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Noise influence on spike activation in a Hindmarsh–Rose small-world neural network

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Abstract

We studied the role of noise in neural networks, especially focusing on its relation to the propagation of spike activity in a small sized system. We set up a source of information using a single neuron that is constantly spiking. This element called *initiator* x_o feeds spikes to the rest of the network that is initially quiescent and subsequently reacts with vigorous spiking after a transitional period of time. We found that noise quickly suppresses the initiator's influence and favors spontaneous spike activity and, using a decibel representation of noise intensity, we established a linear relationship between noise amplitude and the interval from the initiator's first spike and the rest of the network activation. We studied the same process with networks of different sizes (number of neurons) and found that the initiator x_o has a measurable influence on small networks, but as the network grows in size, spontaneous spiking emerges disrupting its effects on networks of more than about $N = 100$ neurons. This suggests that the mechanism of internal noise generation allows information transmission within a small neural neighborhood, but decays for bigger network domains. We also analyzed the Fourier spectrum of the whole network membrane potential and verified that noise provokes the reduction of main θ and α peaks before transitioning into chaotic spiking. However, network size does not reproduce a similar phenomena; instead we recorded a reduction in peaks' amplitude, a better sharpness and definition of Fourier peaks, but not the evident degeneration to chaos observed with increasing external noise. This work aims to contribute to the understanding of the fundamental mechanisms of propagation of spontaneous spiking in neural networks and gives a quantitative assessment of how noise can be used to control and modulate this phenomenon in Hindmarsh−Rose (H−R) neural networks.

Keywords: neural networks, noise, stochastic processes, small-world networks, discrete Fourier analysis

(Some figures may appear in colour only in the online journal)

1. Introduction

The control and flow of information in the brain are fundamental to the understanding of high-level processes like attention, visual perception and other complex functions [1, 2]. Despite enormous system complexity, it has been found that information can be sourced by a single neuron even in major sensory pathways [3]. These distinct units can trigger a chain of events that contribute to complex perception episodes or high-level motor responses. This is known to happen for example with feature detector neurons [4] in lower animals, and similar phenomena are observed in mammals' neurons which hold a higher hierarchical position in the visual system—those that respond to complex patterns and activate elaborate responses [5].

Despite the great volume of experimental facts that point toward noise as a contributing factor in signal transmission in brains [6–8] or generally in non-linear networks [8], we do not have yet a universally accepted theoretical framework to quantitatively evaluate the effects of noise on neurons' operations and the higher level network functions.

Here we want to contribute to this problem with a study in which we emulate a triggering event in the brain through a single active neuron which we call *initiator* x_o; in controlled noise conditions, we study the flow of spike activity along the network to evaluate the role of noise amplitude on the signal propagation. In the first part of this study, we elucidated in a quantitative manner the role of noise and we found for the first time a linear relationship between the amplitude expressed in decibels and the delay interval between the first spike of initiator x_o and network activation. Moreover, studying the dependence with network size, we found that as the network grows in dimension, the spiking activity starts earlier until the role of the initiator is completely suppressed beyond about $N = 100$ neurons. In the second part of the research, we characterize the network especially focusing on its spectral characteristics, and most particularly on its frequency dependence with noise and size. It is found that high levels of noise introduce chaos in the network and that, again, the network size seems to replace the function of external noise for simulations with a constant noise level and variable network sizes.

2. Models and methods

We used a Newman−Watts small-world network to simulate the neural network. A realistic neural network is neither regular nor completely random [9] and this network has both deterministic and random properties [10]. The simulation of relatively small artificial networks can be a good metaphor for real brains, for example, in 2003 Izhikevich demonstrated that a network of simulated spiking neurons exhibited collective waves and frequencies [11] in a range similar to the human brain.

To simulate the neurons we used the Hindmarsh−Rose (H−R) model [12, 13], changing the intensity of noise and the network size to elucidate the role of spiking frequencies in function of various parameters. Even though the precise arrangement of neural connections in real brains is not known, small-world network models are widely used to simulate known

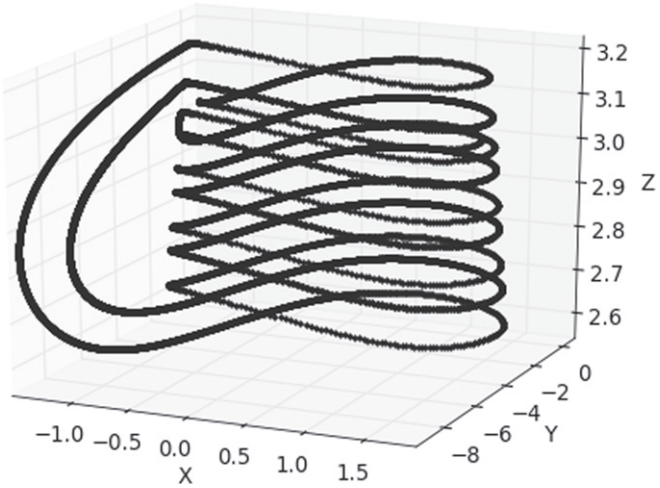


Figure 1. Simulation of a H−R model for a single neuron not affected by noise or connections. This shows the dynamical behavior of the membrane potential. The variable *x(t)* represents the difference of voltage between extracellular and intracellular potentials. Spiking variable *y(t)* instead describes the rate of change of the fast ion channels (sodium ion, potassium ion, etc) and *z(t)* has similar meaning for the slow ion channels (calcium).

statistical properties of the brain's neural connections, reproducing a high clustering coefficient and a low shortest path [14–17]. The network's random connection probability of the small-world structure has been kept to $p = 0.4$ as a reasonable value with enough random connections to induce a fast diffusion of spikes, but still small enough to represent real biological systems. This network structure and p values are considered reasonable in numerous literature studies, for example Bassett and Bullmore's work [15] where regions of the human brain are found to have clustering coefficients as low as 0.14 equivalent to $p > 0.4$, or also Zheng and Lu [18] and Ozer *et al*'s works [19] which find values of $0.1 < p < 0.6$ in small-world neural networks. To measure the global behavior of the network we monitored the integral of the total membrane potential. This is considered to be analogous to the electroencephalogram in a real brain. By means of Fourier analysis, we found that this collective signal has distinct frequency peaks which are compatible with values observed in biological systems. This fact is not trivial since neuronal activities of a complex network are very difficult to predict analytically.

