Аналіз синаптичної передачі сигналів на основі кінетичної моделі

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ANALYSIS OF THE SYNAPTIC SIGNAL TRANSMISSION BASED ON A KINETIC MODEL

We will analyze the peculiarities of the nerve signal transmission through a synaptic cleft (a contact between two neurons). The corresponding analysis is performed using a kinetic model that is based on a system of nonlinear differential equations of the first order and makes it possible to calculate the number of activated receptors on the postsynaptic membrane and the amount of mediator in the synaptic cleft. The model combines simplicity and functionality, which allows obtaining the qualitative results comparable to available experimental data and the results of other theoretical studies. It has been shown that the model correctly describes the process of signal transmission through the synaptic cleft at a qualitative level. Exact (numerical) and approximate (analytic) solutions for the number of activated receptors on the postsynaptic membrane and the amount of a mediator in the synaptic cleft are obtained and analyzed. The stability of stationary states is considered and proved in the framework of the proposed model, which confirms the self-consistency of the model and the possibility of its use for simulating the signal transmission through the synapse.

K e y w o r d s: synapse, mediator, receptor, exocytosis, impulse.

It's clearly a budget. It's got a lot of numbers in it.

George W. Bush

1. Signal Transmission through the Synapse

The study of the synaptic signaling has a long history [1–9]. However, a number of questions remain unanswered. Among them are tasks ranging from determining the characteristics of molecular mechanisms and identifying the methods of their cooperation with regard for the diffusion processes and the presence of chemical reactions [5, 9–14] to develop a complete analytic theory of synaptic transmission of information as such [5, 9]. The reason is that the signal transmission process itself is a complicated phenomenon and involves various mechanisms and a series of non-trivial interactions [15–22]. Therefore, the number of models and approaches used to study the process of nerve impulse propagation is constantly growing, and specialists from various fields take an active part in research. In this sense, it can be argued that the relevant topic is interdisciplinary. This article is a continuation of a series of studies [23–28], which were initiated in work [5], and, for which, the interdisciplinarity factor was the key one. This

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circumstance made it possible to formulate and develop several original models that allowed us to obtain interesting and promising results in the direction aimed at studying the synaptic transmission of information [28].

As is known, a synapse is a contact between two neurons. The synaptic cleft itself is confined between the presynaptic and postsynaptic membranes. The mediator, a special active substance that interacts with receptors on the postsynaptic membrane and activates them, is released from the presynaptic membrane into the synaptic cleft. The activation of the postsynaptic membrane receptors results in the generation of a nerve impulse, which propagates along a neuron to the next synaptic contact [3, 5, 8]. It is clear that, in this case, the description of the signal transmission process is extremely simplified, but it is sufficient for understanding the simulation principles used in the kinetic model that is considered in the paper.

2. Kinetic Model

Hence, the process of impulse transmission through a synapse is a complicated and multifactor phenomenon, which involves many mechanisms of various types and characters [9]. It is quite difficult to be modeled in the framework of a single approach. In the models proposed to describe the synaptic signal transmission, only a few factors are usually singled out, and attention is focused just on them. For example, there are models of mediator exocytosis into the synapse (see, e.g., works [8, 9, 26, 29]), models of mediator diffusion in the synaptic cleft [25, 28, 30], or models describing the activation of the postsynaptic membrane [5, 25, 26, 28].

It is worth noting separately the models that are based on the universal character of various phenomena and processes (first of all, in the theory of liquids and phase transitions), which makes it possible to analyze the process of synaptic impulse transmission at a higher abstraction level, thus reducing such a consideration to the analysis of physical systems (see, e.g., works [5, 25, 28, 31–34]). But even with such approximations, the models remain difficult for the theoretical analysis. Therefore, extremely valuable are those successful approaches that are based on relatively simple models and, at the same time, allow obtaining valid results, even if they are of a general character [5]. Such models include the kinetic model of the synaptic information transmission. It was created by summarizing available experimental data and the results of theoretical studies.

