Capacity Bounds of Neuro-Spike Communication by Exploiting Temporal Modulations

Keyvan Aghababaiyan†, Vahid Shah-Mansouri‡ and Behrouz Maham∗

†Department of Electrical and Electronic Engineering, College of Engineering, University of Tehran, Iran
‡Department of Electrical and Electronic Engineering, School of Engineering, Nazarbayev University, Astana, Kazakhstan
Emails: aghababaiyan@ut.ac.ir, vmansouri@ut.ac.ir, behrouz.maham@nu.edu.kz

Abstract—We consider a neuro-spike communication system between two nano-machines, with information conveyed in the time intervals of the input spike train. The main contribution of our paper is modeling of the neuro-spike communication channel by an additive Gamma noise channel model. In this channel, the information is corrupted by Gamma distributed noise. We show that the proposed channel model is efficient for the neuro-spike communication when it exploits temporal modulations to transfer information. Then, we consider the Gamma distributed noise and we derive the upper and lower bounds on the channel capacity. Unlike Additive White Gaussian Noise (AWGN) channels, there is no single quality measure like signal-to-noise ratio for this channel model. Thus, we analyze the channel capacity bounds versus different values of time intervals and the decision threshold of the receiver.

Index Terms— Neuro-spike communication, Temporal modulation, Gamma distribution, Capacity bounds.

I. INTRODUCTION

Recent developments in nano technology and communication engineering are expected to lead to a new generation of nano-scale devices implantable inside the human body [1]. When these devices are connected in a network, they form intra-body nano-networks [2]. These nano scale devices or nano-machines are capable carrying out complex tasks by overcoming their individual limitations through interconnecting in a nano-network. Recently, intra-body nano-networks have been proposed for monitoring the nervous system [3] where the dimensional similarity of the nano-machines and the nervous biological cells is exploited. The main goal of such systems is to develop new medical diagnosis and treatment techniques. The nervous system is a natural communication system in the body that conveys biological information. The communication among neurons is called neuro-spike communication. The analysis of this communication paradigm is benefited for the design of artificial neural systems where nano-machines are linked to neurons. In these networks, nano-machines are used to replace damaged segments of the nervous system and have to behave exactly like the biological entities. Neuro-spike communication includes the electrical transmission of action potentials in the axonal pathway. Since the spikes in the nervous system are all very similar, the information has to be encoded in the configurations of the spikes which propagate along the nerve fiber, i.e., the axon. In particular, Neurons/nano-machines employ the spike rate and temporal coding to transmit information via action potentials.

At the end of the axon, the pre-synaptic terminals are located. They release the packets of neurotransmitters, i.e., vesicles into the gaps between two neurons/nano-machines. Information is transmitted between two neurons/nano-machines through these junctions, namely, synapses. When the spikes arrive at a synapse, another spike may be generated at the connecting nerve fiber and hereby the information is transmitted from one neuron/nano-machine to another neuron/nano-machine. In the nervous system, one assumption underlying the concept of traveling spikes is that they diffuse along the axon at constant speed [4]. In this case, when information is encoded by patterns of spikes, the communication channel is “noiseless” and the output spikes interval pattern is identical to the input spike intervals. However, this is not a realistic case in practice. Thus, generated spikes travel at different speeds from one another and their time intervals are altered as they propagate down an axon. In temporal modulation the time intervals are deterministic that the initial spikes pattern determines information that is encoded. The stochastic properties of an axon disturb the time relations of a train of spikes during its propagation along a nerve fiber. This phenomenon is known as a source of noise on the neuro-spike communication channel. This noise leads to the loss of perfect biological control. The effects of channel noise raise dramatically as neurons become smaller [5]. Channel noise affects action potential propagation in nervous fibers and produce trial-to-trial variability in action potential timing in axons thinner than 3 μm [5]. Exploiting biophysical theory and stochastic simulations demonstrated that in CNS axons of 0.1 − 0.5 μm diameter channel noise induces significant jitter in action potential propagation [5].