The H−R [20] system is characterized by three independent variables that represent the membrane potential and the two ion channel currents. A single neuron model is described by the following differential equation:

$$\begin{cases} \dot{x} = y - ax^3 + bx^2 - z + I \\ \dot{y} = c - \mathrm{d}x^2 - y \\ \dot{z} = r[s(x - \chi) - z]. \end{cases} \tag{1}$$

In this equation, x is time dependent and represents the membrane potential, whereas y and z are often called the spiking and bursting variables, respectively [21]. These variables are all expressed in arbitrary units and do not have a direct translation in biologically-realistic parameters [20].

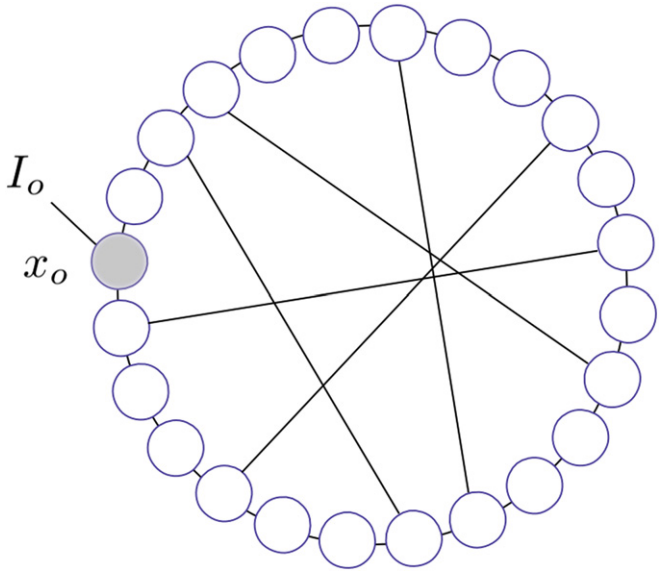


Figure 2. A sketch of the network structure. Each neuron is represented by a circle and simulated by the H−R model. The lines represent neural connections to neighbors' neurons. The random connections that cross the circle are those due to the p parameter of the small-world structure. The grayed neuron indicated by the symbol x_o is a special *initiator* that has a constant external current input I_o and it is used to stimulate the whole network and to study the diffusion of spikes in the system in the presence of noise. In our simulations the connections are changed randomly at every run, however the small-world structural parameters are maintained constant. The cross-connections parameter p is kept at $p = 0.4$ (for image clarity in this sketch p is lower). In some experiments the number of neurons is also varied.

Figure 1 shows a plot of the three variable phase cycle that induces the spike. The choice of the eight parameters in the model results in the kind of neuron simulated. Here we fixed them as $a = 3$, $b = 3$, $c = 1$, $d = 5$, $s = 4$, $r = 0.00$, $\chi = -1.6$ determined in order to produce neuronal-bursting dynamics comparable to realistic neurons of the 'chattering' type. The *initiator* neuron x_o introduced above has a continuous input stimulus of $I = 3$ (a.u.) and starts at $t = 0$ with the initial values $x(0) = 0.3$, $y(0) = 0.3$, $z(0) = 3$. Other authors [22] use similar values of input currents, ranging from about $I = 2$ to $I = 10$. Other neurons' initial current values are set to zero. The neuron is called the *initiator* because of its role as the single source of information in the network. In other terms, the *initiator* x_o is the only neuron that provides stimulated spikes and these are thought of as the single source of information in the network. That is, in the absence of external noise, if other neurons are spiking it is because directly or indirectly they receive the information to do so from x_o. Our study investigates throughout the role of noise in this context. The entire network is connected as a small-world system, with the exception of x_o which receives input from the neighboring neurons and stimulates accordingly the network structure, see figure 2 for a sketch of the configuration used.

The integration of the differential equation (1) is done with an Euler method, a time consuming but straightforward integration method. The time step is set to $\Delta t = 0.01$ ms [23] and has been verified to not introduce instabilities (the slight increase of Δt does not provoke appreciable variation in the network response).

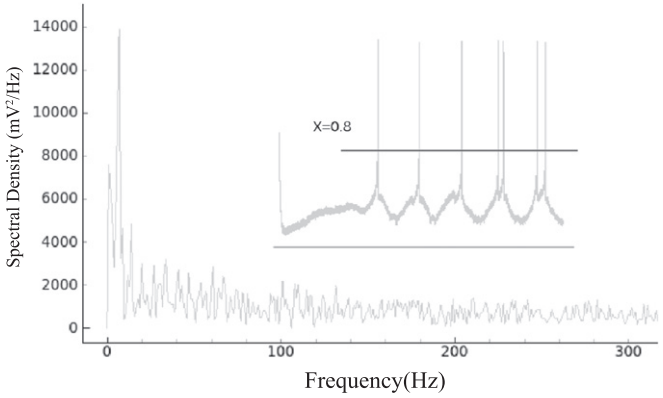


Figure 3. The Fourier transformation of the membrane signal of a single neuron inserted in a $p = 0.4$ network of $N = 10$ and with a SNR of $dB = 40$. This is an example of the frequency signature of a single neuronal response. The inset shows the actual spike plot where the Fourier transformation was calculated from; the horizontal axis represents 1000 msec of time. The bar at $x = 0.8$ indicates the threshold used to separate the baseline activity from the action potential. The neuron is chosen at random and it is not connected to the initiator neuron x_o.

