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Linear Approximation of the Nonlinear Hodgkin–Huxley Model Using First-Order Taylor Expansion for Local Neuronal Behavior Analysis

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Keyword

Hodgkin–Huxley model, Linearization, Taylor series expansion, Neuronal dynamics, Stability analysis

Abstract

The Hodgkin–Huxley model is one of the most important mathematical models used to describe neuronal membrane dynamics and action potential generation. However, the nonlinear structure of the model makes analytical investigation difficult, particularly near equilibrium conditions. This study presents a mathematical linearization of the Hodgkin–Huxley model around the resting membrane potential using first-order Taylor series expansion. The equilibrium point of the system was determined, and perturbation variables representing small deviations from equilibrium were introduced. The sodium, potassium, and leakage currents were then linearized by evaluating partial derivatives at the equilibrium point. In addition, the gating-variable equations were linearized to obtain a complete system of coupled linear differential equations. The resulting model preserves the essential local behavior of the original nonlinear system while providing a mathematically tractable framework for studying neuronal dynamics. The derived linearized system enables the application of analytical methods such as stability analysis and local dynamic response analysis. Overall, the study demonstrates the importance of linearization in simplifying complex neuronal models and facilitating mathematical investigation in computational neuroscience and biomedical engineering.

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التقريب الخطي لنموذج هودجكن–هاكسلي غير الخطي باستخدام متسلسلة تايلور من الدرجة الأولى لتحليل السلوك العصبي المحلي

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الكلمة المفتاحية: *نموذج هودجكين-هكسلي، الخطية، توسيع سلسلة تايلور، ديناميكيات الخلايا العصبية، تحليل الاستقرار*

الملخص

يُعد نموذج هودجكن–هاكسلي من أهم النماذج الرياضية المستخدمة لوصف ديناميكية الغشاء العصبي وتوليد جهد الفعل. إلا أن الطبيعة غير الخطية للنموذج تجعل التحليل الرياضي المباشر أمرًا معقدًا، خصوصًا بالقرب من نقاط الاتزان. تهدف هذه الدراسة إلى خطيّة نموذج هودجكن–هاكسلي حول جهد الغشاء في حالة الراحة باستخدام متسلسلة تايلور من الدرجة الأولى. تم تحديد نقطة الاتزان للنظام، ثم تعريف متغيرات اضطراب تمثل الانحرافات الصغيرة عن حالة الاتزان. بعد ذلك تم خطيّة تيارات الصوديوم والبوتاسيوم والتيار المتسرب من خلال حساب المشتقات الجزئية عند نقطة الاتزان. كما تم خطيّة معادلات متغيرات البوابات للحصول على نظام كامل من المعادلات التفاضلية الخطية المترابطة. حافظ النموذج الناتج على السلوك المحلي الأساسي للنظام غير الخطي الأصلي مع توفير إطار رياضي أبسط لتحليل ديناميكية الخلايا العصبية. كما أتاح النموذج الخطي تطبيق أساليب تحليلية مثل تحليل الاستقرار وتحليل الاستجابة الديناميكية المحلية. وتُبرز الدراسة أهمية الخطيّة في تبسيط النماذج العصبية المعقدة وتسهيل دراستها رياضيًا في مجال علوم الأعصاب الحاسوبية والهندسة الطبية الحيوية.

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1. Introduction

The quantitative study of neuronal activity represents a fundamental intersection between mathematics and medicine. Neurons communicate through electrical signals known as action potentials, which arise from complex interactions between ionic currents across the cell membrane. These processes are essential for normal physiological function and are closely linked to a wide range of neurological disorders, including epilepsy and neurodegenerative diseases (Kandel et al., 2021, pp. 150–165; Dayan & Abbott, 2001, pp. 15–22).

Among the mathematical models developed to describe neuronal dynamics, the Hodgkin–Huxley model remains one of the most influential. Introduced in 1952, this model provides a detailed representation of ionic currents through voltage-gated channels using a system of nonlinear differential equations (Hodgkin & Huxley, 1952, pp. 500–510). It captures the dynamics of sodium and potassium conductances and explains how these contribute to the generation of action potentials. Despite its biological accuracy, the

nonlinear structure of the model presents significant challenges for analytical investigation (Izhikevich, 2007, pp. 10–15; Ermentrout & Terman, 2010, pp. 45–50).

From a mathematical perspective, nonlinear systems such as the Hodgkin–Huxley model are difficult to analyze due to the absence of explicit solutions and the complexity of their dynamics. In biomedical contexts, it is particularly important to understand neuronal behavior near equilibrium, where small perturbations may either decay or grow. However, direct analysis of nonlinear systems is often not feasible, requiring approximation techniques such as linearization (Khalil, 2002, pp. 120–125; Strogatz, 2018, pp. 135–140).

2. Problem Statement

The Hodgkin–Huxley model is formulated as a system of coupled nonlinear differential equations involving the membrane potential and gating variables. While this formulation accurately represents the underlying biological processes, it introduces significant mathematical complexity. In particular, the nonlinear interactions between variables make it difficult to obtain analytical solutions or to directly assess system stability (Hodgkin & Huxley, 1952; Khalil, 2002).

