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ORIGINAL PAPER

The effects of temperature on the dynamics of the biological neural network

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Abstract
The nerve cells are responsible for transmitting messages through the action potential, which generates electrical stimulation. One of the methods and tools of electrical stimulation is infrared neural stimulation (INS). Since the mechanism of INS is based on electromagnetic radiation, it explains how a neuron is stimulated by the heat distribution which is generated by the laser. The present study is focused on modeling and simulating the conditions in which deformed temperature related to the Hodgkin and Huxley model can be effectively and safely used to activate the neurons, the fires of which depend on temperature. The results explain ionic channels in the single and network neurons, which behave differently when thermal stimulation is applied to the cell. It causes the variation of the pattern of the action potential in the Hodgkin-Huxley (HH) model. The stability of the phase-plane at high temperatures has lower fluctuations than at low temperatures, so the channel gates open and close faster. The behavior of these channels under various membrane temperatures shows that the firing rate increases with temperature. Also, the domain of the spikes reduces and the spikes occur faster with increasing temperature.

Keywords Neural network · Neuronal spiking · Action potential · Hodgkin and Huxley · Temperature effect

1 Introduction

An action potential is one of the most essential features of the nerve cells. Using precise measurements of the action potential, scientists can examine the mechanisms of representing the behavior of the nervous system of organisms. To understand the transmitted messages between nerve cells in the network, it is necessary to study action potential patterns. So, the accurate understanding of action potential enables us to know the neuronal communication procedure, because the messages are the action potential patterns. In other words, the action potential is considered as a signal that carries data in different areas of

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the nervous system. It is assumed that this transmission does not change along the axon's path. Information is transmitted from one location to another using the frequency and the pattern of the action potential. It can also be called a spike, or a nerve impulse. General characteristics are common in the action potential in all animals. Therefore, the action potential behavior of some animals can be generalized to some other animals. One of these animals that play an essential role in the formation of the biophysical model to justify the action potential is squid, because squids have large enough axons to manipulate and use their specially built glass electrodes on [1].

The action potential varies under environmental factors such as temperature. The first experimental studies on the effect of temperature on the behaviors of the action potential were carried out by Sjodin and Mullins [2] and Guttman [3]. The observations of Sjodin and Mullins in stimulated pulses for one millisecond showed that the excitability threshold decreased with increasing temperature. At the same time, Gutman used the results of pulses obtained in 100 milliseconds and found that it raised. Electrical stimulation of axons was based on mathematical modeling by Hodgkin and Huxley. In 1950, Hodgkin and Huxley conducted studies on the axon of a squid, which led to the formation of a model for the action potential. They studied the ionic current by placing an electrode inside the cell and injecting current to measure the current of ions and cell membranes based on stimulation current. After several experiments and numerous studies, they obtained precise equations for how ionic current changes.

In the HH model, the membrane's capacity does not change with temperature. Therefore, in this study, the membrane capacity is considered constant. Also, the single neuron and then a network of neurons have been studied such that the membrane potential is related to the temperature [1, 4].

However, in some studies [5–6] the effect of temperature on the HH model has been investigated but the effects of temperature changes on the number of spikes and action potential value in the repetitive firing model have not been determined. The study of temperature variation on the behavior of ion channels by Kaung et al. [8] indicates that the spiking threshold in the HH model occurs in a minimal temperature(T_c) in the range of 4 $^\circ C$ to 25 $^\circ C$. Also, Yuan et al. [9] studied a pattern for the effects of the various environmental temperature as a sinusoidal function on the cell spiking. They observed that the shape of action potential has been changed by rising temperature from $-18\,^\circ C$ to $18\,^\circ C$.

Numerical and analytical methods have been improved to heat transfer during Infrared neural stimulation (INS) [10–13]. Since the INS is dependent on thermal changes, proper knowledge of the distribution of heat in the nervous system may improve the production of optical devices for stimulating the neuron [14]. Norton et al. [15] developed an analytical method by considering a Green's function for thermal changes during the INS. They studied the cochlea nerves and showed the temperature variations activate the neurons and concluded that the minimum temperature T_c increases and also a minimum temperature rate of change dT_c/dt are required for neural stimulation. Xu et al. [16] found that temperature changes with thermistors could control neuronal dynamics. Also, Zhang et al. [17] used thermistors to control the dynamics of the neural network to consider the fluctuation of temperature on the neuronal network behavior.

This paper addresses temperature exposure effects on single and network neurons based on HH biophysical model. First, the spiking patterns of a single neuron under the different values of the temperature are reviewed. Second, the action potential and phase-plane are discussed for the one-dimensional neural network. The results for a single neuron reveal that by increasing environmental temperature from $T = 0\,^\circ C$ to $T = 18\,^\circ C$ the spiking occurs faster and the average of the action potential magnitudes decrease. These results were also observed

at the neural network scale. Also, the results show that the Stable Steady-Points (SSP) gap approach to its lowest value by raising the size of the neural network and the influence of temperature on it. The biophysical basis of this phenomenon, along with its biological significance, is explored and discussed in the context of ion channels gating dynamics. Finally, the concluded results are compared with the experimental results.

2 Temperature effects on Hodgkin-Huxley model

The HH model [1] is introduced as one of the simplest models but most efficient to justify the action potential of nerve cells. The HH model is defined as the set of differential equations to describe the membrane potential behaviors. In general, the dynamical equations of the HH model are described by the following set of equations [19]:

$$\begin{aligned}
\frac{dV}{dt} &= (I_e - I_{ion})/C_m \\
\frac{dm}{dt} &= (m_\infty(V) - m)/\tau_m(V,T) \\
\frac{dh}{dt} &= (h_\infty(V) - h)/\tau_h(V,T) \\
\frac{dn}{dt} &= (n_\infty(V) - n)/\tau_n(V,T),
\end{aligned} \tag{1}$$

where V is the membrane potential, I_e is the stimulation current, I_{ion} is the ionic current through the channels, C_m is the capacitance of the membrane, T is defined as the cell temperature, n is the activation variable of the potassium channel, m and h are the variables for activating and deactivating of the sodium channel, m_∞, h_∞, n_∞ are the steady-state values for m, n and h and τ_m, τ_h, τ_n represent the time constants of the gating variables m, n and h as a function of V and T. Also m, n, and h play the channel gates controlling parameters role in the differential equations. In the specific case of single neuron [19, 20], the HH model is introduced by the time-dependent membrane potential as the following first-order nonlinear differential equation:

