

A Mathematical Model of a Neuron with Synapses based on Physiology

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The neuron, when considered as a signal processing device, its inputs are the frequency of pulses received at the synapses, and its output is the frequency of action potentials generated- in essence, a neuron is a pulse frequency signal processing device. In comparison, electrical devices use either digital or analog signals for communication or processing, and the mathematics behind these subjects is well understood. However, in regards to pulse frequency processing devices, there has not yet been a clear and persuasive mathematical model to describe the functions of neurons. It goes without saying that such a model is very important, not only for understanding neuron and neural system behavior, but also for undeveloped potential applications in industry. This paper proposes a method for obtaining the mathematical relationship between the input and output signals of a neuron based on physiological facts. The proposed method focuses on the currents across the postsynaptic membrane of each synapse, and the key is to recognize that the net charge across the whole membrane of a neuron over each action potential cycle must equal to zero. By analyzing the relationship between the input of a synapse and the currents across the postsynaptic membranes, a dynamic pulse frequency model of the neuron can be obtained. Here, we show that the transfer function of a neuron depends on the function of the postsynaptic current of each synapse in resting state, which can be found by detecting the postsynaptic current when a pulse is received at the synapse. The transfer function of a typical neuron generally includes addition and subtraction of feedthrough terms and/or first order lag functions. To focus on the most basic characteristics of a neuron, accommodation, adaptation, learning, etc. are not discussed in this paper.

If a neuron is considered as a signal processing unit, the synapses and dozens of different types of neurotransmitters discovered thus far that can act on transmitter-gated ion channels in synapses must be examined. Thus far, many neuron models were proposed for explaining the active principle of a neuron¹⁻⁶, such as the famous Hodgkin-Huxley (H-H) model^{1,2}. However, until now, none of these models has considered the function of the synapses. Therefore, those models cannot explain the basic signal processing characteristics of the neuron in principle.

It would seem to be very difficult to find a function to explain the characteristics of each type of transmitter-gated ion channel. However, all of these channels can be considered as having only one primary function, which is to allow the flow of ions through the postsynaptic membrane. This function is easy to satisfy because the ions move passively down its chemical potential which arises from the differential concentrations of ions between the two sides of the membrane. Therefore, each of these ion channels can be modeled as a time-varying conductance. The functions of the time-varying conductance can be obtained from the functions of the currents through the postsynaptic membrane, and the total of the currents in a synapse is the postsynaptic current, which can be experimentally obtained using common methods in the neurophysiology field.

The following section introduces the principle of the action potential as basic background for this paper. Since only the transient signal processing characteristics of a neuron is discussed, the intracellular and extracellular ion concentrations can be assumed to be constant, and the effects of active transports such as ion pumps can be ignored^{1-6,9}.