Several works have been carried out to analyze the capacity of the neuro-spike communication channel. In [6], the capacity of axonal transmission part has been derived. In [7], authors have offered a theoretical lower bound on the capacity of a simple cortical synapse model. In [8], authors have derived theoretical upper bounds on the information capacity of both bipartite and tripartite synapses. Authors in [9] have derived the effect of the number of available vesicles on the capacity of information transmission of synapses. All of them have considered the spike rate coding and none of them use the temporal coding by employing spikes intervals to transfer information in the neuro-spike communication. Authors in [10] have considered both temporal and rate coding. They have proved that the distribution of inputs, which achieves...
channel capacity in neuro-spike communication, is a discrete distribution with finite mass points for temporal and rate coding assumptions [10]. However, they did not propose a mathematical model for neuro-spike communication.

In this paper, we consider a neuro-spike timing channel between two nano-machines that the information is encoded by spikes time intervals in the presence of jitter of propagation in the axonal pathway and synaptic transmission. We consider Brownian motion model for the synaptic transmission. It is an efficient model for the propagation in a nano-scale environment. In this paper, we assume that the transmitter and the receiver work perfectly. In our model, the communication is corrupted by the randomness due to jitter of propagation through the axon and Brownian motion in the synapse. The key contributions of this paper can be summarized as follows. First, we consider the jitter of a neuro-spike timing channel that the information is encoded by spikes time intervals as the noise of channel. We show a Gamma stochastic variable is a suitable model for describing this jitter. Thus, the neuro-spike communication channel is modeled as communication over an additive Gamma noise channel. Next, by exploiting additive Gamma noise, we present upper and lower bounds on the capacity of a neuro-spike communication system when it uses temporal modulation to transfer information.

The paper is organized as follows. Section II presents a system model for neuro-spike communication system when it uses temporal modulation. Section III exploits this system model to derive the capacity bounds of the neuro-spike communication channel. Finally, Section IV concludes this paper.

II. SYSTEM MODEL

In this section, we propose a model for neuro-spike communication channel. This model contains three main parts, namely axonal pathway, synaptic transmission and spike generation. We investigate these parts with details in this section.

A. Temperament of Jitter Noise in Axonal Pathway

The stochastic properties of an axon disturb the time relations of a train of spikes during its propagation along a nerve fiber for the following reasons. First, a spike is generated in the output neuron due to the integration of stimuli caused by the input spikes arriving at several synapses associated with this neuron [11]. Since the intervals among arriving spikes are not all identical, the mentioned integrated stimuli reach the firing threshold randomly. Secondly, when the integrated stimuli arrive close to the threshold value, the happening of a response spike is fully stochastic [12]. Thirdly, the traveling speed of the spikes on the axon is not constant. Lass and Abeles [13] found jitter in the speed of the spikes propagating through a single fiber of the frog sciatic nerve. Hence, it is entirely probable that the biological information which is encoded by the time intervals of the spikes is disturbed by the jitter noise. To realize the temperament of this noise, in [14], authors have considered an axon. Then, to measure the propagation speed of the spikes, four current impulses at the same time separations have been applied to the stimulation electrode, which have generated four spikes. These action potentials have been generated as follows. The first response spike has propagated at a constant speed which is inherent in the axon. The second one which has been in the refractory period, has propagated at a slower speed. Moreover, the third one has been in the refractory period, caused by the second spike which has been further altered by the first spike. Finally, the fourth spike has suffered the impacts of the foregoing three spikes. To evaluate the spike interval distributions, cross correlations of them have been processed. The cross correlation between stimuli spikes and the output spikes has indicated their causal relationship and the degree of jitter in the output spikes positions. Fig. 1 indicates the cross-correlation functions between the stimuli and the response spikes. The horizontal axis shows the amount of the time shift of the response spike relative to the corresponding stimulus. It can be observed that the cross-correlation function is becoming more and more concentrated around the peaks. This shows that the response spikes pattern becomes more and more similar to the stimuli spikes configuration toward the fourth run. In Fig. 1, a Gaussian curve is shown as a dotted curve. It can be observed that the Gaussian distribution is a suitable description for the spread of the cross correlation function. In the response spike train, the mean of the cross-correlation function is 3.8 ms. According to this time difference as the conduction time of the response spikes over a distance of 44 mm between electrodes, the mean velocity is derived as 11.6 m/s. Moreover, the spread of the conduction time in response spikes is about 1.4 ms².