$$\begin{aligned}
C_m \frac{dV}{dt} = &- \bar{g}_{Na} m^3 h (V - V_{Na}) - \bar{g}_K n^4 (V - V_K) \\
&- \bar{g}_L (V - V_L) + I_e,
\end{aligned} \tag{2}$$

where the first term ($\frac{dV}{dt}$) shows membrane current. Let's consider the membrane capacity as $C_m = 1\mu F/cm^2$, and the maximum channels conduction as: $\bar{g}_{Na} = 120mS/cm^2$, $\bar{g}_K = 36mS/cm^2$, $\bar{g}_L = 0.33mS/cm^2$. Also V_L, V_{Na} and V_K are the equilibrium potential which are calculated by the Nernst equation [27]. The sentence $\bar{g}_L(V - V_L)$ is called the leak current, that comprises mostly chloride and other voltage-independent ion channels. To calculate the action and rest potential related to each of the ion channels, the Nernst equation is used more precisely. Calculations on the HH model showed that the probability of open and closed channels are n^4 for potassium and m^3h for sodium. Let's use an assumption to visualize the modes of a gate as bimodal states such that, when a channel is turned off, the gate is closed, and one can say its state is changed from open mode 1 to closed mode 0 in a specific time period. o justify the dynamic of the gate's behavior, some new constant parameters α_i and β_i (opening and closing rates) should be defined, whereas i refers to ionic

channels type. Now the dynamics of each of m, n and h is determined by the difference between open, and close modes [19] as the functions of α_i and β_i:

$$\frac{dx}{dt} = \alpha_x(V)(1-x) - \beta_x(V)x, x = m, h, n. \tag{3}$$

Also, by using interpolation HH model results with experimental data, [19] one can find the coefficients α and β:

$$\begin{aligned}
\alpha_n &= \Phi(T)0.01\frac{10-V}{e^{(10-V)/10}-1}\\
\beta_n &= \Phi(T)0.125e^{\frac{-V}{80}}\\
\alpha_m &= \Phi(T)0.01\frac{25-V}{e^{(25-V)/10}-1}\\
\beta_m &= \Phi(T)4.0e^{\frac{-V}{18}}\\
\alpha_h &= \Phi(T)0.07e^{\frac{-V}{20}}\\
\beta_h &= \Phi(T)0.1\frac{1}{e^{(30-V)/10}+1},
\end{aligned} \tag{4}$$

where V is the potential at temperature T and $\Phi(T)$ is the temperature dependence of the membrane parameters. It's proving that the properties of membranes such as flexibility, inharmonious-ability, and penetrability depend on temperature, electromagnetic field, and pressure. However, the HH model predicts that the channels are sensitive to temperature change as well [22].

In a recent investigation, scientists used an alternative method to electrical stimulation by low-intensity infrared light, such that the temperature increase led to the more electrical excitability of neurons [21]. This occurs because infrared light is well-absorbed by tissue to yield a photo-thermal influence at appropriately high wavelengths. Furthermore, there are several biophysical properties of electrically excitable cells that have temperature sensitivity either explicitly or implicitly. An example of these biophysical processes is the gating kinetics of ion channels, maximum conductances of each ion channel, and equilibrium potentials. Temperature-sensitive components of the HH model include the rate constants, maximum membrane conductivity, and the Nernst potential. Neuronal electrical activity is dependent on membrane patch temperature, and the change in the electrical activity by membrane patch temperature occurs by modulating the rate of opening and closing of ion channels.

The effect of temperature on the action potential in the HH model is introduced as a temperature scaling factor [23] Q_{10}, which is an estimation of rate coefficient increase concerning $10°C$ temperature change alteration:

$$Q_{10} = \left(\frac{X_1}{X_2}\right)^{(T_1-T_2)/10},$$

where X_1 and X_2 are biophysical parameters at temperatures T_1 and T_2 such as temperature, electrical charges, pH and pressure. To create the desired model, T_2 refers to a reference temperature at which the electrophysiological examination temperature occurs. The factor Q_{10} was applied to all α and β values of the gating variables n, m, and h to incorporate temperature sensitivity for them. In this study, the reference temperature is chosen as $T_2 = 6.3\,°C$ for the considerations on the HH model [22]. According to the article [23], from the Law of Arrhenius it is deduced that $X_1/X_2 = 3$, $\Phi(T)$ was defined as follows:

$$\Phi(T) = Q_{10} = 3^{(T-6.3)/10}. \tag{5}$$

3 Results

Equations 3 and 4 were numerically solved using the Euler method with the time step $\Delta t = 0.001\,ms$. From $t = 1\,ms$, the membrane potential starts to rise and firing is obtained. Figure 1 is spiking time patterns at different temperatures, $T = 6\,^{\circ}C$, and $T = 18\,^{\circ}C$, in which the current stimulation was kept constant at $I = 100\mu A$. The number of spikes rises by increasing temperature and the average of potential magnitudes decrease with temperature by steps $\Delta T = 0.5\,^{\circ}C$ from a range of $0\,^{\circ}C$ to $18\,^{\circ}C$. Figures 2 and 3 display these res ults.

There is a two-dimensional system that can be studied on the (n, V) phase-plane. Figure 4 shows the phase diagram for a single neuron at the different temperatures with constant stimulation current. The neuron can be remodeled from equilibrium to train of action potentials and this evolution is compatible with a change of phase portrait. However, if the stimulation current is considered as a constant, the cell's dynamics completes the loop by increasing the temperature more regularly and faster (Fig. 4).

