Range Expansion in Neuro-Spike Synaptic Communication: Error Performance Analysis

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Abstract. In this paper, a neuro-spike synaptic cooperative communication channel model is exploited. In the considered model, a neuro-spike relay (NSR) is placed in the synaptic gap of two neurons to extend the range of communication. For the analysis, a time-slotted channel is exploited, where we transmit a binary bit in each time-slot for the transmission of information from the pre-synaptic neuron called neuro-spike source (NSS) to the post-synaptic neuron called neuro-spike destination (NSD). Further, the considered model is analyzed in terms of the probability of detection and probability of false alarm. Moreover, the effect of ISI due to the transmission of molecules from the previous time-slots, and noise arises from unintended neurons are also considered in the analysis. Furthermore, the closed-form expression for the end-to-end probability of error is also computed for the cooperative link. Above all, the analytical expressions are validated using Monte-Carlo simulations.

Keywords: Neuro-spike communication · synaptic gap · neuro-spike relay · Poisson distribution

1 Introduction

A molecular communication (MC) is one of the trending communication technologies in recent days, in which the characteristics of molecules can be used to transmit the information from one end to other [1]. Few examples of an MC systems are cell-cell signaling [2], bacterial communication [3], and synaptic or neuro-spike communication [4]. The neuro-spike communication is one of the widely investigated forms in molecular communication [5–7]. In this, both electrochemical impulse and neurotransmitter are used for the transmission of molecules from neuro-spike source (NSS) to neuro-spike destination (NSD) neurons. Inside the neuron, the information is transmitted in the form of electrochemical impulse which is called as axon potential and is transmitted to the end of a neuron.

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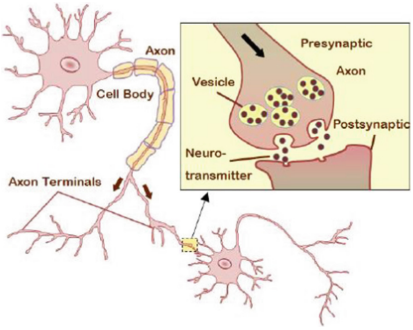


Fig. 1. Basic structure of neuron [8].

Neurons communicate with one another at junctions called *synapses*. At a synapse, one neuron sends a message to a target neuron-another cell. Mostly, synapses are chemical, i.e., these synapses communicate using chemical messengers. In a few cases, synapses are electrical, i.e., in these synapses, ions flow directly between the cells. At a chemical synapse, an action potential triggers the pre-synaptic neuron to release neurotransmitters. These molecules bind to receptors on the postsynaptic cell and make it more or less likely to fire an action potential. The pre-synaptic and post-synaptic neurons have a gap in between which is known as the synaptic cleft. The synaptic communication has nerve cell as a major part and it basically consist of three parts:

- Dendrites:- These are the projections of neuron that receive signal from other neuron. It acts as sensor or receptor of a basic communication system. When a random stimulus is applied to neurons its dendrites fires and generates an electrical signal which propagates through the axon to its tail. Dendrites branches as they move towards their tip just behave like tree branches.
- Axon:- Each neuron in our body has a cable like structure which is very thin. It is several times thinner than human hair. It acts as a carrier for the electrical pulse generated at the dendrites. Depending upon the types of neuron axon length varies.
- Axon terminal:- At this end, packets of neurotransmitter are released in the synaptic cleft. The released neurotransmitter diffuses in the synaptic cleft follows brownian motion. Brownian motion is the random motion of particles. The basic structure of neuron is demonstrated in Fig. 1.

It is very well known fact that neuro-degenerative diseases are incurable in many cases [9]. For instance, connections of neurons are lost which may cause death of a person. Further, in the recent experiments, it was seen that loss of neuron connectivity increases the average gap of two neurons, which is the primary cause of Alzheimer's disease. Size of neuron is found to be very small and it ranges from 4 to 100 μm [10]. In neuro-spike communication, usually, out of one hundred billion nerve cells in the human brain, each one of them communicates with thousand others. The stronger connection between neurons can be made if a neuro-spike relay (NSR) is placed in between the NSS and NSD.

