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Capacity Analysis of Neuro-Spike Communication System for Nanonetworks

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Abstract

Molecular communication is a new paradigm to solve the problems of conventional communication system, such as capacity or energy consumption. The neural network is one of the molecular communication mechanism valid for higher animals and considered as a highly advanced information transfer network in terms of capacity, reliability and energy consumption. In this paper, we provide the capacity of neuro-spike communication system, which is inspired from neural network. The neuro-spike communication consists of axon propagation, vesicle release and neurotransmitter diffusion. Through modeling and analysis of the above three parts, the capacity of the neuro-spike communication can be obtained. Numerical results show the trends of the capacity for main parameters, such as bandwidth, absolute refractory period and distance. The capacity is converged to certain value even though bandwidth of input signal becomes larger and larger. As a result, it can be seen that refractory period is a key parameter in neuro-spike communication system.

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Keywords: Molecular communication; neuro-spike communication; body area sensor network; neural network; nanonetworks

1. Introduction

Nanonetworks are the new paradigm of future networks, that are based on the interaction and communication for nano-scale sensors each other[1]. Based on prior studies, there are two kinds of nanonetworks. The first is a method that uses the electromagnetic wave, which is similar to current communication system[2]. The second is a molecular communication using the molecule as a carrier of information transmission, such as calcium signaling, molecular motors, pheromones and neurotransmitters[3]. However, since the power consumption and bio capability of nanonetworks are important features[1], molecular communication is more preferred in nanonetworks in view of the power consumption and the bio capability.

In the molecular communication, nano-sensors send the information by using the molecules, and there are many researches of bio inspired approach for molecular communication[4]. The *neuro-spike communication* is one of the molecular communication mechanisms, which is inspired from nerve signal transmission of nerve cells, neurons. The neurons can be found in many higher animals including humans. The neural networks can be considered as the systems which have evolved optimally, in terms of information transmission and energy consumption in the body[5]. The neuro-spike communication mimics the signaling process of the neural system.

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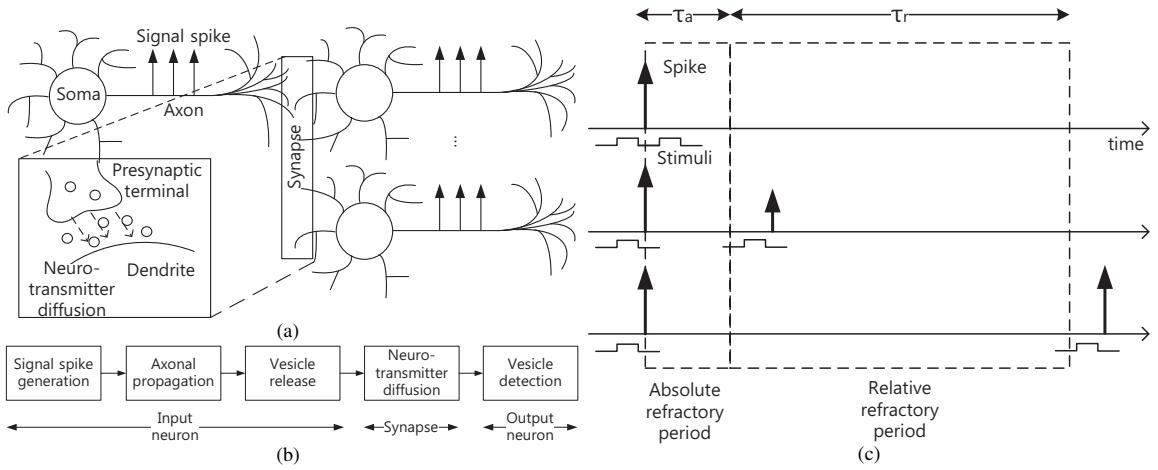

Fig. 1. Neuro-spike communication system and its characteristics (a) Structure of neuro-spike communication system; (b) Block diagram of neuro-spike communication system; (c) Diagram for absolute and relative refractory period.

The neuro-spike communication can be applied for communication that occurs in the body. A number of nano-scale sensors will be embedded in the human body, and these sensors will communicate with each other or with sensors outside. Then, it will be possible to transmit and receive a large amount of collected biometric information by using the neuro-spike communication system. Because the neuro-spike communication with high bio-capability is based on the bio-inspired approach, it does not affect the human body due to its high bio-capability.