The temperature evolution in the HH model changes the three components: the equilibrium potential, the maximum membrane conductivity, and the voltage-dependent rate constants α_i and β_i. These changes were checked in the constant domain range. With this evolution, the system was also investigated in the equilibrium potential. In fact, taking into account the temperature changes at the potential of equilibrium, the number of spikes does not change; it only causes the height difference in the membrane potential. Meanwhile, when changes were applied to the ion conduction domain, it has no significant development

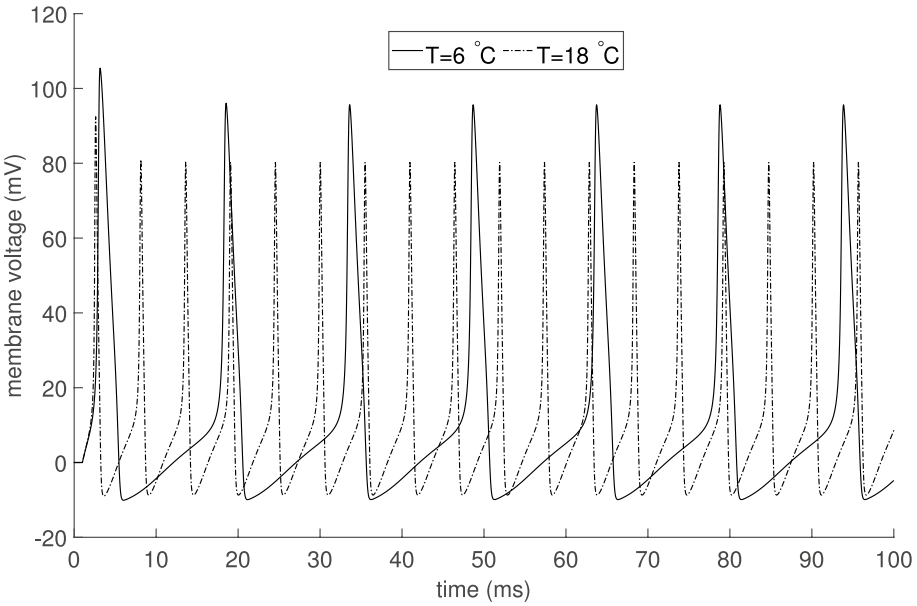


Fig. 1 The time course of membrane potential at environmental temperatures $T = 6\,^{\circ}C$, and $T = 18\,^{\circ}C$

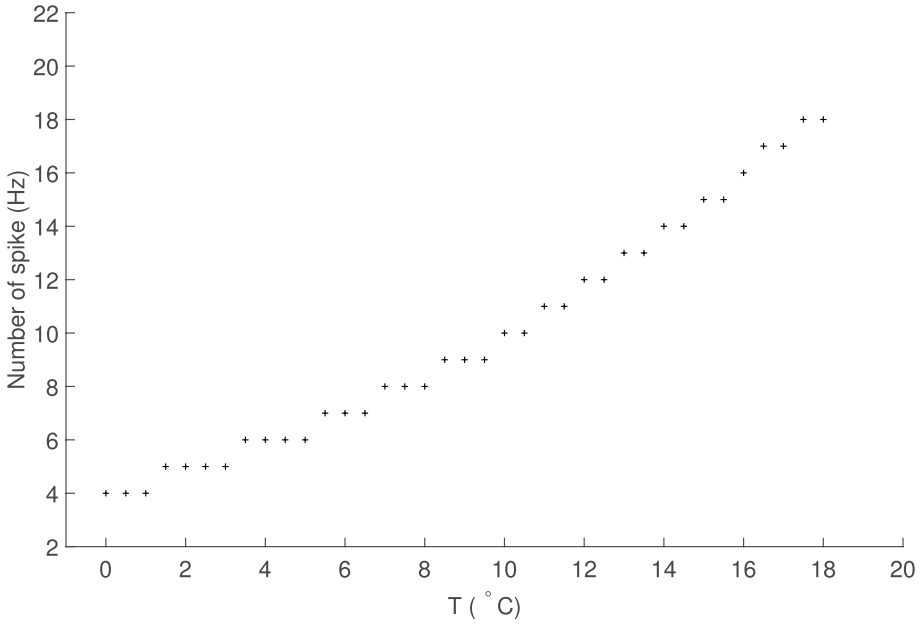


Fig. 2 The spike rate of a single neuron rises as a function of temperature under a constant stimulation current

on the pattern of spikes [18]. Figures 2 and 3 illustrate the temperature evolution in the α and β constant parameters cause significant changes in the height and number of spikes.

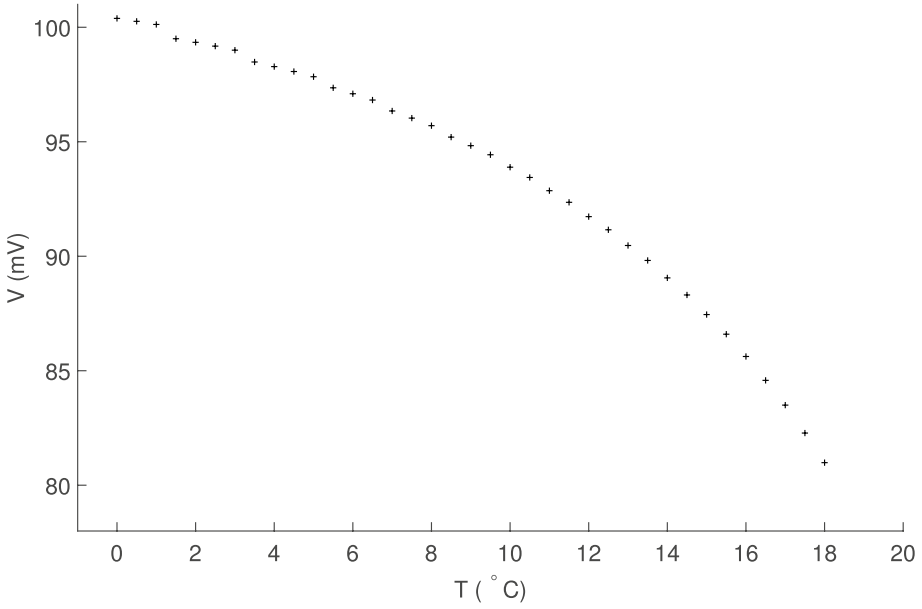


Fig. 3 Under a constant stimulation current, action potential decreases with temperature