A neuro-spike-communication system consists of three-part, i.e., axonal pathway, spike generation, and synaptic transmission. Our study mainly focuses on synaptic communication where glutamate molecules act as a carrier of information from one neuron to another. When axon potential reaches the tail of transmitting neurons, a number of vesicles diffuse in the synaptic gap. These molecules follow Brownian motion in the synaptic cleft and reach the dendrites of the destination neuron. When the received number of molecules at post-synaptic neurons exceeds the required decision threshold, the receiver confirms that the particular bit of information is successfully received.

The communication between two neurons gets weaker because of the increase in average path length between them. This increase in average path length occurs due to death or progressive degradation of nerve cells. Several works have studied synaptic communication in different aspects. For instance, in [11] authors have discussed capacity analysis of neuro-spike communication using temporal modulation. Further, authors have discussed the role of ISI in synaptic communication in [12]. The interfacing of nano-machine and neuron communication has been provided in [13]. Moreover, in [14], the joint optimization of input spike rate and decision threshold at the receiver to maximize achievable bit rate has been exploited. Authors have discussed the diffusion-based model for synaptic communication in [15].

The error probability for a cooperative communication system consisting of K receivers in a diffusive medium was provided in [16]. In [17], authors have discussed ML detection for a cooperative communication system in the diffusive channel. In [18], authors have discussed the optimal positioning of a relay machine in the cooperative diffusive molecular communication channel. In [19], authors have discussed characterizing the three-dimensional diffusive molecular communication channel with an absorbing receiver. In [20], authors have discussed optimal receiver design for diffusive molecular communication channels with flow and additive noise. In [21], authors have analyzed a communication network consisting of a single transmitter and receiver incorporated by multiple relays in between them. In [22], authors have considered a passive receiver that receives molecules without changing its characteristics, and further, they can diffuse in the environment. In [23,24], authors have assumed that molecular concentration remains uniform throughout the channel and source and destination nano-machine can count the number of molecules within the spherical receiver boundary. Note that, the concentration of neuro-transmitter attenuates with the distance [25]. Thus, neuro-spike direct communication between source and destination may leads to the information loss due to the degraded neuro-transmitters. One way to reduce the imapct of degradation on information loss is by adding the additional nodes (known as neuro spike relay) between NSS and NSD which can increase the communication channel length.

To the best of the author's knowledge, no authors have considered the error performance of cooperative neuro-spike communication. Based on the above research gap and motivations, in this paper we consider range expansion using

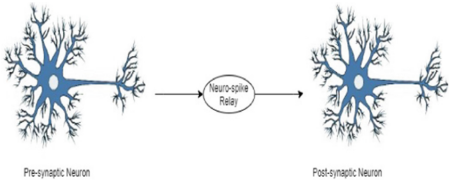


Fig. 2. Two neurons assisted by a NSR.

the relay-assisted method in neuro-spike synaptic communication. In this context, the following are our key contributions through this work:

- First time in the literature, for the range expansion the relay-assisted neuro-spike Synaptic communication is considered.
- We consider the effect of re-uptake probability and spill-over effect for calculation of results.
- The impacts of inter-symbol-interference (ISI) and unintended molecules from other sources are also considered.
- Further, the considered system is presented in the form of equivalent binary channel and is analyzed in terms of probability of detection and probability of false alarm.
- Moreover, the expressions for average probability of error considering ISI is derived. We evaluate the error performance of the system considering various environment parameters, e.g., diffusion coefficient of neuro transmitter.

Above all, the analytical expressions are verified using Monte-Carlo simulation.

The rest of the paper is organized as follows. In Sect. 2, we present a system model of the cooperative neuro-spike communication system. Also in this section, we provide the preliminaries required for neuro-spike communication. The formulation of analytical frameworks for direct and NSR-assisted communication links is provided in Sect. 3. Numerical results are examined in Sect. 4. At the end, we conclude our paper in Sect. 5.