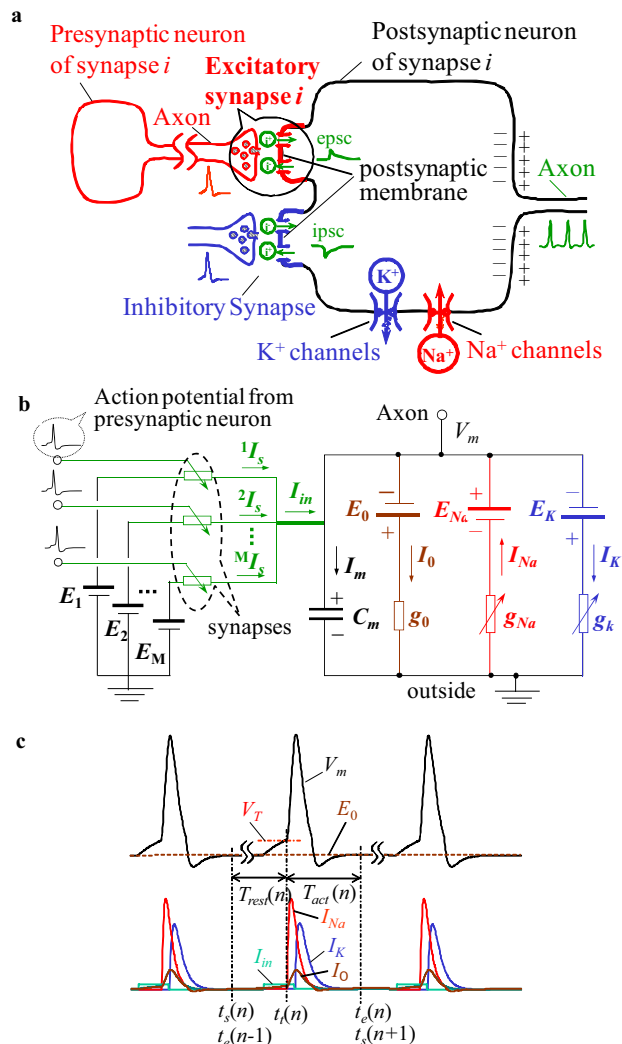


Figure 1 | The relationship between membrane potential and ion currents across the membrane. **a**, Illustration of the electrical mechanism of a neuron. **b**, Equivalent circuit of a neuron. C_m represents the membrane electrical capacitance, and the voltage V_m across the capacitance represents the membrane potential. The conductance g_{Na} , g_K , and g_0 represent the membrane permeability of Na^+ , K^+ , and the aggregate membrane permeability of other ions (mostly Cl^-). E_{Na} and E_K are the chemical potentials of Na^+ and K^+ . E_0 is the resting membrane potential or resting potential. It is defined as the membrane potential when there is no net current across the membrane. I_{in} is the total current from transmitter-gated ion channels of all synapses, and I_s ($m=1,2, \dots, M$) represents the current through the postsynaptic membrane of the m^{th} synapse. E_1, E_2, \dots, E_M represents the chemical potentials of each corresponding ions, ex. if E_m is the chemical potential of Na^+ , $E_m = E_{Na}$. **c**, The membrane potential caused by the ion currents across the membrane: I_{Na} is the current through the Na^+ channel, I_K is the current through the K^+ channel, I_0 is the leakage current, which includes all ions flowing through the membrane without channels. Note that none of the currents mentioned above are postsynaptic currents. $T_{rest}(n)$ is the resting state period, and $T_{act}(n)$ is the action state period of the n^{th} action.

Fig.1a illustrates the electrical mechanism of a neuron. When an action potential from the presynaptic neuron arrives at a synapse, ion currents will flow into or out of the postsynaptic membrane. If the synapse is an excitatory synapse, the ion current flow is positive (ex. the flow of Na^+ ions into the cell) and results in membrane depolarization. Conversely, if the synapse is inhibitory, the ion current flow is negative (ex. the flow of Cl^- ions into the cell) and results in membrane hyperpolarization. If the stimulations at the excitatory synapses are continuous or strong enough to result in membrane

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potentials greater than the action potential threshold V_T of the neuron, then most of the Na^+ channels on the membrane will open and Na^+ ions will flow into the neuron in the direction of the chemical gradient. Subsequently, the membrane potential increases sharply and an action potential is generated. Since the K^+ channels also open shortly afterwards, K^+ ions flow out of the cell in the direction of the chemical gradient. Thus, the membrane potential goes back down quickly, and most of the ion channels become closed. The H-H equivalent circuit, as depicted by Fig.1b, can explain this process.

Fig.1b is an H-H equivalent circuit of a neuron that is improved with the addition of synaptic circuits. To simplify the discussion, other types of ion channels, such as the Ca^{2+} channel, are not considered.

The membrane potential of a neuron depends on the ion currents across the membrane. Fig.1c illustrates the ion flows based on the equivalent circuit of a neuron shown in Fig.1b. To analyze the principle of an action potential, the input current I_{in} is set as square waves, and each square wave is sufficient to cause an action potential. For my analysis of the action potential frequency of a neuron, I divided the time for one action cycle into two periods. The start of the first period $T_{rest}(n)$ for the n^{th} action potential, is defined as the time $t_s(n)$, when the neuron first reaches the resting potential E_0 after the end of the refractory period of the $n-1^{\text{th}}$ action potential. The end of the first period $T_{rest}(n)$, is defined as the time $t_r(n)$, when the membrane potential reaches threshold for the n^{th} action potential. Because in period T_{rest} , the neuron is in the resting state, T_{rest} will be referred as the resting period.

The start of the second period $T_{act}(n)$, is defined as the time $t_a(n)$. The end of the second period, defined as $t_e(n)$, is when the membrane potential first reaches the resting potential E_0 after the end of the refractory period of the n^{th} action potential. Note that $t_e(n-1) = t_s(n)$, and the total time $T(n)$ of the action cycle is $T(n)=T_{rest}(n)+T_{act}(n)$. In period T_{act} , the neuron is in the action state (includes the refractory period), so T_{act} is referred as the action period.