From a physiological perspective, understanding neuronal behavior near equilibrium is of critical importance. Many neurological conditions arise from small deviations in membrane potential or ion channel dynamics, which may lead to significant changes in neuronal activity. However, due to the nonlinear nature of the model, predicting the evolution of such perturbations remains challenging (Kandel et al., 2021).

Therefore, there is a need for a mathematical approach that simplifies the system while preserving its essential behavior near the equilibrium point. Linearization provides such an approach by approximating the nonlinear system with a linear one, allowing for a more tractable analysis of local dynamics. The central problem addressed in this study is the derivation of a linear approximation of the Hodgkin–Huxley model that accurately captures its behavior near equilibrium.

3. Objectives of the Study

The main objective of this study is to develop a rigorous mathematical framework for the linearization of the Hodgkin–Huxley model. This involves transforming the nonlinear system into a linear one that can be analyzed using standard mathematical techniques.

The specific objectives are as follows:

1. Present the Hodgkin–Huxley model in a mathematical form suitable for analysis.
2. Determine the equilibrium point corresponding to the resting state of the neuron.
3. Introduce perturbation variables representing small deviations from equilibrium.

4. Apply first-order Taylor series expansion to linearize the system.

4. Previous Studies

The Hodgkin–Huxley model has been extensively studied since its introduction and remains a cornerstone of mathematical neuroscience. The original work by Hodgkin and Huxley (1952) provided the first quantitative description of ionic currents in neurons and established a nonlinear framework for modeling action potentials (Hodgkin & Huxley, 1952, pp. 500–544).

Subsequent research has expanded the mathematical analysis of neuronal systems. Izhikevich (2007) examined neuronal dynamics using nonlinear dynamical systems theory, highlighting the importance of excitability and oscillations (pp. 10–20). Similarly, Koch (2004) explored the biophysical basis of neuronal computation, emphasizing the role of ion channel dynamics (pp. 25–35). Gerstner et al. (2014) further extended these concepts to network-level modeling, integrating biological realism with computational frameworks (pp. 50–60).

From a broader mathematical perspective, Keener and Sneyd (2009) provided a comprehensive treatment of physiological systems using differential equations (pp. 120–130), while Ermentrout and Terman (2010) focused on stability and qualitative analysis in neuronal models (pp. 45–55). More recent studies have also investigated advanced modeling approaches, including neuron–glia interactions and their role in neural signaling (Tewari et al., 2016, pp. 1–5; Poznanski, 2020, pp. 1–6).

The technique of linearization has been widely developed in nonlinear system analysis. Khalil (2002) introduced systematic methods based on Taylor expansion and Jacobian matrices (pp. 120–130), while Strogatz (2018) demonstrated how linearization can be used to analyze stability near equilibrium points (pp. 135–145). These methods form the theoretical foundation of the present study.

5. Action Potential as a Basis for Mathematical Modeling

The action potential is a rapid variation in membrane potential resulting from ion movement through voltage-gated channels. At rest, the membrane maintains a stable potential due to ion concentration gradients. When stimulated, sodium channels open, causing depolarization, followed by potassium channel activation, which restores the membrane potential through repolarization (Kandel et al., 2021, pp. 150–165; Dayan & Abbott, 2001, pp. 15–22).

From a mathematical perspective, the action potential can be interpreted as the response of a dynamical system in which the membrane potential evolves over time under ionic currents. These currents can be decomposed into sodium, potassium, and leakage

components, allowing the system to be represented using differential equations (Hodgkin & Huxley, 1952, pp. 500–506).

This abstraction enables the translation of physiological processes into mathematical form, forming the foundation for the Hodgkin–Huxley model and subsequent analytical methods such as linearization (Izhikevich, 2007, pp. 10–15; Keener & Sneyd, 2009, pp. 120–125).

6. The Hodgkin–Huxley Model and Its Mathematical Formulation

The model represents neuronal membrane dynamics using an electrical circuit analogy, in which the lipid bilayer is modeled as a capacitor and ion channels are represented as conductive pathways. This analogy allows the application of fundamental electrical principles to biological systems, enabling a quantitative description of membrane behavior.

In this framework, sodium (Na^+), potassium (K^+), and leakage channels are modeled as parallel branches, each contributing to the total membrane current. Such circuit-based representations are widely used in computational neuroscience due to their ability to bridge physical intuition and biological complexity (Koch, 2004; Dayan and Abbott, 2001).

