

Integrative connectionist learning systems inspired by nature: current models, future trends and challenges

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Abstract The so far developed and widely utilized connectionist systems (artificial neural networks) are mainly based on a single brain-like connectionist principle of information processing, where learning and information exchange occur in the connections. This paper extends this paradigm of connectionist systems to a new trend—integrative connectionist learning systems (ICOS) that integrate in their structure and learning algorithms principles from different hierarchical levels of information processing in the brain, including neuronal-, genetic-, quantum. Spiking neural networks (SNN) are used as a basic connectionist learning model which is further extended with other information learning principles to create different ICOS. For example, evolving SNN for multitask learning are presented and illustrated on a case study of person authentication based on multimodal auditory and visual information. Integrative gene-SNN are presented, where gene interactions are included in the functioning of a spiking neuron. They are applied on a case study of computational neurogenetic modeling. Integrative quantum-SNN are introduced with a quantum Hebbian learning, where input features as well as information spikes are represented by quantum bits that result in exponentially faster feature selection and model learning. ICOS can be used to solve more efficiently challenging biological and engineering problems when fast adaptive learning systems are needed to incrementally learn in a large dimensional space. They can also help to better understand complex information processes in the brain especially how information processes at different information levels interact. Open questions, challenges and directions for further research are presented.

Keywords Connectionist learning systems · Artificial neural networks · Spiking neural networks · Evolving spiking neural networks · Multiple task learning · Multimodal audio-visual information processing · Gene-spiking neural networks · Computational neurogenetic modeling · Quantum spiking neural networks

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1 Integrative adaptive processes in the brain and integrative connectionist learning systems

1.1 The brain as an integrative adaptive system

The brain is a dynamic information processing system that evolves its structure and functionality in time through information processing at different hierarchical levels as shown in Fig. 1: quantum-, molecular (genetic)-, single neuron-, ensemble of neurons-, cognitive-, evolutionary (Kasabov 2007).

At the quantum level, particles, that make every molecule in the material world, are moving continuously, being in several states at the same time that are characterised by probability, phase, frequency, energy (Feynman et al. 1965). These states can change, evolve under certain conditions following the principles of quantum mechanics.

At a molecular level, RNA and protein molecules evolve in a cell and interact in a continuous way, based on the stored information in the DNA and on external factors, and affect the functioning of a cell (neuron) (Crick 1970; Dow et al. 1995; Collado-Vides and Hofestadt 2002).

At the level of a synapse and a single neuron, the internal information processes and the external stimuli in their interplay cause the synapses to change (learning) and the neuron to produce a signal that carries information to be transferred to other neurons (Freeman 2000; Arbib 2003), which is a continuous, evolving process.

At the level of neuronal ensembles, all neurons operate in a “concert”, defining the function of the ensemble through continuous learning, for instance perception of spoken words (Cooper et al. 2004).

At the level of the whole brain, cognitive processes take place in a life-long incremental multiple task/multiple modalities learning mode, such as language and reasoning, and global information processes are manifested, such as consciousness (Grossberg 1982; Chalmers 1996; Taylor 1999; Arbib 2003).

At the level of a population of individuals, species evolve through evolution, changing the genetic DNA code for a better adaptation (Darwin 1859).

The information processes at each hierarchical level from Fig. 1 are very complex and difficult to understand as they evolve all the time, but much more difficult to understand is the interaction between the different levels and how this interaction affect the learning in the brain. It may be that understanding the interaction through its modeling is the key to understanding each level of information processing in the brain and perhaps the brain as a whole. Building computational models that integrate principles from different information levels and in particular, models that are based on connectionist learning, may well be the

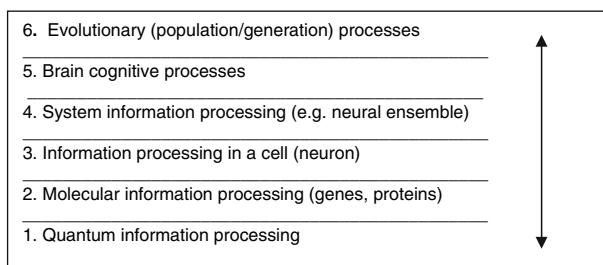


Fig. 1 Levels of integrative evolving information processes in the brain

key towards solving efficiently complex problems. *These models are called here Integrative Connectionist Learning Systems (ICOS)* as described next in the paper.

1.2 Integrative connectionist learning systems (ICOS)

Principles from several levels of information processing and learning as shown in Fig. 1, have been already used to create hybrid connectionist learning systems, to name only few of them:

- evolutionary artificial neural networks (ANN) (Holland 1975; Goldberg 1989; Fogel et al. 1990; Yao 1993; Fogel and Corne 2003);
- adaptive, cognitive ANN models (Carpenter and Grossberg 1990, 1992; Carpenter et al. 1991; Kasabov 1996; Kohonen 1997; Arbib 2003);
- evolving connections systems (ECOS) (Kasabov 2001, 2007; Kasabov and Song 2002);
- quantum-inspired (QI) evolutionary optimization (Han and Kim 2002) and ANN models (Ezhov and Ventura 2000; Narayanan and Meneer 2000; Venayagamoorthy and Gaurav 2005; Kasabov 2006).

Despite the developed so far connectionist systems, there are still no efficient connectionist learning methods for modeling complex brain functions, for adaptive learning of a large number of objects in a multi-dimensional space, for dynamic learning of multiple models in a multidimensional and changing environment, such as an ontology (Ashburner et al. 2000; Kasabov 2002, 2007; Gottgtroy et al. 2006), and for flexible decision support systems (DSS) for solving global and complex problems in a dynamically changing environment, where principles from different information levels (Fig. 1) are properly used in their dynamic interaction and interdependency. Using, for example evolutionary algorithms to train an ANN and adjust the connection weights according to a fixed data set given in advance, does not reflect the nature of learning in the brain (Fogel et al. 1990).

