# Extended Spiking Neural P systems with Excitatory and Inhibitory Astrocytes

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**Summary.** We investigate an extended model of spiking neural P systems incorporating astrocytes and their excitatory or inhibitory influence on axons between neurons. Using very restricted variants of *extended spiking neural P systems with excitatory and inhibitory astrocytes* we can easily model Boolean gates like NAND-gates as well as discrete amplifiers.

## 1 Introduction

In this paper we integrate several models describing the functioning of the human brain based on the biological background. New models in the area of neural computation were introduced based on the observation that neurons send electrical impulses (also called *spikes*) along axons to other neurons, e.g., see [4], [11], [12]. P systems (membrane systems) were introduced as a formal model describing the hierarchical structure of membranes in living organisms and the biological processes in and between cells (an introduction to this field can be found in [17], for the actual state of the art in this area we refer the reader to [23]).

Combining the ideas of P systems and spiking neurons, a new variant of so-called tissue P systems (see [13]) called spiking neural P systems was investigated, e.g., see [8], [18]. An extended version of spiking neural P systems allowing to send different informations along the axons between two neurons was investigated in [1]. In spiking neural P systems (see [8]), the contents of a neuron consists of a number of so-called spikes. The rules assigned to a cell allow us to send information to other neurons in the form of electrical impulses – spikes – which are summed up at the target cell; the application of the rules depends on the contents of the neuron. In [1], an extended version of this original model of spiking neural P systems was

introduced based on some other observations from biology; for example, the spikes coming along different axons may cause effects of different magnitude. In [3], the role of inhibitory axons in extended spiking neural P systems was investigated (the arrival of spikes in the neuron affected by spikes along an inhibitory axon is inhibited).

Until recently, *astrocytes*, a sub-type of macroglia have been understood as star-shaped glial cells spanning around neurons in the central nervous system (CNS). Their main function was suspected to be the metabolic support of the neurons with glucose and nutrients as well as metabolic support for endothelial cells for keeping the blood barrier. They also were found to play a critical role in the neuronal survival and differentiation or neurite outgrowth. For more details see [19]. More recent biochemical literature, however, has put forward the idea that astrocytes have an important role in the plasticity of the CNS namely the synaptogenesis [7]. Astrocytes were also found to influence the concentration of neuroactive substances [14] and may serve as intermediaries in neuronal regulation of blood flow [16]. It also has become apparent that astrocytes themselves form an information-transmitting network by passing elevations of Calcium (Ca2+) [5][6]. It has been found that Ca2+ elevations in astrocytes modulate neuronal excitability and synaptic transmission. On the other hand, astrocytes are shown to be influenced by neurotransmitters [20] that might influence Ca2+ concentrations indicating that astrocytes might discriminate between different levels of neuronal activity [19]. It is also suggested that astrocytes may respond to synaptic activity in local domains [15] only and that these local domains may also discriminate between neurotransmitters (see [19]). Hence, a complex feedback loop of neuronal modulation exerted by astrocytes can be postulated.

The influence of *astrocytes* in the functioning of the human brain has also been investigated in [21], where to the interaction between the networks of neurons and astrocytes in addition the influence of the capillary system in connection with the networks of neurons and astrocytes was modelled. Based on the biological background, but without claiming to model it in a decent way, we develop a model of membrane systems incorporating some specific features of complex systems consisting of two interacting networks of neurons and astrocytes. For the signals sent from one neuron to another one, we base our model of *extended spiking neural P systems with excitatory and inhibitory astrocytes* on the ideas of (extended) spiking neural P systems and add the concept of astrocytes influencing the signals along the axons. For the astrocytes themselves, we assume their membrane potential to be changed according to external inputs which may either come from neural cells or the firing intensity and frequency along the axon. We shall assume two thresholds; then in the excitatory case, the effect of the astrocyte on the axon it controls is as follows:

If the membrane potential is below the first threshold, then there is no effect of the membrane potential of the astrocyte on the controlled axon. If the membrane potential is between the lower and the upper threshold, then the signals along the controlled axon are affected in an excitatory (amplifying) way. Yet if the membrane

potential goes beyond the upper threshold the effect turns to be an inhibitory one, decreasing the weight of the signals coming along the axon. In this way, the astrocyte network acts as a complex control mechanism on the network of the neural cells and is itself regulated by this network of neural cells. Hence, these networks of neurons and astrocytes form a complex system. We do not model the influence of the capillary system which is described as a third part of such a complex interacting system of networks as described in [21].