Since the shape of the membrane potential during the action period is almost always the same, and T_{act} is almost always constant, the frequency of action potentials can be considered to be dependent solely on the characteristics of ions flowing through the membrane during resting period T_{rest} .

Because most neurons can be considered as a pulse frequency processing device, the intervals between pulses need to be broad enough, relative to the width of the pulse, to explain the wide range of signal values. So, ideally,

$$T_{rest}(n) \approx T(n) \gg T_{act}(n) \quad (n = 0, 1, 2, \dots) \quad (1)$$

From Fig.1c it is easy to realize that the charge flowing into a neuron in period T_{rest} is equal to the charge flowing out of the neuron in period T_{act} . In Fig.1b,

$$I_m(t) = I_{in}(t) - I_0(t) + I_{Na}(t) - I_K(t) \quad (2)$$

In the resting period $T_{rest}(n)$, because the neuron is in the resting state, the ion channels on the membrane, except those on the postsynaptic membrane, are closed, i.e. $I_{Na}(t)$ and $I_K(t)$ are close to 0, so the total ion current flow into the neuron are the sum of the total currents $I_{in}(t)$ from the synapses and the leakage currents $I_0(t)$. From Fig.1b, Fig.1c, and equation (2) the following equations are obtained.

$$q_{in}(n) = C_m(V_T - E_0) = \int_{t_s(n)}^{t_r(n)} I_m(t) dt = \int_{t_s(n)}^{t_r(n)} I_{in}(t) dt - \int_{t_s(n)}^{t_r(n)} I_0(t) dt \quad (3)$$

Where, $q_{in}(n)$ is the total charge of ions which flow into the neuron in the period $T_{rest}(n)$, and V_T is the threshold for action potential. Because C_m , V_T , E_0 are constant, $q_{in}(n)$ is also constant. Additionally, in the period $T_{rest}(n)$, the membrane potential is close to the resting potential E_0 , therefore the leakage currents I_0 can be considered to be small enough to be insignificant.

Here, we define the frequency of action potentials of a neuron in the period $T(n)$ as $f_{out}(n)=1/T(n)$, and thus equation (3) becomes,

$$C_m(V_T - E_0)f_{out}(n) = \frac{1}{T(n)} \int_{t_s(n)}^{t_r(n)} I_{in}(t) dt \quad (4)$$

The average current of $I_{in}(t)$ in the period $T_{rest}(n)$ is,

$$I_{in}(n) = \frac{1}{T_{rest}(n)} \int_{t_s(n)}^{t_r(n)} I_{in}(t) dt \quad (5)$$

From (4) and (5), the following equation can be obtained.

$$\begin{aligned} f_{out}(n) &= \frac{1}{C_m(V_T - E_0)} \frac{T_{rest}(n)}{T(n)} I_{in}(n) \\ &= \frac{1}{C_m(V_T - E_0)} \left(1 - \frac{T_{act}(n)}{T(n)}\right) I_{in}(n) \end{aligned}$$

T_{act} is almost constant. From equation (1), if T_{act} is small enough, i.e. $T_{act}(n)/T(n) \rightarrow 0$, the following equation is obtained.

$$f_{out}(n) = \frac{1}{C_m(V_T - E_0)} I_{in}(n) \quad (6)$$

When $\sum_{m=0}^{n-1} T(m) < t \leq \sum_{m=0}^n T(m)$, define (Like D-A transform)

$$f_{out}(t) = f_{out}(n), \quad I_{in}(t) \approx I_{in}(n)$$

Thus (6) can be explained as

$$f_{out}(t) = \lambda I_{in}(t) \quad (7)$$

Where $\lambda = C_m^{-1}(V_T - E_0)^{-1}$ is a constant.

From Fig.1b equation (7) can be explained as

$$f_{out}(t) = \lambda \sum_{m=1}^M I_s(t) \quad (8)$$

Where M is the number of synapses of the neuron, and $I_s(t)$ depends on the characteristics of the synapse and the frequency of the action potentials of the presynaptic neurons. Notice $I_s(t)$ is the current through the postsynaptic membrane of the synapse in the resting state of the neuron.

