Emergent creativity in declarative memories

A. Horzyk
Dept. Automatics and Biomedical Engineering
AGH University of Science and Technology
Cracow, Poland
horzyk@agh.edu.pl

J. A. Starzyk and Basawaraj
School of EECS
Ohio University,
Athens, OH, USA
{starzykj, basawaraj.basawaraj.1}@ohio.edu

***Abstract*—This paper presents a structural organization of declarative memories using a new model of spiking neurons. Using this model we propose a self-organizing mechanism to build episodic and semantic memories on the cognitive level. Neurons in this approach represent symbolic concepts that are stored and associated with each other based on the observed events in the environment. We demonstrate that the associative neuron and their synaptic connections yield memories that are capable to recognize sequences of events and therefore can be a foundation for episodic memories. We also demonstrate that semantic memories build using these neuron models are capable to store the knowledge and respond in a novel, creative way to input stimuli. The paper presents the developed associative neuron model, neural network organization that results from input sequences, and simulation results.**

Keywords — associative neurons; declarative memory; episodic memory; semantic memory; emergent creativity.

I. Introduction

Declarative memories are used in cognitive systems to store past episodes, experiences and knowledge that can be cognitively explained [1]. They are an essential functional component of any cognitive system, supporting understanding, planning, anticipation, procedural learning, and cognitive support for desired actions. Declarative memories can be divided into episodic and semantic memories that complement each other in their operations and concept forming. Concept forming results from observations of activities in the external world, where sensory inputs activate lower levels of the sensory neurons hierarchy and objects are recognized and categorized using so called symbol grounding process [2]. Full treatment of building object representations and symbol grounding is beyond the scope of this paper, thus in further discussion we assume symbolic representations of the perceived objects, actions and goals.

Semantic memory is a repository of knowledge in a cognitive system, and its structure gradually emerges from the learning process. Neurons in the semantic memory build synaptic connections between associated concepts representing their relationships between each other and relations to cognitive agent objectives [3]. Episodic memory uses the concepts stored in the semantic memory to register the personal history of events located in time and space [4]. Episodic memory supports learning in the semantic memory by providing a recollection of past events. In return, semantic memory provides a wider context and understanding of the observed scenes, and using associations to the observed events it may trigger associated events that could suggest alternative solutions to a problem, guiding a cognitive system to a successful completion of its goals.

Semantic memory is critical for creative approach to problem solving. By focusing attention on a specific concept or idea, it may create new ideas and allow evaluation of thoughts related to an intrinsic goal of the cognitive system. Such an approach was suggested in the motivated learning cognitive architecture (MLECOG) [5] that used attention focus, attention switching, intrinsic motivation and goal creation to solve machine's problems. Semantic and episodic memories are essential functional components of the MLECOG architecture. Their functionalities were described in [5] in relation to other functional blocks like sensory and motor processing, working memory, motivations and goal creation, and motor control, however there was no specific recommendation for how these memories should be organized.

In this paper we provide a unifying approach to organization of episodic and semantic memory, based on a new model of associative neurons used in a self-organizing structure of these memories. An associative neuron model has been proposed in [6] and was used to obtain synaptic connections between concepts presented to the system in input sequences (e.g. sentences represented by sequences of words). A neural network that resulted from the process of associative neuron synaptic connection building demonstrated emergent creativity, and was able to classify, categorize and answer questions related to sequences used during a training process. This associative neuron model was modified in this work by changing neuron's activation threshold to reflect frequency of its use, adopting neurons self-organization to requirements of episodic memory, considering influence of fanout on neuron activation, and introducing synaptic fatigue.

We demonstrate how such a modified associative neuron can be used to build an integrated declarative memory and test its properties. Organization of semantic and episodic memories and their internal dynamics are different, however they share the same model of neurons and self-organizing principles. We demonstrate emerging creativity of the obtained memories and show how they cooperate.

II. Associative Neuron

A. Associative Neuron

Artificial neural networks (ANN) are usually treated as universal nonlinear approximation systems that implement projection $y = f(x)$, where x is an input vector and f is a

nonlinear function of many variables [7]. The function f computes an output on the basis of the weighted sum of inputs and can be used for classification, regression or prediction. Artificial neurons can be connected in various ways however the most popular is a multilayer structure. The biological nature of neurons is rarely used in computational intelligence. On the other hand, spiking neurons, that better model biological neurons, are seldom used in computational intelligence because of the difficulties to train them and model efficiently [8].

The fundamental question about modelling neurons is what impact a neuron should have on its surroundings defined by the connected elements and the other neurons that are close in space. There are no general rules in artificial neural networks that conditionally define plasticity of neurons, provide new neurons or new connections and change the structure or parameters of already created neurons and synapses. Most methods adjust a structure experimentally, adding new elements when the result of training are insufficient or removing them when a network overfits [7]. In this work, we use a biologically plausible mechanism which is defined by activations of neurons and time that elapses between these activations. The proposed associative model of neuron focuses on these properties of biological neurons that enable them to cooperate in representation of frequent combinations of input signals. Input signals can have an external sensor origin or be internally produced as a result of neural activations. The associative model also defines a role of a neuron in a neural network structure. In this model, each neuron represents these combinations of input signals which activate it. The combinations of input signals can be spread over time. It means that input signals are successively added over time. This is balanced by automatic recovery processes that relax or refract neurons over time. In this model the neuron's activation threshold models sensitivity of a neuron to various combinations of input stimuli. The sensitivity of each neuron allows them to specialize and be not reactive to many combinations of input stimuli except those which the neurons represents and reacts to. Variable and conditionally updated thresholds representing sensitivity of neurons also allow for controlling the set of input combinations that are represented by neurons. Rising of this threshold enables to specialize a function that a neuron plays in its neural structure. Thus, the main idea of the associative model of neurons is to enable neurons to represent various input combinations, specialize neurons in their representation and connect neurons to emphasize their spatio-temporal associations. In this paper, new rules and equations for modelling plasticity of thresholds and changing the sensitivity of neurons are proposed and adapted.