Our new model could be used for the representation of artificial neural networks, especially for self-organizing feature maps; yet in contrast to analytic models of such variants of neural networks, our model works in a discrete manner, but on the other hand, is based on a graph-like structure and not on a (usually two-dimensional) grid. An example of such a two-dimensional artificial neural network based on biological observations of the complex networks of neurons and astrocytes in the human neocortex can be found in [2]. Moreover, our model of neurons and axons with the axons being influenced by astrocytes in an excitatory or inhibitory way and the neurons influencing the astrocytes, is also related with the specific model of Petri nets with range arcs as described in [9] where process arcs influence transitions in an activating or inhibitory way and this influence depends on the number of tokens in a place which makes enabling a given transition possible.

In this paper we do not focus on applications as the possibility for modelling artificial neural networks as self-organizing feature maps (e.g., see [10]) for specific application tasks. Instead we show the potentials of our model to formalize discrete functions, e.g., networks of logical gates. Moreover, we exhibit the computational completeness of our model.

## 2 Extended Spiking Neural P Systems with Excitatory and Inhibitory Astrocytes

For the basic elements of formal language theory needed in the following, we refer to any monograph in this area, in particular, to [22]. We just list a few notions and notations: $V^*$ is the free monoid generated by the alphabet $V$ under the operation of concatenation and the empty string, denoted by $\lambda$, as unit element. $\mathbb{N}_+$ denotes the set of positive integers (natural numbers), $\mathbb{N}$ is the set of non-negative integers, i.e., $\mathbb{N} = \mathbb{N}_+ \cup \{0\}$. The interval of non-negative integers between $k$ and $m$ is denoted by $[k..m]$. By $REG(\mathbb{N})$ and $RE(M)$ we denote the sets of subsets of $\mathbb{N}$ that are regular and recursively enumerable, respectively.

The basic elements of membrane computing are taken from [17]; comprehensive information can be found on the P systems web page `http://psystems.disco.unimib.it`. Moreover, for the motivation and the biological background of spiking neural P systems we refer the reader to [8].

An *extended spiking neural P system with excitatory and inhibitory astrocytes* (of degree $m \geq 1$) (in the following we shall simply speak of an *ESNPA system*) is a construct

$$\Pi = (m, n, S, R, U)$$

where

- $m$ is the number of *neurons*; the neurons are uniquely identified by a number between 1 and $m$ (obviously, we could instead use an alphabet with $m$ symbols to identify the neurons);
- $n$ is the number of *astrocytes*; the astrocytes are uniquely identified by a number between $m+1$ and $m+n$;
- $S$ describes the *initial configuration* by assigning an initial value (of spikes) to each neuron as well as an initial value (membrane potential) to each astrocyte;
- $R$ is a finite set of *rules* of the form $\left(i, E/a^k \to P\right)$ such that $i \in [1..m]$ (specifying that this rule is assigned to cell $i$), $E \subseteq REG(\mathbb{N})$ is the *checking set* (the current number of spikes in the neuron has to be from $E$ if this rule shall be executed), $k \in \mathbb{N}$ is the "number of spikes" (the energy) consumed by this rule, and $P$ is a (possibly empty) set of *productions* of the form $(l, w)$ where $l \in [1..m+n]$ (thus specifying the target neuron or astrocyte), $w \in \mathbb{N}$ is the *weight* of the energy sent along the axon from neuron $i$ to neuron or astrocyte $l$;
- $U$ is a finite set of *rules* of the form $(r, p, q, h, h', f, f', f'')$ such that $r \in [m+1..n]$ and $p, q \in [1..m]$ (specifying that this rule is assigned to astrocyte $r$ and influencing the axon between the neurons $p$ and $q$), $h, h' \in \mathbb{N}$, $h \leq h'$ are thresholds, and $f, f', f''$ are functions $\mathbb{N} \to \mathbb{N}$ changing the energy $w$, sent along the axon from $p$ to $q$, to $w'$ as follows: if $w < h$, then $w' = f(w)$, if $h \leq w \leq h'$, then $w' = f'(w)$, if $w > h'$, then $w' = f''(w)$.