Discussion (1): In the above analysis, the leakage current I_0 was considered to be small enough to be neglected. In the standpoint of energy efficiency, the smaller g_0 is, the better. However, if g_0 is too small, the membrane potential cannot easily be kept at the resting membrane potential. Since the K^+ channel causes the membrane potential during after-hyperpolarization to be lower than the resting potential, if it cannot let the potential return to the resting potential quickly, the generation of high frequency action potentials will be difficult.

To answer this contradiction, I considered that there are 3 possibilities in a neuron. First case is that the efficiency of ion pumps is variable. This means that when the membrane potential is lower than the resting potential (in T_{act} period), the efficiency becomes higher, and causes the membrane potential to return to the resting potential more quickly. Second case is to let g_0 to be a variable value. For example, g_0 may have a small value in T_{rest} period, and become a large value in T_{act} period. Third case is that g_0 has different values depending on the direction of current flow, like a diode (i.e g_0 is small during depolarization and g_0 is large during hyperpolarization). Because the resting potential is close to the reverse chemical potential of chloride, considering the second and third cases, I speculate that there exists a type of chloride channel that opens during the refractory period, and/or the membrane is highly permeable to chloride ions flowing out, and relatively impermeable to chloride ions flowing in.

Of course, all of the three cases do not influence the above equations because the variations of the ion pumps or g_0 are all in action period T_{act} .

Discussion (2): In equations (1) and (6), the action pulse width T_{act} is considered small enough compared with the action cycle T . However, in cases of high frequency output, the $T_{act}/T \rightarrow 0$ simplification will not hold, and the relative refractory period will become shorter i.e. T_{act} is

not a constant. Therefore, in the high frequency domain, nonlinear problems will become apparent. To address this complex problem, one possible approach may be to introduce a sigmoid function, similar to a neural network using the Back-Propagation method¹². However, for the sake of simplicity, this paper only considers the linear period of the function of neurons, i.e. we assume cases where $T_{act}/T \rightarrow 0$.

Modeling of synapses: Fig.2 shows the mechanism of synapses. When an action potential traveling along the axon arrives at a synapse, some synaptic vesicles in the presynapse will move towards the presynaptic membrane and release an amount of neurotransmitter into the synaptic cleft. The neurotransmitters bind to transmitter-gated ion channels on the postsynaptic membrane and allow specific ions to flow through the channels. The flow of ions will cause membrane depolarization (excitatory synapse) or hyperpolarization (inhibitory synapse). All of these transmitter-gated ion channels can be considered as having one primary function, which is to allow the flow of ions through the postsynaptic membrane. This function is easy to satisfy because the ions move passively down its chemical potential.

In this paper, we divided the neurotransmitters into two types. If the amount of transmitter, emitted by a single pulse is rapidly removed from the synaptic cleft before the next pulse arrives, we call this type of transmitter the type I neurotransmitter. Thus, this type of neurotransmitter has the characteristic that previous action potentials from the presynaptic neuron do not affect the postsynaptic membrane current of the subsequent action potentials. Most Type I neurotransmitters are released from small vesicles (Fig.2)^{7,9}.

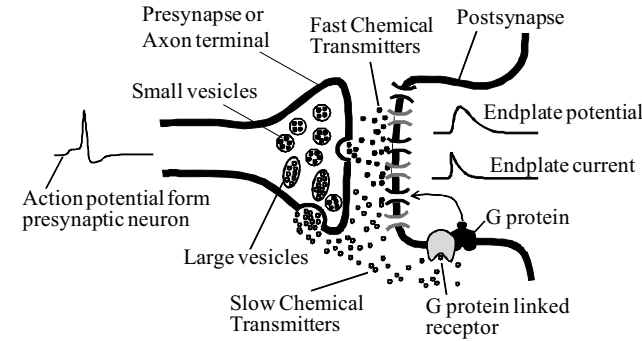


Figure 2 | A typical mechanism of a synapse⁸

If some of the transmitters from a previous pulse remain in the synaptic cleft after the subsequent pulse, then we call this type of transmitter the type II transmitter. Thus, previous pulses can affect the present postsynaptic current. Most Type II neurotransmitters are called slow chemical transmitters, and considered to be released from large vesicles^{8,7}.

In the case of type I neurotransmitters: If we define q_s as the amount of charge that flows through the postsynaptic membrane in the resting period $^{post}T_{rest}$ (T_{rest} of the postsynaptic neuron) of postsynaptic neuron caused by a type I transmitter emitted by 1 pulse of presynaptic neuron, then the current through the postsynaptic membrane is

$$I_s(t) = q_s f_{in}(t) \quad (9)$$

Where $f_{in}(t) = 1/^{pre}T(t)$ is the input signal, which are the frequency of the action potentials of the presynaptic neuron at the synapse.