Figure 3 shows the Fourier transformation of the membrane signal of a single neuron inserted in a $p = 0.4$ network. The network itself is intentionally chosen to be smaller ($N = 10$) and with a minimal noise level (signal-to-noise ratio (SNR) $dB = 40$) in order to be an example of the frequency signature of the neuron response. The neuron position is chosen at random far from the active x_o. Interestingly, we can observe the emergence of peaks at 10 Hz; these frequencies in the averages membrane potential can be thought as analogous to α waves in real brains. This shows biological plausibility in simulations as already established for example by Izhikevich *et al* and Palva and Palva [24, 25].

To generate the network, firstly, we create a regular network with a ring over the N vertices. Each vertex stands for a neuron. Then, every vertex in the ring is connected with its nearest neighbors at both sides. In all our tests, $k = 1$; this means that each neuron is connected with two neighbors, one on its left and the other on its right. We say that the network is of dimension $k = 1$ (if $k = 2$ each neuron has four neighbors, two on the left and two on the right). Starting from this regular network structure, we create random shortcuts by adding connections pointing to other neurons chosen at random with probability p ($p = 0.4$ in our case). In this way there are on average Nkp shortcuts in the network. Each vertex v_i ($i \in \{1, 2, 3 \ldots N\}$) can be connected with any other with probability p. For vertex v_i, a uniform distribution random number in the range zero to one is generated and compared with p. If the number is lower than p, we selected another vertex v_j ($i \neq j$) randomly and add a shortcut between v_i and v_j. A similar test is done to the adjacent neuron until all neurons are examined (from v_1 to v_N). Neurons have no connections with themselves and two neurons can be coupled by a unique segment; a single neuron can receive more than one connection from different neurons [26].

A good representation of biologically-plausible noise is controversial, however since the central limit theorem guarantees that a good number of arbitrary distributed sources converge to a Gaussian signal, we opted for this type of noise [27–29]. The intensity is modulated using different values of SNR defined in decibels as $dB = 10 \log_{10}\left(\frac{A_s}{A_n}\right)^2$, where A_s is the amplitude

of the signal and A_n that of the noise. We use this definition because it is standard and logarithmic, however we have to empathize that bigger values of *dB* mean a less noisy network.

The random variable is generated with this formula:

$$\phi(X,\,\mu,\,\sigma) = \frac{1}{\sigma\sqrt{2\pi}} e^{\left(\frac{-(X-\mu)^2}{2\sigma^2}\right)}. \tag{2}$$

Here X is a flat pseudo-random value generated internally by the numerical library *numpy* [30, 31], μ is the mean value around which the Gaussian is centered and σ is the corresponding standard deviation. In our experiment, μ was fixed as 0 and σ as 1 [32, 33]. We use

$$\begin{aligned} S &= \frac{1}{N}\Sigma_i x_i, \\ \eta &= \sqrt{\frac{S}{10^{dB/10}}}, \\ \varepsilon &= \eta\phi(X,\,\mu,\,\sigma). \end{aligned} \tag{3}$$

N is the total number of neurons, x_i the membrane potential of neuron i, η the noise amplitude and ε the final Gaussian noise random variable. It results in being distributed along a Gaussian curve, with an intensity proportional to η that contains the decibel parameter that is varied in the tests [34]. The intensity of noise is calculated at each time loop, so several neurons receive the same noise intensity for the same time step. This is an approximation of a realistic network where noise does not have spatial specificity.

The differential equation that describes the time evolution of a single neuron x_i in the system is:

$$\begin{cases} \dot{x}_i = y_i - ax_i^3 + bx_i^2 - z_i + \varepsilon_i + g_i \sum_{j=1}^{N} a_{ij}(x_i - x_j) + I_o \\ \dot{y}_i = c - \mathrm{d}x_i^2 - y_i \\ \dot{z}_i = r\,[s(x_i - \chi) - z_i]. \end{cases} \tag{4}$$

This formula is equivalent to equation (1) but extended for a system of N neurons; variables and parameters have the same meaning explained previously. The index i represents each of the N neurons in the network. The parameter ε_i is the Gaussian noise of neuron i, updated at each time loop. To compute the input current that each neuron receives, we calculate $g_i\Sigma_{j=1}^{N} a_{ij}(x_i - x_j)$. The sum is mediated by an adjacency matrix a_{ij} that stores the connection between the vertices. In it, a_{ij} is 1 if there is a connection between the neurons i and j, otherwise it is 0. x_i and x_j are the membrane potential of neurons i and j. g_i is the coupling strength [35, 36] that is normalized to the number of connections of neuron i. For the integration of this equation, we also used Euler method. In this way:

$$g_i = \frac{1}{\sum_{j=1}^{N} a_{ij}}. \tag{5}$$

In this formula, N is the number of neurons in the network. If we do not impose this normalization, the network will have an unbalanced influence of signals coming from the neighbors' neurons depending on the number of connections [37, 38].