Existing connectionist models still fail to solve generic problems of computational intelligence such as:

- “*the curse of dimensionality*”, i.e. in a large dimensional space existing feature selection algorithms related to ANN models fail to select an optimal set of input variables and to modify this set optimally when new data arrives;
- “*the curse of the local optimum*”, i.e. the models usually reach a local, rather than global optimum solutions;
- “*the curse of multiple modality and multiple task learning*”, i.e. it remains difficult to dynamically integrate multiple models in order to discover common patterns/relationships, features, to make the models share knowledge in order to improve the learning processes (Kasabov 2006, 2007).

We introduce here ICOS, as an extension to the connectionist and the hybrid connectionist systems, being characterized by several principles.

1.2.1 *The principle of integration through interaction*

The principle of *Integration Through Interaction (ITI)* between hierarchical levels of information processing states that information processes at different levels in the information hierarchy interact and influence each other. For example, quantum-, genetic- and

synaptic-information principles are interdependent as they operate on hierarchically embedded objects (e.g. particles, genes, synapses, neurons) even at different time scales.

Below an exemplar *ICOS* model is presented that is based on the *ITI* principle:

A future state Q' of a particle or a group of particles (e.g. ions, electrons, etc.) depends on the current state Q and on the frequency spectrum E_Q of an external signal, according to the Max Planck famous equation:

$$E = h\nu \quad (1)$$

$$Q' = F_Q(Q, E_Q). \quad (2)$$

A future state of a molecule M' or a group of molecules (e.g. genes, proteins) depends on its current state M , on the quantum state Q of the particles, and on an external signal E_M :

$$M' = F_M(Q, M, E_M). \quad (3)$$

A future state N' of a neuron, or an ensemble of neurons will depend on its current state N , on the state of the molecules M , on the state of the particles Q and on external signals E_N

$$N' = F_N(N, M, Q, E_N). \quad (4)$$

And finally, a future neuronal state C' of the brain will depend on its current state C and also on the neuronal N , on the molecular M , and on the quantum Q states of the brain:

$$C' = F_C(C, N, M, Q, E_C). \quad (5)$$

The above theoretical *ICOS* model is based on the following assumptions from Physics, Biology and Cognitive Science:

- A large amount of atoms are characterised by the same quantum properties, possibly related to the same gene/protein expression profile of a large amount of neurons characterised by spiking activity that can be represented as a function.
- A large neuronal ensemble can be represented by a single local field potential (LFP) function.
- A cognitive process can be represented, at an abstract level, as a function F_C that depends on all lower levels of neuronal, genetic and quantum activities.

1.2.2 The principle of evolvability

As the processes shown schematically in Fig. 1 *evolve* (develop, unfold, change) in time through *interaction* between each other, *ICOS* have to have these two principles in their functionality.

The principle of *evolvability* states that a system evolves its structure and functionality through incremental learning from data streams and active interaction with the environment, thus continuously improving its performance (Kasabov 2002, 2007). The evolving process is based on incremental forming of local clusters of data and developing local functions. The evolved local structures and functions represent meaningful patterns that can be interpreted as new information. This principle is fundamental for the brain at all functional levels from Fig. 1 as already discussed. This principle has already been used to create simple ANN, one of them being the evolving connectionist systems (ECOS) (Kasabov 2002, 2007).

ECOS are adaptive, incremental learning and knowledge representation systems, that evolve their structure and functionality from incoming data, where in the core of a system is a connectionist architecture that consists of neurons (information processing units) and connections between them (Kasabov 2001, 2007; Kasabov and Song 2002).

An ECOS learns continuously in time and adapts its structure and functionality through a continuous interaction with the environment and with other systems. The adaptive/evolving learning is defined through:

1. A set of evolving rules.
2. A set of parameters (“genes”) that are subject to change during the system operation.
3. An incoming continuous flow of information, possibly with unknown distribution.
4. Goal (rationale) criteria (also subject to modification) that are applied to optimize the performance of the system over time.

ECOS learning algorithms are inspired by brain-like information processing principles as follows (Kasabov 2002, 2007):

1. ECOS evolve in an open space, where the dimensions of the space can change.
2. They learn via incremental learning, possibly in an on-line mode.
3. They learn continuously in a lifelong learning mode.
4. They learn both as individual systems and as an evolutionary population of such systems.
5. They use constructive learning and have evolving structures.
6. They learn and partition the problem space locally, thus allowing for a fast adaptation and tracing the evolving processes over time.
7. They evolve different types of knowledge representation from data, mostly a combination of memory-based and symbolic knowledge.

At any time of the ECOS continuous incremental learning, meaningful rules can be derived from the structure, which rules represent clusters of data and local functions associated with these clusters, e.g.:

IF <data is in cluster N_{cj} , defined by a cluster center N_j , a cluster radius R_j and a number of examples N_{jex} > THEN <the output function is F_c >

The local rules represent locally learned knowledge.

1.2.3 The principle of multiple modality/multiple task learning

This principle states that a connectionist system consists of multiple interrelated modules that learn different but related information modalities or/and tasks and share information in a dynamic way to speed up the learning process and to improve the system performance and the system adaptation.

One particular type of ANN—spiking neural networks (SNN) is used in this paper for the purpose of building ICOS that are based on the above principles. Section 2 presents a recently developed architecture of an evolving SNN (ESNN) for multimodal learning of audio-visual data and illustrates it on a biometric person authentication case study task. Section 3 introduces ESNN based on both brain and gene information processing principles and illustrates them on a computational neurogenetic modeling task. Section 4 introduces for the first time the main principles of quantum-ESNN, which are ESNN where spikes are represented as quantum bits and quantum Hebbian learning rule is applied.

Future trends and challenges for ICOS are discussed in each section as well as in the concluding Sect. 5.