In the contemporary artificial models of neurons used for engineering computations, we used to sequentially process computations in the discrete steps during which weights are updated [7]. Biological neurons work and update their states concurrently and asynchronously in time [9, 10]. Moreover, many of internal neuron processes are temporal, which enables biological neurons to take into account a context of previous stimulations for associated in time events that should have an impact on parameters and plastic changes in neurons. Artificial neurons use various artificial rules to connect neurons or construct an artificial neural network structure. Biological neurons are plastic accordingly to their activities and their frequencies in time [9, 10]. Plasticity allows biological neural networks to automatically and conditionally update their structures and parameters towards representing processed data. Contemporary computational models of neurons usually sever this natural ability of biological neurons. In this work, we try to propose a new associative model of neurons that is conditionally plastic, works and updates its synaptic connections concurrently and in real time. In our model, each associative neuron is in one of six states (Fig. 1): resting, charging, relaxing, activation, absolute refracting, or relative refracting, which are decided by its internal excitation level and possible external stimulations.

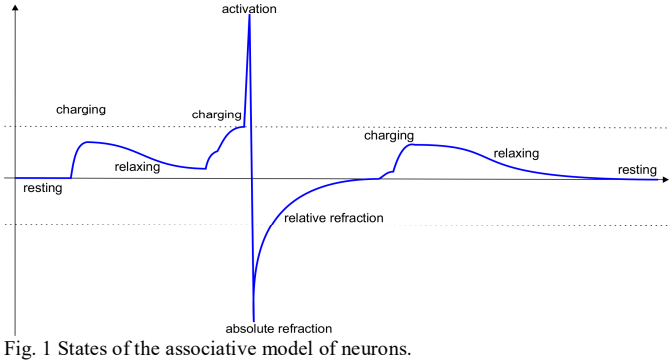

Fig. 1 States of the associative model of neurons.

Equations (1), (2), and (3) evaluate an associative neuron excitation level respectively during charging, relaxing, and refraction periods.

$$X_{N_i}^{t+\Delta t} = X_{N_i}^{t} + \left[\sum_{N_m \to N_i}\left(X_{N_m}^{t} \cdot w_{N_m,N_i}\right)\right] \cdot sin\left(\frac{\pi \cdot \Delta t}{2 \cdot \Delta t^{C}}\right) \quad (1)$$

where t is the time when the presynaptic stimulation started to influence postsynaptic neuron N_i, Δt – is the interval started from time t when neuron N_i started its charging $t < \Delta t \leq t + \Delta t^{C}$, Δt^{C} is the period of time necessary to charge and activate postsynaptic neuron N_i after stimulating synapse between N_m and N_i neurons (here $\Delta t^{C} = 20$ms), w_{N_m,N_i} is the synaptic permeability – a component of the synaptic weight.

$$X_{N_i}^{t+\Delta t} = X_{N_i}^{t} \cdot \frac{1}{2} \cdot \left(1 + cos\left(\frac{\pi \cdot \Delta t}{X_{N_i}^{t} \cdot \Delta t^{R}}\right)\right) \quad (2)$$

where $t < \Delta t \leq t + \Delta t^{R}$, Δt^{R} is the maximum period of time during which postsynaptic neuron N_i relaxes and returns to its resting state after its charging that was not strong enough to activate this neuron (here $\Delta t^{R} = 300$ms),

$$X_{N_i}^{t+\Delta t} = X_{N_i}^{t} \cdot \frac{1}{2} \cdot \left(1 + cos\left(\frac{\pi \cdot \Delta t}{\left|X_{N_i}^{t}\right| \cdot \Delta t^{F}}\right)\right) \quad (3)$$

where $t < \Delta t \leq t + \Delta t^F$, Δt^F is the maximum period of time during which neuron N_i finishes its refraction after activation and returns to its resting state (here $\Delta t^F = 60$ms).

The synapse model introduced in this paper distinguishes presynaptic and postsynaptic neuron influences that determine a final synaptic weight:

$$w = b\, c\, m \tag{4}$$

where

- b is the behavior factor that determines how the synapse influences the postsynaptic neuron ($b = 1$ when this influence is excitatory and $b = -1$ when is inhibitory),
- c is the synaptic permeability that specifies how strongly the input stimulation influences the postsynaptic neuron considering elapsed time between activations of pre- and postsynaptic neurons,
- m is the multiplication factor that determines how strongly this stimulation should influence the postsynaptic activity due to the frequency and importance of the association defined by training sequences and their repetitions.