2 System Model

As shown in Fig. 2, we consider a relay-assisted neuro-spike communication system, in which a pre-synaptic and a post-synaptic neurons are placed at a particular distance as a source and destination, respectively. To enhance the range of communication, a neuro-spike relay is also placed in between the pre-synaptic and post-synaptic neurons. Let us consider a binary information transmission between pre-synaptic and post-synaptic neurons. And to do so, a time-slotted channel is assumed, where pre-synaptic neuron transmits K binary information bits towards the post-synaptic neuron. Let us consider the binary information sequence of length K bits which is to be transmitted, is presented by $\{x[1], x[2], \ldots, x[i], \ldots, x[K]\}$, where $x[i] \in \{0, 1\}$, $\forall i$. Thus, for K bits, total 2^K

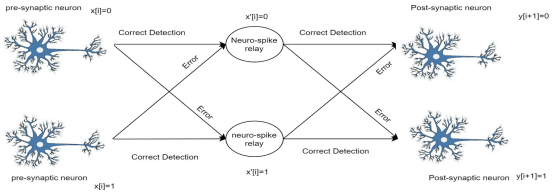


Fig. 3. System model for two neurons cooperated by a NSR.

symbols can be encoded and transmitted from the source. We assume that the priory probability of a particular bit being transmitted as 0 and 1 be α_0 and α_1, respectively. For relaying, a decode and forward strategy is used, in which pre-synaptic neuron transmits a particular bit in the i^{th} time slot, later on, that bit is received and decoded first at the NSR, subsequently, the decoded bit is forwarded to the destination in the same time slot. At the end, the information bit is received and decoded at the post-synaptic neuron, eventually, in $(i+1)^{th}$ time slot. Therefore, the transmission of complete K bits of information from source to destination neurons takes $(K+1)$ time slots.

We consider a binary concentration shift keying (BCSK) for the transmission of information sequence, i.e., the pre-synaptic neurons and NSR transmit $Q_T[i]$ and $Q_C[i]$ number of molecules, respectively, for the transmission of binary bit 1, and zero molecules are used for transmission of binary bit 0 at the beginning of i^{th} time slot. Further, it is assumed that source neurons and neuro-spike relay emits the different types of molecules, such that there is no self-interference at the neuro-spike relay [26,27]. Due to the random movement of molecules from pre-synaptic to post-synaptic neurons, the molecules may reach to the post-synaptic neuron out of order, eventually, leads to the stochastic channel behaviour. One of the possible ways to characterizes the stochastic behaviour of channel between the pre-synaptic and post-synaptic neurons is by using transition probability matrix. The transition probability matrix for the links from source to NSR, NSR to destination, and source to destination can be described as below

$$\begin{bmatrix} P_{00}^{sr} & P_{01}^{sr} \\ P_{10}^{sr} & P_{11}^{sr} \end{bmatrix}, \begin{bmatrix} P_{00}^{rd} & P_{01}^{rd} \\ P_{10}^{rd} & P_{11}^{rd} \end{bmatrix}, \text{ and } \begin{bmatrix} P_{00}^{sd} & P_{01}^{sd} \\ P_{10}^{sd} & P_{11}^{sd} \end{bmatrix},$$

respectively, here P_{lm}^{sr}, P_{lm}^{rd} and P_{lm}^{sd} $(\{l,m\} \in \{0,1\})$ are the transition probabilities corresponding to the transmitted symbol l and received symbol m from source to relay, relay to destination, and source to destination, respectively (Fig. 3).