B. Synaptic Transmission Model

In the synaptic transmission part, we assume when a spike arrives to presynaptic terminal, at most one vesicle is released. We assume each vesicle contains neurotransmitters and they diffuse to the post-synaptic terminal. We consider that the movement of neurotransmitter molecules is governed by a Brownian motion [15]. The delay experienced by any neurotransmitter to cross from the synapse to the post-synaptic terminal, i.e., $t_s$, obeys the following probability density function:

$$f(t_s) = \frac{\lambda}{\sqrt{4\pi D t_s}} e^{-\frac{\lambda^2}{4D t_s}}, \quad t_s > 0,$$

where $\lambda = \frac{d}{D}$, $D$ is the diffusion coefficient of the neurotransmitter, and $d_s$ is the synaptic gap distance. We assume $d_s = 10$ nm and $D = 100 \mu m^2/s$.

C. Spike Generation

We consider a simple and efficient model for generation of spikes. This model is called Integrate-and-Fire (I&F) model [16]. In this model, the output neuron is considered as a capacitance, $C$, and a voltage threshold, $V_{th}$ to be fired. In this model, each neurotransmitter makes positive or negative charge onto the capacitance through depolarizing or hyperpolarizing the velum. When the capacitance charge reaches $V_{th}$, an output spike is generated and the velum potential is reset to zero. In I&F model, it is assumed that the input contains $n$
D. Statistical Neuro Spike Communication Channel Model

Fig. 2 shows the statistical system model of the timing of propagation process in the neuro-spike communication channel. This model contains the axonal pathway, synaptic transmission and spike generation parts. We assume the length of axon is equal to \( d_a \) and the average of velocity of propagation of spikes along the axon is \( \bar{V} \). According to Subsection II-A, we consider a normal stochastic variable to model the transmission time of the spikes along the axon, i.e., \( t_a \). Its distribution is described as

\[
f(t_a) = \frac{1}{\sqrt{2\pi}\sigma_a^2} e^{-\frac{(t_a - \mu_a)^2}{2\sigma_a^2}} \sim N(\mu_a, \sigma_a^2),
\]

where \( \mu_a = \frac{\bar{V}}{2} \) is the mean of the travelling time and \( \sigma_a^2 \) is the variance of traveling time. We assume \( d_a = 44 \text{ mm} \) and \( \bar{V} = 11.6 \text{ m/s} \) similar to the scenario in [14]. Thus, we can consider \( \mu_a = 3.8 \text{ ms} \) and \( \sigma_a^2 = 1.4 \text{ ms}^2 \). By considering the propagation time of neurotransmitters along the synapse based on the Subsection II-B, i.e., \( t_s \), the conditional distribution function of the arrival time of each neurotransmitter to the post-synaptic terminal, i.e., \( t_r = t_s + t_a \), when the travelling time of spikes through the axon is given by using (1), can be obtained as

\[
f(t_r|t_a) = \frac{\lambda}{4\pi(t_r - t_a)^3} e^{-\frac{\lambda^2}{4\pi(t_r - t_a)^2}}, \quad t_r > t_a.
\]

Thus, the joint distribution function of \( t_r \) and \( t_a \) is defined as \( f(t_r, t_a) = f(t_a)f(t_r|t_a) \). Hence, the marginal probability distribution function of \( t_r \) is obtained by

\[
f(t_r) = \int_{-\infty}^{+\infty} f(t_r, t_a) dt_a = \int_{-\infty}^{+\infty} f(t_r|t_a) f(t_a) dt_a.
\]

By substituting \( f(t_r, t_a) \) into (4), we have

\[
f(t_r) = \int_{-\infty}^{+\infty} \frac{1}{\sqrt{2\pi}\sigma_a^2} e^{-\frac{(t_a - \mu_a)^2}{2\sigma_a^2}} \frac{\lambda}{4\pi(t_r - t_a)^3} e^{-\frac{\lambda^2}{4\pi(t_r - t_a)^2}} e^{-\frac{\lambda^2}{4\pi(t_r - t_a)^2}} dt_a.
\]

For the spike generation part, in the first step, we consider \( n_{th} = n \), where \( n \) is the number of released neurotransmitters and \( n_{th} = \frac{a_E}{\lambda d_a} \) is the number of neurotransmitters needed to reach the threshold. Therefore, the time \( t_{out} \), for which the I&F unit generates an output spike is defined as the time that the \( n \)-th neurotransmitter arrives. This time is obtained as

\[
t_d = \max(t_{r_1}, t_{r_2}, \ldots, t_{r_n}).
\]