2 Evolving spiking neural networks (ESNN) for multimodal learning on the case study of audiovisual person authentification

2.1 Spiking neuronal models

Spiking models of a neuron and of neural networks—spiking neural networks (SNN), have been inspired and developed to mimic more biologically the spiking activity of neurons in the brain when processing information (Gerstner and Kistler 2002; Arbib 2003; Izhikevich 2003; Kasabov 2007; Wysoski et al. 2006). SNN represent information as trains of spikes, rather than as single scalars, thus allowing use of such features as frequency, phase, incremental accumulation of input signals, time of activation, etc. (Gerstner and Kistler 2002). This could potentially result in a much higher number of objects (patterns) stored in a model and more flexible processing.

Neuronal dynamics of a spiking neuron are based on the increase in the inner potential of a neuron (post synaptic potential, PSP), after every input spike arrival. When a PSP reaches a certain threshold, the neuron emits a spike at its output (Fig. 2). A wide range of models to simulate neuronal activity have been proposed (Integrate-and-Fire, Spike Response Model, Hodgkin–Huxley Model) (Gerstner and Kistler 2002; Izhikevich 2003; Izhikevich and Desai 2003).

One model—the spike response model (SRM) of a neuron (Gerstner and Kistler 2002; Izhikevich 2003) is described below. It is used here to build an ESNN architecture. A neuron i receives input spikes from pre-synaptic neurons $j \in \Gamma_i$, where Γ_i is a pool of all neurons pre-synaptic to neuron i . The state of the neuron i is described by the state variable $u_i(t)$ that can be interpreted as a total postsynaptic potential (PSP) at the membrane of soma—Fig. 2. When $u_i(t)$ reaches a firing threshold $\vartheta_i(t)$, neuron i fires, i.e. emits a spike. The value of the state variable $u_i(t)$ is the sum of all postsynaptic potentials, i.e.

$$u_i(t) = \sum_{j \in \Gamma_i} \sum_{t_j \in F_j} J_{ij} \varepsilon_{ij} (t - t_j - \Delta_{ij}^{ax}). \quad (6)$$

The weight of synaptic connection from neuron j to neuron i is denoted by J_{ij} . It takes positive (negative) values for excitatory (inhibitory) connections, respectively. Depending on the sign of J_{ij} , a presynaptic spike generated at time t_j increases (or decreases) $u_i(t)$ by an amount of $\varepsilon_{ij} (t - t_j - \Delta_{ij}^{ax})$. Δ_{ij}^{ax} is an axonal delay between neurons i and j which increases with Euclidean distance between neurons.

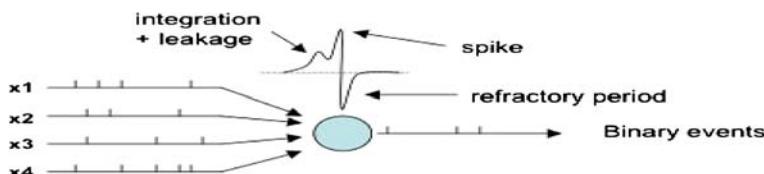


Fig. 2 A schematic representation of a spiking neuron model

The positive kernel $\varepsilon_{ij}(t - t_j - \Delta_{ij}^{ax}) = \varepsilon_{ij}(s)$ expresses an individual postsynaptic potential (PSP) evoked by a pre-synaptic neuron j on neuron i . A double exponential formula can be used

$$\varepsilon_{ij}^{\text{synapse}}(s) = A^{\text{synapse}} \left(\exp \left(-\frac{s}{\tau_{\text{decay}}^{\text{synapse}}} \right) - \exp \left(-\frac{s}{\tau_{\text{rise}}^{\text{synapse}}} \right) \right). \quad (7)$$

The following notations are used above: $\tau_{\text{decay/rise}}^{\text{synapse}}$ are time constants of the rise and fall of an individual PSP; A is the PSP's amplitude; *synapse* represents the type of the activity of the synapse from the neuron j to neuron i , that can be measured and modeled separately for a *fast_excitation*, *fast_inhibition*, *slow_excitation*, and *slow_inhibition*, all integrated in the formula. These types of PSPs are based on neurobiology and have been used for the development of computational neuro-genetic models (Benuskova and Kasabov 2007a, b), where the different synaptic activities are represented as functions of different proteins (neuro-transmitters and neuro-receptors).

External inputs from the input layer are added at each time step, thus incorporating the background noise and/or the background oscillations. Each external input has its own weight $J_{ik}^{\text{ext_input}}$ and amount of signal $\varepsilon_k(t)$, such that:

$$u_i^{\text{ext_input}}(t) = J_{ik}^{\text{ext_input}} \varepsilon_{ik}(t). \quad (8)$$

It is optional to add some degree of Gaussian noise to the right hand side of the equation above to obtain a stochastic neuron model instead of a deterministic one.

2.2 Evolving spiking neural networks (ESNN)

SNN models can be built with the use of the above spiking neuron model. Spiking neurons within a SNN can be either excitatory or inhibitory. Lateral connections between neurons in a SNN may have weights that decrease in value with distance from neuron i for instance, according to a Gaussian formula while the connections between neurons themselves can be established at random. SNN can be used to build biologically plausible models of brain functions.

Evolving SNN (ESNN) evolve/develop their structure and functionality in an incremental way from incoming data based on the following principles (Wysoski et al. 2006; Benuskova and Kasabov 2007a, b; Kasabov 2007):

- (1) New spiking neurons are created to accommodate new data, e.g. new output classes, such as faces in a face recognition system.
- (2) Spiking neurons are merged if they represent the same concept (class) and have similar connection weights.

In Wysoski et al. (2006) an ESNN architecture is proposed. In this architecture the change in a synaptic weight is achieved through a simple rule:

$$\Delta w_{j,i} = \text{mod}^{\text{order}(j)} \quad (9)$$

where $w_{j,i}$ is the weight between neuron j and neuron i , $\text{mod} \in (0,1)$ is the modulation factor, $\text{order}(j)$ is the order of arrival to neuron i of a spike produced by neuron j . For each training sample, we use the *winner-takes-all* approach, where only the neuron that has the highest PSP value has its weights updated.