The presynaptic influence is determined by the synaptic efficiency δ_{N_m,N_i} of a synapse between neurons $N_m \rightarrow N_i$ which is defined as:

$$\delta_{N_m,N_i} = \sum_{\{(N_m,N_i)\in S^n \in \mathbb{S}\}} \left(\frac{1}{1+\frac{\Delta t^A - min(\Delta t^C, \Delta t^A)}{\Delta t^R}} \right)^{\gamma} \tag{5}$$

where

- Δt^A is the period of time that lapsed between stimulation of synapse between N_m and N_i neurons and activation of postsynaptic neuron N_i during training,
- Δt^C is the period of time necessary to charge and activate postsynaptic neuron N_i after stimulating synapse between N_m and N_i neurons (here $\Delta t^C = 20$ms),
- Δt^R is the maximum period of time during which postsynaptic neuron N_i relaxes and returns to its resting state (here $\Delta t^R = 300$ms),
- γ is a context influence factor changing the influence of the previously activated and connected neurons on the postsynaptic neuron N_i (here equal to 4).
- S^n is a training sequence during which activation of presynaptic neuron N_m and postsynaptic neuron N_i were observed,
- $\mathbb{S}$ is the set of all training sequences used for adaptation.

Using (5) the synaptic permeabilities are computed for all outgoing synapses by one of the following methods:

Linear permeability formula

$$c = \frac{\eta}{2\eta - \delta} \tag{6}$$

Square root permeability formula

$$c = \frac{\sqrt{\eta\delta}}{\sqrt{\eta\delta} + \eta - \delta} \tag{7}$$

Quadratic permeability formula

$$c = \frac{\eta\delta}{\eta\delta + \eta^2 - \delta^2} \tag{8}$$

Proportional permeability formula

$$c = \frac{\delta}{\eta} \tag{9}$$

Power permeability formula

$$c = \left(\frac{\delta}{\eta}\right)^{\frac{1}{k}} \tag{10}$$

where

η is a number of activations of a presynaptic neuron N_m during training, δ is a synaptic efficiency computed for this synapse (5), and k>1 is an integer.

The multiplication factors are computed by postsynaptic neurons for all incoming synapses in cases when postsynaptic neuron thresholds are big and presynaptic activity of neurons conducted by single weights are not enough to achieve these thresholds due to the training sequences and order of elements represented by the following neurons that should be activated automatically. If the context of presynaptic activity of neurons is unique and represents a full subsequence of any training sequence (Fig. 5) it should be able to activate the neuron representing the next element in this sequence. If it does not it means that the existing connections are too weak and should be increased. Thus, synapses between neurons are multiplied and strengthen by the postsynaptic neurons that were supposed to be activated but were not. A simple rule is used: If the neuron is not activated by previously activated neurons that represent the first part of a sequence ($S_1 \rightarrow \cdots \rightarrow S_L$), then the synaptic weights between all activated presynaptic neurons $N_{S_1}, \ldots, N_{S_L}$ (representing the context) and this neuron $N_{S_{L+1}}$ should be increased. In order to correctly compute necessary multiplication of synaptic connections between presynaptic neurons and the postsynaptic neuron we have to compute a postsynaptic neuron total excitation $X_{N_i}^{S_1+\cdots+S_L}$.

The multiplication factors are computed after the following formula:

$$m = \frac{\theta_{N_i}^t}{X_{N_i}^{S_1+\cdots+S_L}} - \frac{x^{LAST}}{2} \tag{11}$$

where

- $\theta_{N_i}^n$ is the activation threshold of postsynaptic neuron N_i (here $\theta_{N_i}^n = 1$),
- x^{LAST} is the last postsynaptic stimulation made by activated presynaptic neurons to the postsynaptic neurons,

The only limitation for computing multiplication factor is

$$m \leq \theta_{N_i}^t \tag{12}$$

Hence, if any of the computed multiplication factors after (11) is bigger than the threshold of the postsynaptic neuron it is reduced to it appropriately ($m = \theta_{N_i}^t$).

B. Threshold Increase

The development, maturation, and growth of cerebral cortical interneurons were studied in [11]. The morphological study revealed that large interneurons had significantly more branching material in the postnatal brains than their prenatal neurons (Fig. 2). These increases of dendritic span and branching provide larger receptive areas which may improve the

development of connections in functional intracortical columns. Increase in the neuron size and its dendritic span corresponds to a larger number of ions that must be delivered to a neuron to activate it. In addition, a neuron that is more frequently activated grows, while the one that is not activated shrinks reducing a minimum number of ions required for its activation. Thus, we can reasonably assume that the size of the soma can grow as more connections are made to a neuron.

Thus we propose an associative neuron model which increases its activation threshold when the neuron is more frequently activated. This changes the sensitivity of neurons to input stimulations with larger charge needed to activate a neuron again. Subsequently, only combinations of stronger or more frequent stimuli will activate a neuron. This leads to specialization of such neurons and limits input combinations that can activate them. Such a process is not destructive because specialization of neurons enables them to be more specific, and react more adequately to a situation. Rejected frequent combinations by already specialized neurons are represented by smaller neurons and thus automatically an input data space is represented more precisely by a larger number of neurons.

From neurobiology and neuroscience we know that neurons can have various sizes of soma and various numbers of dendrites and axonal terminals [9, 10]. Neuroscience does not satisfactorily explains the functional aspects of these differences, which include neurons sensitivity for various spatio-temporal combinations of input stimuli, ability to specialize neurons in reacting for groups of such combinations, creation of multiple connections between the same neurons to strengthen associations between represented classes by these neurons etc. [6, 12].

