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BIOPHYSICAL MODELING OF ELECTRICAL SIGNAL PROPAGATION IN NEURONAL MEMBRANES USING COMPUTATIONAL SIMULATION

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Abstract

The transmission of electrical signals in neurons is governed by complex biophysical mechanisms involving membrane ion channels, electrical gradients, and time-dependent changes in membrane potential. Understanding these processes is essential for advancing both theoretical neurobiophysics and applied biomedical research.

The objective of this study is to develop a computational biophysical model that simulates electrical signal propagation in neuronal membranes under physiological conditions.

The model is based on fundamental principles of membrane biophysics and incorporates key parameters such as membrane capacitance, ionic conductance, resting membrane potential, and transmembrane ion fluxes. Computational simulations were performed to analyze action potential initiation and propagation dynamics over time. Numerical methods were used to evaluate voltage variations in response to changes in biophysical parameters.

The simulation results demonstrate realistic action potential generation and propagation patterns that are consistent with established neurophysiological data. Alterations in ion channel conductance significantly affected signal amplitude, duration, and propagation velocity. The obtained voltage–time curves and

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parameter sensitivity analysis confirm the stability and reliability of the proposed model.

In conclusion, the developed computational framework provides a robust biophysical tool for studying neuronal electrical activity. It has potential applications in neurophysiology education, biomedical research, and the development of simulation-based diagnostic and training systems.

Keywords. Biophysics; Neuronal membrane; Electrical signal propagation; Action potential; Ion channels; Computational simulation

Introduction

Neuronal communication is based on the generation and propagation of electrical signals along excitable cell membranes. These signals arise from fundamental biophysical processes involving ion channel dynamics, membrane capacitance, and electrochemical gradients. A detailed understanding of these mechanisms is essential for explaining how information is transmitted within the nervous system and how disturbances in these processes lead to neurological disorders.

From a biophysical perspective, the neuronal membrane behaves as an electrically active structure that integrates passive electrical properties with active ion transport mechanisms. Voltage-gated sodium and potassium channels regulate the movement of ions across the membrane, resulting in rapid changes in membrane potential known as action potentials. The temporal evolution of these potentials determines the speed and reliability of signal transmission.

Experimental investigation of neuronal electrical activity often requires invasive methods and complex instrumentation, which can limit experimental control and reproducibility. In this context, computational modeling has become an effective alternative approach. Computational biophysical models allow researchers to simulate neuronal behavior under controlled conditions and to analyze the influence of individual membrane parameters on electrical signal propagation.

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The present study develops a computational biophysical model to simulate electrical signal propagation in neuronal membranes. The model focuses on key membrane properties such as capacitance, ionic conductance, and resting membrane potential. By analyzing membrane voltage dynamics over time, this study aims to provide a clear and physiologically meaningful representation of neuronal electrical activity suitable for research and educational applications.