In the framework of the kinetic model proposed earlier in the series of works [5, 23–26], the process of signal transmission through the synaptic cleft was described by the following system of nonlinear differential equations of the first order:

$$\frac{da}{dt} = k_1(R - a(t))m(t) - k_2\, a(t), \tag{1}$$

$$\frac{dm}{dt} = f(t) - k_1(R - a(t))\, m(t). \tag{2}$$

Here, R denotes the total number of receptors on the postsynaptic membrane, $a(t)$ is the number of activated receptors on the postsynaptic membrane at the time t, $m(t)$ denotes the amount of a mediator in the synaptic cleft, and $f(t)$ is a function that describes the intensity of mediator release into the synaptic cleft. The first term on the right-hand side of Eq. (1) and the second term on the right-hand side of Eq. (2) describe the interaction of the mediator with inactive receptors on the postsynaptic membrane (see, e.g., works [5, 25, 28]). At the same time, the assumption was made that, after the interaction with the receptors, the mediator is removed from the synaptic cleft. This is an evident approximation, but it has no qualitative effect on the final result (see, e.g., works [23–28]).

On the one hand, this model is quite simple. On the other hand, it describes the general process of signal transmission through the synapse rather well, as is evidenced by the results obtained on the basis of this model and its modifications (see, e.g., work [28]). At the same time, it should be noted that the previously obtained results were most likely focused on the analysis of specific solutions for the synapse functioning modes. Such an approach has practical value, but then the properties of the system as such recede into the background. This paper is designed to close the relevant gaps. It contains some qualitative analysis of the model at a general qualitative level, which is combined with a quantitative analysis of separate partial or limiting cases. In so doing, we apply standard approaches and techniques inherent to the theory of stability and the theory of self-organization.

3. Stationary Cases

First of all, let us analyze the results given by the model for stationary cases or, more precisely, for the system states before and after the impulse transfer. Such an analysis is important in view of several circumstances. First of all, this concerns how adequate the model is with respect to the real situation, because it describes a substantially simplified process of impulse transmission through the synapse. So, it is important to make sure that the obtained results are reliable and consistent.

Therefore, before the impulse arrival at $t = 0$, the system is in an unexcited state with $a(0) = 0$ (no activated receptors), $m(0) = 0$ (no mediator in the cleft), and $f(0) = 0$ (no process of mediator exocytosis into the cleft). This state is consistent with the system of equations (1)–(2), being its partial solution. To test the system stability, let us assume the presence of some small deviations $\delta a(t)$ and $\delta m(t)$ for the number of activated receptors and the amount of a mediator in the cleft, respectively. We consider the indicated parameters to be small and write system (1)–(2) in the linear approximation with respect to those parameters as follows:

$$\frac{d\delta a}{dt} = k_1 R\delta m(t) - k_2\delta a(t), \tag{3}$$

$$\frac{d\delta m}{dt} = \delta f(t) - k_1 R\delta m(t), \tag{4}$$

where $\delta f(t)$ denotes the variation (a small deviation) of the function $f(t)$ describing the intensity of attractant introduction into the system. Actually, $\delta f(t)$ is responsible for random acts of mediator exocytosis into the synapse.

The solution of the linear system of equations (3)–(4) can be written in the following form:

$$\delta a(t) = \frac{k_1 R}{k_1 R - k_2} \times$$

$$\times \left(\exp(-k_1 Rt) \int_0^t \delta f(\tau) \exp(k_1 R\tau) d\tau + \right.$$

$$+ \exp(-k_2 t) \int_0^t \delta f(\tau) \exp(k_2\tau) d\tau +$$

$$\left. + \left(A + M - \frac{k_2 A}{k_1 R} \right) \exp(-k_2 t) - M \exp(-k_1 Rt) \right), \tag{5}$$

$$\delta m(t) = M \exp(-k_1 Rt) +$$

$$+ \exp(-k_1 Rt) \int_0^t \delta f(\tau) \exp(k_1 R\tau) d\tau, \tag{6}$$

where $\delta a(0) = A$ denotes the initial deviation of the number of activated receptors, and $\delta m(0) = M$ the initial deviation of the mediator amount. Solutions (5)–(6) asymptotically tend to zero provided that the characteristic time of the period of random mediator exocytosis into the synapse (denoted by t_0) is substantially shorter than the observation time t, i.e., if $t_0 \ll t$ holds. In addition, a sufficient stability condition is the asymptotic $\delta f(t) \sim \exp(-\alpha t)$ for any value $\alpha > 0$. In this sense, it can be considered that the stationary zero solution of system (1)–(2) is stable.