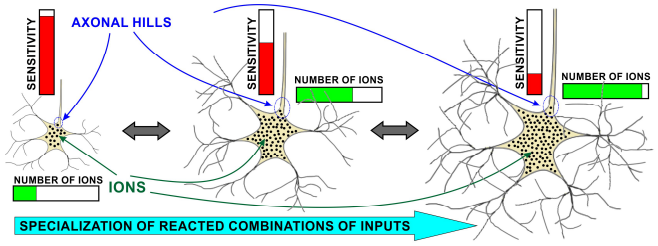

Fig. 2 When a neuron grows its soma gets larger and requires more charges to be activated. In associative neurons this translates to increase of the activation threshold. More frequently activated neurons have usually also more connections.

In this paper we model some of these functional aspects of neurons and use them in machine learning algorithms to adapt active associative neural graphs. Biological neuron bodies have different shapes and sizes [10] so the bigger neuronal bodies need to be stronger or more frequently stimulated to achieve activation thresholds (Fig. 2). Moreover, bigger neuron bodies have larger surfaces that can have more built in ion channels which can accelerate ion flux processes. Thus bigger neurons charge, relax and refract usually faster than smaller ones [10, 13].

Neurons should be activated in a proper sequence to the activations of other neurons to represent the subsequent elements of the trained sequence. Thus only a full previously activated context for each element should activate the neuron representing this element. Hence, we need to adapt the neuron sensitivity to make its activation possible only when stimulations from all presynaptic neurons representing this context come.

$$X_{N_i}^{S_1+\cdots+S_{L-1}} \le \theta_{N_i}^t \le X_{N_i}^{S_1+\cdots+S_L} \tag{13}$$

where $X_{N_i}^{S_1+\cdots+S_L}$ represents the excitation achieved for the total previous context represented by stimulations $S_1, \ldots, S_L$ coming from neurons $N_{S_1}, \ldots, N_{S_L}$; $X_{N_i}^{S_1+\cdots+S_{L-1}}$ represents the excitation achieved for the total previous context without the last element of sequence that should charge the neuron above its threshold $\theta_{N_i}^t$. Thus, when postsynaptic neuron $N_{S_{L+1}}$ representing the S_{L+1} sequence element is activated too early its threshold should be increased to exceed its current excitation level:

$$\theta_{N_i}^t = X_{N_i}^{S_1+\cdots+S_K} + \varepsilon \tag{14}$$

where ε is a small number, e.g. $\varepsilon = \theta_{N_i}^t / K^2$.

C. Axon Growth

As a growing neuron requires more charges to be activated, a growing axon also requires more charges to activate an increasing number of postsynaptic neurons. This can be compared to a fan-out issue in logic gates that determines load-driving capabilities of a gate. The larger fan-out the longer it takes for a gate to charge its output.

The simple way of introducing the fan-out effect is to normalize synaptic permeabilities in the described associative neuron model. However, this would increase the importance of weak synapses, which is not desirable. In order to avoid such increase, we scale the synaptic strength by the norm of all synaptic permeabilities of the presynaptic neuron. Thus, if the fan-out problem is considered, the associative neuron excitation level during charging is evaluated using activities of all presynaptic neurons as follows:

$$X_{N_i}^{t+\Delta t} = X_{N_i}^t + \left[\sum_{N_m \to N_i} \left(X_{N_m}^t \cdot \frac{\left(w_{N_m,N_i}\right)^2}{\|w_{N_m}\|} \right)\right] \cdot sin\left(\frac{\pi \cdot \Delta t}{2 \cdot \Delta t^C}\right) \tag{15}$$

Modification of the associative neuron excitation level to (15) is needed in episodic memory to distinguish sequences that begin with the same subsequence as discussed in section IV.

D. Synaptic Fatigue

Synaptic fatigue [11, 14] is a form of short changes in synaptic plasticity that lowers firing activities of a postsynaptic neuron. Frequent stimulation of the same sensory neurons results in habituation and lowers the neuron's response. It acts as a form of negative feedback that physiologically controls neurons' activity. Synaptic fatigue is caused by a temporary depletion of synaptic vesicles that store neurotransmitters released at the synapse as a result of repetitive neuronal stimulation. The neurotransmitters propagate the signal to the postsynaptic neuron. It takes between 1 and 40 seconds for neurotransmitter to be released into the synaptic cleft and return to presynaptic cell for reuse. If the presynaptic vesicles are released into the synaptic cleft faster than they are returned for reuse, synaptic fatigue starts to increase. Fig. 3 shows a typical central nervous system synapse.

