Echo State Networks with Artificial Astrocytes and Hebbian Connections

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Abstract. For the last few decades, the neuroscientific research has highlighted the importance of astrocytes, a type of glial cells, in the information processing capabilities. By dynamic bidirectional communication with neurons, astrocytes regulate their excitability through a variety of mechanisms. Traditional artificial neural networks (ANNs) are connectionist models that describe how information passes throughout layer of neurons abstracting from low-level mechanisms. However, very little research has addressed artificial astrocytes and their incorporation into ANNs. In this paper, we present an echo state network (ESN) extended with astrocytes which influence the neurons by fixed or Hebbian connections. By systematic analysis we investigate their role on five classification tasks and show that they can outperform the standard ESN without astrocytes. Although the model with fixed astrocytic weights yields from none to little improvement, the model with Hebbian weights from astrocytes to neurons is significantly superior.

Keywords: Glial cells · Astrocytes · ESN · Classification · Computational model

1 Introduction

Firstly identified in the 19th century, glial cells (often called glia) significantly contribute to the total brain mass with around 50% and their glia:neuron ratio in mammalian brains about 1:1 [3]. Although considered as non-functional and supportive units for over more than a decade, recently they gained lots of attention, as the emerging evidence indicates their role as active and equally important components compared to neurons. By interacting and cooperating with each other in nervous systems, they both take a significant part in various neurophysiological processes. Several functions of the glia are well characterized, including maintenance of homeostasis, being inevitable in the development of the central and peripheral nervous system, forming structural foundations that hold neurons, providing metabolic support and so on. However, the full characterization of their active roles and exact mechanisms still remain unresolved.

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The population of glia is commonly subdivided into four major groups: (1) astrocytes, (2) oligodendrocytes, (3) microglia and (4) their progenitors NG2-glia. According to recent evidence, astrocytes, the most abundant and probably the most complex group, play significant role in cognitive functions, traditionally attributed solely to neurons, such as learning and memory, information transfer and processing [2,8]. Although not being able to be excited electrically and generating action potentials as neurons do, they are incorporated in network *glial syncytium* where upon being excited they propagate Ca^{2+} signals throughout gap junctions. They are characterized by having the resting membrane potential of $\sim -80\,\text{mV}$ close to Nernst equilibrium for potassium ions (K^+) [5] and express both ionotropic and metabotropic glutamate receptors [19] allowing them to be highly sensitive to neuronal activity.

In order to better understand these low-level mechanisms, computational modelling is often employed which recently has become an essential part of neuroscience. Such models may provide testable predictions for processes that are built upon these mechanisms such as neuronal regulation, or synaptic plasticity. A better knowledge about astrocyte–neuron cooperation may also provide building blocks for studying the regulatory capability of glial syncytium on a larger scale. Computational models of ANNs extended with astrocytes may not only be used as an interesting novel concept, but mainly can provide space for hypotheses to explain the potential roles of glia in biological neuronal circuits and networks.

In the previous study, we investigated the role of astrocytes as neuronal regulators in a feedforward ANN [7]. The proposed models were superior to the traditional multi-layer perceptron on the same datasets, however, this was not the case for all of them. In addition, we detected unique astrocytic regimes in terms of output distributions that were different for each problem. In this paper, we transfer the same model of astrocytes to ESNs and systematically investigate their role using five classification tasks from UCR time series classification archive [4]. In addition, we incorporate and analyze Hebbian learning as a form of plasticity for astrocytic weights. The paper is organized as follows. Section 2 includes the related background and work. In Sect. 3, we describe proposed model in depth. In Sect. 4, we provide the experimental results. Section 5 concludes the paper.

2 Related Background

Computational neuroscience distinguishes two modeling paradigms: biophysical and connectionist. While the former focuses on physical and chemical properties of a biological system using various mathematical methods, the latter makes a significant reduction in the complexity of low-level mechanisms which in turn may lead to better comprehension of the system from a higher level. Despite the plethora of biophysical models of astrocytes per se and neuron-astrocyte coupling, connectionist modeling has so far been out of scientific interest. For an overview of biophysical models, we recommend reviews in [18,21,22].

