An. Ştiinţ. Univ. Al. I. Cuza Iaşi. Mat. (N.S.)
Tomul LXVI, 2020, f. 2

Age-structure in neuronal models

Carmen-Oana Tarniceriu

Abstract We review in this paper few age-dependent population models that are currently in use in the field of Computational Neuroscience. The general form of the mainly used models and the interpretation of the notions in the special case of neuronal population are given. Moreover, some special connections that can be made with a biophysical model are presented.

Keywords Renewal theory · Age-structured equations · Fokker-Planck equations

Mathematics Subject Classification (2010) 60H10 · 60K05

1 Introduction

"Age" is an unusual notion in the context of neural dynamics structuring variables. It is actually used to express the time that has passed since a neuron has emitted an electrical signal (*spike*). That is why, in literature, these models can be found under the name of *time elapsed models* or *refractory densities models* [14, 15, 6, 18, 19].

A spike is generated at the single cell level by including both internal mechanisms as well as network influences. A population's of neurons activity is next defined as the limit of the number of spikes registered in a very short amount of time. Considering as the main hypothesis that the time elapsed since the last spike is a good predictor of a population of neurons activity, the consequent dynamics of a population of neurons can be given in the form of an age-structured population. The key element in these models is the *age-dependent death rate*, or, equivalently, *hazard rate*. The complexity of the functional form of this rate highly influences the population's dynamics, allowing successful simulations of various neural activity patterns. Although simple in form, the age-dependent models of neural populations provide a powerful tool in predicting the non-stationary dynamics of large population of neurons, such as oscillations.

The main difficulty in using the age structured models in this setting is to realistically connect the hazard rate to biophysical features of the neural cell, such as its membrane potential. To this account, in this survey, we will underline some connections between an age structured model and a model of a neural population that is structured by the membrane's potential variable.

For the start, we will present some age-structured models of neuronal populations currently in use in the field of Computational Neuroscience. We briefly remind the main considerations that lead to particular forms of the hazard rate and we end by reviewing the connections that can be made between an age-structured model and a model of a population of neurons structured by the membrane's potential.

2 Age-structured models of populations of neurons

2.1 Not-connected homogeneous populations

The introduction of age-structured models to describe the neural populations dynamics was based on the renewal hypothesis: the influence of the past activity over the state of a neuron at the present moment goes back only up until the last firing time (spike) [14].
That permitted the introduction of the survivor function i.e. the probability that a neuron "survives" without firing only as a function of the *time elapsed since the last spike*, or, what we are going to call from now on, *age*, $P(a)$.
Due to the characteristics of a neuron's activity, the survivor function should obey to the following restrictions:

$$\lim_{a\to\infty} P(a) = 0,$$

since a neuron should fire at some point, eventually, and

$$P(0) = 1,$$

since age zero actually means the neuron just fired, therefore the probability to spike again in the same moment is zero.
Closely related to this function, is the *distribution of inter-spike intervals*, which is given by:

$$ISI(a) = -\frac{\mathrm{d}}{\mathrm{d}a}P(a),$$

which, in turn, allows the introduction of the *mean inter-spike interval duration*:

$$T = \int_0^\infty aISI(a)\,\mathrm{d}a.$$

Finally, the *hazard rate* (*stochastic intensity of firing* or *age dependent death rate*) is defined as

$$\rho(a) = \frac{ISI(a)}{P(a)}. \tag{2.1}$$

Considering now a large population of neurons receiving the same (constant) input, their dynamics are described then by ([15])

$$\frac{\partial}{\partial t}n(t,a) + \frac{\partial}{\partial a}n(t,a) + \rho(a)n(t,a) = 0, \tag{2.2}$$

where by $n(t,a)$ it is denoted the density of neurons having at time t the age a.