A *configuration* of the ESNPA system is described as follows:

- for each neuron, the actual number of spikes in the neuron is specified;
- for each astrocyte, the actual membrane potential of the astrocyte is specified.

A *transition* from one configuration to another one now works as follows:

- for each neuron $i$, we first check whether we can "activate a rule" $\left(i, E/a^k \to P\right)$, i.e., if the current value of spikes in neuron $i$ is in $E$; waiting to be executed; then neuron $i$ "spikes", i.e., for every production $(l, w)$ occurring in the set $P$ we put the corresponding package $(l, w)$ on the axon from neuron $i$ to neuron $l$ or astrocyte $l$, respectively;
- if there is a rule $(r, i, l, h, h', f, f', f'') \in U$, the energy $w$ in a package $(l, w)$ on the axon from neuron $i$ to neuron $l$ is modified according to this rule to $(l, w')$ as described above;
- for each neuron $l$, we now consider all eventually modified packages $(l, w')$ on axons leading to neuron $l$; we then sum up all weights $w'$ in such packages and add this sum to the corresponding number of spikes in neuron $l$;
- for each astrocyte $l$, we now consider all packages $(l, w)$ on axons leading to astrocyte $l$; we then sum up all weights $w$ in such packages and take this sum

as the new membrane potential for astrocyte $l$ (i.e., we forget the previous potential).

After having executed all the substeps described above in the correct sequence, we obtain the description of the new configuration. A *computation* is a sequence of configurations starting with the initial configuration given by $S$.

An ESNPA system can be used to generate sets of numbers from $RE(\mathbb{N})$ as follows: A computation is called *successful* if it halts, i.e., if for no neuron, a rule can be activated. We then consider the contents, i.e., the number of spikes, of a specific neuron called *output neuron* in halting computations. According to [8], we can also take the distance between the first two spikes in an output neuron to define the number it computes. For generating $k$-dimensional vectors of non-negative integers, we have to designate $k$ neurons as *output neurons*.

In the following, we shall use ESNPA systems to compute discrete functions, especially we shall exhibit how Boolean functions can be computed by using NAND-gates. When computing functions, we assume external input signals arriving in some designated *input neurons* as well as several *output neurons* for sending out the computed function with a spike indicating the signal 1 and with no spike being sent out indicating the signal 0.

The rules $\left(i, E/a^k \to P\right)$ in the examples given in the succeeding section will be of a very special form, i.e., we always have $E = \left\{a^k\right\}$, hence, we can omit $E$. Moreover, the productions $(l, w)$ in $P$ have the same weights for all $l$ occurring in $P$, and even the sets $P$ are the same for all rules $\left(i, E/a^k \to P\right)$ for each $i$; hence, we can indicate such rules as in Figure 1 where the rule $a^k \to a^l$ in neuron $p$ means that $k$ spikes are consumed in neuron $p$ and $l$ spikes are sent to every neuron $q$ if there exists an axon from $p$ to $q$. Moreover, $a^m$ in neuron $p$ indicates the initial value of $m$ spikes in this neuron.


**Fig. 1.** Representation of simple rules in neurons.

The specific effect of very special astrocytes is depicted in Figures 2 and 3: in Figure 2, the influence of an *excitatory astrocyte* $r$ on an axon between two neurons $p$ and $q$ is depicted: $\geq k|f$ in astrocyte $r$ means that if $x \geq k$ spikes are sent out from neuron $p$ then $f(x)$ spikes will reach neuron $q$, whereas for a number of spikes $x < k$ no spike will reach $q$. On the other hand, in Figure 2, the influence of an *inhibitory astrocyte* $r$ on an axon between two neurons $p$ and $q$ is depicted: $\leq k|f$ in astrocyte $r$ means that if $x \leq k$ spikes are sent out from neuron $p$ then

$f(x)$ spikes will reach neuron $q$, whereas for a number of spikes $x > k$ no spike will reach $q$.