Since adaptation characteristics are not discussed, it can be assumed that the amount of transmitter emitted into the synaptic cleft caused by a single pulse is constant. In addition, we assume that the same amount of transmitter in a synapse causes the same amount of charge to flow through the postsynaptic membrane. Therefore, the value of q_s is almost constant.

In the case of type II neurotransmitters: Assuming that the amount of type II transmitter emitted from the synapse caused by each pulse

from the presynaptic neuron is constant, and the same amount of transmitter causes the same ion current function. Then the total current through the postsynaptic membrane $I_s(t)$ at the time of m^{th} pulse received the synapse can be explained as (Fig.3), i.e.

$$I_s \left(\sum_{i=0}^m {}^{pre}T(i) \right) = I_s(0) + \sum_{j=0}^m I_p \left(\sum_{i=0}^{m-j} {}^{pre}T(m-i) \right) \quad (11)$$

Here, $I_p(t)$ is defined as the time-varying postsynaptic current function for a single pulse received at the synapse¹⁰. Furthermore, time $t=0$ is defined as when one pulse is received at the synapse.

Defining $f_{in}(j) = 1/^{pre}T(j)$, (11) becomes,

$$I_s \left(\sum_{i=0}^m {}^{pre}T(i) \right) = I_s(0) + \sum_{j=0}^m I_p \left(\sum_{i=0}^{m-j} {}^{pre}T(m-i) \right) f_{in}(j) {}^{pre}T(j) \quad (12)$$

When

$$\sum_{k=0}^{m-1} {}^{pre}T(k) < t \leq \sum_{k=0}^m {}^{pre}T(k) \quad (13)$$

if $^{pre}T(k)$ is short enough, equation (12) becomes

$$I_s(t) = I_s(0) + \int I_p(t - \tau) f_{in}(\tau) d\tau \quad (14)$$

Equation (14) is a convolution integral function. $I_p(t)$, which is named the "one pulse postsynaptic current," can be approximately quantified by experiments. Fig.4 shows an example of such an experiment⁷. When providing a stimulus at a synapse, the trace of the current on the probe A is the function of a one pulse postsynaptic current $I_p(t)$.

Actually, the type I neuron can also be explained by (14). In this case, $I_p(t) = k_1 \delta(t)$ (15) since subsequent pulses do not add together, we can substitute $I_p(t)$ with an unit impulse function $\delta(t)$, and k_1 is a constant which depends on the kind of the transmitter and the synapse.

From (8) and (14), the following equation which explain the relationship of input and output is obtained.

$$f_{out}(t) = f_{out}(0) + \lambda \sum_{m=1}^M \int_0^M I_p(t - \tau) {}^m f_{in}(\tau) d\tau \quad (16)$$

So, Laplace transform of (16) is,

$$F_{out}(s) = \lambda \sum_{m=1}^M {}^m I_p(s) {}^m F_{in}(s) \quad (17)$$

The transfer function of the neuron from the m^{th} synapse to the axon ${}^m G(s)$ is

$${}^m G(s) = \lambda {}^m I_p(s) \quad (18)$$

Where ${}^m I_p(s)$ can be obtained using the method showed in Fig.4. $\lambda = C_m^{-1}(V_T - E_0)^{-1}$ is a constant, where C_m is the capacitance of the membrane, V_T is the threshold for action potential, and E_0 is the resting potential. Because most neurons have the same V_T and E_0 in the short term, and the value of C_m is constant, therefore λ is also a constant value. Furthermore, since C_m is related to the area of the membrane, the smaller the neuron cell (smaller membrane area), the bigger the gain for the transfer function of the neuron. From (17), a neuron model can be explained as Fig.5 (a). The mark \pm in Fig.5 means that excitatory synapses are denoted with the symbol $+$, whereas inhibitory synapses are denoted with the symbol $-$.

Discussion(3): Equation (17) shows that the transfer function of a neuron from a synapse to the axon depends on the characteristics of the postsynaptic current of the neuron. That means that the signal processing functions of a neuron depends on the type of transmitters emitted by the presynapse. By adjusting the types of neurotransmitters emitted, different signal processing functions can be composed. This characteristic highlights a very important principle; that is when structuring an equivalent circuit of a neuron, by adjusting the synapse circuit to vary the shape of the one pulse synaptic current, almost any transfer function can be realized.