2.1 Preliminaries

Neuro-spike communication consists of three processes, namely the Vesicle release, diffusion, and ligand-receptor binding. Vesicles are a group of neuro-transmitters enclosed inside a closed volume of thin membrane and it is located

just behind the pre-synaptic membrane of the pre-synaptic or transmitting neuron. When the axon potential reaches the neuron tail, the bundle of glutamate molecules released diffuses in the synaptic cleft. In the synaptic gap, information transmission can be modeled in the form of molecular communication for which the study can have two different approaches; first, microscopic approach [28], in which the focus is on the probability of arrival of a single molecule. And the second is the macroscopic approach, in which the molecular concentration is considered at the receiver corresponding to the impulse response of the channel [29]. In this paper, we use a macroscopic model in which transmitted molecules in the cleft follow a Brownian motion. The concentration of molecules at a different location in the three-dimensional space is defined by Fick's equation.

$$\frac{\partial C(x,y,z,t)}{\partial t} = D\nabla^2 C(x,y,z,t) \; ; \; t \geq 0 \; , \tag{1}$$

where D is the diffusion coefficient, ∇^2 is the Laplacian operation, $(x, y, z) \in \mathbb{R}^2 \times [0, L]$, and L is the gap between the tail of transmitting neurons and head of receiving neurons. Let the concentration of glutamate molecules at a time t in the three-dimensional space is denoted by $C(x, y, z, t)$. At $t = 0$, concentration of glutamate molecules is describe by the equation:

$$C(x,y,z,0) = N_{glu}\,\delta(x,y,z) \; , \tag{2}$$

where N_{glu} is an initial number of glutamate molecules within the vesicle. It is also assumed that there is no flux of glutamate molecules between pre-synaptic and post-synaptic membranes. In recent studies, it is observed that all the molecules in the vesicles of a pre-synaptic neuron do not reach the dendrites of a post-synaptic neuron [30]. Some molecules diffuse long enough to actuate neurons placed outside the synaptic cleft, even sometimes, these molecules can actuate neighboring neurons. This activity of actuating non-intended neurons is called *spill-over* and having significant physiological effects on synaptic transmission. Considering *spill-over* effect and reuptake process[1], the solution of (1) in terms of Fourier series is obtained as [11]

$$C(x,y,z,t) = \frac{N_{glu}}{4\pi LDt} \times \exp\left(-\frac{(x^2+y^2)}{4Dt}\right) \times \left[1+2\sum_{1}^{N}\left[(1-p_u)^n \cos\left(\frac{n\pi z}{L}\right)\exp\left(Dt\left(\frac{n\pi}{L}\right)^2\right)\right]\right], \tag{3}$$

where n represents number of Fourier modes taken for the evaluation of (3).

Remark: When molecules are transmitted from the pre-synaptic neurons, a part of the transmitted glutamate molecule is absorbed by the pre-synaptic terminal for recycling. Therefore, when re-uptake effect is considered, the above solution of the differential equation can be modified. Here, the coefficients of the

[1] The reuptake process is defined as the re-absorption by a neuron of a neurotransmitter following the transmission of a nerve impulse across a synapse [25].

Fourier series get modified based on reuptake probability, i.e., when $p_u = 0$, then no molecules are absorebd by the pre-synaptic membrane, and if $p_u = 1$, then all the molecules which hit the pre-synaptic membrane are absorbed.

Now, we defined a substantial terminology in the context of neuro-spike communication known as *spike generation*. The *spike generation* occurs when the potential of the membrane of a destination neuron reaches the threshold it fires a spike in the form of an electrical signal and propagates via the axon of the post-synaptic neuron. The neurotransmitter that arrives at the post-synaptic neurons either gets reflected from the membrane or gets bonded with the receptor to form ligand-receptor complex [31]. The binding process is modeled in such a way that receptors regularly sample in a small volume, V_e. A receptor can either be in a bound or unbound state. If a neurotransmitter is in the unbound state and it samples at least once in small effective volume, V_e, then it goes to the bound state. Further note that binding of glutamate is also a reversible process, i.e., the ligand may dissociate with the receptor and may get rebounded again. Binding probability is calculated first by considering a single neurotransmitter in the synaptic cleft, i.e., $N_{glu} = 1$. Probability of finding that neurotransmitter in the cleft at any time t is obtained by integrating the concentration function $C(x, y, z, t)$ for spatial coordinates space (x, y, z) and is given by

