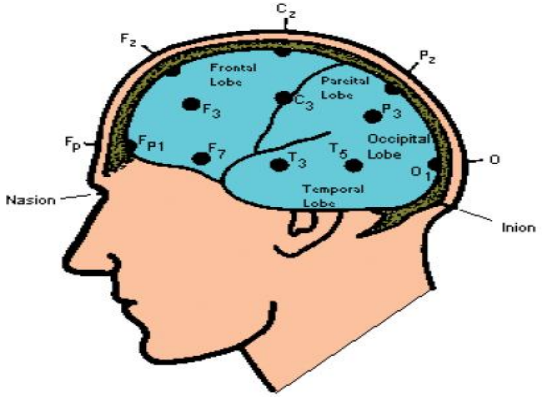
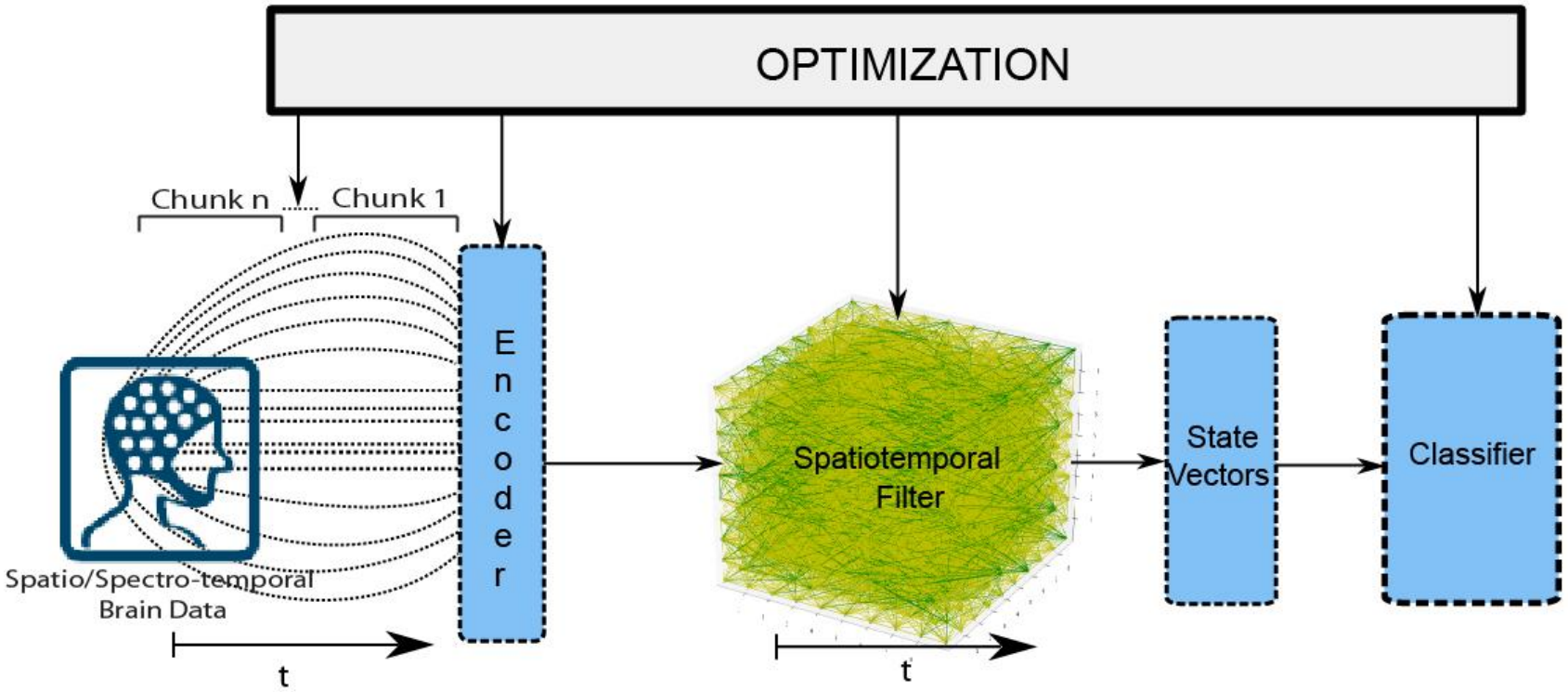
Modelling EEG data