4. General Properties

The following fact is also interesting. System (1)–(2) can be used to obtain the equation

$$\frac{d(a(t) + m(t))}{dt} = f(t) - k_2 a(t). \tag{7}$$

After the integration and taking the conditions $a(0) = a(\infty) = 0$ and $m(0) = m(\infty) = 0$ into account, we get the following:

$$\int_0^\infty f(t) dt = k_2 \int_0^\infty a(t)\, dt. \tag{8}$$

In a certain sense, it is a balance equation, according to which the area under the curve $a(t)$ describing the time dependence of the number of activated receptors is determined by the total amount of a mediator introduced into the synaptic cleft. Therefore, if the function $f(t)$ changes to $f(t)+\delta f(t)$, with $\int_0^\infty \delta f(t) dt = 0$, this correction does not change the integral characteristic $\int_0^\infty a(t) dt$. The integral $\int_0^\infty a(t) dt$ also remains unchanged under the transformation $f(t) \to \lambda f(\lambda t)$.

Generally speaking, the complicated character of the model is associated with its nonlinearity. In turn, the nonlinearity describes the effect of receptor saturation on the postsynaptic membrane. It occurs, if the number of activated receptors approaches the total number of receptors on the postsynaptic membrane. However, if this is not the case (and such a regime is quite feasible and "working"), then the system of model equations becomes substantially simpler. In particular, if the relationship $R \gg a(t)$ holds,

then neglecting the terms proportional to the product $a(t)m(t)$ in the system of equations (1)–(2), we obtain a linear system of differential equations of the first order,

$$\frac{da}{dt} = k_1 R m(t) - k_2 a(t), \tag{9}$$

$$\frac{dm}{dt} = f(t) - k_1 R m(t). \tag{10}$$

This system has an analytic solution. In particular, for the initial conditions $a(0) = 0$ and $m(0) = 0$, we have the following solutions for system (9)–(10):

$$a(t) = \frac{k_1 R}{k_1 R - k_2}\left(\exp(-k_2 t)\int\limits_0^t f(\tau)\exp(k_2\tau)d\tau - \right.$$

$$\left. - \exp(-k_1 R t)\int\limits_0^t f(\tau)\exp(k_1 R\tau)d\tau\right), \tag{11}$$

$$m(t) = \exp(-k_1 R t)\int\limits_0^t f(\tau)\exp(k_1 R\tau)d\tau. \tag{12}$$

In effect, this is a solution of system (5)–(6) at $A = M = 0$.

Using solutions (11)–(12), it is possible to determine, along with other quantities, the conditions (moments of time), when the number of activated receptors and the amount of a mediator in the cleft are maximum. In particular, the number of activated receptors is maximum at the time moment T_a that is a solution of the algebraic equation (hereafter, we assume that $f(0) = 0$):

$$\frac{k_2}{k_1 R - k_2}\left(\exp\left(-k_2 T_a\right)\int\limits_0^{T_a} f(\tau)\,\exp\left(k_2\tau\right)d\tau + \right.$$

$$\left. + \exp\left(-k_1 R T_a\right)\int\limits_0^{T_a} f(\tau)\,\exp\left(k_1 R\tau\right)d\tau\right) =$$

$$= \exp\left(-k_1 R T_a\right)\int\limits_0^{T_a} f(\tau)\,\exp\left(k_1 R\tau\right)d\tau. \tag{13}$$

Accordingly, to determine the time moment T_m, when the mediator amount in the cleft is maximum, we have the following equation:

$$f(T_m) = k_1 R\,\exp(-k_1 R T_m)\int\limits_0^{T_m} f(\tau)\exp(k_1 R\tau)d\tau. \tag{14}$$

Unfortunately, in the general case, the above equations (13) and (14) can be solved only numerically.

5. Quantitative Analysis

For the quantitative analysis of system (1)–(2), it is necessary to normalize the quantities. For this purpose, let us redefine $a \to Ra$, $m \to Rm$, and $t \to \frac{t}{k_1 R}$. We get the following system of equations:

$$\frac{da}{dt} = (1 - a(t))m(t) - k\,a(t), \tag{15}$$

$$\frac{dm}{dt} = \varphi(t) - (1 - a(t))\,m(t), \tag{16}$$

where $k = \frac{k_2}{k_1 R}$ and $\varphi(t) = \frac{f\left(\frac{t}{k_1 R}\right)}{k_1 R^2}$. Then the solution of the corresponding linear system looks like

$$a(t) = \frac{1}{1-k}\left(\exp\left(-kt\right)\int\limits_0^t \varphi(\tau)\,\exp\left(k\tau\right)d\tau - \right.$$

$$\left. - \exp\left(-t\right)\int\limits_0^t \varphi(\tau)\,\exp\left(\tau\right)d\tau\right), \tag{17}$$

$$m(t) = \exp\left(-t\right)\int\limits_0^t \varphi(\tau)\,\exp\left(\tau\right)d\tau. \tag{18}$$

It is obvious that the general solution depends considerably on the character and intensity of transmitted impulses. This means the qualitative dependence of the solution on the function $\varphi(t)$. However, first of all, the peculiarities of a single impulse passage and the issue of the validity of linear approximation are of interest.

