Noise-delayed decay in the response of a scale-free neuronal network

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\textbf{A B S T R A C T}

Noise-delayed decay occurs when the first-spike latency of a periodically forced neuron exhibits a maximum at particular noise intensity. Here we investigate this phenomenon at the network level, in particular by considering scale-free neuronal networks, and under the realistic assumption of noise being due to the stochastic nature of voltage-gated ion channels that are embedded in the neuronal membranes. We show that noise-delayed decay can be observed at the network level, but only if the synaptic coupling strength between the neurons is weak. In case of strong coupling or in a highly interconnected population the phenomenon vanishes, thus indicating that delays in signal detection can no longer be resonantly prolonged by noise. We also find that potassium channel noise plays a more dominant role in the occurrence of noise-delayed decay than sodium channel noise, and that poisoning the neuronal membranes may weaken or intensify the phenomenon depending on targeting.

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\section{1. Introduction}

A challenging research issue in neuroscience is the understanding the neural coding mechanism in the nervous system. In the past decades, much effort has been devoted to explain how neuronal information is represented by the individual or ensemble neuronal activities \cite{1}. So far many neural encoding mechanisms have been proposed, including the rate coding \cite{2,3}, temporal coding \cite{4,5}, population coding \cite{1,6}, synchrony coding \cite{7}, transient coding \cite{8}, latency coding \cite{9,10}. Among these encoding mechanisms, latency coding is a fast and energy efficient theoretical hypothesis, assuming that neurons may perform information processing with only one spike considering the appearance time of the first spike in response to a stimulus. First-spike latency coding has been studied with experimental protocols in different neuronal structures such as somatosensory \cite{10,11}, olfactory \cite{12}, auditory \cite{13,14} and visual systems \cite{15,16}, and it has been shown that the first spike latency conveys a considerable amount of, or even more, information than those of other spikes. Latency coding has also been suggested as an underlying mechanism for the rapid response process in the nervous system of humans and animals \cite{17}.

Besides these experimental works, many theoretical and computational studies have also been performed to investigate the influence of different biophysical mechanisms shaping the first-spike latency response of neurons \cite{18–26}. In this context, it has been shown that the neuronal noise, as a ubiquitous biophysical component in the nervous system, significantly influences the first-spike latency dynamics of model neurons. Pankratova et al. \cite{18,19} analyzed the impact of noise on the response latency of FitzHugh–Nagumo and Hodgkin–Huxley (H–H) neuronal models driven by a suprathreshold periodic forcing, and obtained a non-monotonic dependence of the mean latency on the noise strength at the frequency boundaries of the suprathreshold spiking regime. More precisely, for small noise levels, spike latency does not
change very much. For moderate noise levels, however, the noise induces an increase in spike latency indicating the delay in signal detection. Finally, for too high noise levels, the system is dominated by the noise and the spike latency gets lower values than the deterministic case. The authors attributed this resonance-like behavior of the spike latency to a phenomenon called “noise delayed decay” (NDD), which was also previously described in potential systems by Matgeña et al. [27], and suggested that their results demonstrated the first example of NDD in excitable systems.

So far, although the NDD has been extensively studied, all the previous works [18,19,21,23,25] considered the subject on the level of single cell. Since the neurons in vivo are embedded in networks of active cells, a naturally arising question to be tackled is that whether the NDD is present at the level of neuronal networks, if yes, how the topological properties of the network influence the phenomenon. To our knowledge, the first attempt has been carried out by Ozer and Uzuntarla [22] to study the NDD phenomenon on the small-world neuronal network. Ozer and Uzuntarla [22] showed that increasing the number of random shortcuts within the network decreases the NDD effect for an intermediate coupling strength, indicating the network structure plays a key role by providing an additional operating regime, that is absent in the regular network. However, that attempt assumed a controlled source of noise that affects the neuron dynamics additively and, in most cases, without concerning of biological reality. However, such assumption is no longer valid in in vivo experiments in actual neural systems, where noise is mainly the result of the inherent activity of the neurons, and therefore not easily can be controlled by the experimentalist [28]. Thus, realistic noise modeling approaches are required to explain the occurrence of NDD in neuronal networks with complex biological mechanisms. From a biophysical standpoint, an important source of noise in neurons is that the stochastic dynamics of voltage-gated ion channels, i.e. random transitions between open and closed states. The fluctuations of the number of open ion channels around the corresponding mean values give rise to random ionic conductance fluctuations. Works by various groups [29–34] have shown how channel noise can modify excitability, cause spontaneous firing and result in variability in spike timing as well as interspike intervals.