There are numerous researches related to the neurons and neuro-spike communication. Axonal propagation functions how signals are transmitted through the axon of neurons were investigated in [6,7,8]. Vesicle release function at the presynaptic terminal was analyzed in [9]. In [10,11], they developed the simulators for nanomachine and neuron networks. However, the proposed models weren't analyzed and didn't provide the system capacity. The synaptic interference channels for single-input single-output (SISO) and multiple-input single-output (MISO) were analyzed in [12], but the axonal propagation wasn't considered. Moreover, the channel modeling of neuro-spike communication was performed in [13]. However, diffusion model of synaptic wasn't analyzed, and also there is no analysis on the capacity of neuro-spike communication system.

In this paper, we derive the channel capacity of neuro-spike communication including the process of axon propagation, vesicle release and neurotransmitter diffusion to confirm its outstanding property. The refractory period is the main parameter in case of analyzing the neuro-spike communication, which affects to generate electric signals. Using the generated electric signals, the presynaptic terminal releases the vesicles with probability related to the magnitude of the signal. The emitted neurotransmitters are diffused through the synapse. Through appropriate modeling and analysis of three processes, it is possible to obtain a channel capacity for neuro-spike communication system.

The organizations of the remaining paper are as follows. In Section 2, we describe neuro-spike communication. In Section 3, we perform the modeling of the neuro-spike communication and derive the capacity. In Section 4, we show the numerical results of the capacity which are obtained in Section 3. In Chapter 5, we make the conclusion of this paper.

2. Neuro-spike communications

A neuron is composed of soma and axon. Soma is the nucleus and the cell surrounding the nucleus of nerve cell, and axon is a long projection which extends from soma. Axon hillock is the part that connects the soma and axon, which generates the electric signals that are sent to the axon. Axon plays a role to transmit generated signals from the axon hillock to presynaptic terminal by positive feedback polarization. The electric signals at the presynaptic terminal, are changed into the chemical signals at the synapse. The excited presynaptic terminal exports molecular vesicle, consisting of many neurotransmitters, which are spread through the synapse by diffusion process. Then, the neurotransmitters reach the dendrite, and excited dendrite generates the electric signals again.

To mimic the signal transmission model for neuronal cells, neuro-spike communication transmits the information via the following process. First, the soma, the core of input neurons, generates signal stimuli. Then, signal spikes are

generated according to the stimuli. The signal spikes move and arrive at the presynaptic terminal through propagation along the axon. The presynaptic terminal determines whether to release the neurotransmitter in the synapse depending on the magnitude of the signal spikes. The released neurotransmitters are diffused through the synapse and arrive at dendrite of the output neuron. The exited dendrite generates the electric signals and arrives at soma of the output neuron. The whole processes of neuro-spike communication can be seen as shown in Fig. 1(a). More detailed analysis about axonal propagation, vesicle release and neurotransmitter diffusion is described in the three sub-sections below.

2.1. Axonal propagation

The axonal propagation of the neuro-spike communication can be categorized into different molecular communication system compared to others. The stimuli with high firing rate generate electric signals corresponding to the portion of the stimuli. On the other hand, the stimuli with low firing rate generate electric signals corresponding to the interval of inter-stimulus, of which magnitude decreases inverse exponentially. That is, interval of inter-stimulus has a significant effect on magnitude of electric signal. Because of exponential property, proper amount of electric signal is generated by stimuli with long interval and small or no electric signal is generated by stimuli with small interval. At this time, we define the interval where the signal is not generated as *absolute refractory period*, and the interval where the magnitude of the signal decreases inverse exponentially as *relative refractory period*. Therefore, the maximum frequency of electric signals is determined by the absolute refractory period of a neuron. Fig. 1(c) shows a diagram for refractory periods.

2.2. Vesicle release

Vesicle release at the presynaptic terminal is a random process. The probability of vesicle release is adjusted by the magnitude of the electric signal arriving at the presynaptic terminal. Despite that electric signal came into the presynaptic terminal, it is possible that the presynaptic terminal may not release the vesicles. In other words, perfect electric signals make the vesicle release with probability 1. However, imperfect electric signals make the vesicle release or not. The magnitudes of the electric signals arriving at the presynaptic terminal are affected by relative refractory period or interval of inter-stimulus. In other words, interval of inter-stimulus determines the vesicle release probability of presynaptic terminal. On the other hand, even though there is no incoming electric signal at the presynaptic terminal, there is very small probability of vesicle release. However, we will not consider this phenomenon in this paper.

