

Noise Induced Oscillations in Recurrent Neural Networks

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Abstract—It has been shown that oscillations can be generated by additive Gaussian white noise in a recurrent Hodgkin-Huxley neuron model. Type 1 oscillation was induced with Stochastic Resonance (SR) by additive Gaussian noise at lower amplitudes, while Type 2 oscillation was observed at higher amplitudes. However, the mechanism of Type 2 oscillation is not clear. In this article, we test the hypothesis through computer simulations that the period of the Type 2 oscillation can be affected by temperature in a recurrent neural network in which the recurrent model is constructed by four Hodgkin-Huxley (HH) neuron models. Each HH neuron model is driven by Gaussian noise and sub-threshold excitatory synaptic currents with an alpha function from another HH neuron model, and the action potentials (spike firings) of each HH neuron model are transferred to the other HH neuron model via sub-threshold synaptic currents. From spike firing times recorded, the inter spike interval (ISI) histogram was generated, and the periodicity of spike firings was detected from the ISI histogram at each HH neuron model. The results show that the probability of spike firings in the Type1 oscillation is maximized at a specific standard deviation (S.D.) of the Gaussian white noise with SR at 6.3, 15.0 and 25.0 °C, while the period of the Type 2 oscillation depends on temperature. It is concluded that the Type1 oscillation can be induced by additive Gaussian white noise on the basis of a synaptic delay in the recurrent HH neuron model, whereas ISIs of the Type 2 oscillation may be determined by refractory periods of HH neuron models.

Index Terms—Sub-threshold Synaptic Transmission, Action Potential, Stochastic Resonance, Hodgkin-Huxley model, Inter-Spike Intervals, Computer Simulation, Temperature, Q_{10}

I. INTRODUCTION

Stochastic resonance (SR) is a phenomenon occurring when coupling deterministic and random dynamics in non-linear systems. This phenomenon can be interpreted as an increase in detecting a low-level input signal in the output of the system, which is caused by an increase in the noise level of the input signal. In neurosciences, SR has been observed in peripheral nervous system [1], [3], [4] and central nervous system [5],[6], [7]. It was reported that the detection of a sub-threshold input signal was improved when a certain level of noise was added.

Neural oscillations have been observed in the central nervous systems such as cerebral cortex [2] and spinal cords. Our recent studies have shown that two types of oscillations can be induced by additive Gaussian white noise in a recurrent Hodgkin-Huxley neuron model. The

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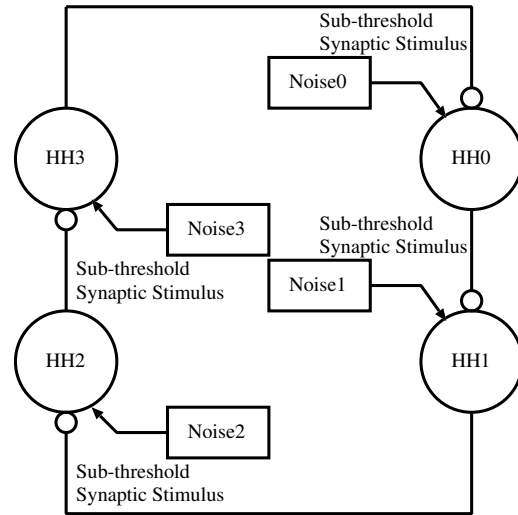


Fig. 1. Recurrent neural network model with four Hodgkin-Huxley (HH) neuron models. Each of HH neuron models is driven by the sub-threshold excitatory synaptic currents and Gaussian white noise.

first type of oscillations (Type 1 oscillation) (about 20 Hz) was induced with SR by additive Gaussian noise at lower amplitudes, while the second type of oscillations (Type 2 oscillation) (about 100 Hz) was generated by some intrinsic characteristics of neurons with noise at higher amplitudes. Although the mechanism of Type 1 oscillation could be attributed to SR, the mechanism of the generation of Type 2 is unknown. In order to study the origin of this oscillation, the effect of temperature on the noise induced oscillations was investigated.

In the recurrent neuron model, Gaussian white noise is added to each of the Hodgkin-Huxley (HH) neuron model and sub-threshold excitatory inputs are applied as synaptic currents with an alpha function. The action potentials (spike firings) of each HH neuron model are coupled to the other HH neuron model via sub-threshold synaptic currents. Using computer simulations at 6.3, 15.0, and 25.0 °C, the inter spike interval (ISI) histogram was generated, and the periodicity of spike firings was detected from the ISI histogram at each HH neuron model. Furthermore, the probability of spike firings in the oscillation period was estimated on the basis of the ISI histogram as the standard deviation (S.D.) of the Gaussian white noise varied.

