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Extended Spiking Neural P Systems with Astrocytes - Variants for Modelling the Brain

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Abstract

We investigate an extended model of spiking neural P systems incorporating astrocytes affected by the activity of spikes along axons and the excitatory or inhibitory influence on axons. Using very restricted variants of *extended spiking neural P systems with astrocytes* we can easily model Boolean gates. Computational completeness thus can easily be obtained for the unrestricted model, whereas the bounded variant can only compute regular functions and generate/accept only regular sets of natural numbers.

1 Introduction

Astrocytes can be seen as an additional network of cells aside the network of neurons, for example in the human brain. For the biological background of the network of astrocytes we refer to [3]. The influence of *astrocytes* in the functioning of the human brain has been investigated in [9], 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 this biological background, in [2] we have developed a model of membrane systems consisting of two interacting networks of neurons and astrocytes. There the model of *extended spiking neural P systems with astrocytes* was based on the ideas of (extended) spiking neural P systems [1] with adding the concept of astrocytes influencing the signals along the axons and with the axons being affected by neurons. In this paper, we assume the astrocytes being affected by the spikes passing along axons.

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 the monograph [8], for the actual state of research we refer to [10]). Combining the ideas of P systems and spiking neurons (e.g., see [6], [4], [7]), spiking neural P systems were introduced in [5]: the contents of a cell (neuron) consists of a number of so-called spikes; the rules assigned to a cell allow for sending information to other neurons in the form of electrical impulses (also called spikes) which are summed up at the target cell; the application of the rules depends on the contents of the neuron. Based on the biological background of astrocytes, we have developed a model of membrane systems incorporating the two interacting networks of neurons and astrocytes in [2] with the astrocytes being affected by the neurons and effecting the transmission of spikes along an axon. In this paper, we consider a model of extended spiking neural P systems with astrocytes where the astrocytes are affected by the spikes sent along axons.

2 Definitions

$\mathbb{N}$ denotes the set of non-negative integers. The interval of non-negative integers between k and m is denoted by $[k..m]$.

For the motivation and the biological background of spiking neural P systems we refer the reader to [5].

An *extended spiking neural P system with astrocytes* (of degree $m \geq 1$) (in the following we shall simply speak of an *ESNPA system*) is a construct $\Pi = (m, n, S, R, \Psi)$ 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 and the

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initial value of the (effector) functions in the astrocytes on the axons.

- R is a finite set of *rules* of the form $\left(i, E/a^k \rightarrow P\right)$ such that $i \in [1..m]$ (specifying that this rule is assigned to cell i), E is the regular *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 $l \in [1..m]$ (thus specifying the target neuron), $w \in \mathbb{N}$ is the *weight* of the energy sent along the axon from neuron i to neuron l;
- $\Psi : [m+1..m+n] \rightarrow F$ where F is a set of recursive functions from $\mathbb{N}^{[1..m]^2}$ to $\{-1, 1\}^{[1..m]^2}$. Ψ is a function which to each astrocyte k, $k \in [m+1..m+n]$, assigns a function g_k taking the number of spikes along each axon as input for g_k and, for every axon (i, j) with $i, j \in [1..m]$, yields a value from $\{-1, 1\}$: -1 means that the activity along the axon (i, j) is inhibited; 1 means that the spikes sent along the axon (i, j) are allowed to pass.

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 k, the actual value of g_k for every axon (i, j) with $i, j \in [1..m]$ is specified.

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

- for each neuron i, we first check whether a rule $\left(i, E/a^k \rightarrow P\right)$ can be "activated ", i.e., if the current value of spikes in neuron i is in E; 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 and only if all values of functions g_k for every astrocyte k, $k \in [m+1..m+n]$, is 1 for the axon (i, l), the energy w in a package (l, w) on the axon from neuron i to neuron l is sent to neuron l;
- for each neuron l, we now consider all packages (l, w) on axons leading to neuron l that could pass 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 k, $k \in [m+1..m+n]$, we compute the new values of g_k for every axon (i, j) by considering every package (j, w) on the axons leading from neuron i to neuron j that was allowed to pass in the preceding substep (w corresponds to the input taken for the axon (i, j) in g_k).

In order to illustrate the definitions given above, we consider the ESNPA $\Pi = (2, 1, S, R, \Psi)$ defined as follows (also see Figure 1):

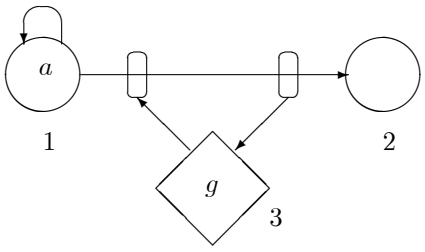

Figure 1: ESNPA system omitting every second spike.

The single rule in R, $R = \{(1, \{a\}/a \rightarrow \{(1, a), (2, a)\})\}$, means that if we find one spike in neuron 1, then this spike is consumed and one spike is sent to neuron 1 as well as to neuron 2.

