

NOVEL COMPUTATIONAL APOPTOSIS-NEUROGENESIS MODEL FOR MULTI-ABSTRACTION LEVEL PERCEPTION

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Abstract-artificial neural network provides a cybernetic model that is similar to human intelligence in terms of parallel processing, generalization and memory stacking on the same neural network. From the era of neurogenesis, research models expect the rules that govern new neuron to depend on old mature circuitry. Other research models show the existence of catastrophic interference associated with new neurons if species is exposed to variable information content environment. In this work, the model developed provides a theoretical framework for a novel attention sensitive neural network as well as an experimental framework revealing the addition of new neuron can be prevented from catastrophic interference phenomena if it undergoes certain structural processes in human brain.

Keywords - Neurogenesis, attention sensitive neural network, computational model

I. INTRODUCTION

Neurogenesis in the brain of adult mammals occurs throughout life, and has been clearly demonstrated at two locations under normal conditions: the subventricular zone (SVZ) of the lateral ventricles and the subgranular zone (SGZ) of the dentate gyrus in the hippocampus. Neurons born in the adult SVZ migrate over a great distance through the rostral migratory stream and become granule neurons and periglomerular neurons in the olfactory bulb. Neurons born in the adult SGZ migrate into the granule cell layer of the dentate gyrus and become dentate granule cells. Recent studies also showed that newborn neurons in the adult brain integrate into the existing circuitry and receive functional input [1]. Adult neurogenesis is regulated by physiological and pathological activities at all levels, including the proliferation of adult neural stem cells (NSCs) or progenitors, differentiation and fate determination of progenitor cells, and the survival, maturation, and integration of newborn neurons. Furthermore, these cells may be required for certain forms of brain function involving the olfactory bulb and the hippocampus, which is important for some forms of learning and memory. Whether neurogenesis occurs in areas of the adult mammalian brain other than the SVZ and SGZ remains controversial [2] and [3]. Adult neurogenesis also occurs in a variety of nonmammalian vertebrates and has been extensively studied in songbirds [4] and [5]. The role of hippocampal neurogenesis is also addressed by computational simulation, based on the computational modeling of hippocampal function. Most computational studies that model hippocampal function are based on its simplified trisynaptic excitatory circuit (dentate gyrus/CA3/CA1). It is proposed

that the dentate gyrus performs pattern separation on inputs from the entorhinal cortex by sending orthogonal and sparse signals to the pyramidal neurons in hippocampal region CA3 via the mossy fiber projection. CA3 serves as a temporary storage site via its recurrent collaterals; CA1 recodes Schaffer collateral inputs from CA3 by competitive learning and plays a role in consolidation by setting up associatively learned backprojections to neocortex to facilitate recall. The pattern separation role of dentate gyrus has been recently demonstrated experimentally by either dentate gyrus-specific lesions or specific deletion of NMDA receptors in dentate gyrus [6-7]. Several computational studies assumed that there was constant turnover of mature neurons in the dentate gyrus as a result of a balance between neurogenesis and apoptosis. Using a simple three-layered network, [8] and [9] found that neurogenesis elicited a more rapid clearance of old memory and enhanced the recall fidelity of new memories. [10] modeled neurogenesis in the dentate gyrus in a more complicated network system simulating the entire hippocampus and found that the constant neuron turnover through neurogenesis helps to create distinct memory traces for highly similar patterns. However, the turnover of mature neurons has not been convincingly demonstrated experimentally in the dentate gyrus. In addition, all of the above models do not distinguish the characteristics of immature neurons from that of mature ones. The work in [11] took the enhanced plasticity of immature neurons into consideration in their theory and proposed that neurogenesis helps the dentate gyrus to avoid catastrophic interference of old memories when adapting to new environments. However, the applicability of this model to the hippocampal system is unclear, as it did not take the sparse coding role of the dentate gyrus into consideration and used a small hidden layer to represent it. Alternatively, the work in [12] suggested that adding plastic immature neurons into the dentate gyrus would hinder its function in pattern separation. They proposed that young neurons may facilitate the formation of temporal association in memory. Together, these computational studies suggest several potential functions of hippocampal neurogenesis and provide some theoretical guidance for future experimental work. In this work, we develop a framework for attention-sensitive neural network that has the ability to handle multiple environmental changes without catastrophic interference. This framework is shown to mimic the actual human brain mental operation and hence has a wider-reaching capabilities than conventional neural network models.