Here, the fraction of neurons that spike is given by the term $\rho(a)n(t,a)$ and, to account for the fact that the density of neurons having at time t age zero is given by the total number of neurons that spiked at that moment, a non-local boundary condition is imposed:

$$n(t,0) = \int_0^\infty \rho(a)n(t,a)\,\mathrm{d}a. \tag{2.3}$$

The integral in the right hand side equation above gives the *population's activity* which is going to be denoted from now on by $r(t)$.
To complete the model, one can take the initial repartition of neurons as known:

$$n(0,a) = n_0(a). \tag{2.4}$$

Given the particularities of a neural population, a supplementary condition expressing the fact that the number of neurons of the population remains constant needs to be imposed. We shall consider here the normalized form of it, namely, supposing that

$$\int_0^{+\infty} n_0(a)\,da = 1,$$

then, at any time $t > 0$, the following should hold:

$$\int_0^{+\infty} n(t,a)\,da = 1. \tag{2.5}$$

Of course, the model above is well known in the field of age-structured population dynamics and much attention it has been given to it up to now. Note that the assumptions that allowed its implementation for neural populations are restrictive, therefore many authors have considered more complex cases to express realistic dynamics of neural networks. We will remind few of them below.

A first more general condition to impose is to consider a time-dependent external input that all neurons in the population receive. Obviously, in this way, the hazard rate becomes time dependent ([15]), which corresponds to the so called non-autonomous case:

$$\rho := \rho(t,a).$$

Of course, in this case, both the survivor function and the inter-spike intervals distribution will become time dependent, i.e. $P := P(t,a)$ and $ISI := ISI(t,a)$, which leads to another relation between the two functions:

$$ISI(t,a) = -\frac{\mathrm{d}}{\mathrm{d}t}P(t,a),$$

where $\frac{\mathrm{d}}{\mathrm{d}t}$ denotes the total derivative with respect to time.

2.2 Inter-connected populations

The next natural step is to consider networks of neurons, which should reflect the fact that the neurons in the population are connected via synapses. In our context, this translates into the fact that every neuron in the population is influenced by the total activity of the network.

The case of large fully connected populations was treated in [18], where the following model has been proposed:

$$\begin{cases} \dfrac{\partial}{\partial t}n(t,a) + \dfrac{\partial}{\partial a}n(t,a) + \rho(a, X(t))n(t,a) = 0 \\ n(t,0) := r(t) = \displaystyle\int_0^\infty \rho(X(t),a)n(t,a)\,\mathrm{d}a \\ n(0,a) = n_0(a). \end{cases} \tag{2.6}$$

Here

$$X(t) = J\int_0^t \alpha(u)r(t-u)\,\mathrm{d}u \tag{2.7}$$

defines the global activity function of the population. The connectivity in the considered population is made via the (non negative) parameter $J > 0$, and the average delay of the transmitted signal is expressed by the function α. As in the previous model, the conservation property of the network is assumed:

$$\int_0^\infty n(t,a)\,\mathrm{d}a = \int_0^\infty n_0(a)\,\mathrm{d}a = 1, \forall t > 0.$$

The well posedness of the model and existence of steady states for different values of the connectivity parameter have been proven. Special behaviours of the solutions of the non-linear model above, in terms of the connectivity parameter, have been analysed in [19].

Also, well-posedness results and asymptotic properties of some modified versions of the model can be found in [4].

Moreover, a micro-foundation of the model starting from a point-processes model has been proposed in [5].

2.3 Finite-size populations

The above models are illustrating the case of infinitely large populations, which is an obvious idealization when talking about biological systems. Under the hypothesis of a finite-size population of connected neurons, there has been derived in [11] an age-structured model for a population of N neurons:

$$\begin{cases} \dfrac{\partial}{\partial t}n(t,a) + \dfrac{\partial}{\partial a}n(t,a) = -\rho(t,a)n(t,a) - \sqrt{\dfrac{\rho(t,a)n(t,a)}{N}}\eta(t,a) \\ n(t,0) := r(t) = \displaystyle\int_0^\infty \rho(t,a)n(t,a)\,\mathrm{d}a + \int_0^\infty \sqrt{\dfrac{\rho(t,a)n(t,a)}{N}}\eta(t,a)\,\mathrm{d}a \end{cases} \tag{2.8}$$

The first equation in (2.8) is a stochastic partial differential that describes the temporal evolution of the finite size refractory density $n(t,a)$. Here, $\eta(t,a)$ is a Gaussian white noise with mean and correlation given by:

$$< \eta(t,a) >= 0, \quad < \eta(t,a)\eta(t',a') >= \delta(t-t')\delta(a-a').$$

The consideration of finite size population hypothesis has been proven to allow the recovery of small finite-size fluctuations that are present at network level and which fail to be captured in the deterministic model. There should be noted that, in the $N \longrightarrow \infty$ limit, the classical deterministic model is recovered.