2.3. Neurotransmitter diffusion

The exited presynaptic terminals release the vesicles. Once presynaptic terminals release the vesicles, many neurotransmitters diffuse through synapses to reach the dendrites of output neurons. At this time, neurotransmitters move by *Brownian random motion* in the synapse. The dendrites of output neuron are excited by arrived neurotransmitters and generate the electric signals to the somas of output neurons.

3. Information capacity of a neuro-spike communication system

In this paper, we consider the neuro-spike communication with single-input and multiple-output neurons, similar to SIMO case, as shown in Fig. 1(a). In order to obtain the capacity of neuro-spike communication, appropriate modeling and analysis of three processes, axon propagation, vesicle release and neurotransmitter diffusion, are needed. The following contents are modeling and analysis for three processes of neuro-spike communication.

3.1. Axonal propagation modeling

In axon propagation, there are two kinds of refractory periods, which are absolute refractory period and relative refractory period[14]. The analysis of refractory periods is as follows.

3.1.1. Absolute refractory periods

We assume that τ_a is the absolute refractory period. As described in section 2, stimuli with the high firing rate, which is faster than absolute refractory period, just generate spikes whose period is τ_a. In other words, stimuli with the high firing rate are sampled with τ_a period and produce spikes corresponding to the sampled stimuli. At this time, if we set the τ to inter-stimulus interval, which is smaller than τ_a, the inter-spike interval becomes τ_a, not τ. Then, the presynaptic terminal with sampled spikes, releases the vesicles onto synapse for each spike respectively. Therefore, vesicle release probability $r_k(\tau)$ has a linear relationship with the interval of the input stimuli, that is $r_k(\tau) = \tau/\tau_a$ for

small value of τ. For example, if τ is 1 ms and τ_a is 10 ms, then vesicles are released every 10 ms. In that case, we can say that vesicle release probability is $1/10$ equivalently.

3.1.2. Relative refractory periods

In the relative refractory period, magnitudes of generated electric signals are decreased inverse exponentially for interval of input stimuli[6]. Moreover, the decreasing speed of the electric signal is proportional to relative refractory period, τ_r and it may be also proportional to absolute refractory period, τ_a.

3.2. Vesicle release modeling

If the electric signals aren't generated as results of the absolute refractory period, the presynaptic terminals don't release any vesicle. On the other hand, if the small electric signals are generated as results of relative refractory periods, the presynaptic terminals release the vesicles with probability $r_k(\tau)$, which is proportional to the magnitude of generated electric signal[9]. Therefore, vesicle release probability can be modeled as a function of interval of input stimuli. The vesicle release probability, $r_k(\tau)$ may be expressed as follows, because the inter-stimulus interval and vesicle release probability have a linear relationship in view of absolute refractory period, and an inverse exponential relationship in view of the relative refractory period.

$$r_k(\tau) = \left(1 - e^{-\frac{\tau}{\tau_a}}\right) \mathrm{U}(\tau) \tag{1}$$

where $\mathrm{U}(\cdot)$ is the unit step function. Then, two boundary conditions are $r_k(0) = 0$ and $r_k(\infty) = 1$. Using (1), if τ goes to +0, $r_k(\tau)$ can be approximated as follows.

$$\lim_{\tau \to +0} r_k(\tau) = r_k'(0)\tau = \frac{\tau}{\tau_a} \tag{2}$$

Here, (2) is well matched with the analysis of vesicle release probability in absolute refractory period. If we assume that neuro-spike system is a bandlimited system with bandwidth W, time interval τ is equal to $1/2W$. Therefore, (1) can be modified as follows.

$$r_k(W) = \left(1 - e^{-\frac{1}{2\tau_a W}}\right) \mathrm{U}(W) \tag{3}$$

where W is a bandwidth of input stimuli, and $\mathrm{U}(1/2W)=\mathrm{U}(W)$. Now the bandwidth of vesicle release, W_v, is limited as follows in terms of the vesicle release probability and bandwidth of input stimuli.