To answer the aforementioned questions, we systematically investigate the NDD in a population of Hodgkin–Huxley neurons by using a realistic approach for the noise in the system. We explicitly model noise as resulting from the stochastic nature of voltage-gated ion channels embedded in the neuronal membrane. In addition, as the underlying topology of the neuron population, we construct a scale-free (SF) complex network because a power law distribution of the degree of neurons has been found applicable for the real brain networks [35]. We examine how topological features of the network, i.e. coupling strength, average connection degree, influence the NDD. By controlling the number of working ion channels in neuronal membranes, we also analyze the contributions of specific channel subunits to the occurrence of NDD in the considered system.

2. Model and methods

To effectively simulate neuronal dynamics on the scale-free networks of neurons, we used the stochastic Hodgkin–Huxley neuron model, being biophysically more realistic one. In the network, the time evolution of the membrane potential of the each coupled neuron is given by the following equation [19]

\[
C_m \frac{dV_i}{dt} = -G_{Na}(m_i, h_i)(V_i - V_{Na}) - G_K(n_i)(V_i - V_K) - G_L(V_i - V_L) + \sum_j \xi_{ij}[V_j(t) - V_i(t)] + s(t),
\]

where \(V_i\) denotes the membrane potential of the neuron \(i = 1, 2, \ldots, N\) (\(N\) is the total number of neurons within the network). \(G_{Na}, G_K\) and \(G_L\) denote sodium, potassium and leakage conductance, respectively. \(C_m = 1 \mu F/cm^2\) is the membrane capacitance. \(V_{Na} = 115\, mV, V_K = -12\, mV\) and \(V_L = 10.6\, mV\) are the reversal potentials of sodium, potassium and leakage currents, respectively. In the model, the leakage conductance is equal to \(G_L = 0.3\, mS/cm^2\), while the sodium and potassium conductance change dynamically in accordance with the following two equations:

\[
G_{Na}(m_i, h_i) = g_{Na}^{max}x_{Na}n_i^2h_i, \quad G_K(n_i) = g_K^{max}x_Kn_i^4
\]

In Eq. (2), \(g_{Na}^{max} = 120\, mScm^{-2}\) and \(g_K^{max} = 36\, mScm^{-2}\) are the maximum sodium and potassium conductance, respectively. \(x_{Na}\) and \(x_K\) are the two scaling parameters that represent the proportion of the working (non-blocked) ion channels to the overall number of sodium and potassium channels, respectively. Unless stated otherwise, we set the scaling factors \(x_{Na} = x_K = 1.0\). Here, we consider that all the neurons in the network are subjected to a strong periodic signal \(s(t) = A\sin(\omega t)\) which is supra-threshold. We set the amplitude of the periodic signal to \(A = 4\, \mu A/cm^2\) as in Pankratova et al. [19] and, set the frequency of periodic signal to \(\omega = 0.13\, ms^{-1} \approx (20\, Hz)\) which is just above the firing threshold (16 Hz) for the amplitude of 4 \(\mu A/cm^2\) [19]. \(\xi_{ij}\) denotes coupling strength between neurons \(i\) and \(j\). If neurons \(i\) and \(j\) are connected then \(\xi_{ij} = \epsilon_j\), otherwise equal to zero. \(m\) and \(h\) denote the activation and inactivation of gating variables for the sodium channel, respectively, whereas the potassium channel includes an activation gating variable \(n\).