II. METHODS

The k -th transmembrane potential, $V_m^{[k]}$, of the recurrent neuron model described by four Hodgkin-Huxley (HH) neuron models was represented for $k = 0, \dots, 3$ (See Figure 1)

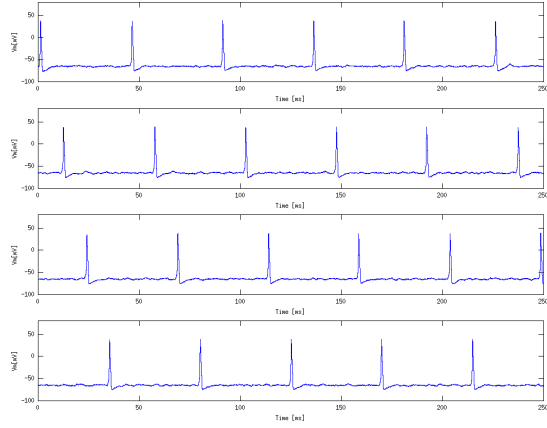


Fig. 2. The transmembrane potentials mV as a function of time (0-250 ms) for four HH neuron models at an S.D. of 40.0 at a temperature of 15 $^{\circ}C$. Type1 oscillation was generated at about 44 ms by this noise level.

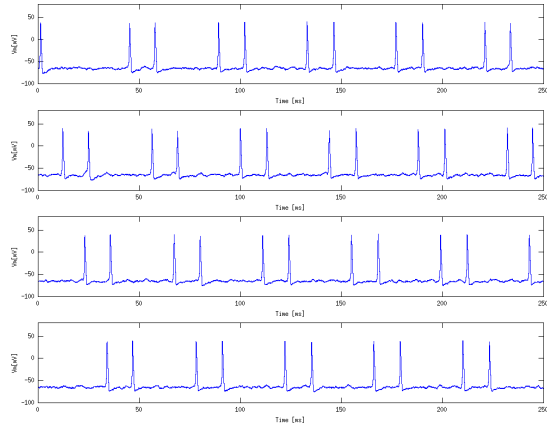


Fig. 3. The transmembrane potentials mV as a function of time (0-250 ms) for four HH neuron models at an S.D. of 60.0 at a temperature of 15 $^{\circ}C$. Inter-spike intervals at about 30 and 15 ms were generated by the intermediate noise level (Type1 and Type2 oscillations).

as follows [8], [9], [10]:

$$C \frac{dV_m^{[k]}(t)}{dt} + g_{Na}(m^{[k]}(t))^3 h^{[k]}(t)(V_m^{[k]}(t) - E_{Na}) + g_K(n^{[k]}(t))^4 (V_m^{[k]}(t) - E_K) = I_{syn}^{[k]}(t) + I_{GWN}^{[k]}(t) \quad (1)$$

where $g_{Na}=120 \text{ mS}$, $E_{Na}=50 \text{ mV}$, $g_K=36 \text{ mS}$, $E_K=-77 \text{ mV}$ and the resting potential was set at -65 mV , and where

$$\frac{dm^{[k]}(t)}{dt} = \alpha_m^{[k]}(V_m^{[k]})(1 - m^{[k]}(t)) - \beta_m^{[k]}(V_m^{[k]})m^{[k]}(t) \quad (2)$$

$$\alpha_m^{[k]} = \frac{0.1(V_m^{[k]} + 40)}{1 - e^{-(V_m^{[k]}+40)/10}} C_r \quad (3)$$

$$\beta_m^{[k]} = 4e^{-(V_m^{[k]}+65)/18} C_r \quad (4)$$

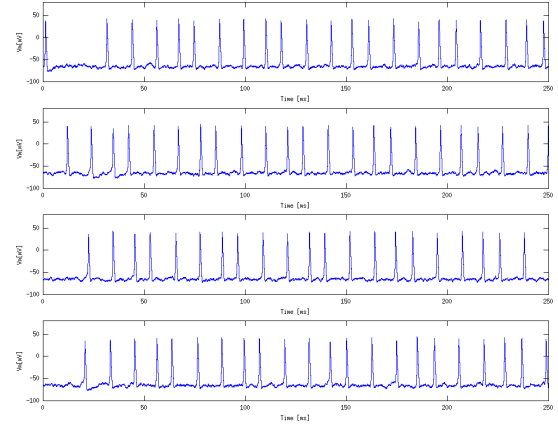


Fig. 4. The transmembrane potentials mV as a function of time (0-250 ms) for four HH neuron models at an S.D. of 100.0 at a temperature of 15 $^{\circ}C$. The noise at an S.D. of 100.0 induced the Type 2 oscillation at about 8 ms spike intervals.