As stated above, in our simulation a neuron x_o receives a specific continuous current stimulus I_o that is set to a value of 3 (a.u.) in all our tests. So, in the first equation of system (4) if $x_i \equiv x_o$, $I_o = 3$; in all other cases $I_o = 0$.

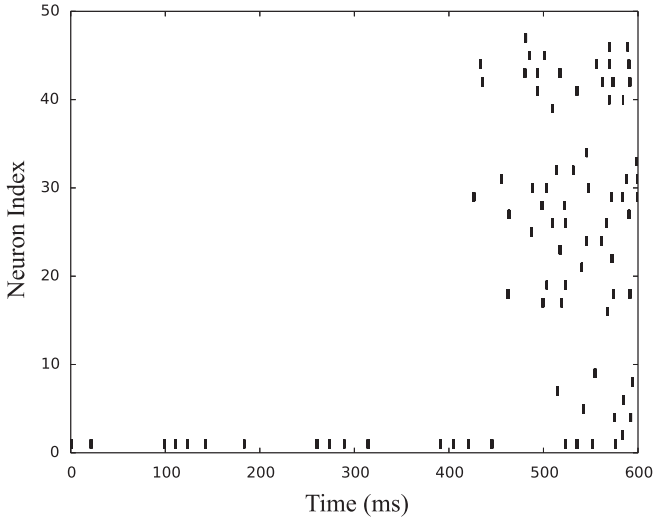


Figure 4. The spike map of a noiseless small-world network of 48 H−R neurons; a sketch of it is outlined in figure 2. The neuron indicated as number 0 is a special one that is connected to an external signal of $I_o = 3$ (a.u.) constant input. It functions as an *initiator* and after a delay of time, provokes the spiking of the whole network. The same experiment reproduced with identical parameters but with no external input ($I_o = 0$), produced no spiking anywhere in the network (blank result not shown).

3. Results

We verified that without the contribution of the special initiator neuron x_o and neuronal noise, the network exhibits no spiking activity and membrane signals settle to a baseline value. However, we verified that with an increasing noise level, the network indeed starts spontaneous spiking, even without any neuronal input.

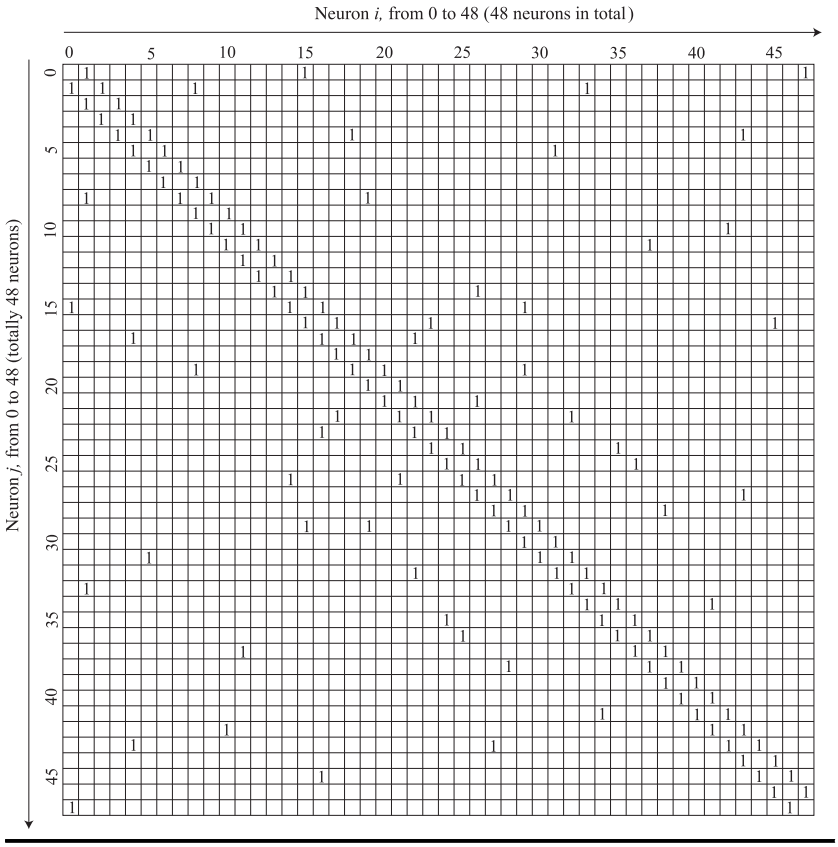
On the other hand, if neuron x_o receives a constant input I_o it becomes active and drives its connected neighbors to spike, and these will induce spiking on their neighbors. The information will propagate until the whole network will be spiking in a random but stable manner.

In the following tests, corresponding to figures from 4 to 7, a network of $N = 48$ neurons was used with connections as in table 1; i and j are the index of neurons. Value 1 in each grid indicates that neuron i is wired with neuron j ($a_{ij} = 1$). An empty grid means there is no connection between neuron i and neuron j ($a_{ij} = 0$).

The noiseless response of such a system is shown in the spike map of figure 4 . This map is generated using an arbitrary threshold to separate the basal random signal from the spikes. After a few tests to evaluate the membrane potential range, the threshold was chosen as $x = 0.8$, see inset of figure 3. When the membrane potential exceeds this value, we assume that the neuron is firing and plot the mark that is shown in figure 4 and the following spike maps. To evaluate the behavior of the network as a whole, we integrate the membrane potential for the entire population of neurons. We use this equation to calculate the parameter $E(t)$:

$$E(t) = \frac{\sum_{i=1}^{N} x_i(t)}{N}. \tag{6}$$

Table 1. An example of network connections. Values of a_{ij} represent the connection strength within the 48 neurons small-world network. The dimension of the network k is 1 and the shortcut probability p is 0.4. Connection strength can be only of value 1 (connected) or zero (not connected). Zero values are not shown for clarity). According to this table, there are in total 60 edges and 22 shortcuts in this network.