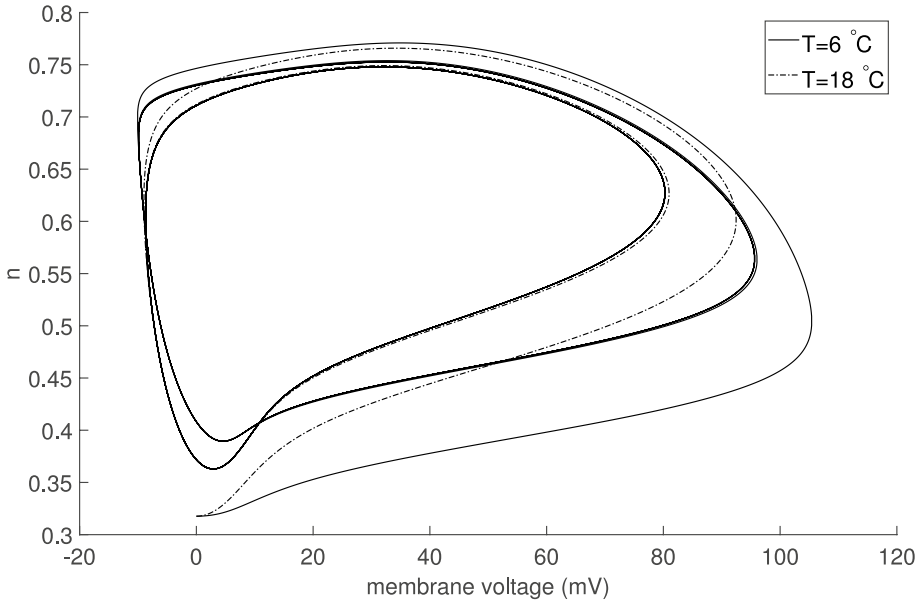


Fig. 4 A plot of gating variable (n) versus the potential (V(mV)) for temperatures $T = 6\,^{\circ}C$ and $T = 18\,^{\circ}C$

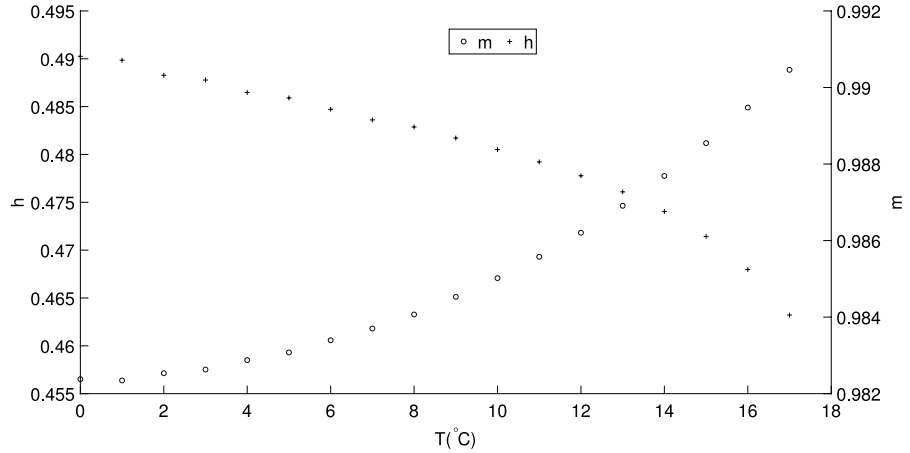
Figure 5 compares the evolution of n, m, and h gates with temperature, when temperature increases, the maximum activated m gate for sodium ion decreases while the inactive gate h increases. One can see the effects of the temperature rise on the potassium channels which decrease the gating variable n.

It was also investigated the situation in which for two temperatures of $T = 6\,^{\circ}C$ and $T = 18\,^{\circ}C$, the variable current injection was considered. Figure 6 shows that by keeping the temperature constant, the number of spikes increases as the current increases. By increasing the excitation current the number of spikes increases for the higher temperature.

4 Temperature effects on neural networks

A neuronal network consists of combinations of tens, hundreds, thousands, and even over 100 million of neurons depending on the size of the network. There are very complicated connections between neurons on the networks such that each neuron can have almost 10^4 connections with other neighboring neurons. The neurons that are responsible for receiving information from the outside are known as "unit" neurons.

A biological neural network is a set of algorithms or circuits of neurons to recognize patterns, and an artificial neural network is designed for solving artificial intelligence problems. In the human brain, types of neurons are very different, and the neural network is a mathematical description of the electrophysiological properties of these neurons. Actually, neurons link to each other using action potential through synapses that interface with axons and dendrites. One of the well-known mathematical models for simulations of the brain is the HH model, which depends on the mathematical parameters to describe the ionic and synaptic conductances, corresponding to the dynamics



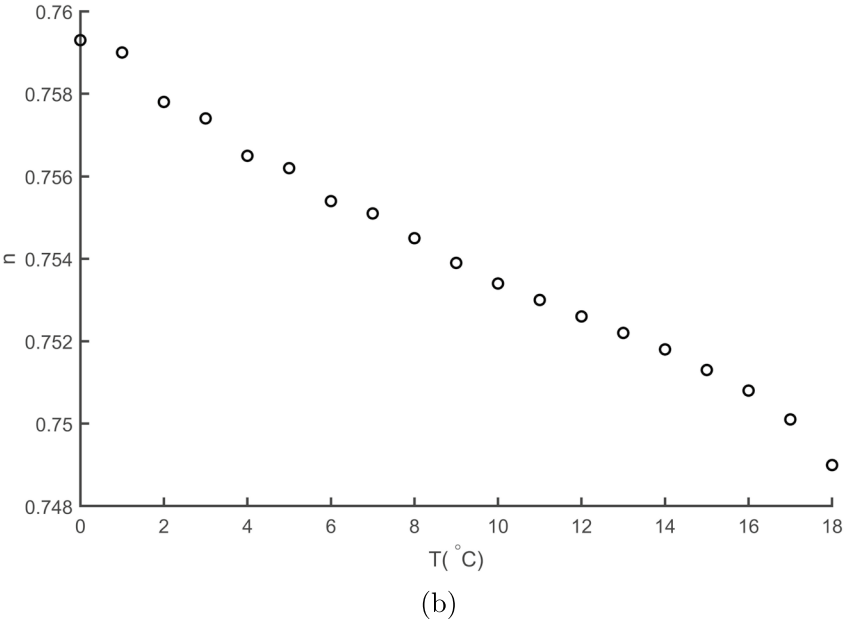


Fig. 5 (**a**) The maximum percentage of gating of m and h gates for sodium channels. (**b**) The maximum percentage of gating of n gates for potassium channels

of ionic channels. The simulation of this model includes difficult computation, which makes the implemented brain networks complex.