The initial configuration S is given by one spike in neuron 1 and no spike in neuron 2 as well as the initial values for g_3 for the astrocyte labelled by 3 set to be 1 for all axons $((1, 1), (1, 2), (2, 1), (2, 2))$. As far as Ψ is concerned (also see Figure 1) we are only interested in the effect of astrocyte 3 on the axon $(1, 2)$ depending on the activities (spikes passing this axon) along this axon which formally means that g_3 is 1 for all inputs and all axons except for the case that g_3 yields the value -1 for the axon $(1, 2)$ if and only if a spike has been sent along axon $(1, 2)$. Hence, in Figure 1 we have only specified the *affector* arc from axon $(1, 2)$ to astrocyte 3 (this is the only input which affects the value of g_3) as well as only one *effector* arc from astrocyte 3 to the axon $(1, 2)$.

If we consider the time evolution of this system we immediately see that after the first spike has passed axon $(1, 2)$, in the next step, this axon is inhibited by the astrocyte 3. For the third time step, this means that the next spike from neuron 1 can again pass the axon, because no spike has passed in the previous step, and therefore, astrocyte 3 does not inhibit the passage of this spike. In sum, we see that a spike can pass from the source neuron 1 to neuron 2 in every odd step, whereas in every even time step, the passage of the spike sent along the axon $(1, 2)$ from the source 1

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is inhibited by the astrocyte 3.

For the sake of conciseness, in the following we restrict ourselves to very simple functions of the form $= h, \leq h, \geq h$ and $\neq h$ and as well as to only specify a subset of the set of axons $[1..m]^2$ as input for the astrocyte k (these are called *affectors*) and excitatory and inhibitory arcs from astrocyte k to axons from $[1..m]^2$ (both will be called *effectors*). According to this convention, those axons which are not specified as affectors have no influence on the value of g_k and for the axons not specified to be effected by the astrocyte k we assume the value of g_k to be 1 in any case. Distinguishing between excitatory and inhibitory effectors means that

- for an excitatory effector, the value of g_k is assumed to be 1 if and only if the sum s of the values of the inputs (affectors) fulfills the condition specified by g_k, i.e., if and only if $s \triangle h$ is true for k being specified as $\triangle h$, $\triangle \in \{=, \leq, \geq, \neq\}$;
- for an inhibitory effector, the value of g_k is assumed to be -1 if and only if the sum s of the values of the inputs (affectors) fulfills the condition specified by g_k, i.e., if and only if $s \triangle h$ is true for k being specified as $\triangle h$, $\triangle \in \{=, \leq, \geq, \neq\}$.

If several astrocytes effect an axon (i, j), then spikes can pass this axon if and only if every astrocyte k effecting this axon yields the value 1 for this axon with the function g_k, i.e., all excitatory effectors are activated (value 1) and all inhibitory effectors are not activated (do not have value -1)

According to the conventions described above the ESNPA from Figure 1 can also be illustrated as in Figure 2.

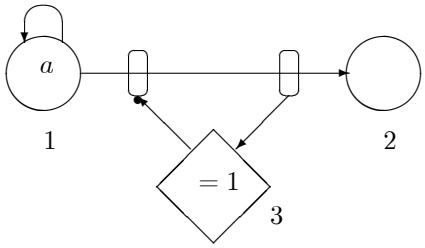

Figure 2: ESNPA system with inhibitory effector.

Figure 2 shows the variant where we use an inhibitory effector, which variant directly corresponds to the definition originally given for this system. On the other hand, Figure 3 shows a system with the same time behaviour yet using an excitatory effector.

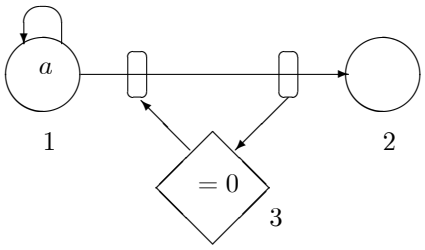

Figure 3: ESNPA system with excitatory effector.

3 Modelling Logical Gates with Simple ESNPA Systems

Even when using only restricted functions in the astrocytes as described in the previous section, i.e., $\triangle h$, for $\triangle \in \{=, \leq, \geq, \neq\}$, for excitatory and inhibitory effectors, we can easily model logical gates. In contrast to the variant investigated in [2], we do not consider neurons as input and output, yet instead the activities along axons are taken as input and effect the activity along an output axon.

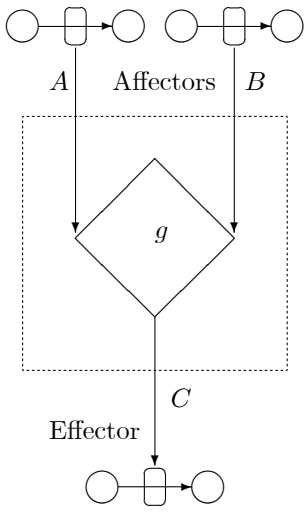

Figure 4: Excitatory astrocyte.