In figure 5 we show the output of the network measured as the average membrane potential calculated with equation (6) for a network of 48 neurons; $p = 0.4$ and $k = 1$. Clearly as the noise increases, the initial transitional behavior gets shorter and shorter. This suggests two things: that noise influences the average baseline of neuronal response and that it also affects the spike rate of the neuronal system.

Figure 6 shows the raster plot for a neuron network with 48 neurons; $k = 1$ and $p = 0.4$ as before, for different noise levels. Again, the initiator neuron x_o is spiking since the beginning, whereas the rest of the network is quiescent and responds with a variable delay that depends on noise level.

The SNR strongly influences the initiation of secondary spiking along the network. Calling S_s the time interval by which secondary spikes are activated, we can interpolate our results with a liner model

$$S_s = \delta\varepsilon + \alpha.$$

where $\delta = 14$ msec per decibel, ε is the noise level in dB and α is an offset, and the linear regression coefficient is better than 0.9. The influence of the initiator x_0 is negligible and the

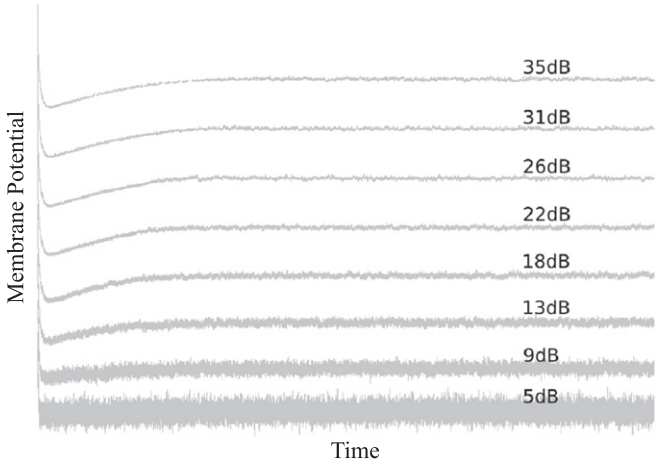


Figure 5. The behavior of the neuron population membrane potential in function of noise level. Noise seems to help the diffusion of spikes along the network. The horizontal axis spans over 1000 msec of simulation calculated in 100 000 steps of 0.01 ms each. The vertical axis is the voltage in arbitrary units. Each simulation is taken on a network of $N = 48$ neurons and averaged over eight different experiments done with same parameters but different random generator seeds. As the SNR grows (less external noise) the initial transition time gets longer indicating a worse diffusion of information across the network.

linear dependence is clear. See figure 7 for a graphical representation of this with a test of several levels of noise. This formula cannot have a general meaning because it is bound to this specific network, however it shows a quantitative relation to modulate the transfer of information with noise intensity in this case.

A network with identical parameters as above was tested in a similar way but changing the size of the network instead of the noise (noise level was kept constant at 20 dB). As shown in figure 8 for small network sizes, the initiator neuron x_o makes the spread of spikes quicker, but it does not seem to strongly influence the transitional delay period after the network grows more than about $N = 100$ neurons.

To analyze the influence of noise to the collective frequencies of the network, we studied the Fourier spectrum of the variable E in formula (6). The Fourier spectrum peaks, if present, are very important for recognizing rhythms and regularities in the entire network that mimic the biological phenomena of slow and fast waves in real brains.

The Fourier transformation presents evident peaks at a strong SNR (top curve, $dB = 35$), whereas the more prominent peaks at α and β seem to decay with increasing noise [24], see figure 9. The Fourier transform data are calculated over a simulation period of 1000 msec, cutting off the first 300 msec to avoid including slow frequencies due to the initial transitional phase discussed above. Interestingly the prominent β peak seems to shift to the right with noise intensity; a very weak effect that is, however, difficult to prove with mathematical means. We studied the total network power average among five different frequency bands, θ, α, β, γ_1 and γ_2. We calculated the Fourier spectrum of a 1000 millisecond simulation as above, then we summed up the power of those five frequency bands with this formula