As far as the connectionist models are concerned, we highlight the work by Ikuta et al. who initially introduced astrocytes[1] into ANN [10]. Their model of an astrocyte served as a chaotic noise generator which was being propagated to the neighboring units and impacted standard neuronal signalling. On the two-spirals classification problem, the performance in terms of mean absolute error and the rate of convergence was superior to the multilayer perceptron (MLP) without astrocytes. The architecture of the model can be described as an extension of MLP with astrocytes regulating neurons on the hidden layer. The authors in their later work investigated various activation functions for astrocytes and various architectures [11–13].

Instead of the neuronal regulation, Porto-Pazos et al. [20] and González et al. [1] focused on synaptic plasticity modulation. They presented an MLP with astrocytes regulating neural transmission on a larger temporal scale (hundreds of milliseconds and seconds) as opposed to fast neuronal and synaptic signaling (milliseconds). Astrocytes were activated by intense neural transmission and consequently regulated synaptic weights with a slow temporal time course. Each neuron was paired with a single astrocyte and each astrocyte only responded to the activity of the associated neuron by modulating its output synaptic weights. Since the model was dependent on various hyperparameters that needed to be fine-tuned, in their following work they presented a method for automatic search of these parameters based on cooperative coevolution [16].

3 Proposed Model and Training Methods

Here we present a novel neural network architecture based on an ESN with the reservoir extended with astrocytes. We first provide a brief overview of ESNs, the training procedure including weights initialization, and then describe the architecture of our model, parameter selection and incorporation of Hebbian learning.

3.1 Echo State Networks

Training traditional recurrent neural networks is considered to be difficult because of limitations of gradient descent methods which tends to be computationally expensive, to have slow convergence and to generally lead to poor local minima. Hence, the full adaption of all network weights is often omitted, yet still yielding excellent performance. This approach serves as a foundation for ESNs which were introduced by Jaeger for nonlinear system identification and time series modeling [14]. They are characterized by having randomly generated input weights and reservoirs with the training only on readout weights. However, in order to work

[1] Originally, authors use term *artificial glia* but we consider *artificial astrocytes* instead, since glia represent the vast majority of non-neuronal cells in the nervous system with multiple functions, whereas only astrocytes are currently considered to play a vital role in information processing tasks.

well, ESNs require delicate tuning of several hyperparameters including the size of the reservoir N, the spectral radius ρ, and input weight scaling τ.

Reservoir activation vectors $\boldsymbol{x}(t) = [x_1(t), ..., x_N(t)]$ and output activations $\boldsymbol{y} = [y_1, ..., y_C]$ for given input pattern $\boldsymbol{u} = [u(1), ..., u(T)]$ are updated according to ESN dynamics given by the formulas

$$\boldsymbol{x}(t) = f(\boldsymbol{w}^{\text{in}} u(t) + \boldsymbol{W}^{\text{res}} \boldsymbol{x}(t-1)) \tag{1}$$

$$\boldsymbol{y}(t) = \boldsymbol{W}^{\text{out}} \boldsymbol{x}(T) \tag{2}$$

with the logistic sigmoid activation function $f(net) = 1/(1 + \exp(-net))$.

For dealing with classification problems, we consider the following training procedure:

1. Generate random input weights $\boldsymbol{w}^{\text{in}}$ and reservoir weights $\boldsymbol{W}^{\text{res}}$ scaled by $\rho/|\lambda_{\max}|$, where $\lambda_{\max}$ denotes the largest absolute eigenvalue of $\boldsymbol{W}^{\text{res}}$ and ρ is manually selected.
2. Run ESN using the training inputs and for each $\boldsymbol{u}_{train}$ collect the last reservoir activation state $\boldsymbol{x}(T)$.
3. Compute the linear readout weights using formula

$$\boldsymbol{W}^{\text{out}} = \boldsymbol{Y}^{\text{tgt}} \boldsymbol{X}^{+} \tag{3}$$

   where $\boldsymbol{Y}^{\text{tgt}}$ is a matrix of concatenated target vectors (in columns) with one-hot encoding and $\boldsymbol{X}^{+}$ is the pseudoinverse matrix of concatenated reservoir activation states from step 2.
4. Use the trained network on new input data $\boldsymbol{u}_{test}$ and decide the class by selecting output neuron with maximum activation

$$\text{class}(\boldsymbol{u}_{test}) = \arg\max_k y_k \tag{4}$$

3.2 Neuron–Astrocyte Coupling – A-ESN Model

Although astrocytes are interconnected within glial syncytium using gap junctions and communicate sharing slow Ca^{2+} signals (as opposed to neuronal firing), we omit this concept for the sake of complexity and start exploring the simplest model of astrocytes. Upon investigating whether they work relatively well and produce favorable results, in the future research we plan to explore more complex and biologically plausible mechanisms. For now, we focus on this simple model.