The postsynaptic threshold (PSP_{Th}) of a neuron is calculated as a proportion $c \in [0, 1]$ of the maximum postsynaptic potential (PSP) generated with the propagation of the training sample into the updated weights, such that:

$$PSP_{Th} = c \max(PSP). \quad (10)$$

The procedure for training the network and creating new neurons is proposed in Wysoski et al. (2006) and is summarized below in a pseudo-code:

```

Until weights converge
For all input vectors in the training set
For each input vector
Create (evolve) a new neuron
Propagate the input vector into the network
Train the newly created neuron using equations (9) and (10)
Calculate the similarity between weight vectors of newly created neuron and existent
neurons
If similarity > Threshold
Merge newly created neuron with the most similar neuron using (11) and (12).

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To merge a newly created neuron with an existing neuron, the weights W of the existing neuron n are updated calculating the average as

$$W = \frac{W_{new} + N_{Frames}W}{1 + N_{Frames}} \quad (11)$$

where N_{Frames} is the number of frames previously used to update the respective neuron. Similarly, the average is also computed to update the corresponding PSP_{Th} :

$$PSP_{Th} = \frac{PSP_{Thnew} + N_{Frames}PSP_{Th}}{1 + N_{Frames}}. \quad (12)$$

Creating and merging neurons based on localized incoming information and on system's performance are main operations in the above architectures that make them continuously *evolvable*.

2.3 ESNN for multimodal learning of auditory and visual information

A detailed architecture of an audiovisual crossmodal integrative ESNN system proposed in Wysoski et al. (2007a, b) is shown in Fig. 3.

The auditory ESNN learning module is described in Wysoski et al. (2007a, b) and the visual ESNN learning module—in Wysoski et al. (2008). In Fig. 3 two neurons (OR and AND) constitute a supramodal layer. Each spiking neuron, similarly to the neurons that compose the ESNNs of individual modalities, has the behavior defined in the above subsection. Having supramodal neurons with modulation factor $mod = 1$ and setting all the incoming excitatory connection weights W to 1, the $PSPTh$ that implements the OR gate for two modalities is equal to 1. The neuron implementing the AND gate receives $PSPTh = 2$. Notice that, it is only possible to set deterministically these parameters because of the properties of the neurons we are using (a neuron can spike only once at any stage of computation).

In this system we effectively model the crossmodal learning and influence through the modification of $PSPTh$ in the layers responsible for decision making within each modality.

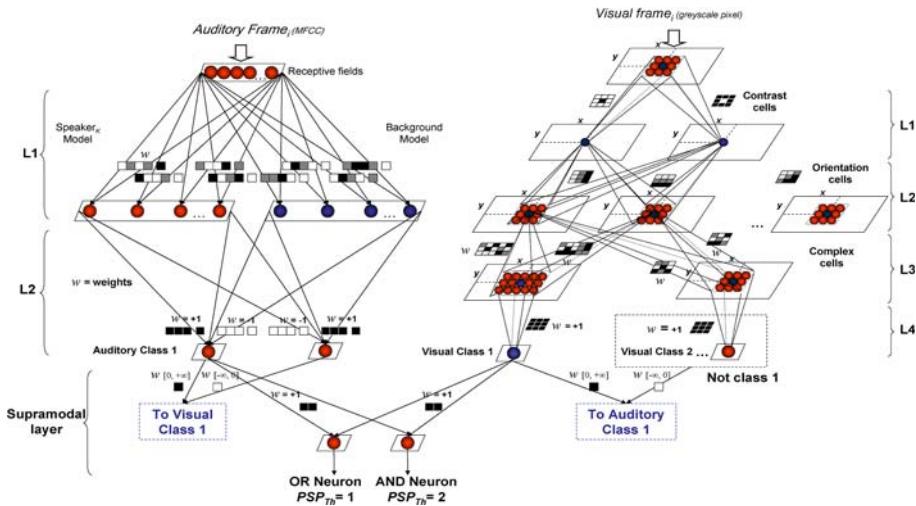


Fig. 3 Crossmodal audiovisual integrative ESNN

More precisely, we modify the PSPTh in layer 1 (L1) neurons in the auditory model and layer 3 (L3) neurons in the visual model. We use the following crossmodal parameters to denote the strength of the crossmodal influences: CMAVexc (audio to video excitation), CMAVinh (audio to video inhibition), CMVAexc (video to audio excitation), CMVAinh (video to audio inhibition), which are implemented with a proportional change in the usual PSPTh values as:

$$PSP_{ThNew} = PSP_{ThOld}(1 + CM_{exc/inh}) \quad (13)$$

where CMexc/inh is negative for crossmodal excitatory influence and positive for inhibitory influence. The crossmodal influence starts from the period one individual modality produces a result and lasts until all modalities finish processing.

Notice that, in the simplest case, setting crossmodal coupling to zero, we will have effectively each of the modality processed separately, with a simple OR/AND fusion of opinions.

2.4 A case study implementation on a person authentication task utilizing audiovisual person information

In Wysoski et al. (2007a, b) an implementation is presented of the integrative learning of audiovisual modalities with a network of spiking neurons and used for evaluation the VidTimit dataset (Sanderson and Paliwal 2004), which contains video and audio recordings of 43 persons. Our test setup deals specifically with the audiovisual person authentication learning problem. Thus, a person is authenticated by a personally trained system based on spoken phrases and the corresponding facial information as the utterance is recorded (captured in frontal view).

Figure 4 shows the best performance obtained on each individual modality and also on the cross-modal mode of operation. While the best total error (TE) for the face authentication is 21%, the auditory authentication is $TE \approx 38\%$ (varying values of L1 PSPTh in the

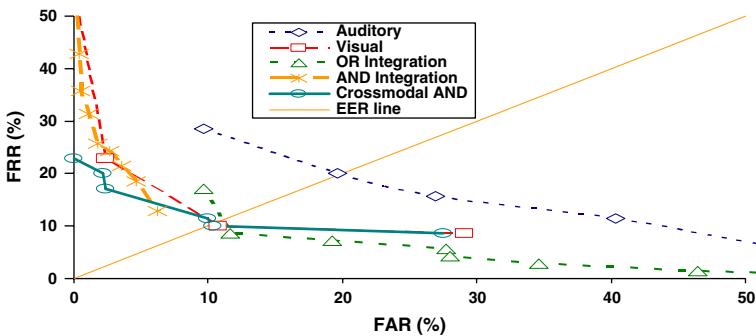


Fig. 4 Performance of individual modalities for different values of auditory (L1 PSPTh) and visual parameters (L3 PSPTh) and performance of the OR and AND integration of modalities with a supramodal layer of spiking neurons. FAR is the false acceptance rate, FRR is the false rejection rate. Comparison between individual modes (auditory and visual) and different integration scenarios in the FAR–FRR plane. EER is the equal error rate, where $\text{FAR} = \text{FRR}$

auditory system and L3 PSPTh in the visual system). Having in mind that the parameters have been optimized by hand, in Fig. 4 we can see the potential advantages of the integration module. When the system needs to operate with low FAR levels (below 10%), the Crossmodal AND provide lower FRR than any singular modality. When the system requires operating with low FRR (below 10%), OR integration can be used instead, which gives lower FAR for the same FRR levels (see Wysoski et al. 2007a, b).

2.5 Challenges for ESNN

The following theoretical aspects and models of ESNN need to be further investigated and developed:

1. Neuronal coding, i.e. how information is efficiently encoded and decoded as spikes. This is to investigate encoding through precise spiking time, data transmission efficiency (maximum data transmission), and minimum processing time.