in which $C_r = 3.0^{(T-6.3)/10}$, where T denotes temperature, and

$$\frac{dh^{[k]}(t)}{dt} = \alpha_h^{[k]}(V_m^{[k]})(1 - h^{[k]}(t)) - \beta_h^{[k]}(V_m^{[k]})h^{[k]}(t) \quad (5)$$

$$\alpha_h^{[k]} = 0.07e^{-(V_m^{[k]}+65)/20} C_r \quad (6)$$

$$\beta_h^{[k]} = \frac{1}{1 + e^{-(V_m^{[k]}+35)/10}} C_r \quad (7)$$

$$\frac{dn^{[k]}(t)}{dt} = \alpha_n^{[k]}(V_m^{[k]})(1 - n^{[k]}(t)) - \beta_n^{[k]}(V_m^{[k]})n^{[k]}(t) \quad (8)$$

$$\alpha_n^{[k]} = \frac{0.01(V_m^{[k]} + 55)}{1 - e^{-(V_m^{[k]}+55)/10}} C_r \quad (9)$$

$$\beta_n^{[k]} = 0.125e^{-(V_m^{[k]}+65)/80} C_r \quad (10)$$

in which the excitatory synaptic current, $I_{syn}^{[k]}(t)$, is expressed as an alpha function by:

$$I_{syn}^{[k]}(t) = g_{syn} C_c \alpha_s(t - \tau_d) e^{-\alpha_s(t - \tau_d)} (V_m^{[mod(k-1,4)]}(t - \tau_d) - E_{syn}) \quad (11)$$

where $g_{syn}=0.2 \text{ mS}$ for sub-threshold stimuli, $\alpha_s=1.0 \text{ ms}^{-1}$, $E_{syn}=0 \text{ mV}$, $\tau_d=10 \text{ ms}$, $C_c = 1.5^{(T-6.3)/10}$, and in which the Gaussian white noise, $I_{GWN}^{[k]}(t)$, has the following statistical properties:

$$\begin{cases} E[I_{GWN}^{[k]}(t)] = 0 \\ E[I_{GWN}^{[k]}(t)I_{GWN}^{[k]}(t + \tau)] = \sigma^2 \delta(\tau) \end{cases} \quad (12)$$

where $E[\]$ and $\delta(\)$ stand for the expectation operation and Kronecker's delta function, respectively, and σ^2 denotes the variance of the Gaussian white noise.

Transmembrane potentials were numerically calculated at a sampling step of 1 ms by simultaneously solving the HH equations (1)-(11) with the Euler method. The spike firing times were determined by detecting when the transmembrane potential reached its maximum value and was greater than

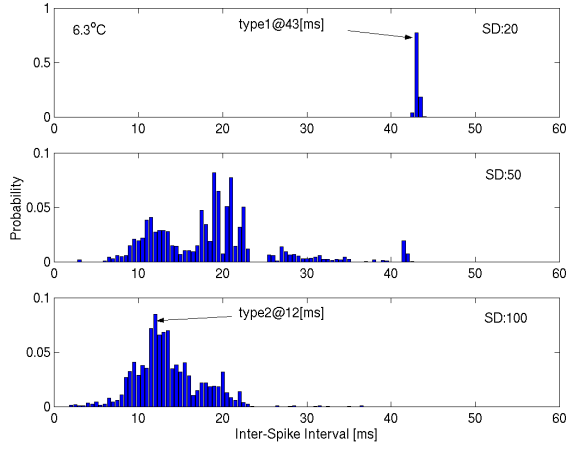


Fig. 5. ISI histograms at S.D.s of 20.0, 50.0, and 100.0 with a bin width of $500 \mu s$ at $6.3^\circ C$. The Type1 oscillation with $43ms$ ISIs was observed at an S.D. of 20.0 in the top trace, while the Type2 oscillation with $12 ms$ ISIs was observed at an S.D. of 100.0.