In the HH model, dynamics of gating variables change over time as a function of membrane potential deterministically [36]. To take into account the stochastic ion channel dynamics, we use the Fox’s algorithm presented in [37] due to its widespread use and computational efficiency. In the Fox’s algorithm, the gating variables are described by the following Langevin generalization [37]:

\[
\frac{dy}{dt} = \phi_y(1 - y_i) - \beta_y y_i + \eta_y(t), \quad y = m, n, h
\]

where \(\phi_y\) and \(\beta_y\) are the voltage-dependent rate functions for the gating variable \(y_i\), defined in units of ms [36,37]. The stochasticity \(\eta_y\), occurring due to the random opening and closing of individual ion channels, is modeled as an independent zero mean Gaussian white noise whose autocorrelation functions are given as follows [37]:
\[
\langle z_m(t)z_m(t') \rangle = \frac{2\alpha_m h_m}{N_{Na}x_{Na}(z_{m} + \beta_{m})} \delta(t - t') \tag{4a}
\]
\[
\langle z_h(t)z_h(t') \rangle = \frac{2\alpha_h h_h}{N_{K}x_{K}(z_{h} + \beta_{h})} \delta(t - t') \tag{4b}
\]
\[
\langle z_a(t)z_a(t') \rangle = \frac{2\alpha_a h_a}{N_{Na}x_{Na}(z_{a} + \beta_{a})} \delta(t - t') \tag{4c}
\]
where \(N_{Na}\) and \(N_{K}\) represent the total numbers of sodium and potassium channels in a given cell membrane area, respectively. The total channel numbers are calculated as \(N_{Na,K} = \rho_{Na,K} \cdot (\text{CellMembraneArea})\). The number of channels per square micrometer of membrane area is \(\rho_{Na} = 60\) for sodium and \(\rho_{K} = 18\) for potassium, respectively [21–23,36,37]. One can easily obtain from Eq. (4) that the intensity of the channel noise depends on the cell membrane area for a given channel density. When the cell size is large enough, stochastic effects of the channel noise become negligible and the collective dynamics approach the deterministic description. However, when the cell size is small, stochastic dynamics of the individual channels have significant effects on the membrane dynamics [23].

Following the procedure in [38,39], we construct a scale-free neuronal network which comprises \(N = 200\) identical H–H neurons. Then, we define the latency to first spike for each neuron as the time of the first upward crossing of the membrane potential past a fixed detection threshold value of 20 mV, which equals to the value used in [19,21,22].

To analyze the response dynamics of the network, we will refer to the mean value of the appearance time of the first spikes for each neuron as the mean response time (MRT). Then, the mean response time (MRT) of the network is computed as follows:

\[
\text{MRT} = \left\langle \frac{1}{N} \sum_{i=1}^{N} t_i \right\rangle \tag{5}
\]

where \(t_i\) is the appearance time of the first spike for the neuron \(i\) within the network. We also compute the standard deviation of the appearance time of the first spike, or temporal jitter as follows:

\[
\sigma = \sqrt{\frac{1}{N} \sum_{i=1}^{N} t_i^2 - \left( \frac{1}{N} \sum_{i=1}^{N} t_i \right)^2} \tag{6}
\]

In Eqs. (5) and (6), \(\langle \cdot \rangle\) represent the averaging process over 50 different network realizations. For each network realizations, we assumed that the initial conditions are the same for all neurons within the network as in [19,21,22].