Further directions have been employed in order to obtain more realistic models that can render observed particular dynamics of the population, as well as to connect them with the biophysical characteristics of the cell. Apart from the use of the renewal theory in the description of neural networks (see also [12]), there has been used the so-called quasi-renewal hypothesis, which leaded to the dependence of the hazard rate to the recent history of spiking [17]. The extraction of the hazard rate from conductance-based neural models has been illustrated in [6, 7], numerical simulations of the age-structured models can be found in [7–10]. Another extension of the age structured models to the case of finite size population with a modified hazard rate has been treated in [21]. For a review of the refractory densities models corresponding to generalized integrate-and-fire neurons we refer to [22].

3 Links with a biophysical model

The age-structured models rely, therefore, on the building assumption that one can take the time elapsed since the last spike as the structuring variable and considering an appropriate age-dependent death rate to capture the probability of spiking for a certain input current. The main problem of such a model is the missing link with biophysical characteristics of the neurons in the considered population.

Up to now, in order to incorporate the mechanisms that influence the single neuron dynamics in the age-structured population models, there have been proposed special forms of hazard rates that have been derived from conductance based single neuron models [6, 7], and we also refer to [22] for a review of refractory density models derived from conductance-based neuron models.

Another way, which is going to be reviewed below, finds a theoretical link between a population of neurons individually described by the noisy leaky integrate-and-fire (NLIF) model, and the age-structured population model (2.2) - (2.4). This has been done by appropriately expressing the stochastic dynamics described by the NLIF model in the equivalent description in the form of Chapman-Kolmogorov equations.

3.1 The Noisy Leaky Integrate-and-Fire model

To fix the framework, we shortly present here the noisy leaky integrate and fire neuron model, for which we mainly refer to [3].

The (normalized) model consists in a stochastic differential equation that gives the evolution in time of the membrane potential of a neuron:

$$\frac{\mathrm{d}}{\mathrm{d}t}v(t) = \mu - v(t) + \xi(t), \tag{3.1}$$

completed with a reset mechanism:

$$\text{whenever} \quad \mathrm{v} \geq 1, \quad \text{then} \quad \mathrm{v} \mapsto \mathrm{v_r}. \tag{3.2}$$

Here μ is a constant external stimulus acting upon the neuronal cell and $\xi(t)$ is a Gaussian white noise process:

$$< \xi(t) >= 0, \quad < \xi(t)\xi'(t) >= \sigma\delta(t-t'),$$

The model consists, therefore, in a deterministic part giving the drift due to the deterministic current μ, and a stochastic part given by the noise term $\xi(t)$, that expresses the influence of the network over the neuron.

The reset mechanism (3.2) says that the neuron will emit a spike whenever the potential reaches the (fix) threshold, which is here normalized to $v = 1$, and once the spike occurred, instantaneously, the potential is reset to another fix value $v_r < 1$, called the reset potential.

3.2 The population density model

Considering now the corresponding population density of neurons having at time t the membrane's potential value v, $p(t,v)$, a population of NLIF neurons can be described by the following equation [1]:

$$\frac{\partial}{\partial t}p(t,v) + \overbrace{\frac{\partial}{\partial v}[(\mu - v)p(t,v)]}^{\text{Drift}} - \overbrace{\frac{\sigma^2}{2}\frac{\partial^2}{\partial v^2}p(t,v)}^{\text{Diffusion}} = \overbrace{\delta(v - v_r)r(t)}^{\text{Reset}}. \tag{3.3}$$

In the equation (3.3), the evolution in time of the density functions is influenced by the two processes present in the single neuronal dynamics (3.1): the drift, which is generated by the deterministic part and the diffusion term which is generated by the action of noise over the individual trajectories. The term in the right hand side describes the reset mechanism: the neurons that spiked re-enter in the potential interval at the reset value $v = v_r$.
The spiking process which is considered to take place whenever the neuron's potential reaches the threshold value $v = 1$, leads to the absorbing boundary condition:

$$p(t,1) = 0, \tag{3.4}$$

and a reflecting boundary condition

$$\lim_{v \to -\infty}\left[(-\mu + v)p(t,v) + \frac{\sigma^2}{2}\frac{\partial}{\partial v}p(t,v)\right] = 0, \tag{3.5}$$

is imposed at the lower one, which states that there is no flux passing through this boundary.