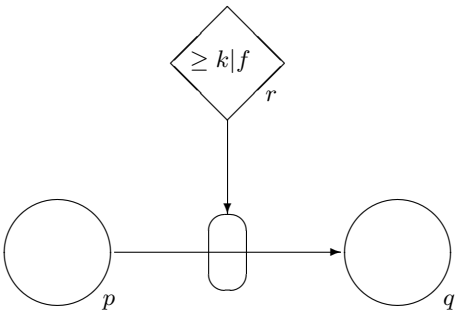


**Fig. 2.** Excitatory astrocyte.

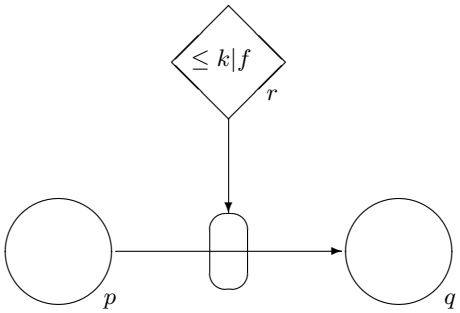


**Fig. 3.** Inhibitory astrocyte.

## 3 Computing with ESNPA Systems

In this section we first exhibit that ESNPA systems working as generators are computationally complete, i.e., able to generate any recursively enumerable set of non-negative integers. Then we show how networks of logical gates can be simulated by using specific ESNPA systems; in fact we describe an ESNPA system representing a NAND-gate. Finally we describe an ESNPA system representing a discrete amplifier.

### 3.1 Computational Completeness

As already the original model of spiking neural P systems was shown to be computationally complete, i.e., able to generate any recursively enumerable set of non-negative integers, with only those features also allowed in the sub-network of neurons in ESNPA systems, we immediately obtain computational completeness for ESNPA systems, too, because just omitting astrocytes gives a sufficiently powerful submodel of spiking neural P system as defined in [8]. Moreover, the model of ESNPA systems as defined above with just omitting the astrocytes is a submodel of extended spiking neural P systems as defined in [1] that again is sufficiently powerful to cover the proofs given there for computational completeness. The additional use of astrocytes would allow for different constructions with the rules for the neurons being even more restricted than in the case of spiking neural P systems, yet we do not go into the technical details of such constructions in this paper.

### 3.2 Networks of Logical Gates

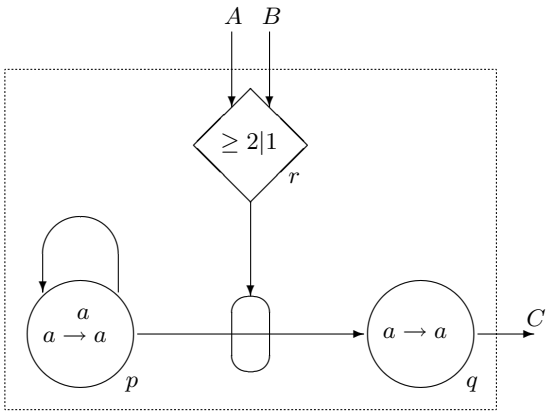


**Fig. 4.** AND-gate.

As is well known, any Boolean function can be obtained by networks only consisting of NAND-gates (and units representing the identity function). The identity function obviously can be obtained by using the same inputs (i.e., $A = B$) in an AND-gate as depicted in Figure 4.