### 3. Results and discussions

Having established that the NDD phenomenon, as previously described in the literature, occurs on the level of a single cell, we now investigate the phenomenon on the level of network. We first examine whether the channel noise is able to induce NDD in a scale-free neural network and, how the key topological properties of the network influence the first-spike latency dynamics of the population in response to a suprathreshold periodic driving. To do so, we set the average connection degree of neurons \(k_{avg} = 4\), and compute the mean response time (MRT) and its standard deviations, henceforth called jitter, as a function of the cell membrane area for five different values of the synaptic coupling strength. Fig. 1a and b feature the obtained results. When the cell membrane area is large, regardless of the level of synaptic coupling strength within the neurons, it is seen that the MRT of the network is very close to the first-spike timing of deterministic single neuron. This is due to the very large number of ion channels embedded in neuronal membranes which impart very weak stochasticity to neurons' dynamics. Thus, all the neurons in the network are locked to the external stimulus and, act synchronously resulting in a nearly zero synaptic current. However, with the decrease in cell size from large values, the influence of intrinsic channel noise and synaptic coupling strength begin to arise. More precisely, for most of the range of coupling strengths of interest in Fig. 1, MRT and the jitter increase substantially as the cell area decreases.
size decreases up to a moderate value. After reaching some maximum, they start to decrease with further decrease in cell size. This increase for a particular range of cell size (noise strength) and then later decrease in MRT indicates a clear signature of channel noise induced NDD in our considered system. Importantly, it is also seen in Fig. 1 that the synaptic coupling strength acts as a scaling factor in determining the NDD level in the network: increased values of $\varepsilon$ decrease the maximums of MRT and jitter. This means that delay in external signal detection can be reduced or minimized in a strongly coupled neuron population, as well as the temporal spiking precision can be enhanced. Indeed, for very large values of $\varepsilon$ (see $\varepsilon \geq 0.04$ curves), MRT and jitter do not change very much for almost full span of membrane area. Similar influences of $\varepsilon$ on NDD were also reported in our previous work [22] where the underlying network topology for the population was a small-world network structure. Thus, we suggest that the high synaptic coupling strength in a neuron population reduces the NDD effect regardless of the network topology.

To gain more insight into the dependence of NDD on scale-free network’s topological features, we further investigate the influence of average connection degree of neurons on NDD. We carried out this investigation because $k_{\text{avg}}$ determines the density of interneuronal links within the network, and it is accordingly an important parameter. For this purpose, we fix the synaptic coupling strength $\varepsilon = 0.01$ so as to lead NDD in MRT against cell membrane area, then calculate the MRT and jitter for different values of $k_{\text{avg}}$. Fig. 2 illustrates the obtained results. It is seen that the impact of increasing $k_{\text{avg}}$ is comparable to the impact of increasing $\varepsilon$ when compared to the MRT and jitter presented in Fig. 1. Namely, although there exists a constant coupling strength within the neurons in the network, increased values of $k_{\text{avg}}$ decreases the MRT and jitter indicating that delay in external signal detection due to the intrinsic noise can be reduced or even completely removed (for $k_{\text{avg}} = 12$, data not shown here), and temporal spiking precision can be enhanced in a scale-free network with high interneuronal link density.

Following above investigations, where we showed that NDD might occur in neural systems as a consequence of ion channel stochasticity and, the network features might play a constructive role in minimizing this effect, we investigate now the relative contributions of different channel subunits on the occurrence of this phenomenon in our considered system. To understand the role of specific ion channels, we constructed a hybrid stochastic HH system for each neuron in the network in which one of the channel populations is stochastic and the other deterministic, and simulated the system for three different cases: (1) Na channels stochastic, K channels deterministic, (2) K channels stochastic, Na channels deterministic, (3) both Na and K channels are stochastic. Notably, we considered a scale-free network configuration with $\varepsilon = 0.01$ and $k_{\text{avg}} = 4$ so as to work in a regime where the NDD is present. The obtained results are shown in Fig. 3. It is seen that regardless of the source of stochasticity from ion channels, NDD still appears as a function of cell membrane area. However, when only the Na channels are stochastic, the maximums in MRT and jitter get lower values, and furthermore, NDD occurs at smaller cell sizes compared to the fully stochastic model. This is due to the difference in total number of sodium and potassium channels for a given size, that is the Na channel density for a given membrane patch area is 3.3 times larger than the K channel density ($p_{\text{Na}}/p_{\text{K}} \approx 3.3$). Based on the previous statistical models on ion channel populations, the size of open channel fluctuations is proportional to $1/\sqrt{N_{\text{ch}}}$, favoring that ion channels with large number of population $N_{\text{ch}}$ impart less fluctuation to the related ionic conductance [34]. Thus, Na channel stochasticity alone is able to provide the required conductance fluctuations at smaller cell sizes for the occurrence of NDD effect. On the other hand, when only the K channels gated stochastically, we see that MRT and jitter curves closely follow the trend of fully stochastic model, indicating that K channels are more dominant than Na channels in determining the NDD in the considered system. This might be due to the difference in timescales of these two types of channels, where the K channels work with slower gating kinetics than the Na channels. Because the membrane capacitance acts as a low pass filter, noise from channels with faster kinetics (Na channels) is more attenuated than the noise from channels with slower kinetics (K channels) [40,41]. Our findings on the contributions of different subunits to NDD are consistent with the previous works reporting that the K channel stochasticity accounts $\approx 75\%$ of the total channel noise [29,41,42] and it is the dominant effect in determining the spike-time reliability [33,42].