Most neural networks are fully connected, this means that neurons in a network are connected to other layers in network.

The connection between neurons is represented by a parameter which is called the weight of the connection. The weight is positive, if one unit excites another one, or it is

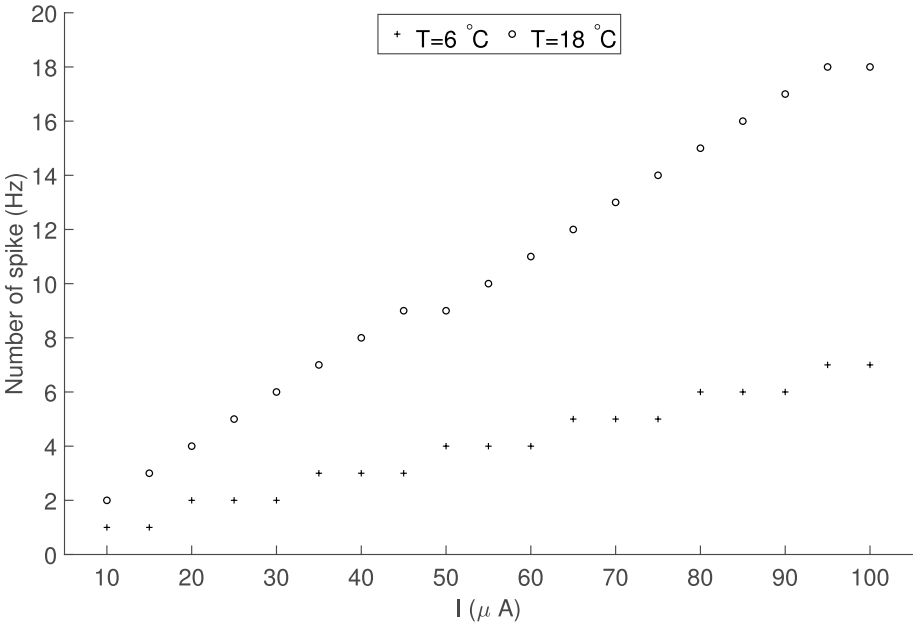


Fig. 6 A plot of the number of spikes during a 100 ms vs current injection for temperatures $T = 6\,^{\circ}C$ and $T = 18\,^{\circ}C$

negative, if one unit suppresses the other one. It is proven that by raising the weight of the connection, the impact of one unit on the other neurons will grow [26].

The HH model is elegantly generalized for a neural network model [24, 25]. Here, it is considered a linear network of N neurons (Fig. 7) with the weight of connection g_i between two neighboring neurons. The weight of connection represents the ability of synaptic connections of the excitatory neurons to each other. Ion channels have some significant rules for this linear network. In general, the HH model introduces the membrane potential difference of a biological neural network as:

$$\begin{aligned} C_m \dot{V}_i(t) + \bar{g}_{Na} m^3 h (V_i - V_{Na}) + \bar{g}_K n^4 (V_i - V_K) \\ + \bar{g}_L (V_i - V_L) + I_{syn_i} = I_e, \end{aligned} \tag{6}$$

where the index i labels the neuron in the network and I_{syn_i} is synaptic current by the following definition:

$$I_{syn} = g_i (V_N - V_i), \tag{7}$$

where $(V_N - V_i)$ is the difference of potential between the membrane of neuron N and neuron i in the network and I_e is the stimulation current. Let's define a network of identical

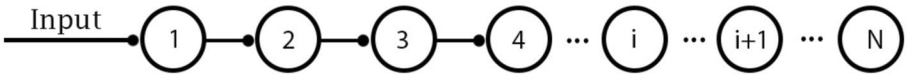


Fig. 7 A linear network of N-neurons

HH cells that are connected with a user-controlled conductance. So, as an example g_i can optionally set to 5 for all neurons [25].

The dynamics of a variable (such as gating variables) in the network mode can be written as:

$$\frac{dx_i}{dt} = \frac{x_\infty(V_i) - x_i}{\tau_x(V_i, T)}, \tag{8}$$

where x contains each of the gating parameters m, h ,and n.

Let's consider two neural networks contain 10 and 50 neurons, and the fifth neuron is selected for each of them to concentrate the simulations on its spiking and the potential changes, as it is shown in Figs. 8 and 9. The rise of temperature changes the dynamics of the HH model to demonstrate a stable oscillatory behavior. The stable oscillations in physical terms are due to a stable limit cycle, seen in Figs. 10 and 11 on the (V, n) phase plane. As Fig. 11 shows there are some small fluctuations in the voltage from the initial resting potential, and it decays back to resetting potential again. By increasing temperature, these fluctuations are obviously reduced. The phase plane was supposed to change when there was any variation in the equation parameters. By increasing the scale of the neural network and the effect of temperature on it, the stable steady-points (*SSP*) distance will be reached to its minimum value (Fig. 11). One can find from Figs. 8 and 9 which introduce potential mappings in terms of time at two temperatures of 6 °C and 18 °C, by raising the temperature, the number of spikes increases and the spiking height of the action potential reduces (Fig. 12).