In our model we consider an astrocyte to play a single role in neuronal regulation. Since it was discovered that mammalian cortices have glia:neuron ratio of about 1:1, as stated in the introduction, in the context of ESN we equip each reservoir neuron with one astrocyte as shown in Fig. 1. We call this model **A-ESN**.

Reservoir activation $x'_i(t)$ takes into account input pattern $u(t)$, previous time step activation vector $\boldsymbol{x}'(t-1)$ and astrocyte activation $\psi_i(t)$ weighted by a single shared weight w^{α}, which is expressed in the vector form as

$$\boldsymbol{x}'(t) = f(\boldsymbol{w}^{\text{in}} u(t) + \boldsymbol{W}^{\text{res}} \boldsymbol{x}'(t-1) + w^{\alpha} \boldsymbol{\psi}(t)) \tag{5}$$

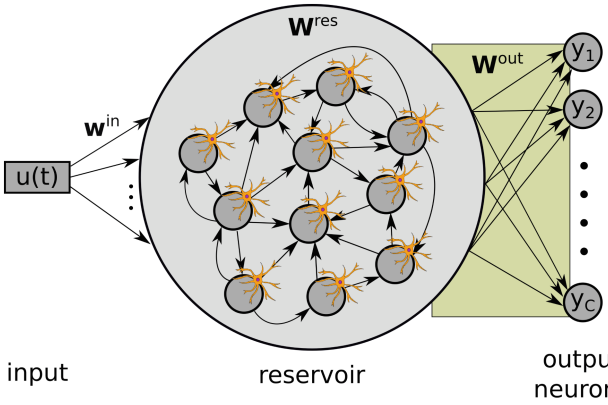

Fig. 1. The architecture of the proposed model, A-ESN, with a reservoir of neurons and astrocytes. Each neuron is paired with an astrocyte that listens to it and regulates its behaviour based on activity. Since we consider the single time series classification problems, we use a single input neuron, N neurons and astrocytes within the reservoir, and C output neurons representing the classes.

Astrocytes $\psi_i(t)$ listen to their associated neurons and when some of the neurons exceed the threshold θ, astrocytes produce the activation value of 1. The rest of them decays by factor γ, as shown in Eq. 6.

$$\psi_i(t) = \begin{cases} 1, & \text{if } \theta < x'_i(t-1) \\ \gamma\psi_i(t-1), & \text{otherwise} \end{cases} \tag{6}$$

This ESN dynamics is graphically depicted in Fig. 2.

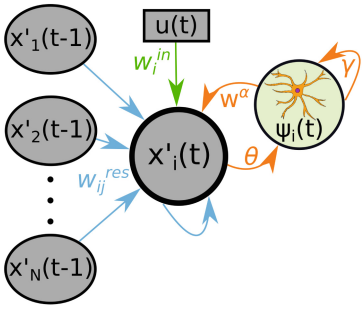

Fig. 2. Neuron–astrocyte coupling. The astrocyte, weighted by w^α, regulates the associated neuron by contributing to its input. When the neuron surpasses the threshold θ, the astrocyte outputs 1 and slowly decays by γ in the next time steps. Blue arrows depict reservoir weights, the green arrow an input weight and orange arrows the astrocyte parameters. (Color figure online)

3.3 Hebbian Learning in A-HL-ESN Model

Since using a single shared weight w^α for all astrocytes may be too constraining, we consider an individual weight for each astrocyte. Although astrocytes are not considered to be able to trigger neuronal action potential, they still modulate their membrane potential by the release of gliotransmitters including glutamate (exciting the neuron) or ATP (inhibiting the neuron) [6]. For that reason we consider randomly generated weights from a uniform distribution $Uni(-1, 1)$.