$$P_e(t) = \iiint_{V_e} C(x, y, z, t)\, dx\, dy\, dz. \tag{4}$$

Let i be an intended information transmission time-slot, in which the pre-synaptic neuron transmits $N_{glu}[i]$ number of molecules towards the post-synaptic neuron. Thus, the total number of molecules at the post-synaptic neuron is given by

$$N^{sd}[i] = N^{sd}_{cr}[i] + N^{sd}_{int}[i] + N^{sd}_{n}[i], \tag{5}$$

where $N^{sd}[i]$ is the total number of molecules received in i^{th} time slot at the receiving neurons. $N^{sd}_{cr}[i]$ is the number of molecules received in the i^{th} time slot at the receiving neuron due to transmission of molecules in the current time slot and it follows Binomial distribution given by $\mathcal{B}(N_{glu}[i]\, x[i]; P^{sd}_{cr})$, where P^{sd}_{cr} is the probability of arrival of molecules in the current time slot.

$N^{sd}_{int}[i]$ is the number of molecules received in the i^{th} time slot due to transmission of molecules from the previous time slot, i.e., from 1^{st} time slot to $(i-1)^{th}$ time slot and these time slots are called as interfering time slot. It follows binomial distribution given by $\mathcal{B}(N_{glu}[i-j]\, x[i-j]; P^{sd}_{int})$, where $j \in \{1, 2, \ldots, i\}$ and P^{sd}_{int} is the probability of arrival of molecules from the previous time slot i.e.$(i-1)^{th}$ slot. N^{sd}_{n} is the number of molecules at post-synaptic neuron due to the background noise which is known as synaptic noise. The main source of the synaptic noise is the background synaptic activity which is due to the multiple access of synapses from thousands of other synapses [32].

Since, for a large number of molecules and small arrival probability, the Binomial distribution can be well approximated as Poisson's distribution [33]. Thus, by applying Poisson approximation, $N^{sd}_{cr}[i]$, $N^{sd}_{int}[i]$ and $N^{sd}_{n}[i]$ can be approximated with average rate $\theta^{sd}_{cr}[i] = N_{glu}[i-j]\, x[i]\, P^{sd}_{cr}$, $\theta^{sd}_{int}[i] = N_{glu}[i]\, x[i-j]\, P^{sd}_{int}$ and $\theta^{sd}_{n}[i]$ respectively.

3 Detection Analysis and Error Probability Computation

Now we calculate the number of molecules obtained at NSR after transmission from source neurons and, eventually, the number of molecules received at destination neuron. We define two binary hypotheses for the transmission of binary symbols 0 and 1, i.e., null and alternate hypotheses, respectively.

3.1 NSR Assisted Communication Between Pre-synaptic and Post-synaptic Neurons

Let us consider an information bit is transmitted from a pre-synaptic neuron to NSR in i^{th} time slot, and is decoded and re-transmitted to the post-synaptic neuron in the same time slot, and later it arrived at the post-synaptic neuron in the $(i+1)^{th}$ time slot. The symbol detection problem for an NSR-assisted system can also be formulated as a binary hypothesis testing problem. The null and alternate hypothesis for the link between the pre-synaptic neuron and NSR can be written as

$$H_0^{sr}[i] : N_n^{sr}[i] + \sum_{k=1}^{i-1}(N_{int}^{sr}[k]), \qquad H_1^{sr}[i] : N_n^{sr}[i] + N_{cr}^{sr}[i]\sum_{k=1}^{i-1} N_{int}^{sr}[k], \tag{6}$$

where $H_0^{sr}[i]$ and $H_1^{sr}[i]$ are binary hypotheses corresponding to transmission of bits 0 and 1, respectively. Thus, the number of molecules $N^{sr}[i]$ under $H_0^{sr}[i]$ and $H_1^{sr}[i]$ follow Poisson distribution [33,34]

$$H_0^{sr}[i] : \mathcal{P}(\theta_0^{sr}[i])\,; \qquad H_0^{sr}[i] : \mathcal{P}(\theta_1^{sr}[i]), \tag{7}$$