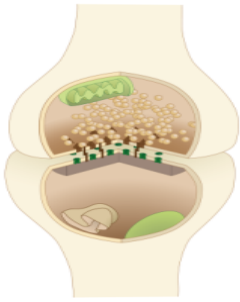

Fig. 3 Synaptic vesicles represented by small circles on the top and postsynaptic receptors shown in postsynaptic neuron at the bottom. Source https://en.wikipedia.org/wiki/Synaptic_fatigue

Synaptic fatigue can affect synapses of many different types of neurons [15]. Although synaptic fatigue existence is widely accepted, the exact mechanisms underlying the phenomenon are not completely understood. By introducing a mechanism similar to synaptic fatigue in our spiking neuron model we obtain better tools to model a declarative memory operation. Synaptic fatigue is simulated in our work by modifying resistance of associative neurons to activation during charging as follows:

$$X_{N_i}^{t+\Delta t} = X_{N_i}^{t} + \left[\sum_{N_m \to N_i}\left(X_{N_m}^{t} \cdot \frac{\left(w_{N_m,N_i}\right)^2}{\|w_{N_m}\|}\right)\right] \cdot S_{N_i}^{t+\Delta t} \cdot sin\left(\frac{\pi \cdot \Delta t}{2 \cdot \Delta t^C}\right) \quad (16)$$

where the neuron sensitivity factor $S_{N_i}^{t+\Delta t}$ is updated as

$$S_{N_i}^{t+\Delta t} = 1 - \left(1 - S_{N_i}^{t}\right) \cdot e^{\frac{-\Delta t}{\Delta t^F}} \quad (17)$$

and Δt^F is the fatigue relaxing time constant during which postsynaptic neuron N_i recovers from the fatigue and approaches its full sensitivity to stimuli (here $\Delta t^F = 20$s).
(16) describes an automatic process of gradual recovery from the fatigue, however each time a neuron is activated its activation sensitivity $S_{N_i}^{t+}$ is lowered using

$$S_{N_i}^{t+} = \frac{1}{\frac{1}{S_{N_i}^{t-}} + \Delta F} \quad (18)$$

where $S_{N_i}^{t-}$ is neuron's N_i sensitivity before activation, $S_{N_i}^{t+}$ is neuron's N_i sensitivity after firing, ΔF is a fatigue factor (here we use $\Delta F = 0.03$).

Fig. 4 shows changes in neuron sensitivity due to fatigue after it was frequently activated.

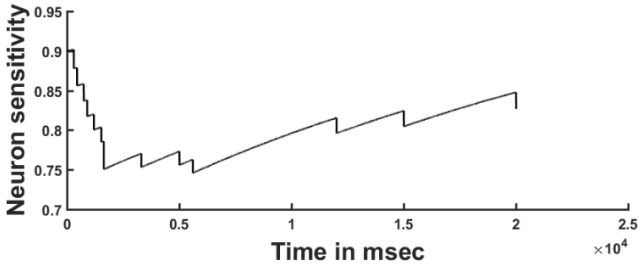

Fig. 4 Changes in neuron sensitivity due to a fatigue factor.

Neuron's initial sensitivity was 0.9 and activation was in discrete time moments equal to [305, 450, 750, 900, 1200, 1530, 1640, 1630, 3300, 5000, 5600, 12000, 15000, 20000] msec. The neuron was frequently activated during first 1630 msec, and during this period of time we see decreasing sensitivity, after which we see its gradual recovery.

III. Semantic Memory

Object representation is obtained in the semantic memory (SM) through a process of symbol grounding [2], associating sensory data with action and reward obtained by the system in its interaction with the environment. The semantic memory investigated in this paper uses an active neuro-associative knowledge graph (ANAKG) – that can represent and associate training sequences of objects or classes of objects [6]. The memory binds objects that appeared in close proximity in the input sequences, providing time domain or spatial associations. The created synaptic connections are weighted, so each association has its own importance. The SM gathers knowledge about the environment and can provide common sense solutions to new situations that were not experienced before, thus exhibiting emergent creativity property. The model demonstrates that memories can be changed if new data is processed or old data is repeated.

A. Structural Organization of Semantic Memory

Symbol grounding and learning of complex motor functions should be based on finding critical perceptual information about objects [16]. In a similar way, we try to improve predictive power of episodic memory by focusing on characteristic features of the observed episode to better predict the next episode. We can accomplish this by selecting most relevant input features with the best prediction accuracy.

Semantic memory results from self-organization of an associative neural network that is a result of adding neurons and their connections described in equations (1)-(17). Fig. 5 illustrates the memory structure that results from the presented training sequences. Nodes on Fig. 5 represent words (concepts) while edges represent spatio-temporal associations.

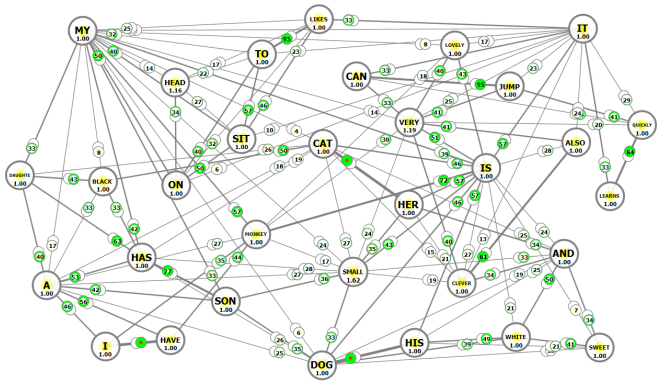

Fig. 5 A sample neuronal structure formed during associative processes for training sequences: "I have a monkey. My monkey is very small. It is very lovely. It likes to sit on my head. It can jump very quickly. It is also very clever. It learns quickly. My monkey is lovely. My son has a small dog. His dog is white and sweet. My daughter has a black cat. Her cat is small and clever."