II. METHODOLOGY

A. Network Models, Assumptions and Equations

Multiple stroop-effect (shown in Fig. 1) describes rotation and indentation change of meaning. The A of Z pattern reflects multiple abstraction level in the same image and its associated interference according to level of view. we have 3 facts all is true, 1-micro we have Z pattern, 2- macro recognizes A pattern and intermediate (Integrated) abstraction level which recognize A pattern of ZZ Sub pattern. In this research, we will not concentrate on the competence between automatic and required attention task but with multiple perception dilemma associated with the allocation of attention for the same object.

Consider we have a multiple abstractive perceptual events vector $E = e_1, e_2, e_3, \dots, e_n$ consider the corresponding human attention vector is $A = a_1, a_2, a_3, \dots, a_n$, assume we have (potential of event vector P). assuming $A=f(P)$, where is a linear function, so for each event e_n we have a_n functionally established from the event potential vector P (model the potential of external event required attention degree -mammals conscious decision -), where the sum of attention at any time instant remains constant.

$$\sum_{k=1}^n a_k = c \quad (1)$$

At any neuron nodes

$$G(x) = \sum_{i=0}^n w_i x_i \quad (2)$$

For encoding layer allocation (Learning /Recall Phase):

Assume the mechanism of neurogenesis/apoptosis during learning happens as follows. For a fixed number of input and output layers, consider n is the number of neurons in encoding layer and K_{en} is size of event e_n . In the recall phase, (attention degradation function/constant). Fig. 2 shows output layer neuron O_i and is calculated by assuming constant attention degradation function per cluster of neurogenesis allocation neuron, assume we have (L) of (C) cluster group in encoding layer then the assumed constant attention degradation vector $A = [a_1, a_2, a_3, \dots, a_n]$.

$$O_i = \sum_{k=1}^q w_{kji} x_{kji} \quad (3)$$

Let c_{ji} be the output of neurogenesis clusters group j to the output neuron O_i . Assume q is the number of neurogenesis neurons in allocated neuron cluster in encoding layer. $q=f(K_{en})$. As a simplification, assume that we have fixed q neurogenesis clusters and neurons group allocated each time. For each group,

$$c_{ji} = \sum_{k=1}^q w_{kji} x_{kji} \quad (4)$$

where w_{kji} is connection weight of the k th neuron of the j th cluster connected to i output neuron, x_{kji} is the synapse connection weight of K neuron in encoding layer of the j th cluster connected to output neuron i . Then, by substitution of (1) into (4), the output calculated can be given as,

$$O_i = \sum_{j=1}^l a_j \sum_{k=1}^q w_{kji} x_{kji} \quad (5)$$

In the learning phase (Cluster Masking Type Learning) For training of cluster j a_j must obey,

$$A \in \{0,1\}, \\ a_i = 1 \quad \forall i=j \text{ and } a_i = 0 \text{ otherwise.}$$

Improved information delivered to higher abstraction recognition layer as result of this attention series transformation is shown in Fig. 3. We will consider attention window A_w that contains equal attention value for one or more abstractive feature (in-scope). For complete analysis and extraction of global picture we consider feature window moves on feature domain in form of attention configuration as, $((\text{feature}_1, \text{feature}_4, \dots, \text{feature}_n), C)g$ where $\text{feature}_1, \text{feature}_4, \dots, \text{feature}_n$, is attention feature window, c is required attention value from the network and g is a configuration number. A series of last attention configuration may set for each seen to determine all abstraction level required in-scope Fig. 4. The set $\{a_1, a_2, a_3, a_4, a_5\}$ this set of configuration take the network attention to specific feature group at a time, the response of the network is a series of responses as attention feature change which is o_{ig} output neuron number i for attenuation configuration g and o_{ig} may considered as two dimensional matrix.



Fig. 1. Multiple stroop-effect. contains both proximity and rotational perceptual change of meaning.