The AND-gate is shown in Figure 4: $A, B$ are the inputs, $C$ is the output; the neuron $p$ is a source sending out one spike in each time step which only reaches neuron $q$ if the axon is *excited* by the astrocyte which reaches the excitatory threshold 2 if and only if both inputs $A$ and $B$ are 1. The notion $\geq 2|1$ in astrocyte $r$ means that only if the sum of input spikes ($A$ and $B$) is $\geq 2$, then one (1) spike

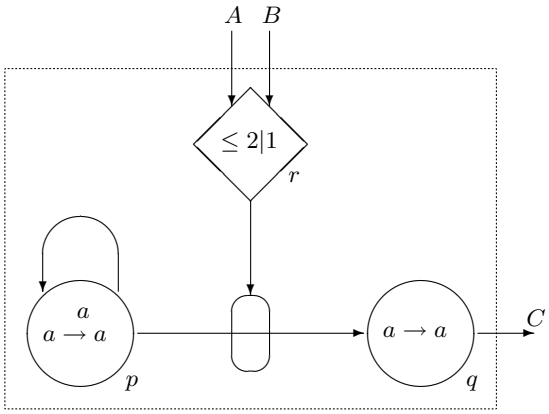


**Fig. 5.** NAND-gate.

is sent to neuron $q$, whereas if less than two input spikes arrive in astrocyte $r$, then no spike will reach the output neuron $q$. If both inputs ($A$ and $B$) represent the same signal, i.e., if $A = B$, then neuron $q$ will get a spike if and only if $A = 1$.

The NAND-gate is shown in Figure 5: again $A, B$ are the inputs, $C$ is the output; the neuron $p$ is a source sending out one spike in each time step which only reaches neuron $q$ if the axon is not inhibited by the astrocyte which reaches the inhibitory threshold 2 if and only if both inputs $A$ and $B$ are 1.

Any network of NAND-gates and AND-gates (only needed as identities keeping a signal as it is for one time step) of depth $n$ yields the result of the computation with a delay of $n$, i.e., given the input at time $t$, the corresponding output appears at time $t + n$.

### 3.3 A Discrete Amplifier

The ESNPA system depicted in Figure 6 represents a discrete amplifier which, as soon as the input from $B$ goes beyond the given threshold $k$, from the input $x$ given at $E$ computes the function $f(x) = nx$ at $C$. We have to remark that the rules $a^l \to a^l$ given in the neurons $p$ and $q$ represent the (theoretically infinite) set of rules $\left\{\{a\}^* / a^l \to a^l \mid l \in \mathbb{N}\right\}$ (for practical applications, an upper bound can be assumed).

## 4 Conclusion

The model we discussed in this paper is already very powerful from a theoretical point of view as elaborated in the preceding section. On the other hand, for specific applications, especially in the area of artificial neural networks and self-organizing feature maps, an extended version where we allow the dynamic evolution of new

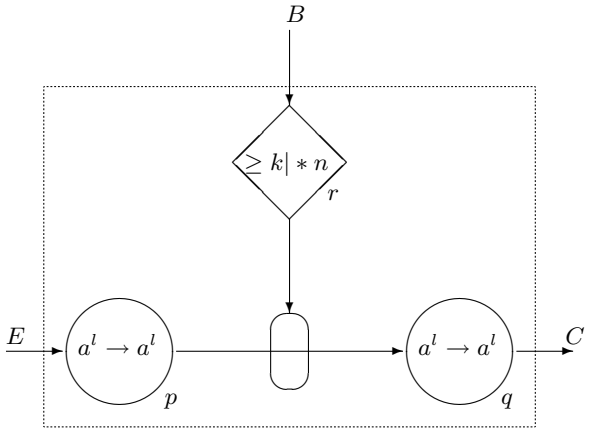


**Fig. 6.** An ESNPA amplifier.

connections between neurons, could be useful; the influence of the already existing astrocytes on these new axons plays an important role.

Another variant to be considered in the future are networks where part of the network may be destroyed which also has an interesting biological background. In this case, the ability of such a complex network to reorganize itself is the most challenging aspect of this variant.

Other variants may allow one astrocyte to influence more than one axon, eventually even in a different way and on the other hand, one axon may be influenced by several astrocytes, again eventually in a different way (in an inhibitory or excitatory way). For example, in this way more complex functions can be described by a single "unit" (circuit).