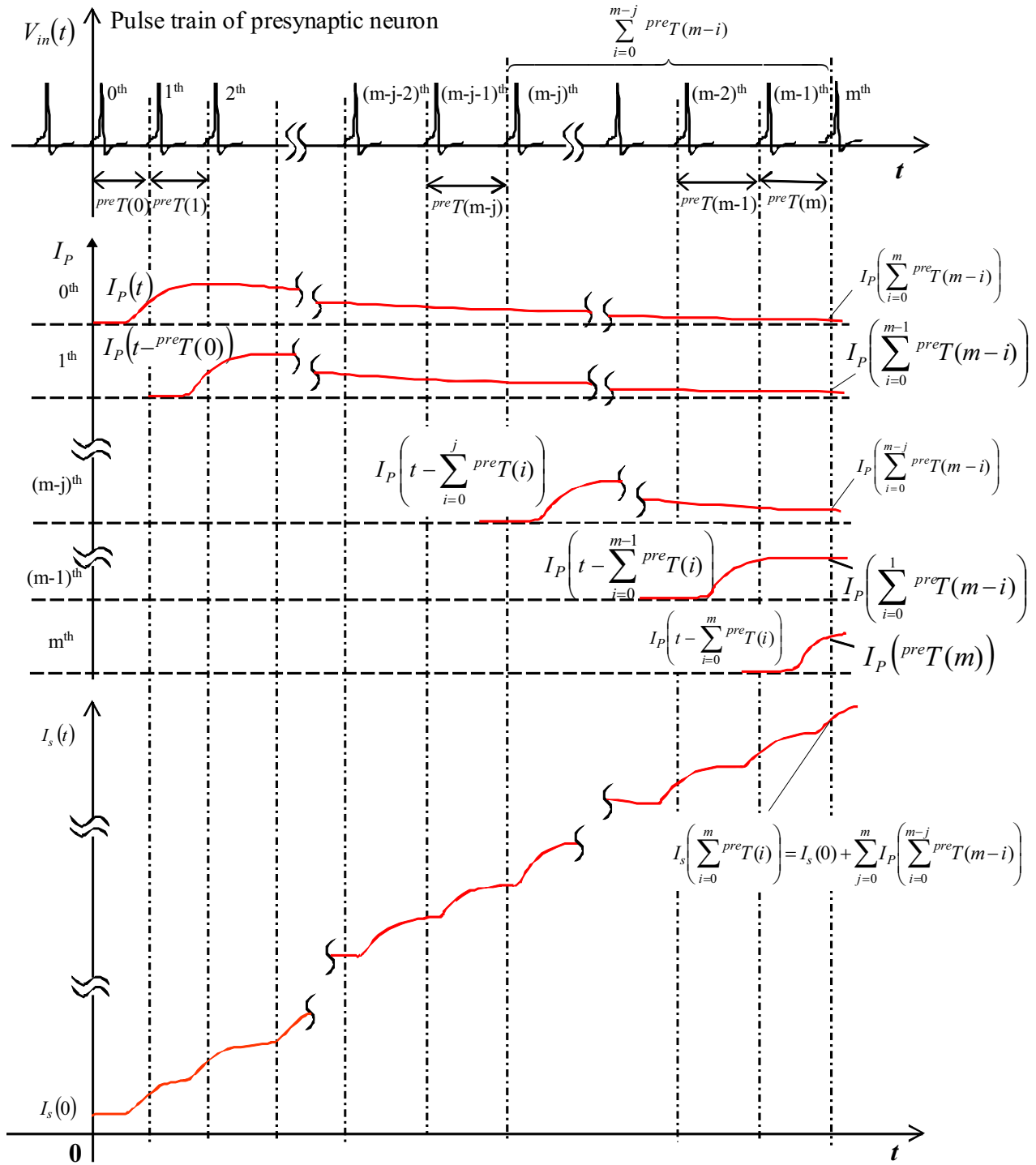


Figure 3 | The relationship between presynaptic action potentials and the amount of type II transmitter in a synapse¹⁰.