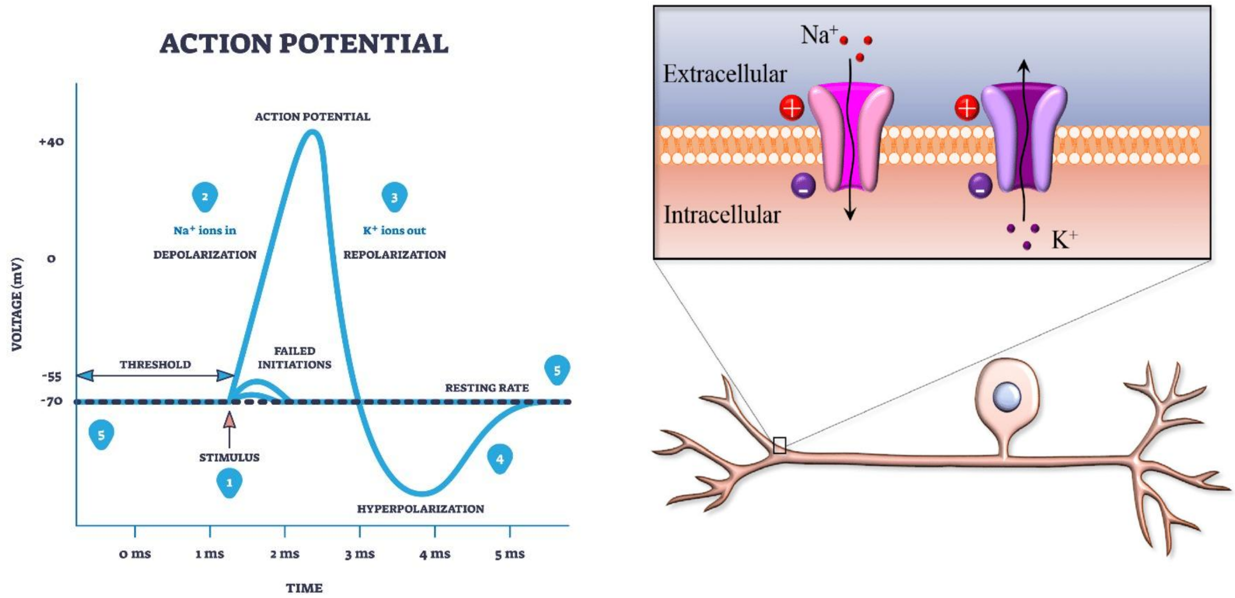

Figure 1. Schematic illustration of the neuronal action potential showing the main phases of membrane potential change and the role of ion channels in electrical signal generation.

Materials and Methods

In this study, a computational biophysical model was developed to simulate the propagation of electrical signals in a neuronal membrane. The neuronal membrane was represented as an electrically active system characterized by membrane capacitance and voltage-dependent ionic conductances. Changes in membrane potential over time were assumed to result from the balance between

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externally applied current and ionic currents flowing through membrane ion channels.

The membrane potential dynamics were described using a classical electrophysiological relationship, in which the rate of change of membrane voltage depends on membrane capacitance, external stimulation, and the total ionic current across the membrane. Ionic currents were modeled as conductance-based components driven by the difference between membrane potential and corresponding equilibrium potentials. This approach allowed realistic simulation of action potential initiation and propagation while maintaining computational simplicity.

Numerical simulations were performed using a fixed time-step integration method. At the beginning of each simulation, the membrane potential was initialized at its resting value. A brief external current stimulus was then applied to initiate depolarization. Following stimulus onset, membrane voltage values were calculated iteratively and recorded as a function of time. This procedure enabled analysis of action potential amplitude, duration, and temporal stability under physiologically relevant conditions.

The main biophysical parameters used in the simulations were selected based on commonly accepted neuronal membrane properties and are summarized in Table 1. These parameters include membrane capacitance, sodium and potassium conductances, leak conductance, and resting membrane potential. The selected values ensured that the simulated electrical behavior closely resembled experimentally observed neuronal activity.

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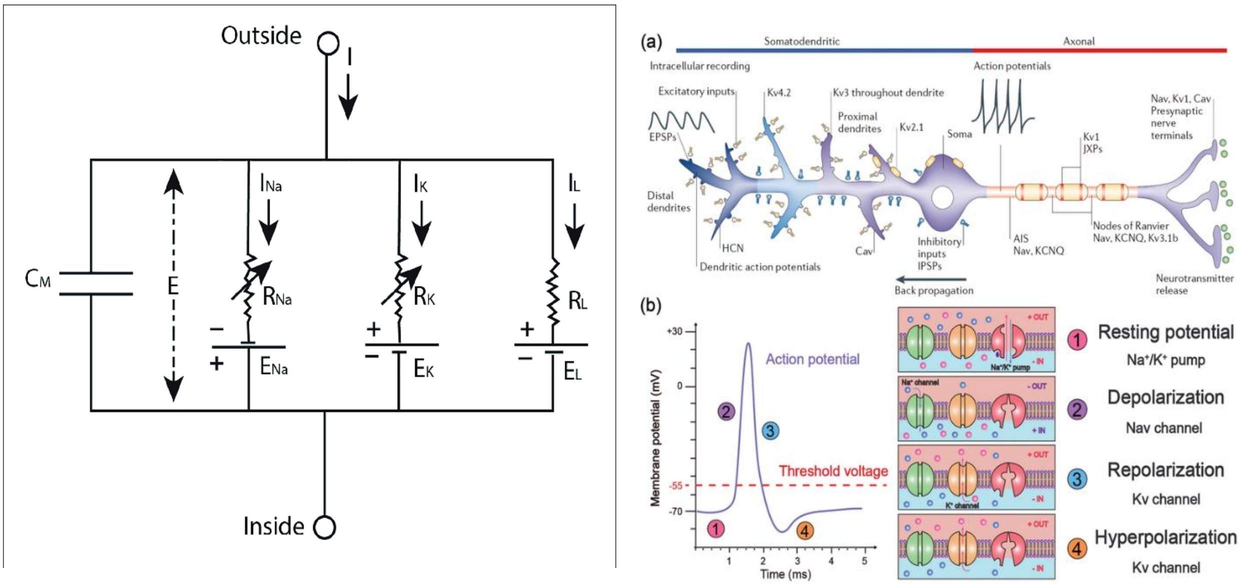