Also, the firing rate of the population, $r(t)$, is defined as the flux at the firing threshold:

$$r(t) = -\frac{\sigma^2}{2}\frac{\partial}{\partial v}p(t,1). \tag{3.6}$$

Finally, an initial condition is given:

$$p(0,v) = p_0(v). \tag{3.7}$$

As before, the fact that the number of neurons in the population remains fix is expressed by the (normalized) conservation property

$$\int_{-\infty}^{1} p(t,v)\,dv = 1, \tag{3.8}$$

which can be obtained for every $t > 0$ by direct integration of the equation (3.3) over the potential values interval, if one imposes that at initial time:

$$\int_{-\infty}^{1} p_0(v)\,dv = 1. \tag{3.9}$$

The Fokker-Planck equation (3.3) with the corresponding conditions was used in literature also to model the probability density function. For more considerations about the the Fokker-Planck equation we refer to [19], [16], [2].

3.3 Forward and backward Chapman-Kolmogorov equations

We deal therefore with a population of NLIF neurons subject to the dynamics described by (3.3)-(3.7).

Since the state of a single neuron is described by a stochastic variable, it is usual to consider instead the equivalent description in terms of the probability density function that gives the likelihood for the neuron to be at a given moment in the state v. We arrive this way to the Chapman-Kolmogorov equations associated to stochastic differential equations. More precise, two conditional probabilities are introduced [13]:

– The conditional probability for a neuron that started at the last firing time s from the corresponding state v_r to be at time t in a state v

$$\varphi(a,v) := \mathcal{P}(t-s, v|0, v_r),$$

where $a = t - s$, and the variables which are kept fix are ignored from the notation;

– The conditional probability for a neuron that started at age zero from a potential value v to"survive" up to the age a:

$$\psi(a,v) := \int_{-\infty}^{1} \mathcal{P}(t-s, w|0, v)\,\mathrm{d}w.$$

It can be shown [13] that the joint probability $\varphi(a,v)$ is solution to the *forward Chapman-Kolmogorov equation*, mainly known as the Fokker-Planck equation:

$$\frac{\partial}{\partial a}\varphi(a,v) + \frac{\partial}{\partial v}[(\mu - v)\varphi(a,v)] - \frac{\sigma^2}{2}\frac{\partial^2}{\partial v^2}\varphi(a,v) = 0, \tag{3.10}$$

which is completed, in our case, with the natural initial condition

$$\varphi(0,v) = \delta(v - v_r). \tag{3.11}$$

and the boundary condition that is due, again, to the firing process at threshold value:

$$\varphi(a,1) = 0. \tag{3.12}$$

A reflecting boundary is also required:

$$\lim_{v\to-\infty}\left[(v-\mu)\varphi(a,v) + \frac{\sigma^2}{2}\frac{\partial}{\partial v}\varphi(a,v)\right] = 0. \tag{3.13}$$

In this case, the inter-spike interval distribution is defined as the probability flux at the firing threshold:

$$ISI(a) = -\frac{\sigma^2}{2}\frac{\partial}{\partial v}\varphi(a,1). \tag{3.14}$$

Starting with (3.14), the survivor function $\mathcal{P}$ as the probability to survive up to age a for a neuron that started at age zero from the position v_r is defined as

$$\mathcal{P}(a) = \int_{-\infty}^{1}\varphi(a,v)\,\mathrm{d}v. \tag{3.15}$$

Also, it takes place:

$$ISI(a) = -\frac{\partial}{\partial a}\int_{-\infty}^{1}\varphi(a,v)\,\mathrm{d}v. \tag{3.16}$$

The conditional probability $\psi(a,v)$ is proven to satisfy the backward Chapman - Kolmogorov equation, which, by a suitable change of variable is written as:

$$\frac{\partial}{\partial a}\psi(a,v) - (\mu - v)\frac{\partial}{\partial v}\psi(a,v) - \frac{\sigma^2}{2}\frac{\partial^2}{\partial v^2}\psi(a,v) = 0, \tag{3.17}$$

with a natural initial condition:

$$\psi(0,v) = 1, \tag{3.18}$$

and the boundary conditions:

$$\psi(a,1) = 0, \tag{3.19}$$

$$\lim_{v\to-\infty}\frac{\partial\psi(a,v)}{\partial v} = 0. \tag{3.20}$$

The biological interpretation of ψ is that of the probability of survival at age a for a neuron that started at initial time from a potential value v [20]. Note that, in this interpretation, the initial condition is natural since the probability of survival of e neuron starting at initial time from a potential $v < 1$ is 1.