In the case where a single impulse passes through the synapse, the function $\varphi(t)$ was chosen in the form

$$\varphi(t) = B\,\exp\left[-\beta(t - t_0)^2\right], \tag{19}$$

where the parameter B determines the signal intensity, the parameter t_0 is the characteristic time of the signal arrival, and the parameter β determines the degree of temporal localization of the signal (actually, its duration). For the indicated $\varphi(t)$-dependence, Fig. 1 illustrates the time-dependences of the number of activated receptors, $a(t)$, calculated for the exact and approximate systems of equations (with the parameter values $k = 2$, $B = 0.5$, $t_0 = 1$, and $\beta = 15$).

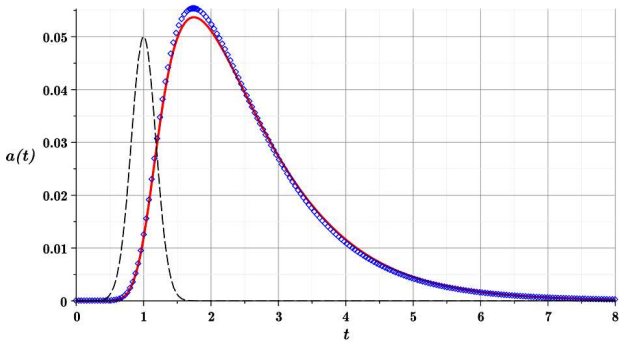

Fig. 1. Dependences $a(t)$ calculated on the basis of the exact (solid curve) and approximate (diamonds) models. The following parameter values were used in calculations: $k = 2$, $B = 0.5$, $t_0 = 1$, and $\beta = 15$. For clarity, the dependence $\varphi(t)/10$ is also plotted (dashed curve)

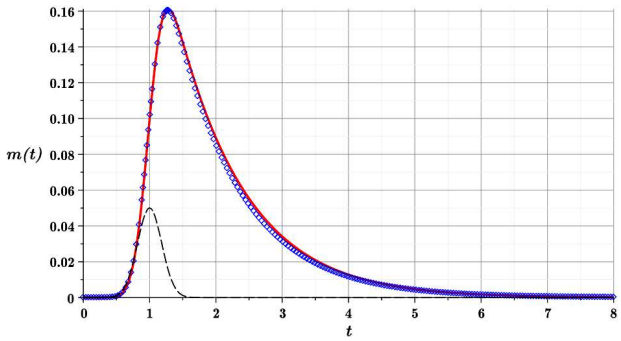

Fig. 2. Dependences $m(t)$ is calculated on the basis of the exact (solid curve) and approximate (diamonds) models. The following parameter values were used in calculations: $k = 2$, $B = 0.5$, $t_0 = 1$, and $\beta = 15$. For clarity, the dependence $\varphi(t)/10$ is also plotted (dashed curve)

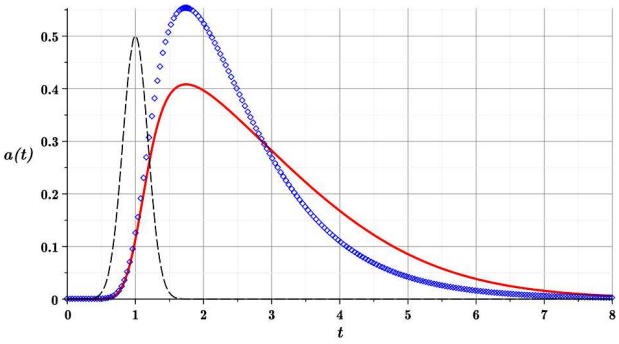

Fig. 3. Dependences $a(t)$ calculated on the basis of the exact (solid curve) and approximate (diamonds) models. The following parameter values were used in calculations: $k = 2$, $B = 5$, $t_0 = 1$, and $\beta = 15$. The dependence $\varphi(t)/10$ is also plotted (dashed curve)