where

$$\theta_0^{sr}[i] = \theta_{int}^{sr}[i] + \theta_n^{sr}[i] = N_{glu}[i]\sum_{j=1}^{i-1} x[i-j]P_{int}^{sr} + \theta_n^{sr}[i], \tag{8}$$

and

$$\theta_1^{sr}[i] = \theta_{int}^{sr}[i] + \theta_{ms}^{sr}[i] + \theta_{cr}^{sr}[i] = N_{glu}[i]\sum_{j=1}^{i-1} x[i-j]P_{int}^{sr} + \theta_{ms}^{sr}[i] + N_{glu}[i]x[i]P_{cr}^{sr}\,. \tag{9}$$

Similarly, number of molecules received from NSR to Post-synaptic neuron can be formulated in term of two binary hypotheses as

$$H_1^{rd} : N_1^{rd}[i+1] = N_n^{rd}[i+1] + N_{cr}^{rd}[i+1] + \sum_{k=2}^{i} N_{int}^{rd}[k]\,, \tag{10}$$

$$H_0^{rd} : N_0^{rd}[i+1] = N_n^{rd}[i+1] + \sum_{k=2}^{i} N_{int}^{rd}[k]. \tag{11}$$

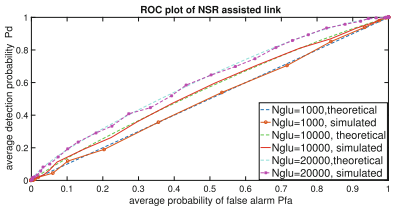


Fig. 4. ROC plot for NSR assisted link

The total number of molecules received at NSD follows the Poisson statistics and can formulated in terms of two hypotheses $H_0^{rd}[i+1]$ and $H_1^{rd}[i+1]$ as

$$H_0^{rd}[i+1] : \mathcal{P}(\theta_0^{rd}[i+1]); \qquad H_1^{rd}[i+1] : \mathcal{P}(\theta_1^{rd}[i+1]), \tag{12}$$

where the expressions for $\theta_0^{rd}[i+1]$ and $\theta_1^{rd}[i+1]$ can be obtained analogously from (8) and (9), respectively.

Now, depending upon the concentration of molecules received at the neurospike relay $N^{sr}[i]$ and at NSD $N^{rd}[i+1]$, the corresponding symbols are decoded. The symbol detection problems at relay and NSD are formulated as

$$N^{sr}[i] \underset{H_0^{sr}}{\overset{H_1^{sr}}{\gtrless}} \eta^{sr}[i]; \quad N^{rd}[i+1] \underset{H_0^{rd}}{\overset{H_1^{rd}}{\gtrless}} \eta^{rd}[i+1], \tag{13}$$

respectively. Here $\eta^{sr}[i]$ and $\eta^{rd}[i+1]$ are the optimum thresholds at relay and NSD, respectively. Let $X[i]$, $X'[i]$ and $Y[i+1]$ are the symbols at NSS, relay and NSD, respectively. In cooperative link, error occurs if any of the links is erroneous, i.e., $X[i] \neq X'[i]$and $X'[i] = Y[i+1]$ or $X[i] = X'[i]$ and $X'[i] \neq Y[i+1]$. Thus, the expression for probability of error in $(i+1)^{th}$ time slot can be written as:

$$P_e[i+1|X_1^{i-1}] = \alpha_1 P_e[i+1|X[i]=1, X_1^{i-1}] + \alpha_0 P_e[i+1|X[i]=0, X_1^{i-1}], \tag{14}$$

where $P_e[i+1|X[i]=1, X_1^{i-1}]$ is the probability of error when 1 was transmitted from the source neuron and $P_e[i+1|X[i]=0, X_1^{i-1}]$ is the probability of error at the receiver in $(i+1)^{th}$ time slot when 0 was transmitted from the transmitter for the given ISI sequence. Now the terms in (14) can be further expanded as