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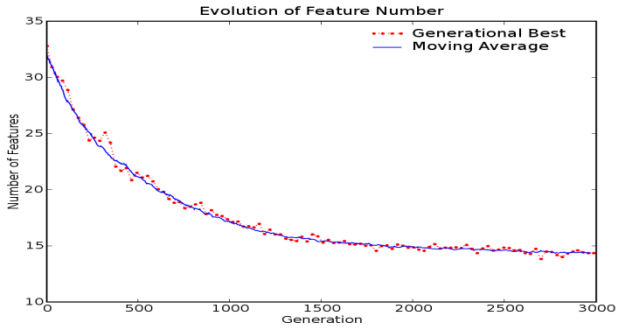
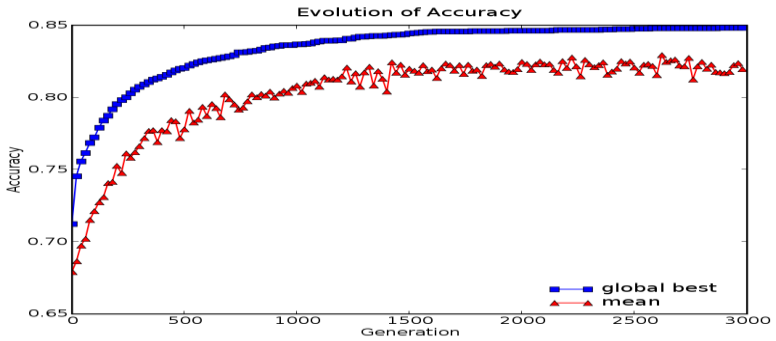
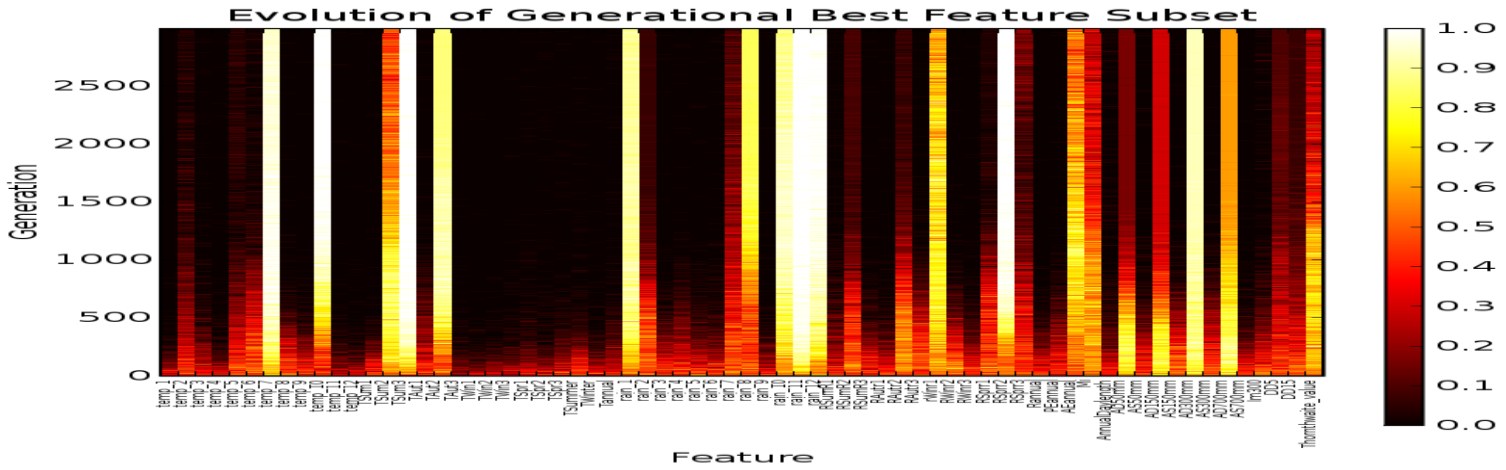
eSNN and QiGA for feature selection in ecological modelling (insect establishment prediction)



Evolution of classification accuracy on the climate data set after 3,000 generations.



Evolution of the features on the climate data set. The best accuracy model is obtained for 15 features .