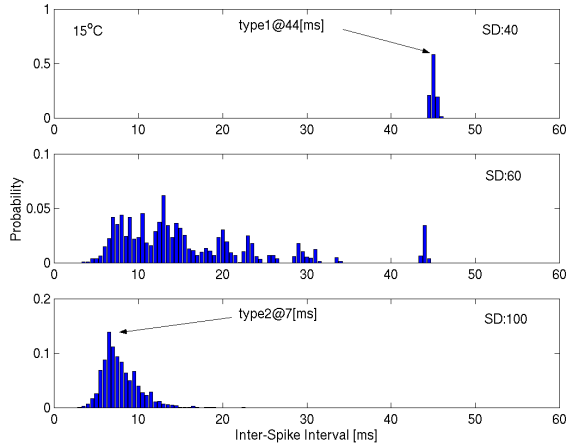


Fig. 6. ISI histograms at S.D.s of 40.0, 60.0, and 100.0 with a bin width of $500 \mu s$ at $15.0^\circ C$. The Type1 oscillation with $44ms$ ISIs was observed at an S.D. of 40.0 in the top trace, while the Type2 oscillation with $7 ms$ ISIs was observed at an S.D. of 100.0.

50% of the peak amplitude of action potentials. The interspike interval (ISI) histogram was generated from spike firing times, where the bin width of the ISI histogram, bw , was set at $500 \mu s$. From those histograms the probability of firing spikes at the oscillation period was estimated to quantitatively evaluate how the added Gaussian white noise can improve sub-threshold synaptic transmission in recurrent HH neuron models. All computer simulations were performed on an IBM compatible PC with a Core 2 Quad Q6600 CPU.

III. RESULTS

The effect of the addition of noise to the network was tested with increasing the S.D. of noise and measuring the resulting spike firing induced. Figure 2 shows the transmembrane potentials $V_m^{[k]}(t)$ in mV as a function of time (0-250 ms) for four HH neuron models ($k = 0, \dots, 3$) at an S.D. of 40.0 at a temperature of $15^\circ C$. Type 1 oscillation with an

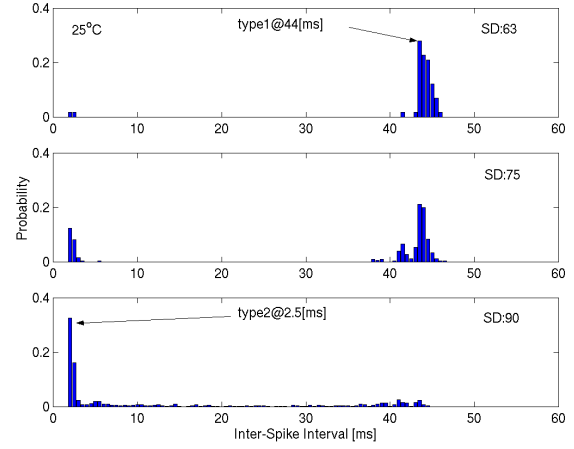


Fig. 7. ISI histograms at S.D.s of 40.0, 60.0, and 100.0 with a bin width of $500 \mu s$ at $25.0^\circ C$. The Type1 oscillation with $44ms$ ISIs was observed at an S.D. of 63.0 in the top trace, while the Type2 oscillation with $2.5 ms$ ISIs was observed at an S.D. of 90.0.

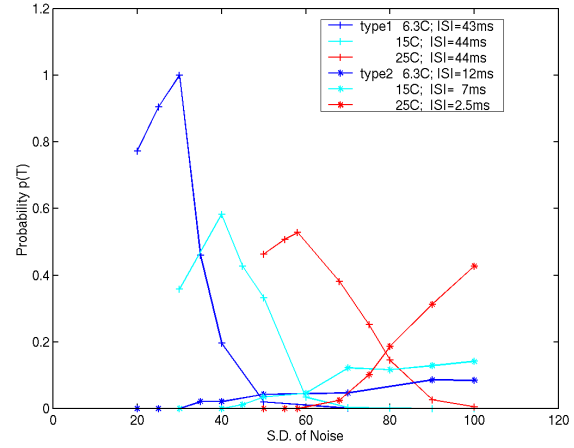


Fig. 8. The probability of spike firings vs. the noise intensity at 6.3 , 15.0 , and $25.0^\circ C$. The probability of the Type1 oscillation was plotted with “+”, whereas that of the Type2 oscillation was plotted with “*”. Typical curve of stochastic resonance was observed at each temperature in the Type1 oscillation, while the probability monotonically increased as the S.D. of Gaussian noise increased.

oscillation period of $44 ms$ was generated and corresponds to the natural oscillations of the network. However, addition of higher amplitude noise (S.D.=40.0) generated doublets and lower spike intervals (Figure 3). Further increase in the S.D. of Gaussian noise to 100 induced the Type 2 oscillation at an oscillation period of $8 ms$ (Figure 4). This noise amplitude is supra-threshold for action potential generation.