$$\begin{aligned}
&P_e[i+1|X[i]=1, X_1^{i-1}]) \\
&= \Pr(N^{sr}[i] < \eta^{sr}[i]|X[i]=1, X_1^{i-1}) \times \Pr(N^{rd}[i+1] < \eta[i+1]|X'[i]=0, X_1'^{i-1}) \\
&+ \Pr(N^{sr}[i] \geq \eta^{sr}[i]|X[i]=1, X_1^{i-1}) \times \Pr(N^{rd}[i+1] < \eta[i+1]|X'[i]=1, X_1'^{i-1}) \\
&= (1 - P_d^{sr}[i|X_1^{i-1}])(1 - P_f^{rd}[i+1|X_1'^{i-1}]) + (P_d^{sr}[i|X_1^{i-1}])(1 - P_d^{rd}[i+1|X_1'^{i-1}]),
\end{aligned} \tag{15}$$

and

$$\begin{aligned}
&P_e[i+1|X[i]=0,X_1^{i-1}]\\
&=\Pr(N^{sr}[i]\geq\eta^{sr}[i]|X[i]=0,X_1^{i-1})\Pr(N^{rd}[i+1]\geq\eta[i+1]|X'[i]=1,X_1'^{i-1})\\
&+\Pr(N^{sc}[i]<\eta^{sr}[i]|X[i]=0,X_1^{i-1})\Pr(N^{rd}[i+1]\geq\eta[i+1]|X'[i]=0,X_1'^{i-1})\\
&=(P_f^{sr}[i|X_1^{i-1}])(P_d^{rd}[i+1|X_1'^{i-1}])+(1-P_f^{sr}[i|X_1^{i-1}])(P_f^{rd}[i+1|X_1'^{i-1}])\,, \qquad (16)
\end{aligned}$$

where $\eta^{sr}[i]$ and $\eta^{rd}[i+1]$ are decision thresholds for NSR and post-synaptic neurons. The terms $P_d^{sr}[i]$ and $P_d^{rd}[i+1]$ denote the probabilities of detection at NSR and receiving neuron, respectively. Also, $P_f^{sr}[i]$ and $P_f^{rd}[i+1]$ denote the probabilities of false alarm at NSR and receiving neuron, respectively, which can be written as

$$P_f^{sr}[i|X_1^{i-1}]=\Pr\left(Y[i+1]=1|X[i]=0,X_1^{(i-1)}\right)=1-\sum_{m=1}^{\lfloor\eta^{sr}[i]\rfloor}\frac{\exp(-\theta_0^{sr}[i])(\theta_0^{sr}[i])^m}{m!}, \qquad (17)$$

and

$$P_d^{sr}[i|X_1^{i-1}]=\Pr\left(Y[i+1]=1|X[i]=1,X_1^{(i-1)}\right)=1-\sum_{m=1}^{\lfloor\eta^{sr}[i]\rfloor}\frac{\exp(-\theta_1^{sr}[i])(\theta_1^{sr}[i])^m}{m!}. \qquad (18)$$

Similarly, the probability of detection and false alarm for the second link, i.e., from NSR to destination neuron can be written as

$$P_f^{rd}[i+1|X_1^{i-1}]=1-\sum_{m=1}^{\lfloor\eta^{rd}[i+1]\rfloor}\frac{\exp(-\theta_0^{rd}[i])(\theta_0^{rd}[i])^m}{m!} \qquad (19)$$

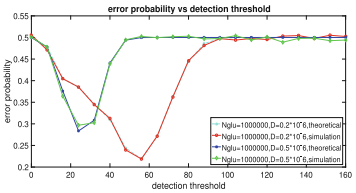
and

$$P_d^{cd}[i|X_1^{i-1}]=1-\sum_{m=1}^{\lfloor\eta^{rd}[i+1]\rfloor}\frac{\exp(-\theta_1^{rd}[i])(\theta_1^{rd}[i])^m}{m!} \qquad (20)$$

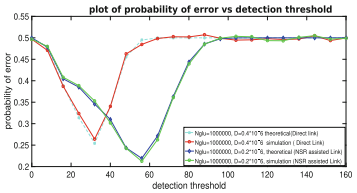
The average probability of error $P_{e,avg}[i+1]$ is obtained by taking average over all the possible realizations of X_1^{i-1}, i.e.,

$$P_{e,avg}[i+1]=\sum_{X_1^{i-1}\in\chi}\Pr(X_1^{i-1})P_e(i+1\mid X_1^{i-1}), \qquad (21)$$

where $\Pr(X_1^{i-1})$ is the probability of occurrence of one ISI symbol and can be evaluated as $\Pr(X_1^{i-1})=\frac{1}{2^{i-1}}$.