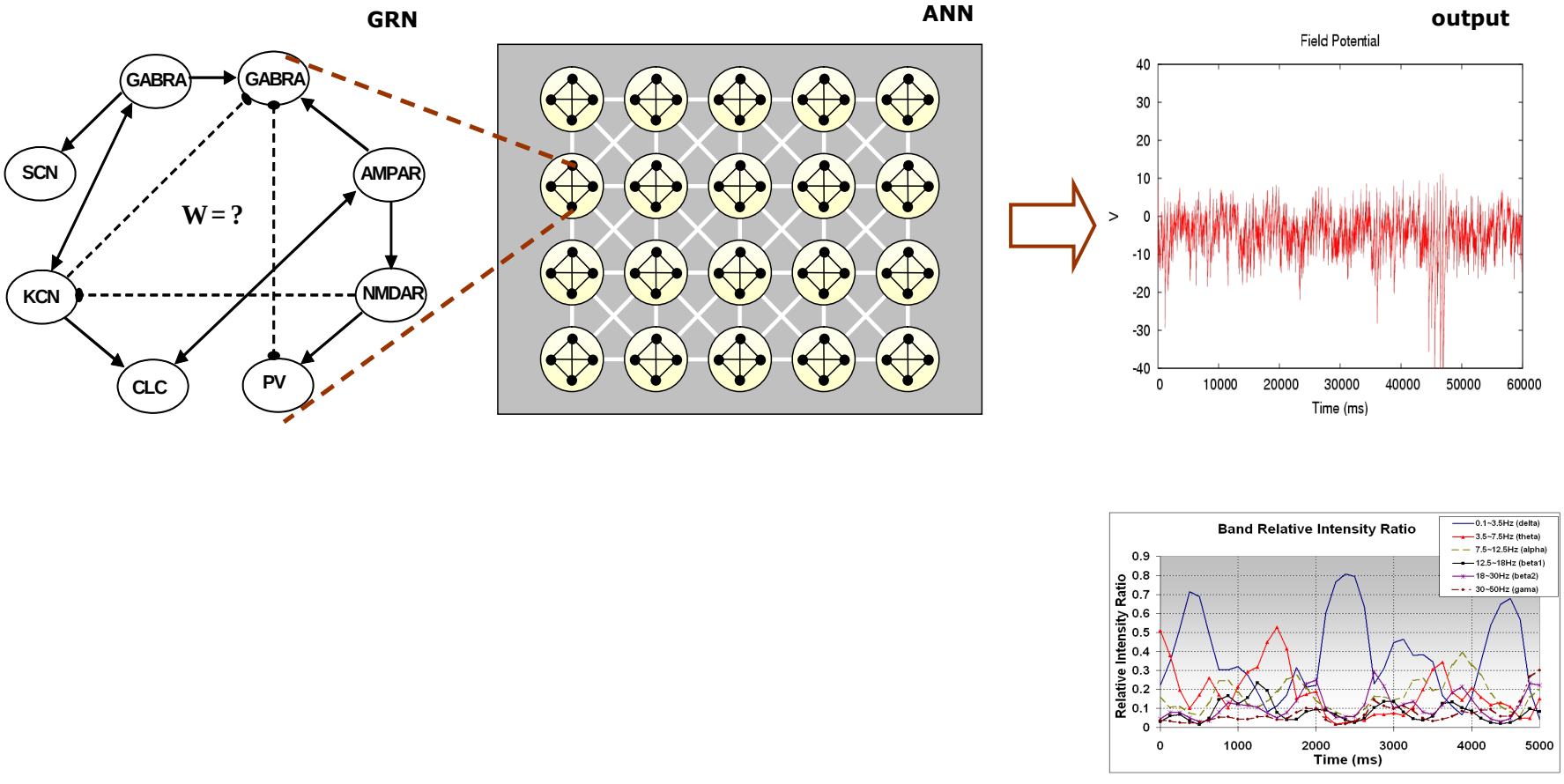


CNGM for modelling and understanding brain diseases

Table 1. Single and multiple genes related to some neurodegenerative diseases and brain abnormalities.