$$W_v = r_k(W)W \tag{4}$$

3.3. Neurotransmitter diffusion modeling

For neurotransmitter diffusion modeling, we assumed that single-input neuron is connected to multiple-output neurons and each diffusion channel follows independent identical distribution. The distribution of neurotransmitters that are exported by excited presynaptic terminal, is determined according to Fick's laws of diffusion in the synapses as follows.

$$\frac{\partial \rho_i(\overrightarrow{p}, t)}{\partial t} = D\nabla^2 \rho_i(\overrightarrow{p}, t) + n_T(t)\delta(|\overrightarrow{p} - \overrightarrow{T}|),\ t > 0,\ i \in \mathcal{I} = \{1, 2, \cdots, N_r\} \tag{5}$$

where $\rho_i(\overrightarrow{p}, t)$ is the distribution of neurotransmitters at the location $\overrightarrow{p}$, the time t and ith synapse, ∇^2 is the Laplacian operator, and $n_T(t)$ is the number of neurotransmitters emitted to each synapse at the location $\overrightarrow{T}$ at the time t. $\delta(\cdot)$ is the Dirac delta function, and D is the particle diffusion coefficient. In addition, neurotransmitters move based on Brownian random motion for given distribution, which is determined by Fick's laws of diffusion. By the property of Weiner process, it might be assumed that the location of a neurotransmitter is independent of that of others. Then the number of received neurotransmitters in ith receiver volume per each measurement, $n^1_{R_i}$ is represented as follow.

$$n^1_{R_i} \sim Poisson\left(\overline{\rho}_i V_R\right) \tag{6}$$

where $\overline{\rho}_i$ is the average of particle distribution at ith receiver, $\overline{\rho}_i = P_H/DdW_v k_B T$ [15] and V_R is the receiver volume. When we assume the receivers have a sphere shape, then the number of received particles in ith receiver volume per time sample, n_{R_i} might be derived as follows.

$$\begin{aligned} n_{R_i} &= \sum_{i=1}^{1/2W_v\tau_p} y_i^1 = B\left(\frac{1}{2W_v\tau_p}, \frac{4}{3}\overline{\rho}_i \pi R_{V_R}^3\right) \\ &= B\left(\frac{D}{2W_v R_{V_R}^2}, \frac{4}{9}\frac{P_H R_{V_R}^3}{W_v D d k_B T}\right) \end{aligned} \tag{7}$$

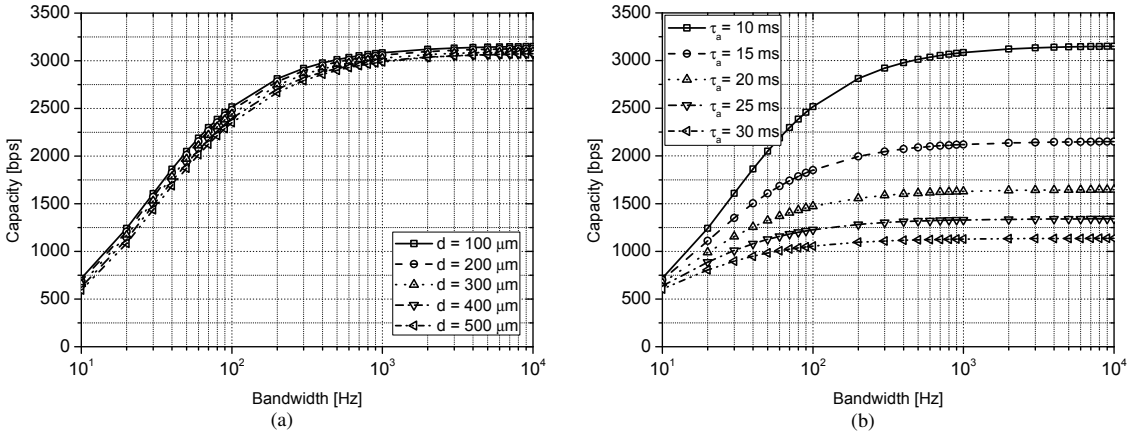

Fig. 2. Capacity of neuro-spike communication system (a) Capacity vs. bandwidth of input signal with varying synapse distance; (b) Capacity vs. bandwidth of input signal with varying absolute refractory period.

where $1/2W_v\tau_p$ is the number of measures how many particles are inside the receiver volume, P_H is the average transmit thermodynamic power for each presynaptic terminal, k_B is the Boltzmann constant, T is the absolute temperature, D is the diffusion coefficient, d is the distance between the presynaptic terminal of input neuron and a dendrite of output neuron, and R_{V_R} is a radius of volume V_R for spherical receiver, i.e.,dendrite, the time interval of quasi-constant particle distribution is represented as $\tau_p = R_{V_R}^2/D$[15] and $B(\cdot)$ is binomial distribution.