B. Testing Semantic Memory

Semantic memories are responsible for linking together information about various dependencies of represented objects and actions performed on them forming knowledge about these objects. It should be possible to retrieve various information from semantic memory for given contexts as well as to

generalize previously learned information or get some new creative responses.

The proposed model of the semantic memories supplies us with all these capabilities. It can learn the most frequent training sequential patterns, recall generalized sequences as well as create new ones. For the sample data from Fig. 5 and initial contexts of stimulation the network we achieve answers shown in Table I. This semantic memory recalls back training sequences when the initial context is unique. In cases when the context is new or not unique we can achieve new or generalized answers. Repetition of not unique contexts enables to force this semantic memory with recalling the most frequent subsequences from the training data.

Table I. Semantic memory answers to various initial contexts.

Initial context / Question	Network Answer
I have (a unique context)	I have a monkey
Her (a unique context)	Her cat is small and clever
His (a unique context)	His dog is white and sweet
My (no unique context)	My
My monkey (repeated 5 times)	My monkey is **small very lovely**
Monkey is (repeated 3 times)	Monkey is very small **lovely**
Cat (repeated 5 times)	Cat is small
Dog (repeated 3 times)	Dog is white
It (repeated 6 times)	It is very lovely
My son and his dog	My son has a small dog and his is white sweet dog
Can I	Can I **have a monkey**

IV. Episodic Memory

Episodic memory registers time domain sequences of episodes, so it can be used for path finding, sequential associative learning (speech, motor control), and recollection of cognitively observed events. Significance of events has a strong influence over the strength and durability of episodic memory. More significant events are easier to recall and are remembered for a longer period of time. Episodic memory gives us time perspective and provides continuity in everyday activities. Episodic memory is believed to be stored in hippocampus [17], a major part of the brain that has structurally different organization of neurons than, responsible for semantic memory, cortex.

A. Structural Organization of Episodic Memory

Several structural models of episodic memory have been proposed [18-21] in the literature in recent years. They differ by the structural organization, storage and retrieval mechanism and properties such as novelty detection, forgetting, anticipation, chunking, etc.

Our model **Błąd! Nie można odnaleźć źródła odwołania.** used a flexible matching mechanism that measures similarity between the learnt and tested sequences. It tolerated errors or presentation order, distortion of the observed scene, and varying time delay. In this work we adopted a simplified version of this memory organization using associative neurons described in Section II. We demonstrate that associative neurons and their semantic connections learning mechanism can be used to formulate episodic memories.

Basic organization of episodic memory is a self-organizing structure of long term memory (LTM) cells shown on Fig. 6. During training, primary neurons P_i that reside in the semantic memory are sequentially activated. A sequence of activations $\{P_1, P_2, P_3, \dots, P_{n-1}, P_n, \}$ stimulate corresponding secondary neurons $\{S_1, S_2, S_3, \dots, S_{n-1}, S_n, \}$ in the episodic memory. Primary neurons represent concepts, so they role in the semantic memory is to represent objects, activities, motivations, or goals and to make associations between concepts.

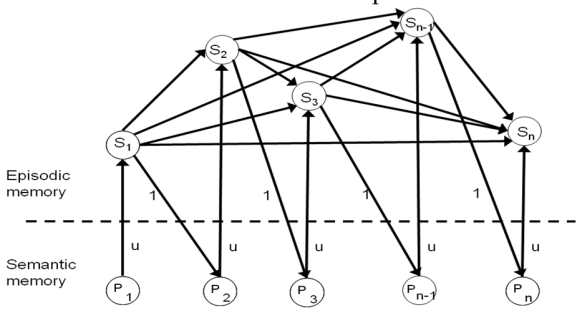

Fig. 6 A model of LTM cell based on associative neurons.

They respond to external stimuli and implement symbol grounding [2], providing understanding of the observed scene. The structure and synaptic strength of all the links between LTM neurons are fixed, so no learning is necessary, and the only thing that differentiates various LTM cells and need to be learned are connections to primary neurons. Secondary neuron activation can be used to make predictions for the next primary neuron that will be activated by the input sequence.

The primary neurons are linked to the corresponding secondary neurons with weights equal to u and the secondary neurons are connected to the primary neurons representing the next element of the sequence using prediction links with weights equal to 1. In addition the secondary neurons are also connected to all the subsequent elements of the episode sequence using weights obtained from any one of (6) - (10).

In this work we have set the weights of links from primary to corresponding secondary neurons u to 1, but they could be normalized to limit the maximum activation to the secondary neurons. The activations of the secondary neurons are computed using (16) – (18).

B. Algorithm for Episodic Memory Retrieval

After the episodic memory was trained, it may be activated during a sequence recognition process. The memory retrieval involves three stages: event detection, episode recognition, and episode recall. Event detection is triggered by activation of primary neurons and neurons in the semantic memory activated by associations with activated primary neurons. Episode recognition is detected by activation of LTM cell neurons. If several LTM cells were activated, the cell that was activated first most likely represent the observed episode. Episode recall provides memory recollection of the rest of the observed episode. The episodic recall plays an important role in learning, by anticipating the expected events. If the anticipation was correct – no learning takes place.

Memory retrieval algorithm is as follows:

- Activate primary neurons P_j^t and the corresponding secondary neurons S_j^t in all LTM cells.
- Compute an activation level of the secondary neurons S_j^{t+i} is computed using (16) - (18).