2. Learning, i.e. changing the connection weights based on incoming data. That relates to creating new evolving learning algorithms where connections and neurons are created incrementally.
3. Model optimization—to explore the principles of minimum energy consumption (minimum number of spikes).
4. Noise robustness—how robust the ESNN are to incrementally introduced noise.
5. Synchronicity versus asynchronicity as a way to define the dynamic behavior of the system.
6. Event-driven (only neurons that receive a spike are updated) versus clock-driven computation (all neurons are updated at a given clock step). The event-driven approach is particularly appropriate for a very large number of neurons with low spiking rate that would increase the patterns stored in the ESNN.
7. ESNN methods and systems to analyze olfactory and gustative sensory information.
8. Creating an ESNN methodology and a software environment to combine systems of different modalities of information to be tested on multimodal biometric case study

problems of face and speech modality for the recognition of a very large number of persons (e.g., more than 10,000).

- 9. Implementing ESNN systems on a massively parallel FPGA hardware machine to achieve several orders of magnitude faster pattern recognition of a large number of objects for real time applications (Johnston et al. 2005).
- 10. ESNN-based large associative visual database memories that perform content-based visual information retrieval. This needs to be tested on a case study of real time moving object recognition of a large number of objects.
- 11. Using ESNN for brain data analysis, modeling and discovery. For example, the brain-gene-simulation ontology (BGSO) (Kasabov et al. 2007) can be further extended with the addition of ESNN simulation models and brain-gene data on learning and memory formation. The BGSO will permit us to search and retrieve relevant data and construct ESNN models for different tasks of modelling and discovery of brain-gene relationships (Benuskova and Kasabov 2007a, b; Kasabov 2007).
- 12. Using ESNN technologies for building embedded systems into robots, cars, human body, etc.

3 Gene-ESNN integrative learning connectionist systems for computational neurogenetic modelling

3.1 General notions

With the advancement of molecular and brain research technologies more and more data and information is being made available about the genetic basis of some neuronal functions (see for example: the brain-gene map of mouse <http://alleninstitute.org>; the brain-gene ontology BGO at <http://www.kedri.info>). This information can be utilized to create biologically plausible ANN models of brain functions and diseases that include models of gene interaction. This area integrates knowledge from computer and information science, brain science, molecular genetics and it is called in (Kasabov and Benuskova 2004; Benuskova and Kasabov 2007a, b) computational neurogenetic modeling (CNGM).

Several CNGM models have been developed so far varying from modeling a single gene in a biologically realistic ANN model (Benuskova and Kasabov 2007a, b), to modeling a set of genes forming an interaction gene regulatory network (GRN) (Benuskova and Kasabov 2007a, b).

3.2 A computational neuro-genetic model (CNGM) that integrates GRN within an ESNN model

The main idea behind the model, proposed in Kasabov and Benuskova (2004), is that interaction of genes in neurons affect the dynamics of the whole ANN through neuronal parameters, which are no longer constant, but change as a function of gene/protein expression. Through optimization of the GRN, the initial gene/protein expression values, and the ANN parameters, particular target states of the ANN can be achieved, so that the ANN can be tuned to model real brain data in particular—Fig. 5.

This idea is further developed and illustrated in Benuskova and Kasabov (2007a, b). The behavior of the SNN is evaluated by means of the local field potential (LFP), thus making

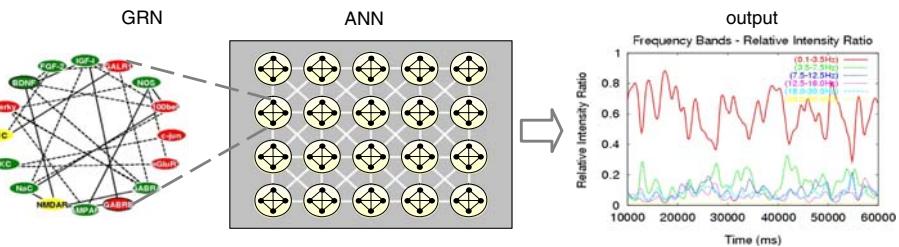


Fig. 5 A CNGM, where a GRN is used to represent the interaction of genes, and an ESNN is employed to model a brain function. The model output is compared against real brain data for validation of the model and for verifying the derived gene interaction GRN after model optimization is applied (from Benuskova and Kasabov 2007a, b)

it possible to attempt modeling the role of genes in different brain states, where EEG data is available to test the model. A standard FFT signal processing technique is used to evaluate the SNN output and to compare it with real human EEG data.

In general, we consider two sets of genes—a set G_{gen} that relates to general cell functions, and a set G_{spec} that defines specific neuronal information-processing functions (receptors, ion channels, etc.). The two sets form together a set $G = \{G_1, G_2, \dots, G_n\}$. We assume that the expression level of each gene is a nonlinear function of expression levels of all the genes in G :

$$g_j(t + \Delta t') = \sigma \left(\sum_{k=1}^n w_{jk} g_k(t) \right). \quad (14)$$

We can also assume for simplicity that: (1) one protein is coded by one gene; (2) relationship between the protein level and the gene expression level is nonlinear; (3) protein levels lie between the minimal and maximal values. Thus, the protein level is expressed by:

$$p_j(t + \Delta t) = \left(p_j^{\max} - p_j^{\min} \right) \sigma \left(\sum_{k=1}^n w_{jk} g_k(t) \right) + p_j^{\min}. \quad (15)$$

The delay constant introduced in the general formula above corresponds to the delay caused by the gene transcription, mRNA translation into proteins and posttranslational protein modifications, and also the delay caused by gene transcription regulation by transcription factors.