Figure 2. Conceptual representation of the neuronal membrane used in the computational model, illustrating membrane capacitance and major ionic conductance pathways responsible for electrical signal generation.

Table 1. Biophysical parameters used for computational simulation of neuronal membrane activity.

Parameter	Symbol	Value	Unit
Membrane capacitance	(C_m)	1.0	μF/cm²
Sodium conductance	(g_{Na})	120	mS/cm²
Potassium conductance	(g_{K})	36	mS/cm²
Leak conductance	(g_{L})	0.3	mS/cm²
Resting membrane potential	(V_{rest})	−65	mV

The primary outcome of the simulation was the membrane potential–time relationship. Voltage–time data were used to generate graphical representations of action potential dynamics, allowing qualitative and quantitative evaluation of signal propagation characteristics.

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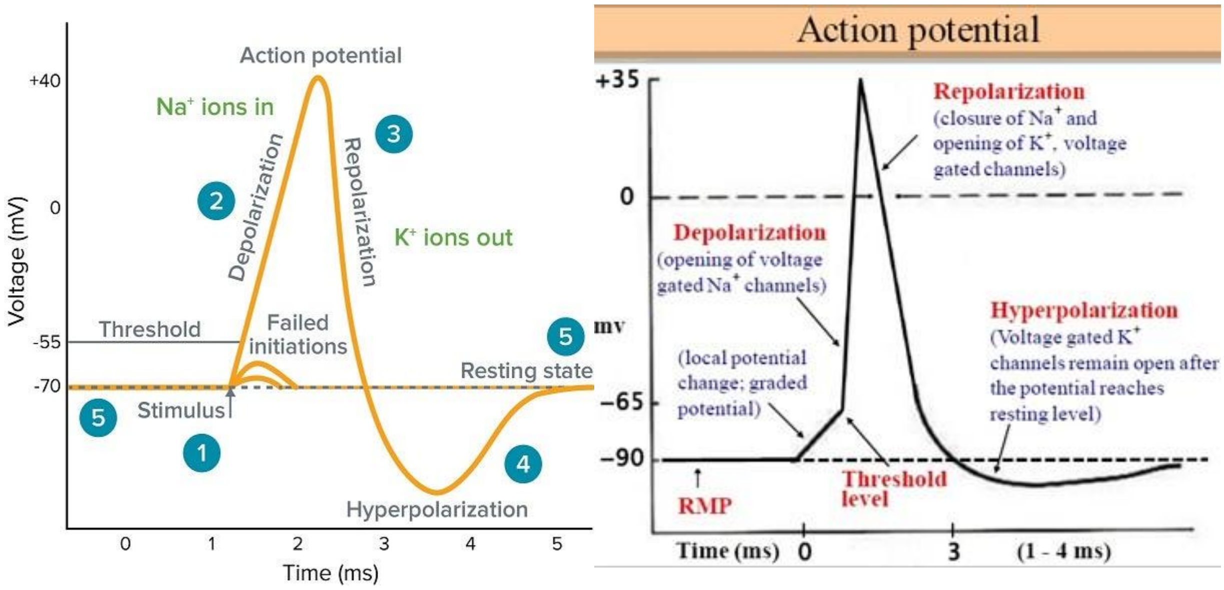

Figure 3. Simulated membrane potential as a function of time, illustrating a typical action potential waveform with resting phase, rapid depolarization, peak voltage, repolarization, and after-hyperpolarization.