Here, it is demonstrated that the temperature can change the action potential patterns of the neuronal network. By keeping the stimulation current constant, it is observed that the width and height of the action potential become smaller by warming up the neuron

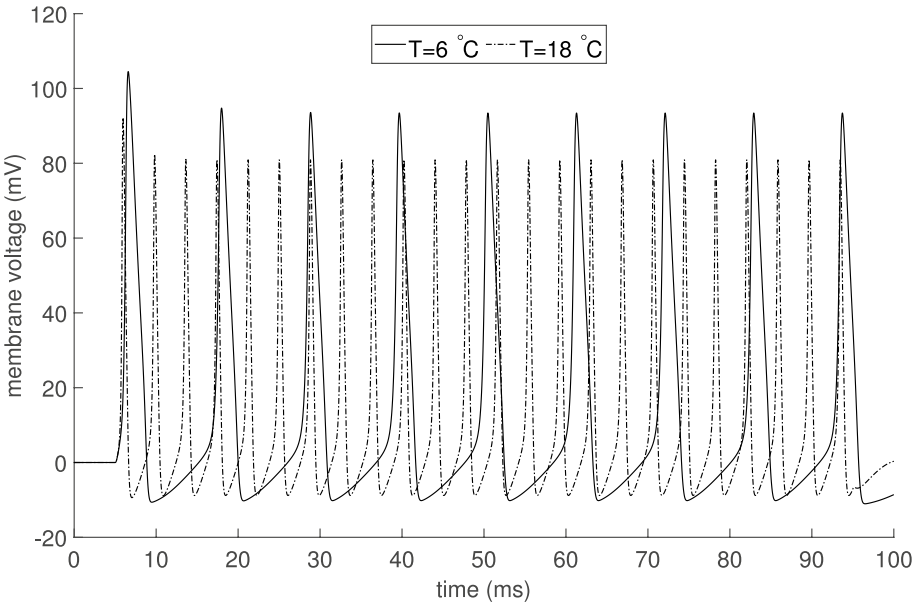


Fig. 8 Responses of the HH 10-neuron network model to applied temperature $T = 6\,°C$ and $T = 18\,°C$

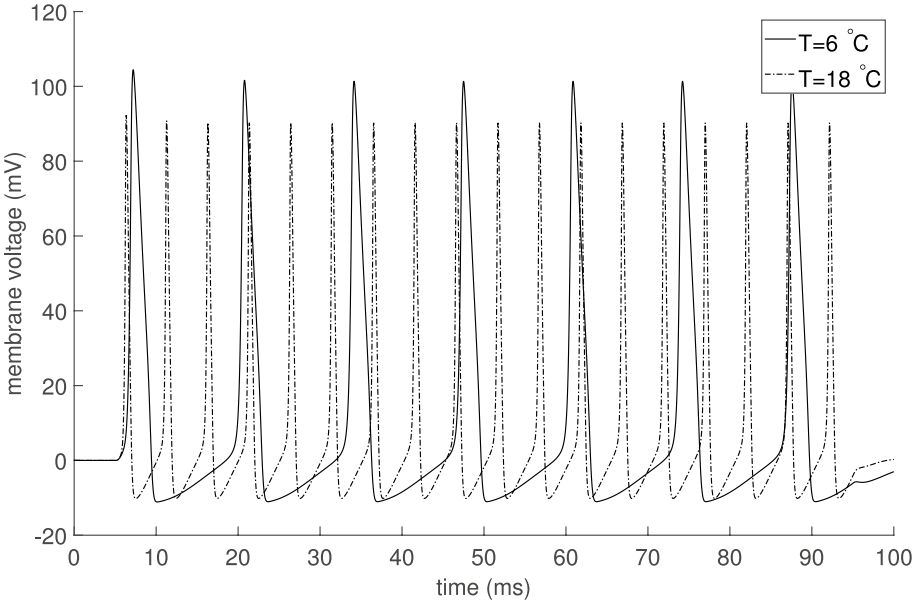


Fig. 9 Responses of the HH 50-neuron network model to applied temperature $T = 6\,^{\circ}C$ and $T = 18\,^{\circ}C$

environment. It is clear that the sensitivity to temperature in the given constant cell membrane parameters m and h is considered a significant evolution like a single neuron. By

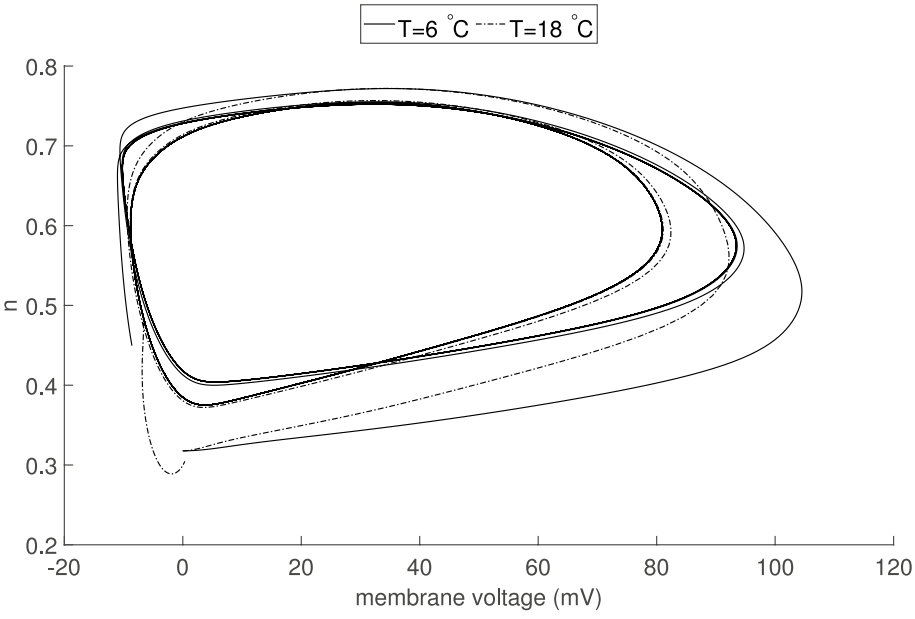


Fig. 10 Shows gating variable (n) versus the potential (V(mV)) at $T = 6\,^{\circ}C$ and $T = 18\,^{\circ}C$ (The number of neurons is 10)