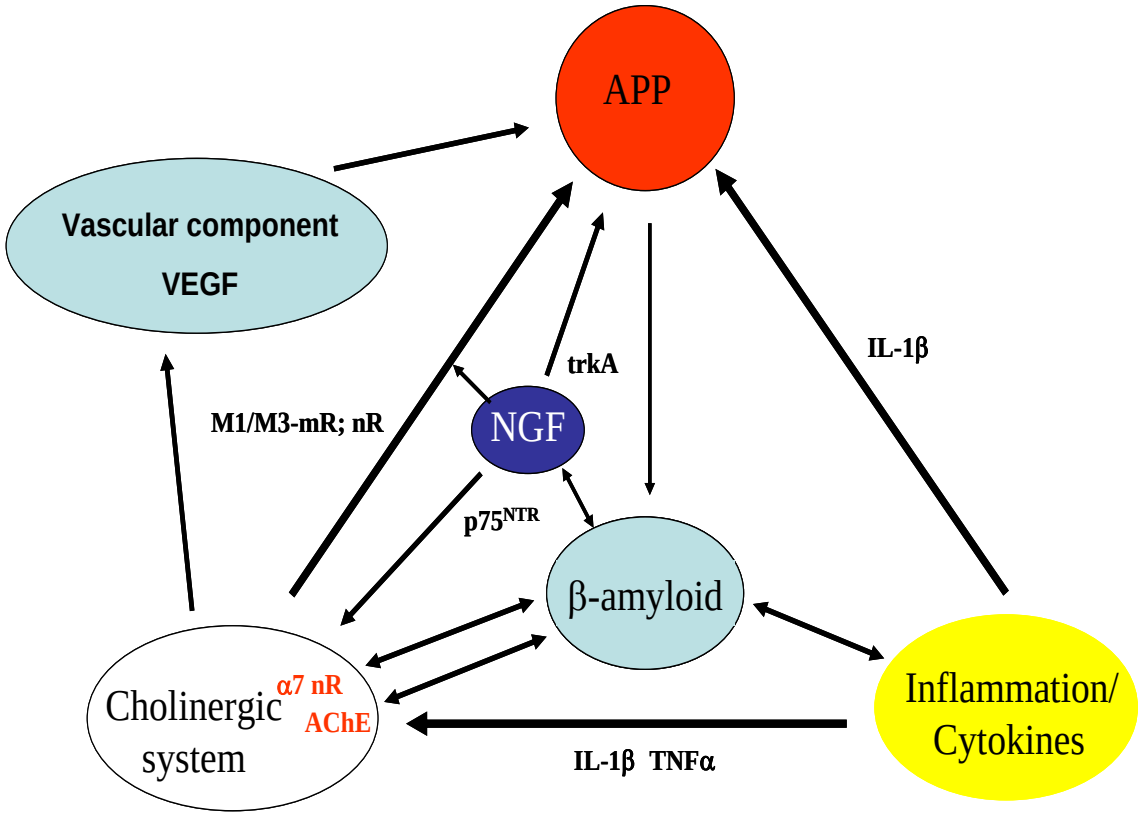
DISEASE	MUTATIONS OF GENES IDENTIFIED SO FAR	LOCATION OF GENES ON CHRO MOSOMES	BRAIN ABNORMALITY	SYMPTOMS	AGE OF ONSET
Alzheimer disease (AD)	PS2 (AD4) PS1 (AD3) unknown unknown	1 14 19 21	plaques made of fragmented brain cells surrounded by amyloid-family proteins, tangles of cytoskeleton filaments	progressive inability to remember facts and events and later to recognize friends and family	71 years
Amyotrophic lateral sclerosis (ALS)	SOD1 (codes for enzyme removing dangerous superoxide radicals)	21	progressive degeneration of motor neuron cells in the spinal cord and brain	loss of motor control which ultimately results in paralysis and death	between 55 and 75 years
Fragile X syndrome	FMR1 (codes for FMRI protein with unknown function)	X	failure of the glutamate synapse formation and elimination	the most common inherited form of mental retardation	1 year
Huntington disease (HD)	HD gene (codes for the protein huntingtin that stimulates expression of BDNF)	4	dilatation of ventricles and atrophy of caudate nucleus and striatum	degenerative neurological disease that leads to dementia	between 30 and 50 years
Rett syndrome	MeCP2 (codes for a protein which controls gene expression in the cell)	X	generalized brain atrophy, decrease in neuronal cell size, increased cell packing density, reduction in cholinergic neurons	loss of purposeful use of hands and speech, wringing hand movements, seizures, mental retardation	6 to 18 months
Williams syndrome	LIM kinase and elastin coding sequences	7	unknown	high competence in language, music and interpersonal relations, with low IQ	At birth

CNGM for modelling epilepsy data



Through optimisation of a GRN within a SNN to match epilepsy data, a GRN model is discovered that points to new gene interactions (with A.Villa and L.Benuskova).