Finally, we examine how the number of working ion channels for a given membrane patch influences the NDD phenomenon. This investigation is carried out because although the intensity of channel noise is related with the total number of ion channels, its actual impact is determined by the number of channels that are open near the threshold for spike firing. Previously, Schneidman et al. [33] reported that there is a short distance in terms of the number of open channels between spiking and non-

![Fig. 2.](image)

The dependence of response time statistics on the average degree obtained by a fixed value of $\varepsilon = 0.01$. The mean response time and the jitter of response latencies (inset) as a function of cell membrane area.
spiking stable states, and that fluctuations due to only a few channels are responsible for the transition between these two stable states. Therefore, controlling the number of working ion channels for a given membrane patch is of great importance to understand the impact of different channel subunits on neuronal dynamics, particularly here on NDD. In this context, some toxins such as tetraethylammonium (TEA), tetradotoxin (TTX) and saxitoxin (STX) are widely used in experiments to block or reduce the number of specific ion channels [43,44]. Here, by using the fully stochastic model for a network configuration by $e = 0.01$ and $k_{\text{avg}} = 4$, we vary the density of one channel type (either $x_{\text{Na}}$ or $x_{\text{K}}$) while keeping the other equal to one. Obtained results are presented in Fig. 4a for potassium and in Fig. 4b for sodium ion channel block. In the case of potassium channel block, MRT of the network decreases gradually with the reduction of working potassium channels, indicating that signal detection performance of the network is improved by potassium channel block. Moreover, although the NDD effect on MRT still appears as a function of cell size for all values of $x_{\text{K}}$, the cell size (noise intensity) at which the maximal MRT occurs shifts towards to the right. It is also seen in Fig. 4a that NDD effect also tends to disappear with the decrease in $x_{\text{K}}$. Notably, we did not consider $x_{\text{K}} < 0.5$ because the response trend of the system does not qualitatively change very much. On the other hand, in the case of sodium channel block, a reduction in working sodium channels influences the MRT oppositely compared to the case of potassium channel block. Namely, decreasing the $x_{\text{Na}}$ always increases the MRT, and the NDD effect first occurs for $x_{\text{Na}} = 1, 0.95$, then disappears for $x_{\text{Na}} = 0.9$, 0.85 and, reoccurs for $x_{\text{Na}} = 0.8$. We did not consider the value of $x_{\text{Na}} < 0.8$, because the applied stimulus is subthreshold for these blocking levels, meaning that it is alone insufficient to initiate spike in deterministic conditions. As a result, the sodium channel block degrades the signal detection capabilities of the scale-free networks as shown in Fig. 4b.

The above underlying effects of the channel blocking on NDD phenomenon can be explained as follows. In potassium channel block case, two main reasons are effective on the emergence of such a result. One of them is that

![Fig. 3](image1.png)

**Fig. 3.** Relative contributions of sodium and potassium channels noise to the occurrence of NDD in a scale-free network obtained by fixed values of $e = 0.01$ and $k_{\text{avg}} = 4$. (a) The mean response time vs. cell membrane area for three different sources of channel noise. (b) The jitter of the response latencies as a function of the cell membrane area for three different sources of channel noise.

![Fig. 4](image2.png)

**Fig. 4.** The dependence of mean response time on cell membrane area in the case of channel blocking. (a) Different levels of potassium channel block. (b) Different levels of sodium channel block.
the reduction of the number of working potassium channels (increasing the potassium channel noise) increases the excitability of the each neuron in the network [45], and causes a diminishment in the threshold level for spiking. Consequently, neurons in the network fire more rapidly than in the unblocked case, thus, the MRT of the network declines. The other reason is that decreasing the \( \kappa \) changes the frequency range of the suprathreshold current stimulus with a fixed amplitude of 4 \( \mu A/cm^2 \) and, causes a shift of the lower boundary to the smaller frequency (for \( \kappa = 0.8 \) the lower boundary is 6 Hz, data not shown here) for the suprathreshold spiking regime. Therefore, the suprathreshold current signal with the amplitude of 4 \( \mu A/cm^2 \) and the frequency of \( \omega = 0.13 \text{ ms}^{-1} \) becomes increasingly suprathreshold, and thus the NDD phenomenon, being more pronounced at the frequency boundaries, weakens and needs more noise (thus small membrane patch areas) to occur.