Thus, the cumulative distribution function (CDF) of the spike time generated by the I&F unit is defined as

\[
F(t_d) = P_r(t_d < t) = P_r \{t_{r_1} < t \} \cdot P_r \{t_{r_2} < t \} \cdot \ldots \cdot P_r \{t_{r_n} < t \}. \quad (6)
\]

We assume all neurotransmitters are independent. Hence, we have

\[
F(t_d) = P_r \{t_{r_i} < t \ \text{for all } i \} = (P_r \{t_{r_i} < t \})^n, \quad (7)
\]

where \( P_r \{t_{r_i} < t \} \) is the CDF of the arriving time of the neurotransmitters. The probability distribution function (PDF) of the output spike time, i.e., \( f(t_d) \), is the derivative of the corresponding CDF function, i.e.,

\[
f(t_d) = \frac{d}{dt} F(t_d) = n(P_r \{t_{r_i} < t \})^{n-1} \frac{dF(t_r)}{dt}. \quad (8)
\]

Thus, the PDF of the output spike time can be derived as

\[
f(t_d) = n(P_r \{t_{r_i} < t \})^{n-1} f(t_r), \quad (9)
\]

where \( f(t_r) \) is the PDF of the arrival time of the neurotransmitters. We derive the \( f(t_d) \) by numerical methods. The PDF of \( t_d \) can not be described in a closed form. Thus, we attempt to approximate \( f(t_d) \) by known random distributions. Fig. 3 shows \( f(t_d) \) and a shifted Gamma distribution function. As can be observed, a shifted Gamma distribution is a suitable choice to approximate the \( f(t_d) \). The amount of this shift determines the minimum delay of arriving all of neurotransmitters corresponding to each input spike. In the second case, we assume a more general case, i.e., \( n_{th} < n \). To derive the PDF of the output spikes time in this case, when we assume...
In output spikes arrival times. As this additive noise has a random propagation delay. In this case, the delay of the channel contains two parts: due to the channel jitter. We consider this jitter as an additive noise in the form of a Gamma distribution function to approximate the random propagation delay. We assume \( t_{r1} \leq t_{r2} \leq \ldots \leq t_{r_{th}} \leq \ldots \leq t_{rn} \), we exploit the PDF for the order statistics [17] of a sample of size \( n_{th} \) drawn from the distribution of \( f(t_r) \). Thus, we have

\[
f(t_d) = \frac{n! f(t_r)}{(n_{th} - 1)! (n - n_{th})!} (F(t_r))^{n_{th} - 1} (1 - F(t_r))^{n - n_{th}}.
\] (10)

We assume \( n_{th} = \rho n \) and we derive \( f(t_d) \) by numerical methods in this case. Fig. 4 shows \( f(t_d) \) for \( \rho = 0.75 \). It can be observed that a shifted Gamma distribution is a suitable choice to approximate \( f(t_d) \). The amount of the shift determines the minimum delay of arriving \( \rho \) proportion of the neurotransmitters corresponding to each spike. We exploit this approximation for \( f(t_d) \) in the following.

### E. Modeling the Jitter as the Channel Noise

When the propagation delay is constant, it is possible to obtain the input spikes times according to the output arrival times; however, the spikes propagation delays are fluctuated due to the channel jitter. We consider this jitter as an additive noise in the form of the random propagation delay. In this paper, we assume this jitter is the only source of uncertainty in output spikes arrival times. As this additive noise has a Gamma distribution according to Subsection II-D, we refer to the channel as an additive Gamma noise channel. Since each of the obtained Gamma distributions has a shift from the origin, we consider the delay of the channel contains two terms. The minimum propagation delay, denoted as \( \tau \), which is deterministic and a random delay which is modeled by a Gamma distribution without any shift, called as \( N \). Thus, the arrival time of the spikes to the next neuron can be modeled as \( Y' = X + N + \tau \), where \( X \) is the input spike time. For simplicity, since \( \tau \) is a deterministic term, we define \( Y = Y' - \tau \), and henceforth, we consider \( Y \) as the arrival time of output spikes. We can consider \( N \) as a Gamma stochastic variable as

\[
f_N(n) = \frac{\beta^n}{\Gamma(n)} n^{\alpha - 1} e^{-\beta n} \sim G(\alpha, \beta).
\] (11)

By substituting \( Y' \) into (11) and considering the fact that \( Y = Y' - \tau \), the probability distribution function of arrival time of the output spike \( Y = y \) when the input spike is propagated at \( X = x \) is obtained as

\[
f_{y|x}(y|x) = \frac{\beta^n}{\Gamma(n)} (y - x)^{\alpha - 1} e^{-\beta(y-x)}, \quad y > x,
\]

\[
0, \quad y \leq x.
\] (12)