Some proteins and genes are known to be affecting the spiking activity of a neuron represented in a SNN model by neuronal parameters, such as fast_excitation, fast_inhibition, slow_excitation, and slow_inhibition. Some neuronal parameters and their correspondence to particular proteins are summarized in Table 1.

Besides the genes coding for the proteins mentioned above and directly affecting the spiking dynamics of a neuron, a GRN model can include other genes relevant to a problem in hand, e.g. modeling a brain function or a brain disease, for example: c-jun, mGLuR3, Jerky, BDNF, FGF-2, IGF-I, GALR1, NOS, S100beta (Benuskova et al. 2006).

The goal of the CNGM from Fig. 5 is to achieve a desired SNN output through optimization of the model parameters. The LFP of the SNN, defined as $LFP = (1/N) \sum u_i(t)$, by means of FFT is evaluated in order to compare the SNN output with the EEG signal

Table 1 Neuronal parameters and related proteins

Neuronal parameter	Protein
Amplitude and time constants of	
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

Abbreviations: psp = postsynaptic potential, ampar = (amino-methylisoxazole-propionic acid) ampa receptor, nmdar = (n-methyl-d-aspartate acid) nmda receptor, gabra = (gamma-aminobutyric acid) gabaa receptor, gabrb = gabab receptor, scn = sodium voltage-gated channel, kcn = kalium (potassium) voltage-gated channel, clc = chloride channel, pv = parvalbumin

analyzed in the same way. It has been shown that brain LFPs in principle have the same spectral characteristics as EEG.

In order to find an optimal GRN within the SNN model, so that the frequency characteristics of the LFP of the SNN model are similar to the brain EEG characteristics, the following evolutionary computation procedure is used:

1. Generate a population of CNGMs, each with randomly, but constrained, generated values of coefficients for the GRN matrix W , initial gene expression values $g(0)$, initial values of SNN parameters $P(0)$, and different connectivity.
2. Run each SNN model over a period of time T and record the LFP.
3. Calculate the spectral characteristics of the LFP using FFT.
4. Compare the spectral characteristics of SNN LFP to the characteristics of the target EEG signal. Evaluate the closeness of the LFP signal for each SNN to the target EEG signal characteristics. Proceed further according to the standard GA algorithm to find a SNN model that matches the EEG spectral characteristics better than previous solutions.
5. Repeat steps 1–4 until the desired GRN and SNN model behavior is obtained.
6. Analyze the GRN and the SNN parameters for significant gene patterns that cause the SNN model to manifest similar spectral characteristics as the real data.

The proposed CNGM modeling framework can be used to find patterns of gene regulation related to brain functions.

3.3 A case study of modeling a brain function

In Benuskova and Kasabov (2007a, b) some preliminary results of analysis performed on real human interictal EEG data are presented. The model performance and the real EEG data are compared for the following relevant to the problem sub-bands: delta (0.5–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz), beta 1 (12.5–18 Hz), beta 2 (18–30 Hz), gamma (above 30 Hz). This particular SNN had an evolved GRN with only 5 genes out of 16 (s100beta, GABRB, GABRA, mGluR3, c-jun) and all other genes having constant expression values. A GRN is obtained that has a meaningful interpretation and can be used to model what will happen if a gene/protein is suppressed by administering a drug, for example.

In ICOS for CNGM new genes can be added to the GRN model at a future time, in addition to the new spiking neurons and connections created incrementally in the ESNN. Developing new ICOS to model brain functions and brain diseases, such as epilepsy, Alzheimer, Parkinson disease, Schizophrenia, mental retardation and others is a challenging problem for a future research (Benuskova and Kasabov 2007a, b).

3.4 Open questions and challenges for ICOS in CNGM

There were some questions that emerged from the first ICOS for CNGM (ICOS-CNGM):

1. How many different GRNs would lead to similar LFPs and what do they have in common?
2. What neuronal parameters to include in an ICOS-CNGM and how to link them to activities of genes/proteins?
3. What genes/proteins to include in the model and how to represent the gene interaction over time within each neuron?
4. How to integrate in time the output activity of the ICOS-CNGM and the genes as it is known that neurons spike in millisecond intervals and the process of gene transcription and translation into proteins takes minutes?
5. How to create and validate an ICOS-CNGM in a situation of insufficient data?
6. How to measure brain activity and the ICOS-CNGM activity in order to validate the model?
7. What useful information (knowledge) can be derived from an ICOS-CNGM?
8. How to adapt incrementally an ICOS-CNGM in a situation of new incoming data about brain functions and genes related to them?
9. Is it possible to create a truly adequate ICOS-CNGM of the whole brain? Would gene-brain maps help in this respect (see <http://alleninstitute.org>)?
10. How can dynamic ICOS-CNGM be used to trace over time and predict the progression of a brain diseases, such as epilepsy and Parkinson's?
11. How to use ICOS-CNGM to model gene mutation effects?
12. How to use ICOS-CNGM to predict drug effects?
13. How ICOS-CNGM can help understand better brain functions, such as memory and learning?
14. What engineering problems can be efficiently solved with the use of a brain-gene inspired ICOS?

4 Quantum evolving spiking neural networks (QESNN)

4.1 The quantum principles of *superposition* and *entanglement*

The quantum principle of *superposition* of states (Feynman et al. 1965; Hey 1999) assumes that a system is in a superposition of all of its possible states at the same time defined by a probability density amplitude and all states can be processed in parallel in order to optimize a global objective function.