- Use the secondary neuron S_j^{t+i} with the strongest activation to find the winning LTM.
- Secondary neuron S_j^{t+i} of the winning LTM cell predicts the next episode.

C. Testing Episodic Memory

Ability to recall past events based on context is a very useful feature in memory. The semantic memory typically provides the context whereas the episodic memory helps in recalling previously encountered episodes. In this simulation, each LTM cell in the episodic memory represents an episode and the individual secondary neurons S_j, represent elements of that episode. Input from the environment triggers primary neurons P_j from the semantic memory, those in turn trigger the corresponding secondary neurons. The semantic and episodic memories were first created from training sequences using associative neurons described in Section II. Subsequently the ability of the episodic memory to recall the correct sequence and differentiate between similar sequences has been tested.

Example 1

The training input file contains the following sequences:

1. A B C D; 2. A B C; 3. B A C E; 4. P Y E J.

The semantic memory is created through consolidation of the training sequences. Each neuron in the semantic memory represents on element (in this case a letter of alphabet) of the training sequence. As the memory is context dependent each element of the sequence simulates its successors, i.e. subsequently learned elements of the memory, through links with weights that reflect their occurrence, frequency and distance (near or far), in the training sequences. The LTM cells in the episodic memory were similarly created, except that each LTM cell was created using only one sequence. Thus, in this example the episodic memory consisted of four LTM cells.

After the memories were created, the sequence "A B C D" was provided as a test sequence using external stimulation to the semantic memory. This stimulation of the semantic memory resulted in activations of its neurons. Strength of activation of these neurons were treated as an output from the semantic memory and was provided as an input to the LTM cells in the episodic memory. That is, the primary neurons {A, B, C, D} in the semantic memory that were activated due to the test sequence activated their corresponding secondary neurons in the LTM cells.

In this example the activation levels of the neurons in LTM cells representing training sequences "A B C D" and "A B C", are of particular interest because they are highly similar to each other and one of them is the same as the testing sequence.

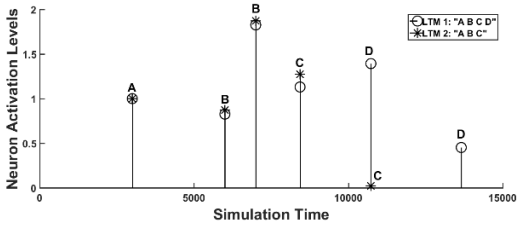

Fig. 7 Activation levels of "winning neuron" in LTM cells 1 and 2.

Fig. 7 shows a comparison of the activation levels of the "winning neuron" in these two LTM cells with symbol o marking LTM1 and * marking LTM2.

When two LTM sequences that have the same initial elements are simulated we expect that the shorter LTM sequence will have higher activation initially, as expected from (15). This was observed in our simulation and clearly visible around 8000 msec. Fig. 7 shows that at this point in time the activation levels of the winning neuron in LTM 2 is higher than the activation level of the winning neuron in LTM 1. Only towards the end, when neuron 'D' is activated the winning neuron in LTM 1 has higher activation than that from LTM 2.

V. Emergent Creativity of Declaratrive Memories

Declarative memories (which are obtained from integration of semantic and episodic memories) are essential functional component of the motivated learning cognitive architecture (MLECOG) [5]. In this section we illustrate how associative declarative memories create new categories, can generalize, and answer new questions.

Table I demonstrates some answers collected with new initial contexts that were not used in training sequences. As a result we obtained answers that are different from the trained ones, yet reflecting the knowledge gained from the training sequences presented in Fig. 5. If there are many possible answers that could be derived from the training sequences the declarative memory chooses the most common answer if the initial context is repeated a few times. Repetition of difficult questions is normal for people who are searching for answers. This kind of memory behaves similarly.

Changing context (Table I) may result in changing the answer based on the training set. This reflects generalization and creativity determined by dynamically modified knowledge gained during training that can be obtained within this kind of declarative memory (Table I). As network develops it changes the way it processes the input data, reflecting its stage of development and ability to generate context based answers.

The declarative memory created and tested for this work had the semantic memory receiving the inputs and the output of the semantic memory was the input to the episodic memory. The semantic and episodic memories were trained with the sample data from Fig. 5 and Table II shows the answers provided by the declarative memory to the same initial context as in Table I. Note that as the individual neurons start their relaxing or refraction phase the winning LTM cells can change. In Table II only the first winning LTM cell is specified. The major advantage of the declarative memory is observed in the response to the last five initial context/questions shown in Table II. The declarative memory was able to generate meaningful responses without necessitating repetitions of inputs, this is because any activation in the semantic memory will stimulate the LTM cells of the episodic memory thus potentially enabling the declarative memory to generate a response.

Table. II Declarative memory answers to various initial contexts.

Initial context / Question	Declarative Memory Answers
I have	I have a monkey
Her	Her cat is small and clever
His	His dog is white and sweet
My	My monkey is lovely.
My monkey[1]	My monkey is lovely.
Monkey is[1]	My monkey is lovely.
Cat[1]	Her cat is small and clever.
Dog[1]	His dog is white and sweet.
It[1]	It learns quickly.