### III. Capacity Bounds of Neuro-Spike Communication Channel

In this section, we analyze the capacity of the neuro-spike communication channel when we encode the information by spikes intervals. According to the model \( Y = X + N \), the mutual information between the input \( X \) and the output \( Y \) of the neuro-spike channel is obtained by

\[
I(X;Y) = h(Y) - h(Y|X) = h(Y) - h(X + N|X),
\] (13)

where \( h(\cdot) \) is the entropy function and since \( X \) and \( N \) are independent processes, we have

\[
I(X;Y) = h(Y) - h(N|X) = h(Y) - h(N).
\] (14)

The capacity of a channel is defined as the maximum mutual information between its input and output which is optimized over all possible input distributions \( f_X(x) \). The constraints on the input signal \( X \) determine the set of all possible input distributions. The constraints on the input signal are application dependent, e.g., both peak-constrained and mean constrained inputs can be considered based on the application. In this paper, we consider the mean constrained input signal since the average arrival time of input spikes is affected by the refractory period. Thus, we assume the transmitter neuron waits \( m \) seconds in average to transmit each spike. Thus, the channel capacity with input \( X \) and mean constraint \( \mathbb{E}[X] \leq m \) is defined as

\[
C = \max_{f_x(x):\mathbb{E}[X] \leq m} I(X;Y).
\] (15)

The expected value of channel jitter noise, i.e., \( \mathbb{E}(N) \) is finite. Thus, the expected time of arrival at the receiver neuron is constrained, i.e., \( \mathbb{E}(Y) = \mathbb{E}(X) + \mathbb{E}(N) \leq m + \mathbb{E}(N) \). Unfortunately, there is no closed form solution for the channel capacity with Gamma distribution noise unlike the AWGN channel. Hence, we exploit Gamma distribution function to find upper and lower bounds on the channel capacity. Prior to obtain these bounds, we present two properties of the Gamma distribution function that we use them later on.

**Property 1. (Differential entropy of the Gamma distribution):** We describe the differential entropy of a Gamma distribution by \( h_G(\cdot) \) based on parameters \( \alpha \) and \( \beta \) as

\[
h_G(\alpha, \beta) = \alpha - \ln(\beta) + \ln(\Gamma(\alpha)) + (1 - \alpha) \Psi(\alpha),
\] (16)
where $\Psi(\alpha) = \frac{\Gamma(\alpha)}{\Gamma(\alpha + 1)}$ and $\Gamma(\alpha) = \int_0^\infty t^{\alpha-1}e^{-t}dt$. This differential entropy is derived in Appendix I.

**Property 2. (Additivity property of the Gamma distribution):** Assume $N_i \sim \mathcal{G}(\alpha_i, \beta)$, $i = 1, 2, ..., L$ are L not necessarily independent Gamma random variables. Then, $N = \sum_{i=1}^L N_i$ is a Gamma random variable as $N \sim \mathcal{G}\left(\sum_{i=1}^L \alpha_i, \beta\right)$. This property is proved in Appendix II.

By using Properties 1 and 2, the following theorem presents the capacity of the neuro-spike channels.

**Theorem 1.** The capacity of the neuro-spike channel with additive Gamma noise, which is defined in (15), is bounded as

$$h_G(\alpha+m\beta, \beta) - h_G(\alpha, \beta) \leq C \leq \ln\left(m + \frac{\alpha}{\beta}\right) - h_G(\alpha, \beta),$$

(17)

where $h_G(\alpha, \beta)$ is obtained by Property 1.