In order to obtain the the capacity of the neuro-spike communication system, it is necessary to consider the the axonal propagation and the vesicle release process. At present, (7) is a function of bandwidth of vesicle, W_v, so it must be changed to the function of bandwidth of the input signal, W. Using (4), the capacity of neuro-spike communication system with axonal propagation, vesicle release and neurotransmitter diffusion is represented as (8) with similar derivation in[15].

$$C = \sum_{i=1}^{N_r} \left\{ \begin{array}{l} 2r_k W \left(1 + \log_2 \dfrac{P_H}{3r_k W k_B T}\right) - 2\log_2(\pi D d) - \dfrac{4d}{3\ln 2}\sqrt{\dfrac{\pi r_k W}{D}} \\ -2r_k W n_{R_i} - 2r_k W \ln\left(r_k W \tau_p\right) - 2r_k W \ln\Gamma\left(n_{R_i}\right) - 2r_k W\left(1 - n_{R_i}\right)\psi\left(n_{R_i}\right) \end{array} \right\} \tag{8}$$

where N_R is the number of receivers, $\Gamma(\cdot)$ is the Gamma function, which is defined as $\Gamma(x) = (x-1)!$, and $\psi(\cdot)$ is the Digamma function, which is given by $\psi(\cdot) = \frac{d}{dx}\ln\Gamma(x) = \frac{\Gamma'(x)}{\Gamma(x)}$. The first line of (8) is related to Fick's laws of diffusion, and the second line is related to the Brownian random motion.

4. Numerical results

Table 1. Parameters values.

Parameters	Value
Average transmit power per a presynaptic terminal, P_H	1 pW
Number of receivers, N_R	4
Distance of synapse, d	100 μ m
Absolute refractory period, τ_a	10 ms
Radius of dendrite volume, R_{V_R}	10 nm
Absolute temperature, T	298 K
Diffusion coefficient, D	$10^{-9} m^2/s$
Boltzmann constant, k_B	$1.380650424 \times 10^{-23} J/K$

In this section, we show the numerical results of the vesicle release probability and capacity. Table 1 provides the common parameter values in the numerical results.

Fig. 2(a) shows the relationship between the bandwidth of input signals or stimuli and capacity for various synapse distance values. The bandwidth of input signals is varied from 10 Hz to 10000 Hz. For all distance values, the capacity tends to converge even though bandwidth becomes larger. We can find the interesting point, a low sensitivity of the capacity for distance. In other word, the capacity is not decreased dramatically even if distance becomes larger. For a distance of 100 μm, capacity converges to about 3150 bps, and for a distance of 500 μm, capacity converges to about 3070 bps.

Fig. 2(b) shows the relationship between the bandwidth of input signals or stimuli and capacity for various absolute refractory periods. The bandwidth of input signals also varies from 10 Hz to 10000 Hz. This result shows that the convergence value of the capacity decreases as τ_a becomes larger. It means that the capacity has a high sensitivity to absolute refractory period. For a τ_a of 10 ms, capacity converges to about 3150 bps, and for a τ_a of 30 ms, capacity converges to about 1135 bps.

5. Conclusions

Molecular communication is a new paradigm for future communication systems. In many molecular communication mechanism, the neural network is considered as a highly advanced information transfer network in views of capacity, reliability and energy consumption. In this paper, we derived the system capacity of neuro-spike communication as a function of bandwidth, absolute refractory period and distance

Numerical results show the relation among capacity, bandwidth, absolute refractory period and distance. As a result, it has been shown that absolute refractory period is one of the most important parameters for neuro-spike communication system. The capacity of neuro-spike communication system has a high sensitivity to absolute refractory period. On the other hand, a low sensitivity of capacity for distance is an interesting point. More realistic analysis of upper bound for neuro-spike communication system will be carried out using the encoding and decoding process in the future.

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