3.4 Connections with the noisy integrate-and-fire neurons population

The above description of the single neuron dynamics lead to the possibility of recovering the NLIF neurons model from the AS model, and the other way around. We stress that this will take place for the hazard rate defined by (2.1).

More precisely, the following two results have been stated in [8], [9]:

Theorem 3.1 *Let p a solution to (3.3)-(3.7) and n a solution to (2.2)-(2.4), and $p_0(v)$ and $n_0(a)$ two corresponding initial densities, respectively. Then if p_0 and n_0 satisfy*

$$p_0(v) = \int_0^{+\infty} \frac{\varphi(a,v)}{P(a)} n_0(a)\, da, \tag{3.21}$$

the following relation holds true for any time $t > 0$:

$$p(t,v) = \int_0^{+\infty} \frac{\varphi(a,v)}{P(a)} n(t,a)\, da. \tag{3.22}$$

Here, $\varphi(a,v)$ is the solution to (3.10)-(3.13) and $P(a)$ is the survivor function.

The inverse transformation is a bit trickier: one can find the solution to the age structured system as a flux at time t through the state a of the neurons that started from the potential value v at the firing time $t-a$, but only for the case $t > a$. For the other case, a global relation between the two solutions has been proven. More precisely, the following result was shown to take place [9]:

Theorem 3.2 *Let p be a solution to (3.3)-(3.7) and n be a solution to (2.2)-(2.4), so that the compatibility condition between the corresponding initial states p_0 and n_0 takes place.*

Then, for any t such that $0 \leq a < t$, the following relation holds true:

$$n(t,a) = -\frac{\partial}{\partial a} \int_{-\infty}^{1} \psi(a,v) p(t-a,v)\, \mathrm{d}v. \tag{3.23}$$

Moreover, for any $a \geq t$, a global relation between the corresponding probabilities at time t takes place:

$$\int_t^{+\infty} n(t,a)\, da = \int_{-\infty}^{1} \psi(t,v) p_0(v) dv. \tag{3.24}$$

Here, $\psi(\cdot,v)$ is the solution of the dual problem (3.17)-(3.20).

The above two results, therefore, show a connection between the two considered models, by making use of the fact that the single neuron dynamics can be expressed in terms of Chapmann - Kolmogorov equations. Note that the the Chapmann - Kolmogorov equations give the evolution of the probability densities functions on a single inter-spike interval. That was possible due to the assumption that the input current present in the NLIF model was considered stationary. The more complex case, where the stimulus considered is time dependent, forces the change of the framework, and some results in this case have been stated in [10].

4 Remarks

The low-dimensionality, the versatility and also the rich dynamics that can be captured by the age-structured models are as many reasons for its use in Computational Neuroscience. The biggest challenge though is to correctly define the hazard function so that, on one hand, to realistically connect it to the biophysical cell characteristics, and, on the other hand, to capture in the resulting dynamics the richness of real networks of neurons' behaviour.

Here we have presented only few of the models existing in the literature, that we found of interest. We did insist on connecting age structured models to a model that is well known in the literature for a set of reasons: First of all, these connections do not come as a surprise: similar statistics of spike are generated by the two models [15]. Second, the age structured model fails to express the influence of some parameters that are present in the NLIF model over the population dynamics. Interestingly, a different way of deriving the age-structured model from a model that has a biological interpretation comes as a consequence. Also, the inverse dependence comes with an advantage: the dynamics of the NLIF population give the evolution of the population with respect to the membrane potential. But knowing the membrane potential does not give any crucial information about the spiking times, since two neurons can have the same potential value but different "ages". Therefore, using an age-dependent model instead comes with obvious advantages from this point of view.