The potential for using quantum computing principles became apparent when Shor (1997) and Grover (1996) published their specialized quantum algorithms that performed exponentially faster than any traditional algorithms.

The smallest information unit in today's digital computers is one bit, existing as state '1' or '0' at any given time. The corresponding analogue on a quantum computer is represented by a quantum bit or qbit (Hey 1999). Similar to classical bits a qbit may be in '1'-state or '0'-state but also in a superposition of both states. A qbit state $|\Psi\rangle$ can be described as:

$$|\Psi\rangle = \alpha|0\rangle + \beta|1\rangle \quad (16)$$

where α and β are complex numbers defining probabilities which of the corresponding state are likely to appear when a qbit is read (measured, collapsed). $|\alpha|^2$ and $|\beta|^2$ give the probability of a qbit being found in state '0' or '1' respectively. Normalization of the states to unity guarantees:

$$|\alpha|^2 + |\beta|^2 = 1 \quad (17)$$

at any time. The *qbit* is not a single value entity, but is a function of parameters which values are complex numbers. After the loss of coherence the *qu-bit* will "collapse" into one of the states $|0\rangle$ or $|1\rangle$.

In order to modify the probability amplitudes *quantum gates* can be applied to the states of a qbit. A quantum gate is represented by a square matrix operating on the amplitudes $|\alpha|$ and $|\beta|$ with the only condition that the operation is reversible. Such gates are: NOT-gate, rotation gate, Hadamard gate (Hey 1999).

Another quantum principle is *entanglement*—two or more particles, regardless of their location, can be viewed as "correlated", undistinguishable, "synchronized", coherent. If one particle is "measured" and "collapsed" it causes for all other particles to "collapse" too. An example is a laser beam consisting of millions of photons.

Quantum systems can be described by a probability density ψ that exists in a Hilbert space.

4.2 Principles of QESNN

Recent research activities focus on using quantum principles for ANN. Considering quantum ANN seems to be important for at least two reasons. There is evidence for the role that quantum processes play in the living brain. Roger Penrose argued that a new physics binding quantum phenomena with general relativity can explain such mental abilities as *understanding, awareness* and *consciousness* (Penrose 1994). The second motivation is the possibility that the field of classical ANN could be generalized to the promising new field of quantum computation. Both considerations suggest a new understanding of mind and brain functions as well as new unprecedented abilities in information processing. Ezhov and Ventura are considering the quantum neural networks as the next natural step in the evolution of neurocomputing systems (Ezhov and Ventura 2000).

Several quantum inspired ANN models have been proposed and illustrated on small examples. In Venayagamoorthy and Gaurav (2005) a quantum evolutionary algorithm is used to train a MLP ANN. Narayanan and Meneer simulated classical and quantum inspired ANN and compared their performance on small scale examples (Narayanan and Meneer 2000). These methods unfortunately did not go beyond a fixed structure ANN and therefore have only illustrative meaning rather than a practical one.

Here a QESNN learning architecture is proposed based on six principles (Q1–Q6). Creating and merging neurons based on localized incoming information and on system's

performance are main operations in the above architectures that make them continuously *evolvable*.

4.2.1 (Q1) A quantum representation of a spike

A spike at any time is both present (1) and not present (0), which is represented as a quantum bit (qbit) defined by a probability wave function. When the spike state is evaluated, it is either present or not present. Representing a spike as a quantum entity is supported by the quantum character of the operation of the *ion channels in the synapses*.

To modify the probability amplitudes of a spike we suggest *the rotation gate* as it seems adequate when considering the cylindrical shape of the ion channels:

$$U(\theta) = \begin{bmatrix} \cos \theta & -\sin \theta \\ \sin \theta & \cos \theta \end{bmatrix}. \quad (18)$$

A spike arriving at a moment t at each synapse S_{ij} connecting a neuron N_i to a neuron N_j is represented as a qbit $Q_{ij}(t)$ with its probability $\beta_{ij}(t)$ for a pre-synaptic spike to occur. A neuron N_j is represented as a qbit vector, representing all m synaptic connections to this neuron:

$$\begin{bmatrix} \alpha_1 & \alpha_2 & \dots & \alpha_m \\ \beta_1 & \beta_2 & \dots & \beta_m \end{bmatrix}. \quad (19)$$

4.2.2 (Q2) A quantum integrate and fire model of a spiking neuron

At a time t all pre-synaptic qbits representing a neuron are collapsed into spikes (or no spikes) and their cumulative input $u_i(t)$ to the neuron N_i is calculated:

$$u_i(t) = \sum_{j=1, m} Q_{ij}(t) w_{i,j}(t) \quad (20)$$

where $Q_{ij}(t)$ is the “collapsed” value of the qbit $Q_{ij}(t)$ and $w_{i,j}(t)$ is the connection weight of the j th synapse.

If $u_i(t)$ is above a spiking threshold $\theta(t)$, a quantum output spike is produced by N_i with a probability $P_i(t)$:

$$\text{If}(u_i(t) > \theta(t)) \rightarrow N_i \text{ Spike}. \quad (21)$$

The probability $P_i(t)$ can be considered as a constant unless there is specific requirement for a more biologically plausible model.

4.2.3 (Q3) A quantum Hebbian learning rule

The probability $\beta_{ij}(t)$ of a pre-synaptic spike to occur increases if both N_i and N_j spike depending on the time interval between the two spikes; or $\beta_{ij}(t)$ decreases otherwise, which changes are calculated using the rotation gate (18).

In a more detailed model, $\beta_{ij}(t)$ will depend on the strength and the frequency of the spikes from N_i , on the distance D_{ij} , on many other physical and chemical parameters that are ignored in this model but can be added if necessary.