The exact relationship of neuronal regulation by astrocytes is still not well understood and we can only guess to which extent is this process plastic and what are the specific mechanisms of plasticity. For that matter we speculate using Hebbian learning which is in great detail described in [9]. The basic principle is that the change of a synaptic weight w_{ji} between neurons x_i and y_j, with the learning rate η, is expressed as

$$\Delta w_{ji}(t) = \eta x_i(t) y_j(t) \tag{7}$$

In our case we apply this rule for the change of the weight w^α between a neuron x'_i and an astrocyte ψ_i. Repeated application, however, may lead to an exponential change of the weight which is not biologically plausible, so this is solved by incorporating some form of stabilization. This is in many cases the normalization of the final weights. We consider Oja's rule [17] which introduces a nonlinear, forgetting factor for the weight change

$$\Delta w_i^\alpha(t+1) = \eta x'_i(t)[\psi_i(t) - x'_i(t) w_i^\alpha(t)] \tag{8}$$

To take into account this new dynamics, we split our training algorithm into two phases: (1) once the unsupervised learning of the weights $\boldsymbol{w}^\alpha$ (Eq. 8) in the reservoir is complete, (2) a supervised learning algorithm (from Sect. 3.1) is applied to the readout weights. Instead of using Eq. 5 for the reservoir update, we consider

$$\boldsymbol{x}'(t) = f(\boldsymbol{w}^{\text{in}} u(t) + \boldsymbol{W}^{\text{res}} \boldsymbol{x}'(t-1) + \boldsymbol{w}^\alpha * \boldsymbol{\psi}(t)) \tag{9}$$

with operator '$*$' denoting the element-wise product of vectors. We call the model with Hebbian learning described here as **A-HL-ESN**.

4 Experiments

For evaluating the performance of our new approach, we consider five classification problems from UCR time series classification archive [4]. We use a standard ESN (without astrocytes) as a baseline and compare it with proposed methods described in the previous section. Using grid search we systematically investigated each hyperparameter (averaged over several instances) and selected the values with the lowest error rate on the testing dataset. Regarding the ranges for each hyperparameter we chose the values presented in Table 1.

Table 1. Hyperparameter value ranges used in the grid search for each dataset.

Parameter	Tested values
N	20 to 500 with step = 20
τ	10, 5, 1, 0.5, 0.1, 0.05, 0.01, 0.001, 0.0001
ρ	0.8 to 1.4 with step = 0.05
w^{α}	–1.0 to 1.0 with step = 0.1
γ	0.0 to 1.0 with step = 0.1
θ	0.0 to 1.0 with step = 0.1

The UCR archive already provides train/test split of the datasets, but we found this rather problematic because of the high risk of overfitting the hyperparameters to a particular test dataset. In order to avoid this, we merged both train and test datasets into a single set and used 5-fold cross-validation instead. To eliminate the random fluctuation in performance, we executed training procedures with random weights, random permutations of datasets and averaged error rates over 100 instances.

Allowing for possibility of imbalanced datasets in which one class is overrepresented with the respect to the others, we use *Matthews correlation coefficient* (MCC) as a metrics for performance evaluation score [15] rather than the mean-squared error, accuracy or F1-score which does not work relatively well on imbalanced datasets. The value MCC = 1 corresponds to a perfect match between model predictions and observations, whereas −1 indicates total disagreement between the two.

In all experiments, we used hyperparameters summarized in Table 2 resulting in the largest MCC on testing datasets.

Table 2. Optimal hyperparameters selected using the grid search for each dataset. Non-astrocytic hyperparameters (N, ρ, τ) were shared in all models on a given dataset.

Dataset	ESN			A-ESN			A-HL-ESN	
	N	ρ	τ	w^{α}	γ	θ	γ	θ
FaceFour	20	0.95	0.05	−0.4	0.1	0.2	0.2	0.2
MoteStrain	120	1.3	0.001	−0.3	0.9	0.5	0.3	0.3
OSULeaf	60	0.95	0.01	0.6	0.6	0.8	0.2	0.1
SwedishLeaf	160	1.4	0.001	−0.4	0.2	0.9	0.2	0.2
ToeSegmentation1	60	1.3	1.0	−0.5	1.0	0.9	0.3	0.1

Results in terms of MCC averaged over 100 simulations are presented in Table 3. It is clear that model with Hebbian connections, A-HL-ESN, significantly outperforms models ESN and A-ESN. Despite having more complex

training procedure and thus higher time complexity, gain in terms of performance is clearly notable. Model with fixed connections, A-ESN, have yielded results equivalent to standard ESN (assuming correct settings of hyperparameters), although it is speculative why on the last dataset (ToeSegmentation1), the error rate is significantly better (MCC of 0.5 ± 0.1 vs 0.32 ± 0.11).