Figure 4 shows the implementation of the logical gate when using an astrocyte with an excitatory effector provided that in any case only one spike in any time step may pass along an axon. Spikes passing along two axons A and B affect an astrocyte which in an excitatory way effects an axon C. Depending on the function g used in the astrocyte, according to table 1, we obtain the corresponding logical gate.

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Table 1 Functions g for implementing logical gates with excitatory and inhibitory effector.

excitatory	inhibitory	logical gate
$= 2$	< 2	AND
< 2	$= 2$	$NAND$
≥ 1	$= 0$	OR
$= 0$	≥ 1	NOR
$= 1$	$\neq 1$	XOR

When using the corresponding functions g in the column for inhibitory effectors in table 1, we obtain the corresponding implementations of the logical gates with inhibitory effectors (see Figure 5), again provided that the affecting axons only allow at most one spike to pass in each time step. In contrast to the excitatory effecting arc, the inhibitory effector is marked with a dot at the end of the arc.

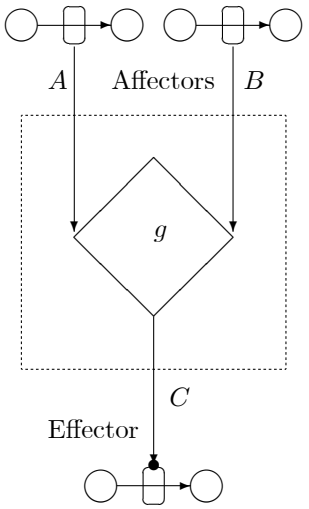

Figure 5: Inhibitory astrocyte.

If more than one spike is allowed to pass along an affecting axon, then we have to use the simulation as depicted in Figure 6, where the inputs from A and B have to be normalized (to 1). In this case, for each input (A and B), we need an intermediate layer with two neurons with the source containing one spike in the beginning and sending one spike to the second neuron (sink) in each step and with an axon leading from the source to the sink (where in each time step any arriving spike is consumed in the next step) which axon is influenced by an astrocyte affected by the input axons A and B, respectively.

4 Computational Power

An ESNPA system can be considered as a computing device as exhibited in the previous section, where we considered logical gates which can be seen as a basic for constructing universal machines. In that sense, ESNPA systems have the same computational power as Turing machines, etc.

This already follows from the results established in [1], where extended spiking neural P system without astrocytes were shown to be computationally complete, i.e., able to compute any partial recursive function on $\mathbb{N}$. Observe that computational completeness was already established for the original model of spiking neural P systems (see [5]). In the papers cited above, spiking neural P systems were especially considered as generating devices, where for the unbounded variants, i.e., without bounding the number of spikes in the neurons, computational completeness could be established for many variants when considering the input to be the number of spikes in a specified input neuron and the result to be the number of spikes in a specific output neuron. As special cases, (extended) spiking neural P systems (with astrocytes) can be considered as generating or accepting devices.

As it was shown in [1] when bounding the number of spikes in all neurons, only regular functions can be computed and only regular sets can be generated/ accepted. If we only consider the restricted functions as used for modelling the logical gates in the previous section, the argumentations there immediately imply that we stay within regular functions/sets when restricting ourselves to ESNPA systems with a bounded number of spikes in every neuron.

The variant of ESNPA systems considered in this paper differs from the variant considered in [2] in such a way that here, the influence of an astrocyte on an axon depends on the activities of spikes along other axons, whereas in the model considered in [2], the astrocytes were influenced directly by the neurons. Which variant might be closer to the biological reality remains as a challenging question for future research.

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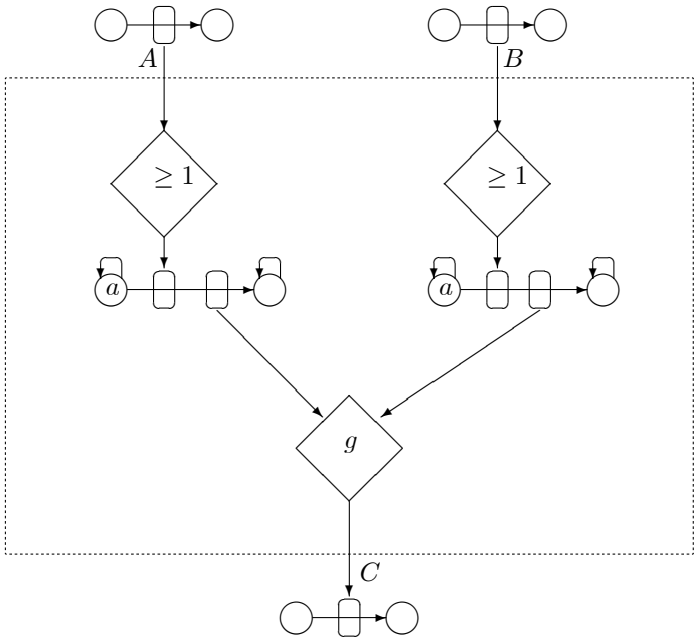

Figure 6: Logical gates with normalization of affector signals.

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