On the other hand, in the case of sodium channel block, decreasing the number of working sodium channels increases the activation barrier for spiking as stated in [45]. At the same time, the suprathreshold periodic stimulus approaches the subthreshold regime and, the sensitivity of the first spike time to the noise increases [23]. For example, in a scale-free network of deterministic HH neurons, the first spike appearance times in response to \( s(t) \) are 9.14, 11.16, 52.62, 53.44, 55.12 ms for \( \kappa = 1, 0.95, 0.9, 0.85, 0.8 \), respectively. In the considered stochastic system here, the MRT of the network increases when compared to the unblocked case due to the increase in spiking barrier. As for the NDD, for both unblocked case and \( \kappa = 0.95 \), the neurons in the network fire in the ascending part of the first cycle of the suprathreshold signal in approximately deterministic conditions (large membrane sizes >100 \( \mu m^2 \)). Then, an intermediate range of the channel noise delays the occurrence of firing events to the later cycles. However, in the presence of very strong channel noise (membrane area <6 \( \mu m^2 \)), neurons fire before the deterministic conditions because the channel noise dominates the neuronal dynamics. As a result, NDD is not observable. For \( \kappa = 0.9 \) and \( \kappa = 0.85 \), all neurons in the network fire in the second cycle of the suprathreshold signal at large membrane sizes. In these two cases, neurons are divided into several groups with the increase in channel noise. Some groups of neurons fire at the first or second cycle of the stimulus, the other groups fire in the latter cycles than the second cycle of the stimulus. As the number of neurons in the former group is greater than those of the latter group, the average MRT declines monotonously with the increasing of channel noise, and thus the NDD is not observable for these sodium block levels. For \( \kappa = 0.8 \), due to the increasing sensitivity of the first spike appearance time to the noise, the NDD phenomenon reappears at an intermediate range of channel noise.

4. Summary

In the present paper, we investigated how the ion channel noise, stemming from the probabilistic nature of the ion channels, affects the first spike latency in response to a suprathreshold stimulus in a scale-free network of stochastic HH neurons. We have obtained that an intermediate range of channel noise degrades the signal detection capability of the scale-free network and causes the emergence of the NDD effect. Then, we analyze the effects of network parameters, which are coupling strength and average degree, on the NDD phenomenon. We have arrived at the result that the network parameters can be used for the enhancement of the signal detection capability of scale-free network by decreasing the MRT and the jitter of the first spike latency. Second, we have studied which channel noise type, sodium or potassium channel noise, plays decisive role in determining the bell-shaped dependence of the NDD on channel noise. We have shown that the potassium channel noise is generally a dominant factor on the occurrence of the NDD phenomenon in large and intermediate cell sizes, but, as depicted in MRT and jitter plots in Fig. 3a and b, the sodium channel noise is more determinative in small cell sizes. Finally, we analyze the effects of the number of the working ion channels, which are open near the threshold, by blocking one channel type while the other channel type is unblocked, on the first spike timing due to the importance stated in Results and discussions section. The results presented in Fig. 4a and b have revealed that, with the increasing of potassium channel block, MRT of the scale-free network decreases and, thus the signal detection capability of the network increases. Moreover, with the reduction of working potassium channels, first spike latency dynamics are dominated by the sodium channels, and thus, the NDD effect appears at small membrane sizes with small amplitudes. On the other hand, with the reduction of working sodium channels, in contrast to the potassium channel block, MRT of the network increases and signal detection capability of the network decreases. In addition, the NDD effect disappears up to \( \kappa = 0.8 \). For \( \kappa = 0.8 \), channel noise suppresses the stimulus effects and the NDD effect reappears.

References