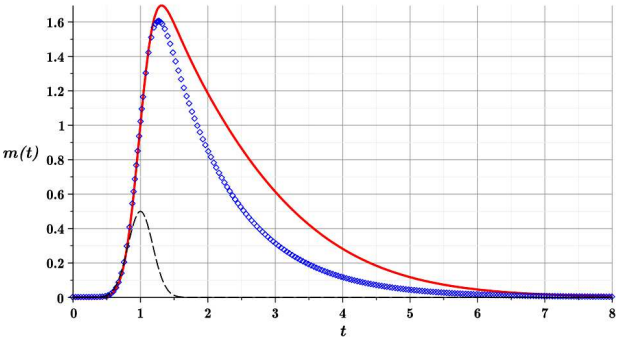

Fig. 4. Dependences $m(t)$ calculated on the basis of the exact (solid curve) and approximate (diamonds) models. The following parameter values were used in calculations: $k = 2$, $B = 5$, $t_0 = 1$, and $\beta = 15$. The dependence $\varphi(t)/10$ is also plotted (dashed curve)

Figure 2 demonstrates the dependences $m(t)$ (the exact and approximate solutions) for the same parameter values ($k = 2$, $B = 0.5$, $t_0 = 1$, and β =15). One can see that, in the considered situation, the approximate solutions give a more than acceptable result (in comparison with the numerical solutions of the original system of equations (15)–(16)). The reason lies in the fact that the amount of a mediator introduced into the synaptic cleft is enough for only a relatively small number of receptors on the postsynaptic membrane (as compared to their total number) to be activated. This result is in the total agreement with the assumption used to obtain the linear approximate system of differential equations (9)–(10).

At the same time, if, for example, the value of the B parameter increases, the difference between the solutions of the approximate linear and original nonlinear systems of equations becomes more noticeable. Figure 3 shows the dependences $a(t)$ for the value $B = 5$ and the same values of other parameters.

The corresponding dependence $m(t)$ is shown in Fig. 4. One can see that, in this case, the linear approximation gives overestimated values for the number of activated receptors and shorter times for the mediator release from the synaptic cleft. That is, in the case where, due to exocytosis, the amount of a mediator in the synaptic cleft is many times larger than the number of receptors on the postsynaptic membrane, the linear approximation is not applicable.

It is also of interest to analyze how the process of receptor activation on the postsynaptic membrane changes, when the $\varphi(t)$ function is varied in such a

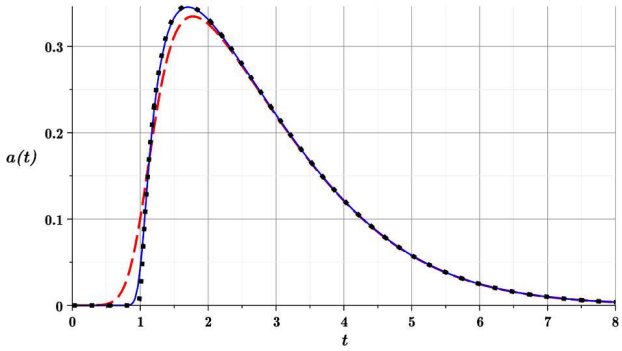

Fig. 5. Dependences $a(t)$ for various values of the parameter $\beta = 10$ (dashed curve), 100 (solid curve), and 1000 (dotted curve). The parameters $k = 2$, $t_0 = 1$, and $B = \sqrt{\beta}$

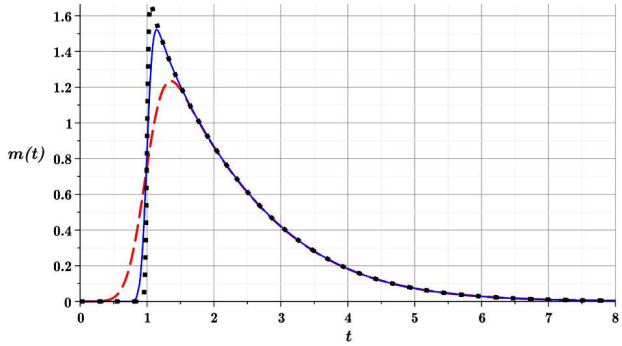

Fig. 6. Dependences $m(t)$ for various values of the parameter $\beta = 10$ (dashed curve), 100 (solid curve), and 1000 (dotted curve). The parameters $k = 2$, $t_0 = 1$, and $B = \sqrt{\beta}$