Table 3. MCC (mean + standard deviation) averaged over 100 simulations on each dataset. In each case, the model A-HL-ESN is superior regarding the performance.

Dataset	ESN	A-ESN	A-HL-ESN
FaceFour	0.44 ± 0.12	0.43 ± 0.13	0.56 ± 0.14
MoteStrain	0.65 ± 0.04	0.67 ± 0.06	0.85 ± 0.03
OSULeaf	0.41 ± 0.06	0.42 ± 0.06	0.57 ± 0.06
SwedishLeaf	0.64 ± 0.03	0.63 ± 0.03	0.84 ± 0.03
ToeSegmentation1	0.32 ± 0.11	0.50 ± 0.10	0.59 ± 0.11

In order to better understand the role of astrocytes with Hebbian connections, we were interested to know how the astrocyte weights develop during learning. For the fully trained models (all 100 instances), we plotted final distributions of the weights w_i^α as depicted in Fig. 3. We can observe that the weight distributions are skewed in the interval (1,2), roughly independent of the dataset, with an exception being MoteStrain, where some of the weights are also between 0 and 1. We may conclude this implies excitatory nature of the astrocytes in terms of neuronal regulation.

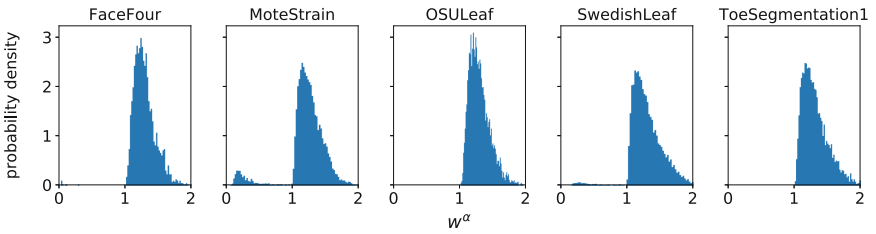

Fig. 3. Distribution of the weights w_i^α in the trained models A-HL-ESN reveals excitatory role of the astrocytes.

5 Conclusion

To advance the modeling of biological neuronal networks, which are inherently recurrent, it is inevitable to focus on models from the same domain. Since training recurrent neural networks is difficult for various problems, we considered ESNs instead. Moroever, the neuroscientific research for the last decades

has highlighted the importance of glial cells in information processing context. Astrocytes regulate neuronal functionality in a variety of ways, particularly by maintaining the concentration of ions and neurotransmitters and by releasing gliotransmitters, and modulating both neuronal excitability and synaptic plasticity. However, limited amount of research has been done in the field of ANNs equipped with artificial astrocytes and basically none on the recurrent models, so the exact role of astrocytes remains speculative.

In our previous work [7], we investigated the role of astrocytes in feedforward models and inspired by positive results, we transferred the same model of astrocytes to ESNs and explored their influence. In addition, we incorporated Hebbian learning for weights between astrocytes and their associated neurons. By systematic analysis of this new dynamics on five classification tasks we found very little contribution of astrocytes with fixed weights, but in case of Hebbian learning the performance yielded significantly positive outcome. By analyzing the final distributions of astrocyte weights, we discovered that astrocytes operate as neuronal excitors by lowering the threshold required for firing. Out of curiosity we also examined various modifications including bipolar activation functions for neurons and astrocytes (with their output activation within the interval (–1, 1)) and swapping astrocytes with neurons in Eq. 8. However, these modifications did not perform that well.

Future research in this area may follow several directions. The activation function for the astrocyte, as formulated in Eq. 6, is definitely not the only one and there are several varieties to be considered. Since Ca^{2+} signalling within glial syncytium operates on a much slower pace as opposed to neuronal firing, it may be beneficiary to incorporate this slow, temporal dynamics into astrocytic behaviour. Although our model of an artificial astrocyte includes slow decay, "firing", however, remained still instant. Despite focusing on the astrocytes as single separate units, it is possible to model glial syncytium and design an astrocytic network of astrocytes connected together, hence fulfilling the biologically plausible spatiotemporal dynamics. Last, but not least, instead of focusing on regulation of neuronal excitability, it is possible to design models that also incorporate rules for synaptic plasticity.

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