[1] Note, unlike in Table I, no repetitions are required or used here

VI. Conclusions

We developed and tested a structural organization of declarative memories using a new model of spiking neurons. Original associative neurons presented in [6] were modified to individually accommodate growth and sensitivity of neurons and their strength of connections that resulted in automatic threshold changes and specialization of neurons. In the presented model the same neurons can be activated by various combinations of input stimuli that represent various contexts of the elements of training sequences. In addition, we introduced neuron's fatigue and showed its effect on declarative memory creativity observed during memory stimulation. We also investigated the effect of synaptic weight sensitivity on overall activity of neurons in the obtained memories.

We tested sequence recognition and associative properties of the obtained self-organizing episodic and semantic memories. Memory organization and all associations were performed on the symbolic level where each neuron's activation represents an object, an action or an idea.

We demonstrated emergent creativity that can be observed in the developed memories. Tested memories were first organized based on a number of training sentences. Subsequently, a number of questions were submitted to the memory. Using knowledge stored in the memory, the memory responded in a novel way within associative context of the questions asked. Thanks to the strengthen associations between neurons, which reflect the frequency of training subsequences, we achieve the ability of the network to generalize.

Future work includes further studies on neuron models developing their ability to adapt to training subsequences that include other sequences. Networks will be enriched with autonomous motivational signals and mechanisms of their automatic associations with symbols and actions represented in declarative memories. Declarative memory structure will be enhanced by feeding back the response of LTM cells to the semantic memory to generate new or generalized relationships.

References

[1] H. Eichenbaum, "The hippocampus and declarative memory: cognitive mechanisms and neural codes," *Behavioural Brain Research,* vol. 127, pp. 199-207, 2001.

[2] M. Taddeo and L. Floridi, "The symbol grounding problem: A critical review of fifteen years of research," *Journal of Experimental and Theoretical Artificial Intelligence,* vol. 17, no. 4, pp. 419-445, 2005.

[3] W. Kintsch, "The role of knowledge in discourse comprehension: A construction-integration model," *Psychological Review*, vol. 95, pp. 163–182, 1988.

[4] E. Tulving, *Elements of episodic memory*. New York, NY: Oxford University Press, 1991.

[5] J. A. Starzyk, J. Graham, "MLECOG - Motivated Learning Embodied Cognitive Architecture," *IEEE Systems Journa*l, vol. PP , no. 99, pp. 1-12, 2015.

[6] A. Horzyk, "How Does Generalization and Creativity Come into Being in Neural Associative Systems and How Does It Form Human-Like Knowledge?", *Neurocomputing*, vol. 144, pp. 238-257, 2014.

[7] S. O. Haykin, *Neural Networks and Learning Machines*. 3rd ed, Upper Saddle River, NJ: Prentice Hall, 2009).

[8] W. Gerstner and W. Kistler, *Spiking Neuron Models: Single Neurons, Populations, Plasticity*. New York NY: Cambridge University Press, 2002.

[9] A. Longstaff, *BIOS Instant Notes in Neuroscience*, New York, NY: Garland Science, 2011.

[10] J. W. Kalat, *Biological Psychology*, Belmont, CA: Wadsworth Publishing, 2012.

[11] M. Prinz, B. Prinz and E. Schulz, "The growth of non-pyramidal neurons in the primary motor cortex of man: a Golgi study," *Histology and histopathology*, vol. 12, no. 4, pp. 895-900, Oct. 1997.

[12] J. A. Starzyk and H. He, "Spatio–temporal memories for machine learning: A long-term memory organization," *IEEE Trans. Neural Netw.*,vol. 20, no. 5, pp. 768–780, May 2009.

[13] B. P. Bean, "The Action Potential in Mammalian Central Neurons," *Nature Reviews Neuroscience*, vol 8, no 6, 2007.

[14] N. S. Simons-Weidenmaier, M. Weber, C. F. Plappert, P. K. D. Pilz, and S. Schmid, "Synaptic depression and short-term habituation are located in the sensory part of the mammalian startle pathway," *BMC Neuroscience*, vol. 7:38, 2006.

[15] H. Zhang, B. Gong, S. Liu, M. Fa, I. Ninan, A. Staniszewski, and O. Arancio, "Synaptic fatigue is more pronounced in the APP/PS1 transgenic mouse model of Alzheimer's disease," *Current Alzheimer Research*, vol. 2, no. 2, pp. 137-140, 2005.

[16] E.J. Gibson, "Perceptual learning in development: Some basic concepts," *Ecological Psychology*, vol. 12, no. 4, pp. 295–302, 2000..

[17] E. T. Rolls, "A computational theory of episodic memory formation in the hippocampus," *Behavioural Brain Research*, vol. 215, no. 2, pp. 180–196, 2010.

[18] L. Shastri, "Episodic memory and cortico-hippocampal interactions," *TRENDS in Cognitive Sciences*, vol. 6, no. 4, pp. 162–168, April 2002.

[19] A. Nuxoll and J. E. Laird, "A Cognitive Model of Episodic Memory Integrated With a General Cognitive Architecture", *Int. Conf. on Cognitive Modelling*, July 2004, pp. 220-225.

[20] J. A. Starzyk and H. He, "Anticipation-based temporal sequences learning in hierarchical structure," *IEEE Trans. Neural Netw.*, vol. 18, no. 2, pp. 344–358, Mar. 2007.

[21] D. L. Wang and B. Yowono, "Anticipation-based temporal pattern generation," *IEEE Trans. Syst., Man Cybern.*, vol. 25, no. 4, pp. 615–628, Apr. 1995.