(a) Probability of error plot of NSR assisted link.



(b) Performance comparison between direct link and NSR assisted link in terms of probability of error

Fig. 5. Probability of error plots for NSR assisted link.

4 Numerical Results

In this section, the Monte-Carlo simulation is used to verify the analytical expressions. The system parameters for the simulation are as $K = 5$, $D_{glu} = 0.1 \times 10^{-9}\mathrm{m}^2/\mathrm{s}$, synaptic cleft width $L = 20\,n\mathrm{m}$, effective volume $V_e = 0.5 \times 0.5 \times 0.5\,n\mathrm{m}^3$, $N = 200$, and reuptake probability $P_u = 0.1$. All the system parameters remain unchanged throughout the simulations until otherwise stated.

The ROC plot for the NSR-assisted link is demonstrated in Fig. 4 considering a large number of transmitted molecules from the source neuron. The ROC is obtained by plotting the values of the probability of detection as a function of the probability of false alarm. First, it can be seen that the theoretical and analytical results for the probability of detection and false alarm are closely matched. Herein, it is observed that as the count of molecules sent from the pre-synaptic neuron increases, the probability of detection increases, and the false alarm probability decreases. Hence, the area under the ROC plot increases, which shows a performance improvement. It can also be observed from this figure that as the received number of molecules increases at the receiver, the area under the ROC plot increases which indicates better detection of the transmitted symbol. This is because detection probability increases with the increment in the value of received molecules. It can be seen that both the analytical and theoretical results match closely.

Figure 5(a) shows the plot of error probability versus detection threshold for NSR-assisted link. It is observed that the error probability achieves its minimum value at a particular value of the decision threshold which is an optimal threshold. It can also be marked that the probability of error increases with the increments in the value of the diffusion coefficient. Further, when the diffusion coefficient of the molecules increases, the total of molecules arriving at the receiver in the current time slot decreases, and the count of molecules at the destination in the interfering time slot increases. This results in an increase in error probability due to high ISI.

Figure 5(b) shows the comparison of direct and relay-assisted transmission systems. For fair comparison, we assume same distance between NSS and NSD in both the cases. Moreover, we ignore the number of resources taken in relay-assisted system. Herein, error probability as a function of the detection threshold is plotted for both transmission systems. For this comparison, all other parameters are kept unchanged, i.e., $Nglu = 300000$, $P_u = 0.1$, and the separation between pre-synaptic and post-synaptic neurons is 20 nm. It is observed that the probability of error is minimum for

the relay-assisted link. Thus, we can conclude that by using NSR, the performance of the system can be improved.

5 Conclusion

In this paper, we considered a cooperative neuro-spike communication system where a neuro-spike relay was placed in the middle of the pre-synaptic and post-synaptic neurons. Pre-synaptic neuron act as a transmitter and post-synaptic neuron act as a receiver. The closed-form expressions for the probability of false alarm, probability of detection, and probability of error were derived for both direct and relay-assisted links. The obtained analytical expressions were also verified using Monte Carlo simulation. In the analysis, we also observed the variation of the ROC plot with a number of glutamate molecules. The variation of error probability with diffusion coefficient is also observed. It was concluded that with an increase in the diffusion coefficient of molecules, the performance of the system decreases. This is due to the increase in ISI and to overcome this issue, we should transmit a large number of molecules. The practical implementation of a cooperative neuro-spike communication system is still a challenging area. Future studies can focus on the treatment of serious health issues like loss of memory power, mood swings, self-neglect, etc.

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