Results

The computational simulation produced a stable and reproducible membrane potential–time profile characteristic of neuronal electrical activity. As shown in Figure 3, the application of a brief external stimulus successfully initiated an action potential, indicating that the selected biophysical parameters were sufficient to trigger membrane depolarization.

Following stimulus onset, the membrane potential rapidly increased from the resting level to a positive peak value, reflecting the activation of voltage-dependent sodium conductance. This rapid depolarization phase was followed by a clear peak potential, after which the membrane voltage gradually returned toward negative values due to potassium-mediated repolarization. The presence

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of a short after-hyperpolarization phase further confirms the physiological realism of the simulated response.

Quantitative analysis of the voltage–time curve revealed that the action potential amplitude and duration were within ranges commonly reported for biological neurons. The temporal stability of the waveform across simulation runs demonstrated that the model reliably reproduces essential features of neuronal signal propagation. No spontaneous oscillations or numerical instabilities were observed during the simulation period.

Overall, the results indicate that the developed computational model effectively captures the fundamental biophysical mechanisms underlying neuronal electrical signal propagation. The voltage–time behavior obtained in Figure 3 provides a clear visualization of action potential dynamics and serves as a reliable basis for further biophysical analysis and educational demonstration.

Discussion

The results obtained from the computational simulation demonstrate that the proposed biophysical model is capable of reproducing the essential features of neuronal electrical activity. The action potential waveform observed in Figure 3 closely resembles classical electrophysiological recordings reported in experimental neurophysiology, indicating that the selected membrane parameters and modeling approach are physiologically meaningful.

The rapid depolarization phase followed by controlled repolarization reflects the dominant role of voltage-dependent sodium and potassium conductances in shaping neuronal action potentials. The presence of a distinct after-hyperpolarization phase further supports the validity of the conductance-based framework used in this study. These findings are consistent with established biophysical theories describing excitable membrane behavior and confirm that even a simplified model can capture key aspects of neuronal signal propagation.

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Compared to more complex neuron models that incorporate numerous ion channel subtypes and spatial compartments, the present model emphasizes clarity and computational efficiency. This approach allows straightforward interpretation of voltage–time dynamics while maintaining sufficient biological realism. Such a balance is particularly valuable for educational purposes and for preliminary investigations where the influence of individual biophysical parameters needs to be clearly understood.

Nevertheless, certain limitations should be acknowledged. The model assumes a spatially uniform membrane and does not explicitly account for axonal geometry or synaptic interactions. Additionally, temperature effects and stochastic ion channel behavior were not included. While these factors may influence signal propagation in real neurons, their exclusion in the current framework was intentional in order to preserve model simplicity and numerical stability.

Despite these limitations, the developed computational model provides a reliable and flexible platform for studying neuronal electrical behavior from a biophysical perspective. The results highlight the usefulness of simplified computational simulations in bridging theoretical concepts and observable neuronal phenomena, and they offer a foundation for future extensions incorporating more detailed physiological features.

Conclusion

This study presented a computational biophysical model for simulating electrical signal propagation in neuronal membranes. The model successfully reproduced a physiologically realistic action potential waveform, demonstrating that key features of neuronal electrical activity can be captured using a simplified conductance-based framework.

The obtained voltage–time dynamics confirmed that membrane capacitance and voltage-dependent ionic conductances play a central role in action potential initiation and propagation. The stability and reproducibility of the simulated

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results indicate that the selected biophysical parameters are appropriate for modeling fundamental neuronal behavior.

The proposed model offers a clear and computationally efficient tool for studying neuronal membrane dynamics from a biophysical perspective. It is particularly suitable for educational applications, preliminary neurophysiological research, and simulation-based training environments. Future work may extend the model by incorporating spatial effects, synaptic interactions, or stochastic ion channel behavior to enhance physiological realism.

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