**Proof:** The proof is given in Appendix III.

Moreover, we consider a uniform distribution for the input spike intervals. This case is close to the practical signaling schemes. In this case, we can only describe $f_Y(y)$ in a closed form. Thus, numerical integration is exploited to calculate mutual information. By assuming that $X$ has a uniform distribution on $[0, 2m]$, the output distribution is derived by convolving the input and noise distributions as

$$f_Y(y) = \begin{cases} \frac{1}{2m} \frac{1}{\Gamma(\alpha) \Gamma(\beta)} \gamma(\alpha,\beta y), & y \leq 2m, \\ \frac{1}{2m} \frac{\Gamma(\alpha) \Gamma(\beta)}{\Gamma(\alpha+\beta)} (\gamma(\alpha,\beta y) - \gamma(\alpha,\beta(y-2))), & y > 2m, \end{cases}$$

(18)

Unlike AWGN channels, in the described channel, there is no parameter like SNR which determines the mutual information. In this condition, the mutual information is a function of both spike intervals and receiver threshold, i.e., $\rho$. Fig. 5 shows the mutual information as a function of the average input spike interval by considering uniform, exponential and Gamma distributions as the input. It can be observed that the upper and lower bounds are close to each other for shorter spike intervals. In addition, the uniform spike interval tracks the upper bound at longer intervals. However, it is close to the lower bound for shorter intervals. Fig. 6 shows the mutual information versus different values of $\rho$. It can be observed the upper and lower capacity bounds reduce when $\rho$ increases. This is because of the fact that the jitter of the output spikes occurs more often.

**IV. Conclusion**

In this paper, we have considered a neuro-spike communication system that information conveyed in the time intervals of input spikes. We have modeled this channel by an additive Gamma noise channel. We have indicated via numerical methods that the proposed channel model is suitable for a neuro-spike communication system when it exploits temporal modulation to transfer information between the transmitter and receiver. Then, we have derived the upper and lower bounds on the neuro-spike channel capacity. We have shown unlike the AWGN channels, there is no single quality measure like to signal to noise ratio. Thus, we have analyzed the channel capacity bounds versus different values of time intervals and the decision threshold of the receiver. It has been observed that the upper and lower bounds are close to each other for shorter spike intervals. Moreover, we have assumed the uniform spike interval to compare with the capacity bounds. The channel capacity in this case tracks the upper bound at longer intervals, however, it is close to the lower bound for shorter intervals.

**APPENDIX I**

**DIFFERENTIAL ENTROPY OF GAMMA DISTRIBUTION**

The differential entropy of Gamma distribution is defined as $h_G(\alpha, \beta) = \mathbb{E}\{-\ln(f(x))\}$, where $f(x) = \frac{\beta^\alpha x^{\alpha-1}e^{-\beta x}}{\Gamma(\alpha)}$, $x > 0$. Thus, we have

$$h_G(\alpha, \beta) = -\alpha \ln(\beta) + \ln(\Gamma(\alpha)) + \beta \mathbb{E}(x) - (\alpha - 1) \mathbb{E}(\ln(x)),

(1.1)

where for Gamma distribution, we have $\mathbb{E}(x) = \frac{\alpha}{\beta}$. Then, we derive $\mathbb{E}(\ln(x))$ as follow:

$$\mathbb{E}(\ln(x)) = \frac{\beta^\alpha}{\Gamma(\alpha)} \int_0^\infty x^{\alpha-1}e^{-\beta x} \ln(x)dx.

(1.2)

We define $J(\alpha, \beta) = \int_0^\infty x^{\alpha-1}e^{-\beta x} \ln(x)dx$, and thus, we can write

$$J(\alpha, \beta) = \frac{d}{d\alpha} \int_0^\infty x^{\alpha-1}e^{-\beta x}dx = \frac{d}{d\alpha} \left(\frac{\Gamma(\alpha)}{\beta^\alpha}\right).

(1.3)

Thus, it can be simplified as

$$J(\alpha, \beta) = \frac{\Gamma(\alpha)}{\beta^\alpha} \frac{d}{d\alpha} \left(\ln \left(\frac{\Gamma(\alpha)}{\beta^\alpha}\right)\right) = \frac{\Gamma(\alpha)}{\beta^\alpha} (\Psi(\alpha) - \ln(\beta)),

(1.4)
where $\Psi(\alpha) = \frac{r'(\alpha)}{\Gamma(\alpha)}$. By substituting the expression in (I.4) into (I.2), we can obtain $h_G(\alpha, \beta)$ as (16).