way that the total amount of the mediator released into the synaptic cleft remains unchanged. In particular, we talk about the synchronous changes of the parameters B and β in dependence (19) such that

$$\int_{-\infty}^{+\infty} \varphi(t)\, dt = \text{const}. \tag{20}$$

The desired effect can be achieved, if we put $B \sim \sqrt{\beta}$. Figure 5 illustrates the dependences $a(t)$ calculated for such a regime. Their counterparts for the amount of the mediator in the synaptic cleft, $m(t)$, calculated for various values of the parameter β are depicted in Fig. 6. One can see that the character of the dependences $a(t)$ does not qualitatively change, when the value of the parameter β changes. This circumstance may be important in the context of the stability of mechanisms governing the signal transmission through synaptic contacts.

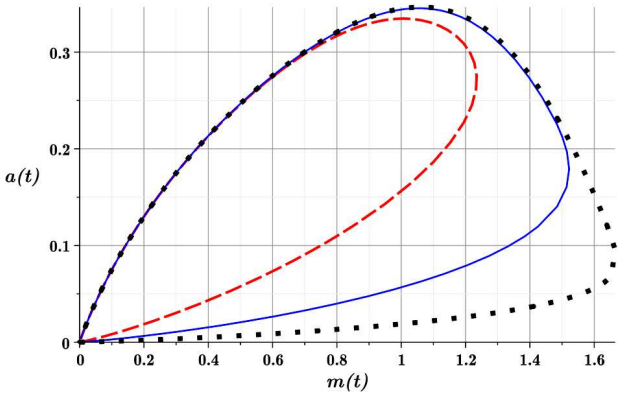

Fig. 7. Parametric dependences $a(t)$ versus $m(t)$ for various values of the parameter $\beta = 10$ (dashed curve), 100 (solid curve), and 1000 (dotted curve). The parameters $k = 2$, $t_0 = 1$, and $B = \sqrt{\beta}$

Also of interest is the parametric dependence between the parameters $a(t)$ and $m(t)$; here, the parameter is the time t. This dependence is presented in Fig. 7. It is clear that it is not unique; each curve is closed, with its beginning and end points at the coordinate origin ($m = 0, a = 0$), because the initial and final states of the system are realized at the zero number of activated receptors and in the absence of a mediator in the synaptic cleft. On the one hand, this result is expected and obvious. On the other hand, it testifies to the self-consistency of the model and the validity of its results, at least at the qualitative level.

6. Conclusions

We have presented the simulation results concerning the process of nerve impulse transmission through the synaptic cleft. The analysis is performed on the basis of a kinetic model. The model is used to calculate, in certain modes, the time dependences of the number of activated receptors on the postsynaptic membrane and the amount of the mediator in the synaptic cleft. The obtained results are in good agreement with the available experimental data and theoretical predictions obtained in the framework of various approaches [9, 28]. It is also shown that, despite its relative simplicity, the kinetic model of signal transmission through the synapse is self-consistent and allows obtaining the reliable results that explain the peculiarities in the passage of impulses through the synapse.

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АНАЛІЗ СИНАПТИЧНОЇ ПЕРЕДАЧІ
СИГНАЛІВ НА ОСНОВІ КІНЕТИЧНОЇ МОДЕЛІ

У статті аналізуються особливості передачі нервових сигналів через синаптичну щілину (контакт між двома нейрона-

ми). Відповідний аналіз виконується з використанням кінетичної моделі, яка ґрунтується на системі нелінійних диференціальних рівнянь першого порядку і дозволяє відстежувати кількість активованих рецепторів на постсинаптичній мембрані та кількість медіатора в синаптичній щілині. Модель поєднує в собі відносну простоту та функціональність, що дозволяє отримувати якісні результати, співставні з наявними експериментальними даними та результатами інших теоретичних досліджень. Показано, що модель на якісному рівні коректно описує процес проходження сигналу через синаптичну щілину. Отримано та проаналізовано точні (числові) та наближені (аналітичні) розв'язки для кількості активованих рецепторів на постсинаптичній мембрані та кількості медіатора в синаптичній щілині. Доведено стійкість стаціонарних станів в рамках моделі, що свідчить про самоузгодженість останньої і можливість її використання для моделювання проходження сигналів через синапс.

Ключові слова: синапс, медіатор, рецептор, екзоцитоз, імпульс.