References

1. Brunel, N. – *Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons*, Journal of Computational Neuroscience, **8** (2000), 183-208.
2. Brunel, N.; Hakim, V. – *Fast global oscillations in networks of integrate-and-fire neurons with low firing rates*, Neural Comput., **11** (1999), no. 7, 1621-1671.
3. Burkitt, A.N. – *A review of the integrate-and-fire neuron model: I. homogeneous synaptic input*, Biological Cybernetics, **95** (2006), 1-19.
4. Cañizo, J.A.; Yoldaş, H. – *Asymptotic behaviour of neuron population models structured by elapsed-time*, Nonlinearity, **32** (2019), no. 2, 464-495.
5. Chevallier, J.; Cáceres, M.J.; Doumic, M.; Reynaud-Bouret, P. – *Microscopic approach of a time elapsed neural model*, Math. Models Methods Appl. Sci., **25** (2015), no. 14, 2669-2719.
6. Chizhov, A.V. – *Conductance-based refractory density approach: comparison with experimental data and generalization to lognormal distribution of input current*, Biol. Cybernet., **111** (2017), no. 5-6, 353-364.
7. Chizhov, A.V.; Graham, L.J. – *Population model of hippocampal pyramidal neurons, linking a refractory density approach to conductance-based neurons*, Phys. Rev. E, **75** (2007), 011924.
8. Dumont, G.; Henry, J.; Tarniceriu, C.O. – *Noisy threshold in neuronal models: connections with the noisy leaky integrate-and-fire model*, J. Math. Biol., **73** (2016), no. 6-7, 1413-1436.
9. Dumont, G.; Henry, J.; Tarniceriu, C.O. – *Theoretical connections between mathematical neuronal models corresponding to different expressions of noise*, J. Theoret. Biol., **406** (2016), 31-41.
10. Dumont, G.; Henry, J.; Tarniceriu, C.O. – *A theoretical connection between the Noisy Leaky integrate-and-fire and escape rate models: The non-autonomous case*, Math. Model. Nat. Phenom., **15** (2020), no. 59.

11. Dumont, G.; Payeur, A.; Longtin, A. – *A stochastic-field description of finite-size spiking neural networks*, PLOS Computational Biology, **13** (2017).
12. Farkhooi, F.; van Vreeswijk, C. – *Renewal approach to the analysis of the asynchronous state for coupled noisy oscillators*, Phys. Rev. Lett., **115** (2015), no. 3.
13. Gardiner, C.W. – *Handbook of Stochastic Methods: for Physics, Chemistry and the Natural Sciences*, Springer, 1996.
14. Gerstner, W. – *Time structure of the activity in neural network models*, Phys. Rev. E., **51** (1995), 738-758.
15. Gerstner, W.; Kistler, W. – *Spiking neuron models*, Single neurons, populations, plasticity, Cambridge University Press, Cambridge, 2002.
16. Knight, B.W. – *Dynamics of encoding in neuron populations: some general mathematical features*, Neural Comput., **12** (2000), no. 3, 473-518.
17. Naud, R.; Gerstner, W. – *Coding and decoding with adapting neurons: a population approach to a peri-stimulus time histogram*, PLOS Comput. Biol., **8** (2012), no. 10, 1-14.
18. Pakdaman, K.; Perthame, B.; Salort, D. – *Dynamics of a structured neuron population*, Nonlinearity, **23** (2009), no. 1, 23-55.
19. Pakdaman, K.; Perthame, B.; Salort, D. – *Relaxation and self-sustained oscillations in the time elapsed neuron network model*, SIAM J. Appl. Math., **73** (2013), no. 3, 1260-1279.
20. Plesser, H.E.; Gerstner, W. – *Noise in integrate-and-fire neurons: from stochastic input to escape rates*, Neural Comput., **12** (2000), no. 2, 367-384.
21. Schwalger, T.; Deger, M.; Gerstner, W. – *Towards a theory of cortical columns: From spiking neurons to interacting neural populations of finite size*, PLOS Comput. Biol., **13** (2017), 1005507.
22. Schwalger, T.; Chizhov, A.V. – *Mind the last spike-firing rate models for mesoscopic populations of spiking neurons*, Current Opinion in Neurobiology, **58** (2019), 155-166.

Received: 14.XI.2020 / Accepted: 15.XII.2020

Author

Carmen-Oana Tarniceriu,
Department of Mathematics and Informatics,
“Gheorghe Asachi” Technical University of Iaşi,
Iaşi, România,
and
Interdisciplinary Research Department - Field Sciences,
“Alexandru Ioan Cuza” University of Iaşi,
Iaşi, România,
E-mail: oana.tarniceriu@tuiasi.ro