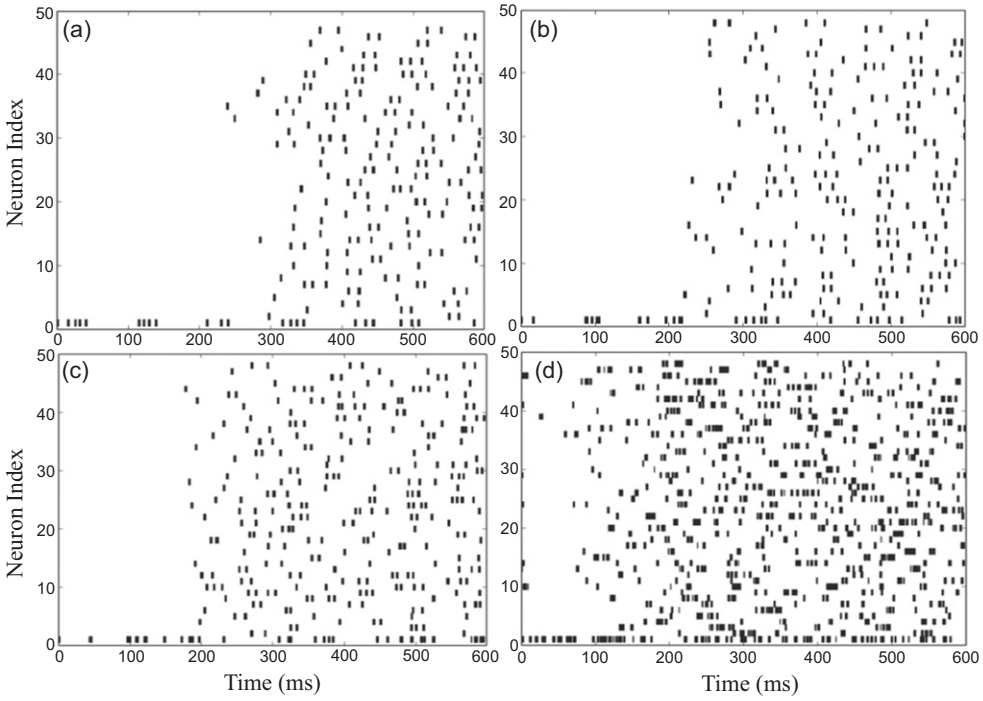


Figure 6. The spike maps of a network with $N = 48$ neurons in different noise conditions. The SNR in each plot is (a) $dB = 35$, (b) $dB = 31$, (c) $dB = 26$ and (d) $dB = 18$. The SNR strongly influences the initiation of regular spiking along the network. On the vertical axis we have the neuron number; the neuron indexed as zero is the special *initiator* neuron x_o. Notice that a better SNR means less external noise. Intermediate dB values were calculated but are not shown.

$$\theta = \frac{1}{N_\theta}\sum_{f=4}^{7}\Psi(f)\mathrm{d}f,$$
$$\alpha = \frac{1}{N_\alpha}\sum_{f=7}^{14}\Psi(f)\mathrm{d}f,$$
$$\beta = \frac{1}{N_\beta}\sum_{f=14}^{30}\Psi(f)\mathrm{d}f,$$
$$\gamma_1 = \frac{1}{N_{\gamma_1}}\sum_{f=30}^{40}\Psi(f)\mathrm{d}f,$$
$$\gamma_2 = \frac{1}{N_{\gamma_2}}\sum_{f=50}^{70}\Psi(f)\mathrm{d}f. \tag{7}$$

Here Ψ represents the vector that contains the module of the fast Fourier transformation array. The elements corresponding to frequencies within the band are added up (θ from 4 to 7 Hz, α 7 to 14 Hz, β 14 to 30 Hz, γ_1 30 to 40 Hz and γ_2 50 to 70 Hz). Each band has a different number of elements, so the value is normalized to the various N_θ, N_α etc, depending on how many elements each band has. Then we plot the results against the noise, as shown in figure 10.

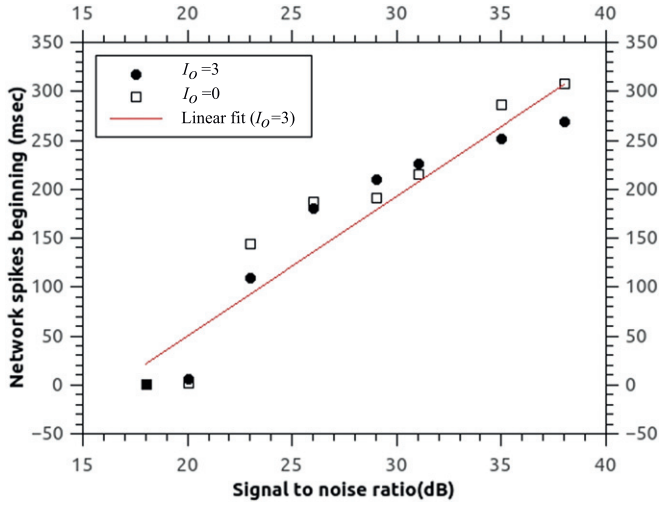


Figure 7. The comparative plot of the delay by which the spiking activity starts in a network with and without an active initiator x_o. Round dots represent the delay with an active ($I_o = 3$) initiator and the squares represent an inactive one. The influence of x_o is negligible and the linear dependence is clear. The red line represents a linear interpolation of $r > 0.9$ and inclination $\delta = 14$ msec per dB.

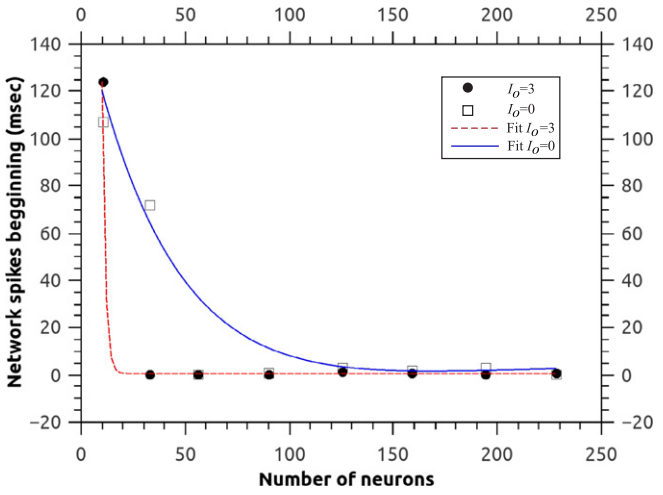


Figure 8. The comparative plot between networks with and without an active spike initiator x_o, in the same fashion as in figure 7, but when the network size is changed. For small network sizes of less than about $N = 100$ neurons the initiator makes a difference and induces a quicker propagation of spike activity. The noise level is kept to 20 dB in all the tests.