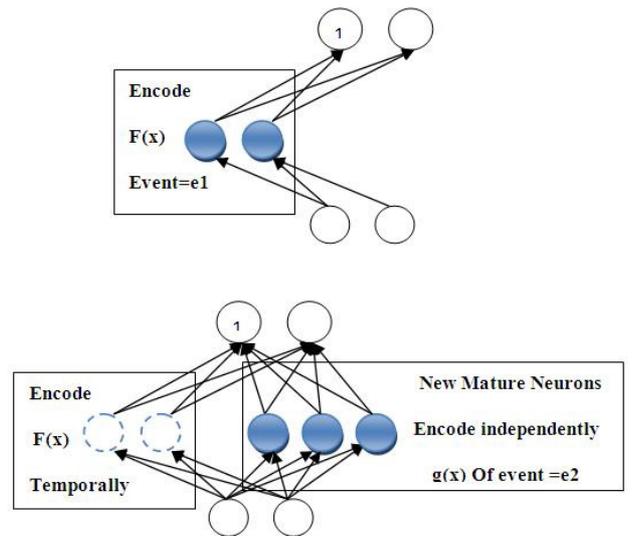


Fig. 2. Proposed mechanism for neuron apoptosis/neurogenesis operation

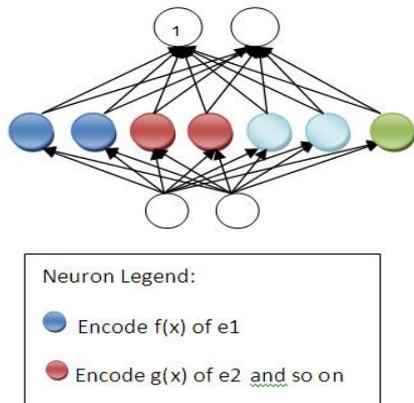


Fig.3. Complete storage of un-correlated multiple perception on one network.

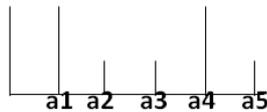


Fig. 4. Predetermined attention configuration scheme for attention vector.

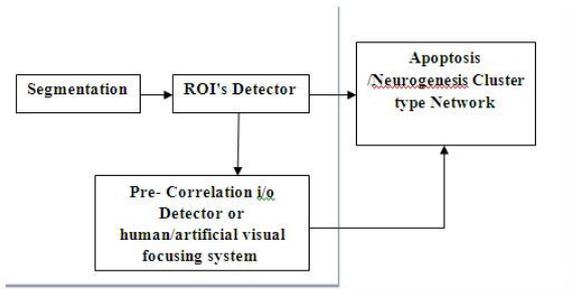


Fig. 5. Complete system block diagram.

B. Experimental Methodology

Two network were constructed, with the old being solid three-layer network and the second as our proposed novel two-cluster neurogenesis-apoptosis network. Experimental simulations were performed using the Matlab 7.6 Neural Network toolbox, where the architecture was build with Matlab to mimic the proposed model. Table 1 Summarizes the experimental conditions and results.

After the first noiseless original image was learned, the noise intensity was varied and added to input images sample with Gaussian distribution and varied mean and variance in single and cluster type network (in noisy data set training phase), the error distribution of network output was plotted. Keeping into account that the experiment must be reproducible under conditions states, we must have sufficiently noisy input data so that mean and variance of classifier error are regarded as a representation of actual error (which should not change observably if the experiment is to be repeated by others). We preserved the same initialization of the network across all trial to provide the experiment the same initial condition for neuron interconnection weights. Single solid network encoding layer n were divided two clustering network each cluster $n/2$. As the number of two clusters input/output interconnection is the same as original solid network, there is no connectivity change that may increase knowledge capacity and interfere with our results. Further study of the effect of experimental conditions such as learning algorithm, number of weight input data, number of images, noise distribution, etc. needs to be conducted in a future work. The complete proposed system block diagram is shown in Fig. 5.

III. RESULTS

A reduced classification error of neurogenesis-apoptosis type comparison with the control is plotted in Fig. 6. Also output probability distribution of error is plotted in Figs. 7 and 8 for both type respectively. Table 1 lists experimental conditions, mean, and variance calculated empirically and reveal strong reduction in both parameters for the proposed neurogenesis-apoptosis network model.

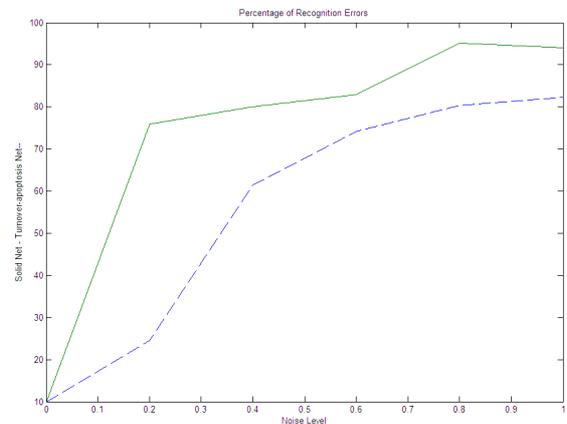


Fig. 6. Percentage classification error for solid network and neurogenesis-apoptosis type (dashed) .