## References

1. A. Alhazov, R. Freund, M. Oswald and M. Slavkovik, Extended Spiking Neural P Systems Generating Strings and Vectors of Non-Negative Integers, in: H. J. Hoogeboom, Gh. Paun, G. Rozenberg (eds), Pre-proceedings of the 7th Workshop on Membrane Computing WMC7, 2006, pp. 88-101.
2. F. Buarque de Lima Neto and P. de Wilde, Venn-like models of neo-cortex patches, *International Joint Conference on Neural Networks*, 2006, pp.89–96.
3. R. Freund and M. Oswald, Extended spiking neural P systems with inhibitory axons, in: M. Sugisaka, H. Tanaka, Proc. *International Symposium on Artificial Life and Robotics*, Beppu, Oita, Japan, 2007.
4. W. Gerstner, W. Kistler, *Spiking Neuron Models. Single Neurons, Populations, Plasticity.* Cambridge Univ. Press, 2002.
5. C. Giaume and K. Mc Carthy, Control of gap-junctional communication in astrocytic networks., *Trends Neuroscience 15*, 1995, pp. 5535–5550.
6. P. Guthrie, J. Knappenberger, M. Segal, M. Bennet, A. Charles and S. Kater., ATP released from astrocyte mediates glial calcium waves.,*J Neuroscience 19*, 1999, pp. 520–528.

7. F. He and Ye. Sun, Glial cells more than support cells?, *Int J Biochem Cell Biol. 39(4)*, 2007, pp. 661–665.
8. M. Ionescu, Gh. Păun and T. Yokomori, Spiking neural P systems, *Fundamenta Informaticae* 71, 2–3, 2006, pp. 279–308.
9. J. Kleijn and M. Koutny, Processes of nets with range arcs, CS-TR 985, School of Computing Science, Newcastle University, Oct. 2006.
10. T. Kohonen, *Self-Organizing Maps*, $3^{rd}$ ed., Springer-Verlag, Berlin, 2001.
11. W. Maass, Computing with spikes, Special Issue on *Foundations of Information Processing* of TELEMATIK 8, 1, 2002, pp. 32–36.
12. W. Maass and C. Bishop (eds), *Pulsed Neural Networks*. MIT Press, Cambridge, 2003.
13. C. Martín-Vide, J. Pazos, Gh. Păun and A. Rodríguez-Patón, A new class of symbolic abstract neural nets: Tissue P systems, in: Proceedings of COCOON 2002, Singapore, *Lecture Notes in Computer Science* 2387, Springer-Verlag, Berlin, 2002, pp. 290–299.
14. S. Mennerik and C. Zorumski, Glial contribution to excitatory neurotransmission in cultural hippocampal cells., *Nature 368*, 1994, pp. 59–62.
15. W. Nett, S. Oloff and K. McCarthy, Hippocampal astrocytes in situ exhibit calcium oscillations that occur independent of neuronal activity., *J Neurophysiol. 87(1)*, 2002, pp. 528–537.
16. R. Parri and V. Crunelli, An astrocyte bridge from synapse to blood flow., *Nat Neurosci 6(1)*, 2003, pp. 5-6.
17. Gh. Păun, *Computing with Membranes: An Introduction*. Springer, Berlin, 2002.
18. Gh. Păun, M.J. Pérez-Jiménez and G. Rozenberg, Spike trains in spiking neural P systems, *Intern J Found Computer Sci*, to appear (also available at [23]).
19. G. Perea and A. Araque, Communication between astrocytes and neurons: a complex language. *Journal of Physiology Paris* 96, 2002, 199–207.
20. J. Porter and Kd. Mc Carthy, Astrocytic neurotransmitter receptors in situ and in vivo., *Prog Neurobiol. (51)*, 1997, pp. 7817–7830.
21. X. Shen and P. de Wilde, Long-term neuronal behavior caused by two synaptic modification mechanisms. *Neurocomputing* 70, 7-9, 2007, 1482–1488.
22. G. Rozenberg and A. Salomaa (eds), *Handbook of Formal Languages* (3 volumes). Springer, Berlin, 1997.
23. The P Systems Web Page, `http://psystems.disco.unimib.it`