In this plot we see that all prominent peaks decrease with noise intensity. This happens because a low level of noise produces the transitional phenomena shown in figures 5 and 6, during which few neurons are spiking, then the network goes to a regime with lower and more

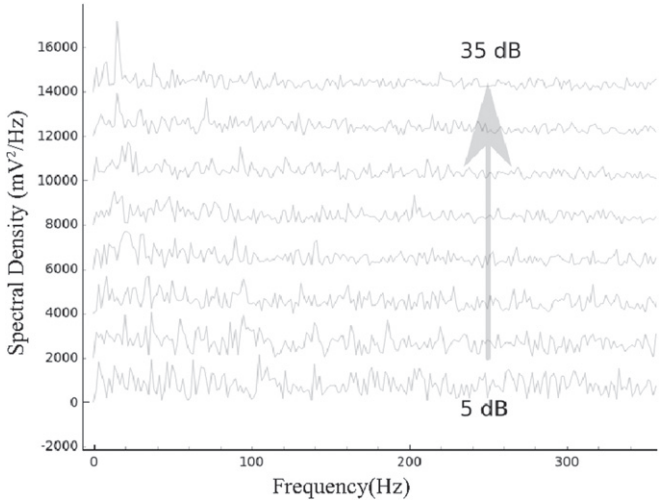


Figure 9. A plot of the fast Fourier transformation of the membrane potential averaged over the whole network. The curves represent its frequency spectrum averaged over the whole network. Each line represents a simulation with different levels of noise. The horizontal axis is in Hz and the vertical in arbitrary units. The latter has a relative meaning since plots are shifted vertically for clarity. These show how the network's characteristic frequencies are influenced by different noise levels. The simulations are produced with a total of $N = 48$ neurons. The small-world network is characterized by a connection probability $p = 0.4$ and dimension $k = 1$ and a spiking *initiator* neuron x_o with input $I_o = 3$ (a.u.). The plot considers only the frequencies at the regime. That is, the simulation is for 1000 msec, the first 300 msec of data are not considered, in order to avoid low frequencies due to the initial baseline variations. Each plot is the average of eight identical simulations executed with different random number seeds. The influence of an active ($I_o = 3$) or passive ($I_o = 0$) neuron x_o is null since we obtain equivalent results in both cases.

distributed frequency peaks. To verify that these patterns are indeed due to the initial transitional phenomena and not to the intrinsic character of the network, we cut off the first 300 msec of simulation and we instead observe a more intricate behavior of the bands' power, as shown in figure 11.

Since each symbol represents a different noise level, the fact that the plots intersect each other indicates that noise produces non-linear effect frequency bands amplitudes in this case. We investigate the network, changing the network size as a paradigm of biological internal noise. The analysis is conducted with other important neuron properties and characteristics kept constant to allow comparison and discussion. Small-world dimension $k = 1$, probability $p = 0.4$ and every simulation is repeated eight times with different random seeds, then averaged as in the previous tests.

As shown in figure 12, each spectrum shows distinct peaks in the α and θ range. Interestingly, this behavior is maintained also for bigger networks. In present conditions, our system is limiting the simulations to these network sizes, but it would be interesting to further increase the number of neurons to see if the diminished lower frequencies peaks phenomenon observed with the external noise increase would be repeated with networks of much higher sizes.

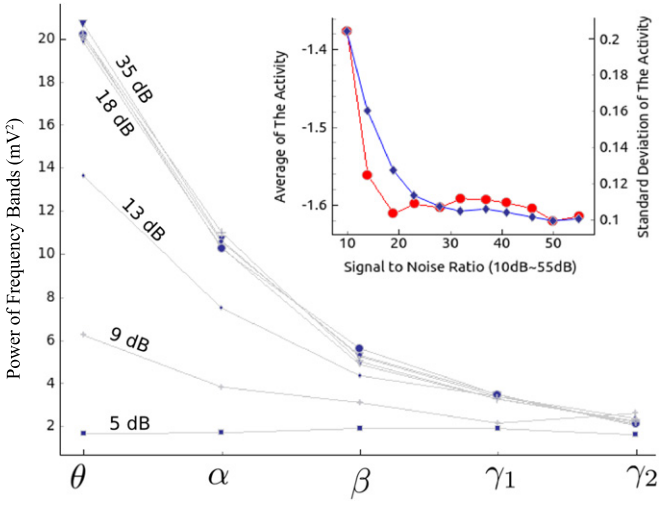


Figure 10. The power of five frequency bands for a network of $N = 48$ neurons. The level of noise is represented by each symbol according to this scheme: square, $dB = 5$, plus, $dB = 9$, diamond, $dB = 13$, triangle, $dB = 18$, triangle (bigger), $dB = 22$, circle, $dB = 26$, circle (bigger), $dB = 31$, plus (bigger), $dB = 35$. The peaks' magnitude seems to keep the same relative order with noise; this effect is caused by the predominance of the initial transition to the regime over the noise influence. The inset shows the average and standard deviation of $E(t)$ in function of noise for the same network. The average potential drops regularly because of the initial transitional period that drives potentials to more negative values. Noise reduces this initial transition, so the curve appears less negative at low dB values. Refer also to figure 5. For reasons of clarity not all the decibel labels are indicated.

4. Conclusions

We analyzed the collective behavior of a H−R small-world network of dimension $k = 1$ and random connection probability $p = 0.4$. The influence of noise is studied, especially concentrating on the effect it has on the propagation of information along the network by studying the membrane average frequencies and spike activity diffusion due to an active neuron that initiates the spiking. To isolate other effects the network is in ideal conditions; identical neurons, no plasticity and no delay time are considered.