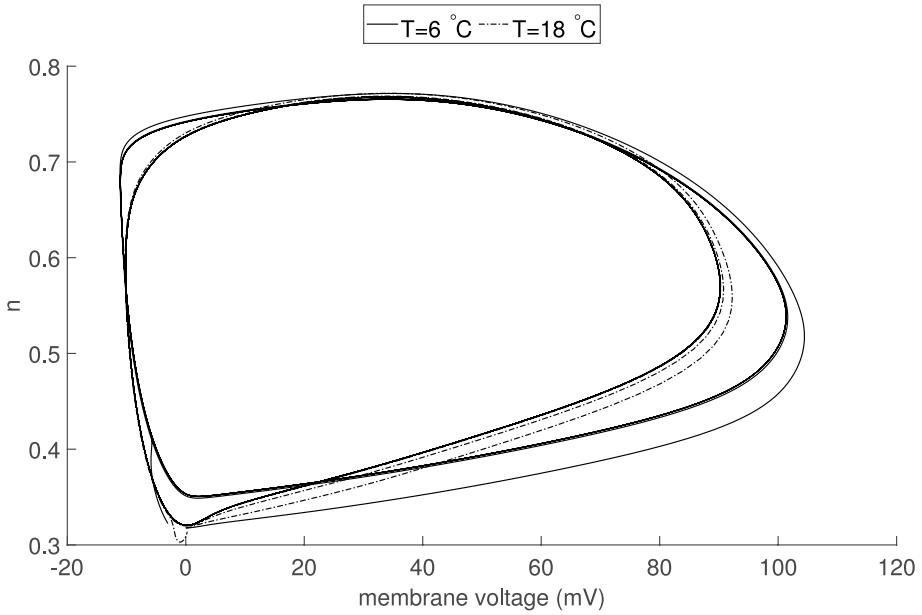


Fig. 11 Shows gating variable (n) versus the potential (V(mV)) at $T = 6\,°C$ and $T = 18\,°C$ (The number of neurons is 50)

changing the temperature from 0 °C to 18 °C, the action potential amplitude decreases, representing that resting membrane conductances start to change membrane potential

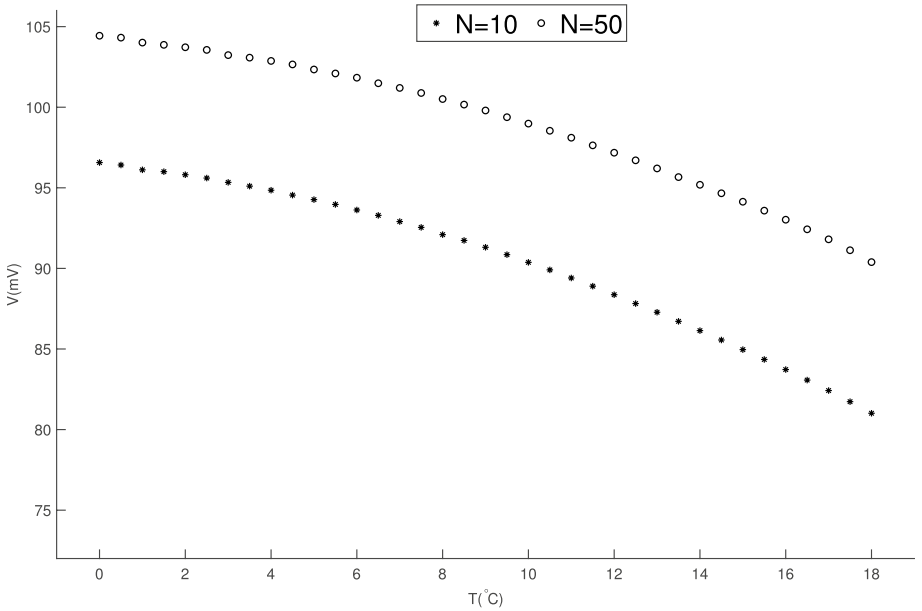


Fig. 12 Under diverse temperature stimulation, it is seen that the potential reduces

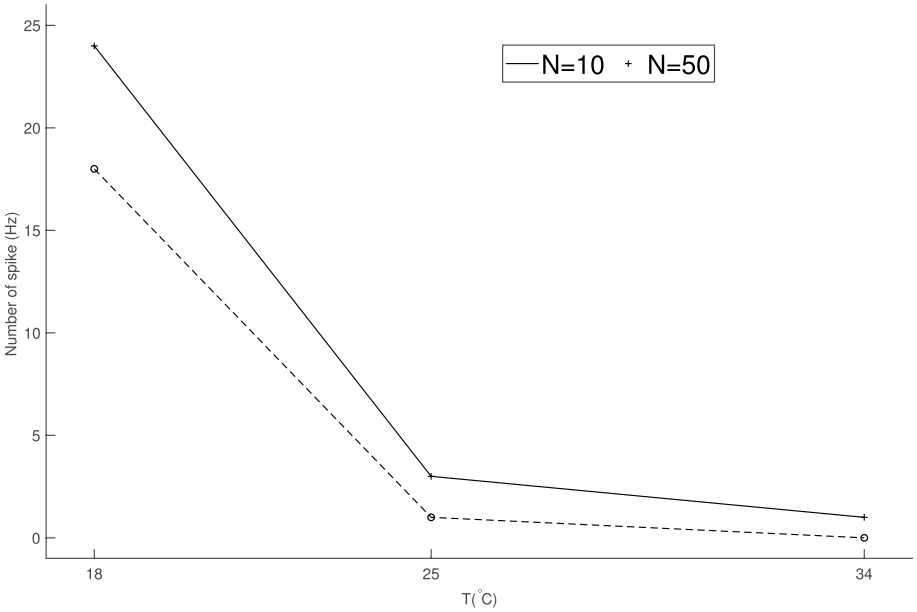


Fig. 13 The number of spikes at $T = 18\,^{\circ}C$, room temperature $T = 25\,^{\circ}C$, and high temperature $T = 34\,^{\circ}C$

performance of any neuron which is chosen from the network of the considered neurons. At room temperature, it is also observed that the number of spikes decreases. At warm temperatures, only one action potential is occurred in the 10-neuron network, while no spike is observed in the 50-neural network (Fig. 13).

The firing patterns of the HH network neuron model can change when the system is under different parameters beyond a certain threshold. The threshold dynamic is investigated by using a diagram of the interspike interval (ISI) as varying the external forcing. In this study, the temperature is an external forcing in the neuronal network. The valuations of ISI during the rising temperature were lesser than those during the falling periods of the temperature change (Fig. 14). The mean interspike interval for each network ($N = 10$ and $N = 50$) decreases like a logarithmic function by increasing the temperature.

To evaluate our theoretical and simulation results, let's consider the experimental studies. Nam Gyu Hyun et al. performed experiments using Aplysia neurons to show that the action potential amplitude decreases by raising the temperature and the spiking frequency parameter increases, which are completely consistent with our simulation results [28].