The effect of temperature was investigated by plotting the ISI histograms at various values of the temperature and various values of the noise amplitude with a bin width of $500 \mu s$ at S.D.s of 20.0, 50.0, and 100.0. The Type 1 oscillation with $43ms$ ISIs was observed at $6.3^\circ C$ at an S.D. of 20.0 (Figure 5 top trace), while an intermediate pattern is generated at an S.D. of 60.0 and Type 2 oscillation with $12 ms$ ISIs was observed at an S.D. of 100.0. When the

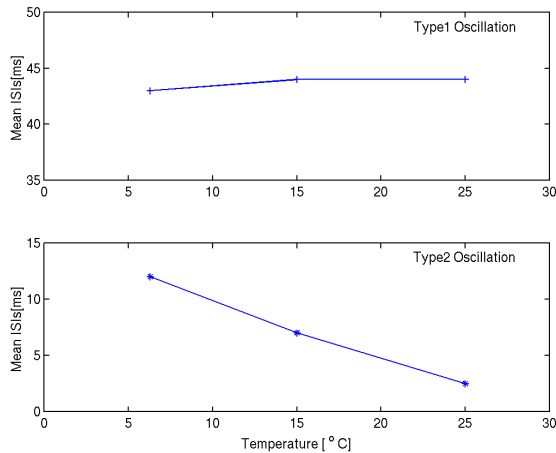


Fig. 9. The periods (Mean ISIs) of the Type1 (top) and Type2 (bottom) oscillations against temperature at 6.3, 15.0, and 25.0 °C. The periods of the Type1 oscillation were found to be insensitive to temperature, while those of Type2 oscillation were dependent on temperature.

temperature is increased to 15.0 and 25, similar patterns are observed but at different values of spike intervals (Figures 6 and 7).

Figure 8 shows the probability of spike firings vs. the S.D. of Gaussian noise at 6.3, 15.0, and 25.0 °C. The probability of the Type1 oscillation was plotted with “+”, whereas that of the Type 2 oscillation was plotted with “*”. Typical curve of SR was observed at each temperature in the Type1 oscillation, while the probability of spike firing in the Type 2 oscillation monotonically increased as the S.D. of Gaussian noise increased. These data show that the SR is responsible for the maximum probability of firing spikes in the Type1 oscillation and not for the Type 2 oscillation, since greater noise amplitudes are no longer within sub-threshold stimuli for Type 2 oscillation.

The inter-spike intervals for Type 1 and Type 2 are plotted as a function of temperature in Figure 9. This plot reveals another significant difference between Type 1 and Type 2 oscillations since Type 1 is independent of temperature while the inter-spike interval of Type 2 oscillation decreases with temperature. These data suggest that Type 1 oscillations are mediated by the network, and that Type 2 oscillations depend on the intrinsic characteristics of HH neuron models.

IV. DISCUSSION AND CONCLUSION

The simulation results show that additive Gaussian noise to a recurrent neural network can induce periodic activity with various noise level. Two types (Type 1 and 2) of oscillations were induced at low noise and high noise amplitudes. By varying the amplitude of the noise and temperature, simulations show that the oscillations have different mechanisms of generation and origin. Type 1 oscillation is a low frequency oscillation that is generated by a low noise amplitude. As the noise amplitude is increased, the probability of spike

generation first increases, reaches a plateau and then decreases as is typical of SR. Type 2 oscillations, however, have higher frequencies and the probability of spike firings increases monotonically with noise amplitude. Therefore, Type 2 oscillations cannot be attributed to SR.

The analysis of the effect of temperature on the generation of the oscillations shows that the Type 1 oscillations are independent of the temperature. The mean value of the period of the oscillation corresponds to the natural frequency of the network and therefore depends directly on the network properties. The mean value of the period of Type 2 oscillations is dependent on the intrinsic properties of the neurons, i.e., refractory periods of HH neuron models due to unavailability of sodium channels, because higher temperature gives rise to faster transition rates of sodium channels (See (3)-(4), (6)-(7)), thereby making refractory periods shorter.

In conclusion, additive noise can generate two types of oscillations, one that is consistent with SR and temperature independent and one that is temperature dependent and relies on the intrinsic properties of the network. These results could be important for understanding the role of noise for generations of normal oscillations such as gamma waves but also abnormal activity such as interictal spikes. In the future, it will be important to employ simulations in more realistic models incorporating different types of neurons, different types of ion channels, and inhibitory synapses.

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