The network has a single active neuron x_o connected to an external stimulus of $I_o = 3$ mA. This neuron is spiking constantly and in the absence of noise it induces the rest of the network to spike after an initial transitional time. Most interestingly, it seems that noise favors the initiation of secondary spikes (the spikes that follow those of x_o), and this spike activity becomes spontaneous and independent from x_o. We found a linear relationship between secondary spikes' initiation time and the SNR in decibels, with an inclination constant of about $\delta = 14$ millisecond per decibel. This phenomenon is instead found to be much weaker or not existent if we increase the network size.

However, for networks of sizes of $N = 100$ neurons or less, the initiator x_o still makes a difference, favoring the quicker spread of spike activity on the network. This suggests that locally-active neurons play a role in the spread of information in confined domains outside which the effect decays with distance. For the sake of simplicity and to avoid complexity this

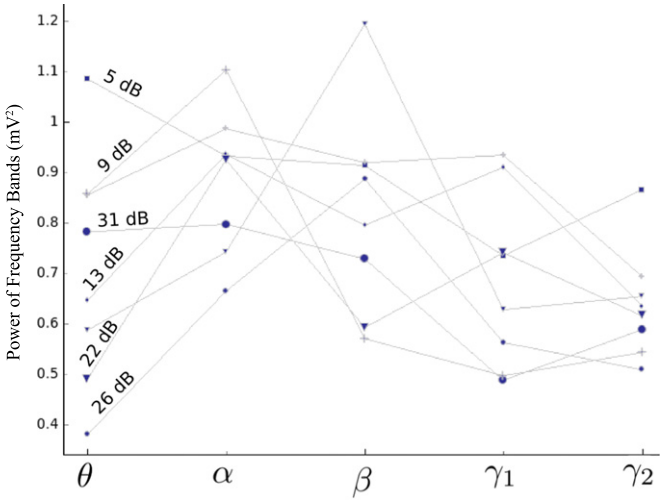


Figure 11. The power of five frequency bands for a network of $N = 48$ neurons at different noise levels as in figure 10. Noticeably, the first 300 msec of simulation were cut off to emphasize the network frequencies at the regime. The various plots cross each other, with no systematic trends. This indicates that the noise is influencing the spectrum relative intensities and seems to shift the peaks' position (see figure 9). For reasons of clarity not all the dB labels are shown. The initiator x_o has negligible influence on these plots, see figure 7.

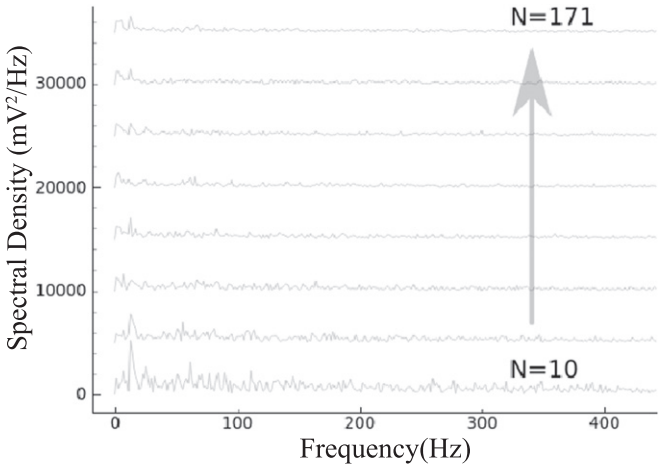


Figure 12. The Fourier transformation of the membrane signal in networks of different sizes and constant 20 dB noise level. Each line is shifted vertically for clarity, in the same fashion as figure 9. From top to bottom, the number of neurons is decreasing (top 171, bottom 10 with steps of 23 neurons). The simulation is run for 1000 msec; the first 300 msec of data are not considered to avoid low frequencies due to the initial baseline variations. Frequencies peaks emerge in all conditions, but are less prominent for networks of bigger sizes. Each plot is the average of eight identical simulations executed with different random number seeds.

result has a limited meaning bound to the specific H−R network studied here, but we intend to verify if this function of noise is a general phenomenon in spiking networks or remains limited to the conditions and network connection structure used here. The importance of noise in neural networks is known, and what we observed reminds us of the phenomena of stochastic resonance in biological systems [6, 39–42].

The influence of noise on frequency has been investigated by Fourier analysis. We have shown that once the transitional delay is past, noise displaces the main frequencies peaks, provoking a slow transition to chaos. Studying the most prominent frequency bands (θ, α, β and γ) we confirmed that noise quenches those biologically-significant peaks in the network. Still, at regime, a less keen transition to chaos is observed: frequency peaks do reduce and tend to disappear, but in a slower and more complex trend that seems to conserve some activity in the *theta* and *alpha* band (as shown in figures 9 and 11).

In real brains, the noise is generated internally, so we scaled up the network and used the network size as a paradigm of the external noise. We found two trends: a reduction in the amplitude of the peaks and a better sharpness and definition of the Fourier peaks, but not the evident degeneration to chaos observed when increasing external noise. This trend must be presumably confirmed with much higher network sizes, and we intend to set up the appropriate computational tools to do that in the near future.

Our work demonstrates that in a H−R network, a single neuron can lead to the excitation of the whole system; we quantitatively studied the influence of noise and have shown that it favors the initiation of secondary spikes in a linear manner, enhancing the propagation of spontaneous spiking activity along the network and canceling the effect of a localized *initiator* neuron that instead maintains its influence in confined domains.

This study gives insights on how networks utilize noise to alter the collective behavior of the system in their operations and brings quantitative assessment on how noise can be used as a criterion to control and modulate the spread of neural activity in H−R neural networks.

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