Recently, Van Hook studied the temperature influence on ion channels function, and synaptic properties in thalamocortical relay neurons [29]. He found that temperature variation has an important consequence on neuronal function and synaptic integration, and it may affect neuronal physiology by acting on the cell's ion channels. Further, it is shown that increasing the temperature led to a general reticence of synaptically-driven spiking performance in thalamocortical relay neurons. Moreover, heating the superfusate caused stimulation of a repairing potassium current and changed the voltage-gated Na^+ and K^+ currents.

On the other hand, the investigation by Kim et al. showed that high temperature can cause different mode transitions in the electrical activities of the neuron [30]. They illustrated the impacts of hyperthermia on the hippocampus in vitro by recording from pyramidal neurons and inhibitory oriens-lacunosum molecular internuncial neurons. Increasing temperatures to 41

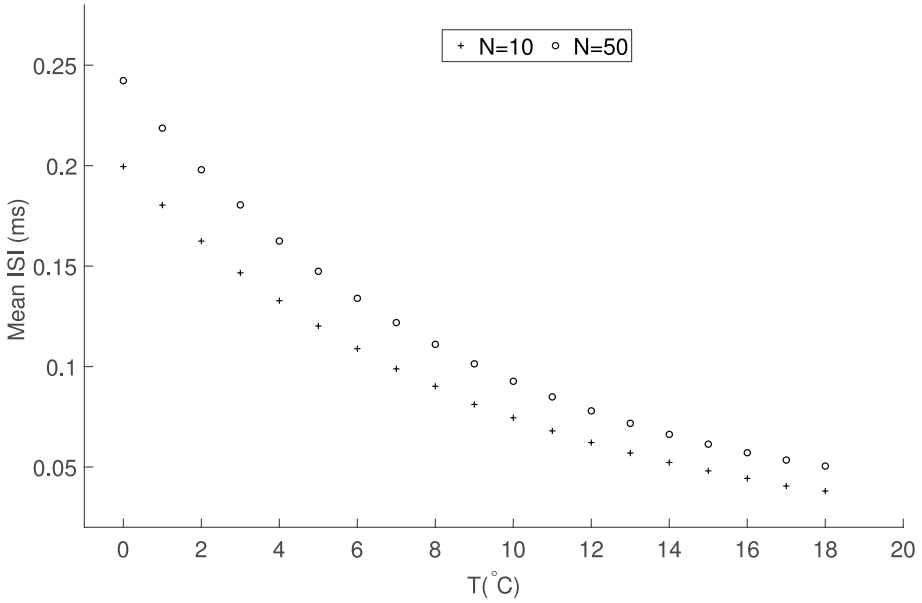


Fig. 14 A bifurcation diagram of the interspike interval (ISI) as a function of the temperature, which varies from $T = 0\,°C$ to $T = 18\,°C$

$°C$ induced depolarization, spontaneous action potentials, decreased input resistance and membrane time constant, and increased spontaneous synaptic activity in most pyramidal cells and oriens-lacunosum molecular interneurons. Their recording of patch-clamp showed that exposure to hyperthermia temperatures caused pyramidal neurons to depolarize and fire spontaneously.

In the study by Graham and et al. on mouse superficial dorsal horn neurons, they discovered that increasing the temperature from 22 $°C$ to 32 $°C$ not only reduces input resistance but also increases the percentage of neurons that do not fire action potentials from 2% to 13% [31]. At higher temperatures, all superficial dorsal horn neurons had lower input resistance and shorter action potentials.

In this study, the results obtained from the simulation of a biological neural network are consistent with Van Hook's results. Also, in the studied network, which is based on the HH model, the results are in agreement with Van Hook's work.

5 Conclusion

The study of the neural network provides a suitable approach to find the moderation of the activities and behavior of neurons concerning external parameters such as temperature. In this work, the effect of temperature on a single neuron and a neural network consisting of identical neurons was investigated based on the HH model. Actually, the neuronal network is a linear one that stimulates adjacent cells by synaptic current, the temperature process can affect the behavior of the network by changing the sodium and potassium ion gates. Temperature can be added by factor Q_{10} in the HH model. In this study, the effects

of temperature exposure on neuronal spike properties of the action potential are systematically investigated. Although the temperature change is not the direct factor for stimulating the cell, it changes the spiking pattern in constant stimulation current.

The simulation of a single neuron shows that the spiking frequency and amplitude of the spikes reduce significantly by increasing the temperature. Due to the temperature changes from $0\,^{\circ}C$ to $18\,^{\circ}C$, the number of spikes increases approximately 2.6 times, and the frequency of the spikes are approximately 1.4 times more. In fact, by increasing temperature, membrane hyper-polarization and depolarization occur faster, so, spikes are produced earlier. The accelerated v-gated K^+ conductance underlie this, as it allows a pull toward K^+ equilibrium to be activated earlier during the spike, thereby truncating the upward Na-dominated phase. By keeping factors such as stimulus current, capacity, conduction, and type of neuron constant, change in the potential pattern of action occurs by all α and β values. This indicates that membrane potential at temperatures of fewer rises more than that of higher temperatures. It is noteworthy that the results obtained in the simulated heat transfer method in this study are compatible with the changes in temperature caused by infrared irradiation.

In two neural networks contain 10 and 50 neurons, according to Fig. 14, the ISI decreased exponentially, which implies that with increasing temperature, spikes occur faster. It should be noted that using the above simulation method at room temperature and $35\,^{\circ}C$, it is observed that the results are consistent with Van Hook's experimental findings.

Another way to look at ion channel behavior during spiking is to examine activation and inactivation states. Plotting the trajectory of the activation or inactivation variable against the membrane voltage generates a phase portrait or phase-plane. At higher temperatures, the phase-plane has a stable limit cycle, indicating stable, repetitive action potential firing with low deviations, while this is not seen at lower temperatures. Finally, the results show that the change of the temperature of the body can widely generate a different pattern of the action potential. A warm body conducted less potassium and sodium ion than a cold body. This causes the messaging of the nervous system to function differently.

It seems that further investigation is needed to study an external magnetic field and variation of temperature effects on a neurological disorder system.

Declarations

Conflicts of interest The authors declare that they have no conflict of interest.

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