Where, $I_P(t)$ is the postsynaptic current function caused by the 0th pulse,

$I_P\left(t - \sum_{i=0}^j preT(i)\right)$ is the postsynaptic current function caused by the number j pulse,

$I_P\left(\sum_{i=0}^{m-j} preT(m-i)\right)$ is the postsynaptic current at time $\sum_{i=0}^j preT(i)$ caused by the number $m-j$ pulse,

$I_s\left(\sum_{i=0}^m preT(i)\right)$ is the total postsynaptic current at time $\sum_{i=0}^m preT(i)$, i.e. $I_s\left(\sum_{i=0}^m preT(i)\right) = I_s(0) + \sum_{j=0}^m I_P\left(\sum_{i=0}^{m-j} preT(m-i)\right)$

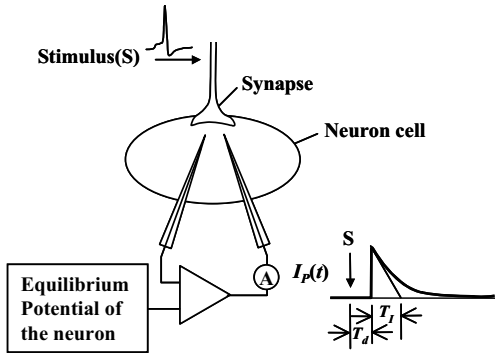


Figure 4 | A method for obtaining the function of a current $I_p(t)$ through the postsynaptic membrane caused by a single pulse⁷, assuming that only one kind of type II neurotransmitter is in the synapse.

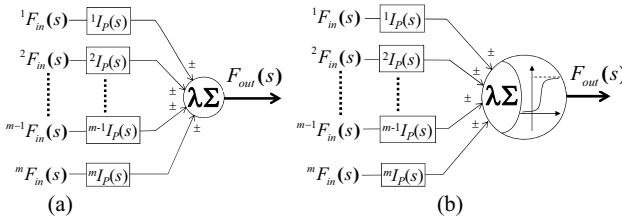


Figure 5 | Neural models

Discussion(4): Equation (14) assumed that the current function through the postsynaptic membrane caused by each pulse received at the synapse is the same. However, in the case when almost all Na^+ channels are open, the postsynaptic currents will become saturated. The saturation problems I feel can be explained by a sigmoid function at each synapse. Because each synapse is connected with the presynaptic neuron's axon, as per discussion (2), we only need one sigmoid function at the output of the model as shown in Fig.5 (b).

Discussion(5): Since the Laplace transform of the one pulse postsynaptic current function is the transfer function of the neuron, if one pulse can trigger enough number of output pulses, the single input pulse can be considered as an impulse signal, and the frequency of output pulses is the impulse response.

Discussion(6): In some cases, the signals of the output and/or input of a neuron can be considered as pulse frequency modulation (PFM) signals. Examples include the neuron fibers from vestibular organs, which has a pulse train at a specific base frequency, and when the head moves, the frequency shifts higher or lower to encode the velocity and direction of head movements. To analyze this problem, we can consider 3 cases. First case is that the input pulse trains of one or more synapses of a neuron have base frequencies, but the output of the neuron has no base frequency (i.e. the base frequency is 0). The second case is that not only do the input pulse trains of the neuron have base frequencies, but also the output pulse train has a base frequency. The third case is that the input pulse trains of the neuron have no base frequency, but the output pulse train has a base frequency.

In fact, in each of the three cases, equations (16) and (17) hold, and the input signals $f_{in}(t)$ or output signal $f_{out}(t)$ are values of the real frequency minus the base frequency.

As for the 1st and 2nd cases, the base frequency pulse train input on the synapses causes a base current through the postsynaptic membrane, and the base current causes the membrane potential to be higher than the resting membrane potential. If the base current is not strong enough to cause an action potential, then this is met by case 1. In contrast, if the base current is big enough to cause action potentials, we have case 2, where the frequency of the action potentials is the base frequency of

output pulse train.

As for the 3rd case, the base output frequency can be caused by some types of channels on the membrane which allow a constant current flow into the neuron. These currents can be included into $I_{in}(t)$ (Fig.1b), so that we can define

$$I_{in}(t) = {}^0I_{in} + \sum_{m=1}^M {}^mI_s(t)$$

where ${}^0I_{in}$ is a constant current that causes a base frequency output pulse train. From (7), the base frequency is $\lambda {}^0I_{in}(t)$.