IV. DISCUSSION

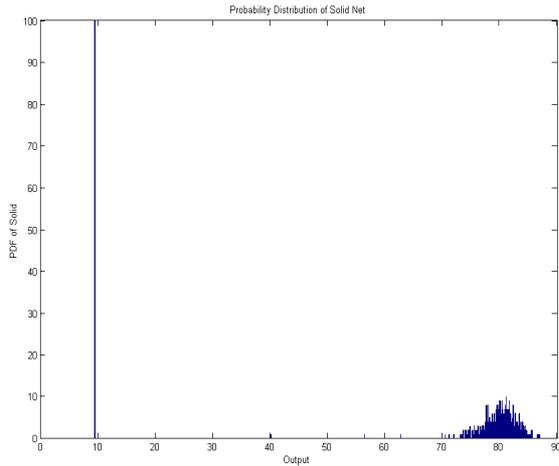


Fig. 7. Probability distribution function of output of solid control network.

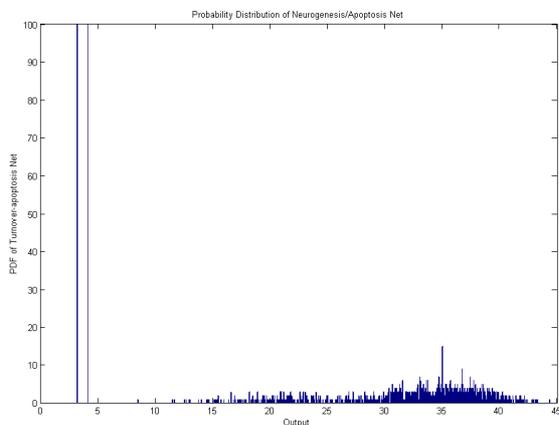


Fig. 8. Probability distribution functions of output of two cluster neurogenesis –apoptosis type network.

Table 1. Experiment condition and classification error output mean and variance for Neuroge-apop type versus the control.

	Solid Large Network	Neurogenesis-Apoptosis
<i>Construction</i>		
No of encoding layer	40	20-20
Size of input (image)	65*54	65*54
Output neuron size	10	10
Additive noise Distr	Gaussian	Gaussian
Additive Noise stat	0 mean var 0->1	0 mean var 0->1
Learning Algorithm	GD	GD
<i>Performance statistics of output class error</i>		
Mean	69.59	27.50
Variance	660.05	140.78

The coding/decoding process of hippocampus area inside human brain is under intense investigation, also the upcoming information for neurogenesis in adult human brain suggested that how new added neuron will acts and behaves. The discussion suggested that the catastrophic interference may resolved by neurogenesis mechanism . In this work, a novel computational model for attention sensitive neural network construct was developed. This model provides theoretical insight about biological neurons interaction after subjected to neurogenesis mechanism and how this mechanism may defeats catastrophic interference as well as draw up higher inferences as they transfers perception attention from one cluster to others. The results of this work may be useful to enhance brain psycho-neurological understanding and provide improvements in artificial intelligent and cybernetic framework. The model has intrinsic characteristics such that data in the second region of interest may require special kind of transfer function for training, initialization and limiting transfer function, the isolation properties of this model will preserve this for each neurogenesis cluster, which enable flexibility for solving problems.

It should be noted that the previous models store each region of interest in separate network. However, with the new model, if input was massively scaled (number of inputs is resolution of large region of interest), or high-resolution robotic image sensor is connected with input layer of the network, a lot of physical resource was consumed. In other work respecting our proposed model if we store all region(s) of interest on single network we have the benefit of sharing input/output neurons as well as the isolation properties , which preserving classification accuracy and solve catastrophic interference dilemma. The model was treated multi-abstractive level perception collected by higher abstractive neurons circuits for global object identification. It is also prevents from catastrophic interference by event isolation (grouping of encoding layer information to the higher abstractive inference network). The minimum cross talk between two different functionally neuron cluster via this isolation structure in learning process observed biologically in cortex boundary between two regions – and subjected to special type of neurotransmitter inhibitor chemical unknown till know. The construction preserves a parallel distribution of perception and the serial perception of image component due to attention fixed amount of resource and focal and movement of human visual system required for fully recognizing external world. We introduce the concept of one network owns multiple skills; this may interprets why neurogenesis integrates into old neuron circuits without catastrophic interference. In other way the model proves to be capable of handling multiple-skills problem with superior performance.

V. CONCLUSIONS

The results have demonstrated that the proposed theoretical model will have better recognition error statistics and intrinsic characteristics. The results also suggested other means for understanding the actual contribution of

neurogenesis operation in human brain, and proposed an ability for building distributed learning algorithm architecture and management of attention in full and semi-isolated neuron cluster, this model will permit building up clever multiple-skills machine in the future.

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