CNGM for Alzheimer's Disease (AD)



CNGM for Modelling and Creation of Cognitive, Emotional Systems

• The CNGM framework allows to represent the complex interaction between parameters and functions at different cognitive levels as follows.

• The lowest ('molecular') level represents sensory information, such as temperature, pressure, odour, sound, image pressure, distance, etc. These 'molecules' can affect the 'genes' in a GRN.

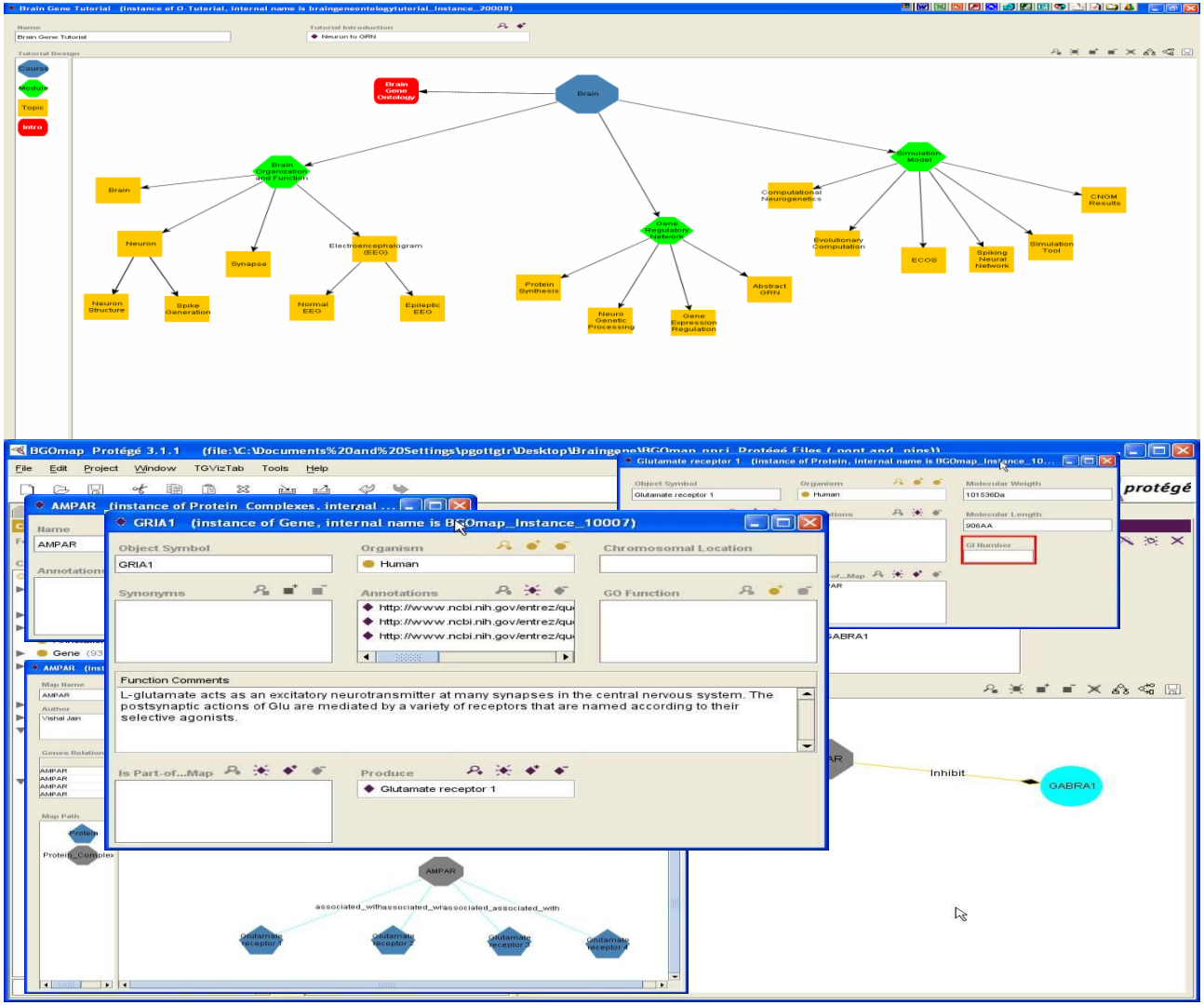
• The GRN model includes parameters that represent neuromodulators (serotonin, dopamine, noradrenalin and acetylcholine) and many other relevant parameters corresponding to the cognitive functions at the highest SNN level. The lowest, sensory information level, can modify the level of the neurotransmitter parameters and other 'genes', but they can be modified also directly through external inputs or through a feedback from the higher cognitive level of SNN.