4.2.4 (Q4) Quantum probabilistic encoding of input information

An input vector $Xp(t)$ presented to the inputs of a QESNN is represented as a qbit vector, each qbit Q_k representing the probability $\beta k(t, t + dt)$ for a spike at the input I_k at any discrete time from the time interval $(t + dt)$. The values $\beta k(t, t + dt)$ depend on the real input values.

Example 1: An input vector $[7, 1, 2]$ will be represented as a vector of probabilities $[\beta 1(t, t + dt), \beta 2(t, t + dt), \beta 3(t, t + dt)] = [0.7, 0.1, 0.2]$.

Example 2: If QESNN inputs represent frequency components of the input vectors, this frequency can be used to calculate the quantum input probabilities.

Example 3: If input signals arrive at the inputs at different times as spikes, e.g. from sensors, there is no need for another encoding of the input information.

4.2.5 (Q6) Quantum input feature selection

A population of n qbit individuals at time t can represent the use of each input variable x_1, x_2, \dots, x_n (Defoin-Platel et al. 2007a, b): 0—a variable is not used in the current QESNN model, 1—the variable is used:

$$Q(t) = \{q_1^t, q_2^t, \dots, q_n^t\} \quad (22)$$

where n is the size of the population and the probability $\beta_i(t)$ defining the use of variable x_i in the current QESNN model. In Defoin-Platel et al. (2007a, b) a versatile quantum inspired evolutionary algorithm is proposed applicable to feature selection for an ANN model as illustrated in the case study below.

4.3 A case study

The proposed in Defoin-Platel et al. (2007a, b) versatile quantum inspired evolutionary algorithm (VQIEA) is applied for feature selection (time lags) of an ANN time series prediction model across hundreds of time series. This algorithm shows a fast convergence towards a global optimum solution—see Fig. 6. It is superior in terms of number of evaluations and accuracy than the classical evolutionary computation algorithms. It is applicable for a large number of variables (e.g. thousands), while the classical exhaustive search algorithm is not practical. For an example of 1,000 variables, it is required 2^{1000} evaluations to be performed using the classical bit-representation if all possible combinations of these variables need to be tested for an optimal solution. If one evaluation is performed for 1 s on a contemporary computer, this means approximately 10^{294} years of testing, which is impractical. Using qbits and the VQIEA, to reach an optimal solution for some class of optimization problems it will require only approximately 1,000 tests. In this respect the VQIEA is exponentially faster than the classical computation.

4.4 Challenges for ICOS and QESNN in particular

Many open questions and challenges need to be addressed in the future related to ICOS and QESNN in particular, such as:

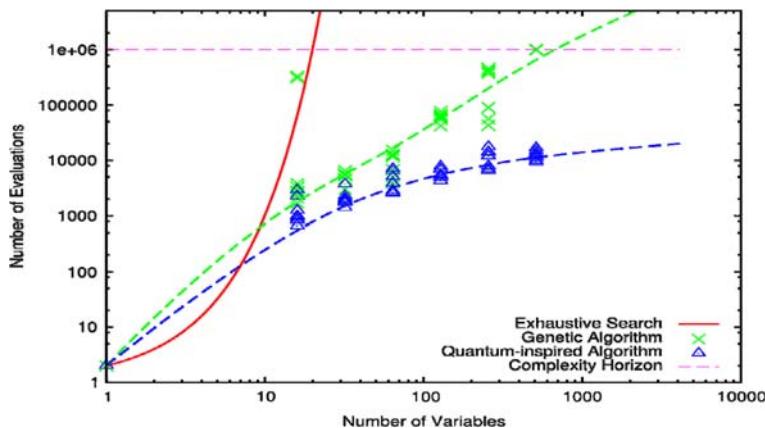


Fig. 6 A quantum inspired evolutionary algorithm based on the superposition principle performs exponentially faster and more accurately than the classical algorithms when evaluating combinations of variables for a modelling task with a large number of variables (from Defoin-Platel et al. 2007a, b)

1. How quantum processes affect the functioning of a living system in general and how can this knowledge be efficiently used for computational modelling?
2. How quantum processes affect cognitive and mental functions?
3. Is it true that the brain is a quantum machine—working in a probabilistic space with many states (e.g. thoughts) being in a superposition all the time and only when we formulate our thought through speech or writing, then the brain “collapses” in a single state?
4. Is fast pattern recognition in the brain, involving far away segments, a result of both parallel spike transmissions and particle entanglement?
5. Is communication between people and between living organisms in general, a result of entanglement processes?
6. How does the energy in the atoms relate to the energy of the proteins, the cells and the whole brain?
7. How to implement the QESNN in order to benefit from their high speed and accuracy? Should we wait for the quantum computers to be realized as hardware some years from now, or we can implement QESNN efficiently on specialized computing devices based on classical principles of physics?

5 Conclusion and future directions

Several reasons can be given in support to the research in integrating principles from quantum-, molecular-, and brain information processing into ICOS. This may lead to a better understanding of neuronal-, molecular-, and quantum information processes. This may lead to new computer devices—million times faster and more accurate than the current ones. At the nano-level of microelectronic devices, quantum processes would have a significant impact and new methods of computation would be needed anyway.

This paper presents some ICOS inspired by principles from different levels of information processing in the brain—including neuronal level, gene/protein level, and quantum level, and argues that models that integrate principles from different levels of information

processing would be useful tools for a better understanding of brain functions and for the creation of more powerful methods and systems of computational intelligence.

Further directions are:

1. Building a brain-gene-quantum ontology system that integrates facts, information, knowledge and models of different levels of information processing in the brain and their interaction.
2. Building novel brain-, gene-, and quantum inspired ICOS models, studying their characteristics and interpreting the results.
3. Applying ICOS to solving complex problems in neuro-informatics and brain diseases, bioinformatics and cancer genetics, multimodal information processing and biometrics.

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