A model of a typical neuron:

The curve $I_p(t)$ shown in Fig.4 is one found commonly in neurophysiology textbooks⁷. $I_p(t)$ can be explained approximately as,

$$I_p(t) = \begin{cases} \rho e^{-\frac{t-T_d}{T_r}} & t \geq T_d \\ 0 & t < T_d \end{cases} \quad (19)$$

Where, the parameter ρ , T_r , and T_d depend on the characteristics of the transmitter and the channels. The transfer function (18) of the neuron through the synapse can be obtained from (19) as follows.

$$G(s) = \lambda \frac{\rho}{T_r s + 1} e^{-s T_d} \quad (20)$$

The upper equation shows that the synapse which has type II neurotransmitter (19) is a first order lag function with a dead time⁷.

If there are any non-linear elements in the neuron caused by type II neurotransmitters, then a different mathematical model of type II neurotransmitters will be required. This can be done as per the experiment in Fig.4.

Using (15), (17) and (20) the whole neuron model can be structured as follows,

$$F_{out}(s) = \sum_{n=1}^N \left({}^n k_p + \frac{{}^n k_I}{s + \frac{1}{T_I}} e^{-s {}^n T_d} \right) {}^n F_{in}(s) \quad (21)$$

Where, N is the number of synapses, and K_p , K_I , T_I are parameters which depend on the transmitters and the transmitter-gated ion channels of each synapse. If learning or adaptation functions of the neuron are ignored, K_p , K_I , T_I are constant. When the synapse is an excitatory synapse K_p , K_I are positive, and when the synapse is an inhibitory synapse K_p , K_I are negative.

From (21) the relationship between inputs and output of the neuron can be explained as in Fig.6. For normalization, and taking into account the action potential frequency limitation of a neuron, the summation unit in Fig.6 can be modeled using a sigmoid function as in Fig.5(b). Compared with most common neuron unit models¹¹, it is clear that the proposed neuron unit model is a dynamic system. When T_d is small enough, and T_I is big enough, (21) can be viewed as a summation of integrator units and feedthrough terms.

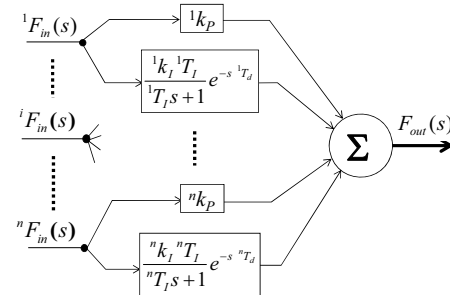


Figure 6 | A dynamic model of a typify neuron

Conclusion

This paper analyzed the physiological mechanisms of a neuron using mathematical techniques and the salient points are as follows:

(1) A neuron is a pulse frequency signal processing device. The transfer function, from a synapse of the neuron to the axon, can be obtained by detecting the characteristics of the postsynaptic current. In other words, the transfer function is the Laplace transform of the time function of the post-synaptic current caused by one input pulse.

(2) If we construct an equivalent circuit of a neuron based on Fig.1b, by adjusting the synapse circuit to vary the shape of the one pulse synaptic current, almost any transfer function can be realized.

(3) A typical neuron model is a dynamic calculation system that has feedthrough terms and/or first order lag functions (integration function with a loss).

(4) The characteristics of the postsynaptic currents and membrane potential in the action period do not contribute to the signal processing results. Remarkably, this means that the shape of an action potential conveys almost no information.

(5) A single pulse can be considered as an impulse signal, when the pulse can cause a strong enough postsynaptic current to trigger action potentials on the postsynaptic neuron.

Future works:

For our next paper, we will demonstrate some neuron equivalent circuits that include synapses. Since the transfer functions of a neuron depends on the characteristics of one pulse postsynaptic currents of the neuron, arbitrary transfer functions of the equivalent circuit can be easily constructed by correctly designing the circuit of the synapse. We will show that these equivalent circuits will have many practical industrial applications.

To focus on the most basic characteristics of a neuron, this paper only discusses the short-term functions. However, our future research will focus on discussing the long-term functions. The long-term functions includes the characteristics of the potential dependent Ca^{2+} channel and Ca^{2+} -activated K^+ channel⁹, the plasticity of the synapses and the growth of a neuron.

In this paper, the transfer functions of the synapses are independent of each other. However, this condition is under the assumption that the transmitters released from a synapse cannot functionally affect neighbouring synapses. In the future, we will investigate if any neuron transmitters do not conform to the above assumption, and if so, how to explain the characteristics of the neuron transmitter using a mathematical model.

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