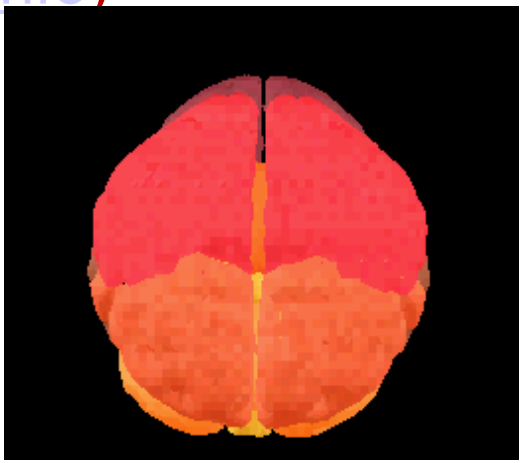
• The spiking patterns of the pSNN are analysed and recognised as a state of cognitive behaviour.

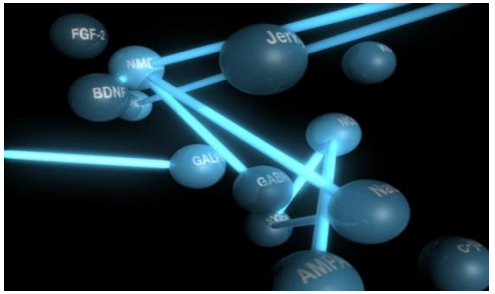
• The structure of the multi-scale CNGM is evolving in both space and time thus allowing for the cognitive system to develop, adapt, learn new functions, change its behaviour and express emotions.

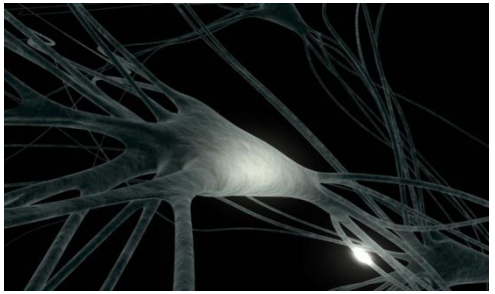
• One of the main features of such pCNGM-based cognitive systems is the *integration of several dynamical systems* that function at different time scales. All these processes will be modelled together in a probabilistic, stochastic way.

Integrated CNGM and brain-gene ontology systems. The KEDRI BGO (www.kedri.info)









5. Future developments

- Modelling evolving connectivity in a large scale CNGM – synaptic ontogenesis;
- Integrated brain-gene ontology with CNGM
- Methods and algorithms for generic tasks of a large scale: finite automata; associative memories;
- Neurogenetic robots;
- Medical decision support systems for personalised risk and outcome prediction of brain diseases:
  - AD;
  - Clinical depression;
  - Stroke and TBI;
  - Bipolar disease (data from the Welcome Trust UK – www.wtccc.org.uk);
- New hardware and software realisations;
- Large scale applications for cognitive function modelling;
- Large scale engineering applications , e.g. cyber security, environmental disaster prediction, climate change prediction, ….
- New Brain- Computer Interfaces (BCI)

KEDRI: The Knowledge Engineering and Discovery Research Institute (www.kedri.info)
Auckland University of Technology (AUT), New Zealand

- Established June 2002
- 4 senior research fellows and post-docs
- 25PhD and Masters students;
- 25 associated researchers
- Both fundamental and applied researc (theory + practice)
- 320 refereed publications
- 5 patents
- Multicultural environment (10 ethnic origins)
- Strong national and international collaboration
- New PhD students are welcome to apply.