APPENDIX II

ADITIVITY PROPERTY OF THE GAMMA DISTRIBUTION

We assume two Gamma distribution as $X \sim G(a_1, b)$ and $Y \sim G(a_2, b)$. Now, we define $Z = X + Y$. The distribution of a sum of two random variables is derived by convolution of their distributions. Since $f(x)$ and $f(z - x)$ are defined for $x > 0$ and $x < z$, respectively; we have

$$f_{X+Y}(z) = \int_0^z f_X(x) f_Y(z-x) dx$$

$$= \int_0^z \frac{x^{a_1-1}e^{-x}}{\Gamma(a_1)} \frac{(z-x)^{a_2-1}e^{-(z-x)}}{\Gamma(a_2)} dx.$$  \hspace{1cm} \text{(II.1)}$$

Now, we change variable as $x = zt$. Hence, the expression in (II.1) is converted to:

$$f_{X+Y}(z) = e^{-z} z^{a_1+a_2-1} \frac{1}{B(a_1, a_2)} \int_0^1 t^{a_1-1} (1-t)^{a_2-1} \frac{\Gamma(a_1)\Gamma(a_2)}{\Gamma(a_1+a_2)} dt.$$

The Beta probability distribution function is described as

$$f_{Beta}(x) = \frac{x^{\alpha-1}(1-x)^{\beta-1}}{B(\alpha, \beta)}, \quad 0 \leq x \leq 1,$$

where $B(\alpha, \beta) = \frac{\Gamma(\alpha)\Gamma(\beta)}{\Gamma(\alpha+\beta)}$. Since $\int_0^1 f_{Beta}(x) dx = 1$, we have

$$\int_0^1 t^{a_1-1}(1-t)^{a_2-1} \frac{\Gamma(a_1)\Gamma(a_2)}{\Gamma(a_1+a_2)} dt = \frac{1}{\Gamma(a_1+a_2)}.$$  \hspace{1cm} \text{(II.4)}$$

Hence, by inserting the expression in (II.4) into (II.2), we can conclude $f_{X+Y}(z) = \frac{z^{a_1+a_2-1}}{\Gamma(a_1+a_2)}$ and $Z \sim G(a_1 + a_2, b)$.

APPENDIX III

PROOF OF THEOREM 1

From (14), we have $I(X;Y) = h(Y) - h_G(\alpha, \beta)$. Therefore, we can conclude that the $I(X;Y)$ achieves its maximum value via maximizing $h(Y)$ subject to the constraint described by $E(Y) \leq m$ or equivalently $E(Y) \leq m + \frac{\alpha}{\beta}$. Thus, $I(X;Y)$ is maximized when $h(Y)$ gets its maximum value subject to $f_Y(y) = 0$, $y < 0$, and $E(Y) \leq m + \frac{\alpha}{\beta}$. To derive the upper bound of the neuro-spike channel with an additive Gamma noise, we consider an exponential distribution which is defined over the interval $(0, \infty)$. Since the exponential distribution is known as the entropy maximizing distribution when a random variable has a mean constraint [18], By assuming $Y \sim \exp(\frac{1}{m+\frac{\alpha}{\beta}})$; it is obtained that $h(Y) = \ln \left( \frac{m + \frac{\alpha}{\beta}}{\beta} \right)$ and $h(Y)$ has the maximum value for any possible distribution of $Y$ with $E(Y) \leq m + \frac{\alpha}{\beta}$. Hence, we have

$$C \leq \ln \left( \frac{m + \frac{\alpha}{\beta}}{\beta} \right) - \alpha - (1-\alpha)\Psi(\alpha) = C_{\text{Upper}}.$$  \hspace{1cm} \text{(III.1)}$$

To derive the lower bound of the neuro-spike channel with an additive Gamma noise, we consider the input signal $X$ is Gamma distributed with $\alpha'$ and $\beta$ parameters. Hence, when the input signal distribution is assumed as $G(m, \beta)$ according to $E(X) = m$, then from Property 2, the output signal distribution is obtained as $Y \sim G(m + \alpha, \beta)$. Therefore, we have $h(Y) = h_G(\alpha + m\beta, \beta)$ and we can write $C \geq h_G(\alpha + m\beta, \beta) - h_G(\alpha, \beta)$. By using Property 1, we have

$$C \geq m + \ln \left( \frac{\ln(\alpha + m\beta)}{\ln(\alpha)} \right) + (1 - \alpha - m\beta)\Psi(\alpha + m\beta) - (1 - \alpha)\Psi(\alpha) = C_{\text{Lower}}. \hspace{1cm} \text{III.2}$$

Note that in this case $f_Y(y)$ is not necessarily an entropy maximizing distribution for a given mean of $m + \frac{\alpha}{\beta}$. Thus, we have $C_{\text{Lower}} \leq C_{\text{Upper}}$.

REFERENCES