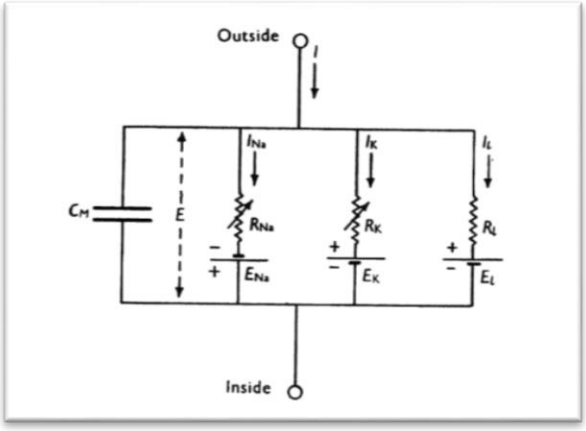

FIGURE 1: An Electrical Circuit Describes the Action Potential

The membrane capacitance C_m accounts for the ability of the membrane to store electrical charge, while ionic channels are modeled as resistive elements or, equivalently, conductances. The leakage channel is typically assumed to have a constant conductance, representing passive ion diffusion across the membrane. In contrast, sodium and potassium channels exhibit voltage-dependent behavior, meaning that their conductances vary dynamically in response to changes in membrane potential. This voltage sensitivity is a key feature underlying neuronal excitability and signal propagation (Kandel et al., 2021; Alberts et al., 2002).

6-1. Physiological Basis and Derivation from Action Potential

The equations were derived using the voltage-clamp technique, which allowed precise measurement of ionic currents while controlling membrane potential. This enabled the separation of sodium and potassium currents and the identification of their roles in action potential generation (Hodgkin & Huxley, 1952, pp. 500–510; Schwiening, 2012, p. 2572).

These experimental observations led to the formulation of mathematical expressions describing how ionic currents depend on voltage and time, forming the basis of the model.

6-2. The Hodgkin-Huxley Equations

The dynamics of ion currents are described using key variables, including the membrane potential (V_m) and conductances of the channels (g_K,g_{Na}, g_l). The main equation governing the membrane potential is:

$$\mathbf{I} = \boldsymbol{C_m}\frac{\boldsymbol{dV_m}}{\boldsymbol{dt}} + \boldsymbol{g_K}(\boldsymbol{V_m} - \boldsymbol{V_K}) + \boldsymbol{g_{Na}}(\boldsymbol{V_m} - \boldsymbol{V_{Na}}) + \boldsymbol{g_l}(\boldsymbol{V_m} - \boldsymbol{V_l})$$

Where:

- I: Total current,
- C_m: Membrane capacitance,
- g_K, g_{Na}, g_l: Conductances for potassium, sodium, and leaky channels,
- V_K, V_{Na}, V_l: Reversal potentials for potassium, sodium, and leaky channels,
- V_m: Membrane potential.

While the leaky conductance (g_l) is constant, potassium and sodium conductances (g_K, g_{Na}) vary with membrane potential, reflecting the channels' gating dynamics.

This model not only provides insights into how action potentials are generated and propagated but also offers a quantitative framework for understanding neural behavior in electrophysiology. (Hodgkin & Huxley, 1952, pp. 500–544)

Changes in Sodium and Potassium Conductance:

Sodium conductance (g_{Na}) is regulated by activation and inactivation particles that move between the intracellular and extracellular sides of the membrane. The activation particles (m) are located inside the membrane, while the inactivation particles (h) are outside. The sodium conductance is expressed as:

$$\boldsymbol{g_{Na}} = \overline{\boldsymbol{g_{Na}}}\,\mathbf{m^3h}$$

Where:

g_{Na} represents the maximum sodium conductance. The behavior of m and h are described by the following differential equations:

$$\frac{dm}{dt} = \alpha_m (V_m)(1 - \mathbf{m}) - \beta_m (V_m)\mathbf{m}$$

$$\frac{dh}{dt} = \alpha_h (V_m)(1 - \mathbf{h}) - \beta_h (V_m)\mathbf{h}$$

The transfer rates of the activation (α_m, β_m) and inactivation (α_h, β_h) particles depend on the membrane potential (V_m) but are independent of time.

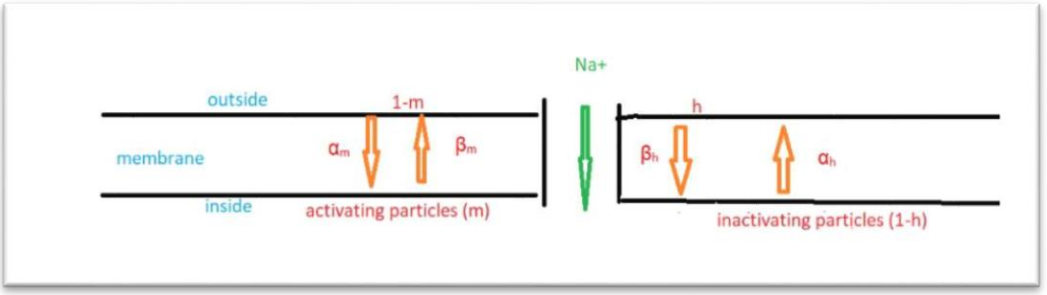

FIGURE 2: Changes in Sodium Conductance

Potassium conductance (g_K) is controlled solely by activation particles (n), located inside the membrane. The potassium conductance is given by:

$$\boldsymbol{g_k} = \overline{\boldsymbol{g_k}}\, \mathbf{n^4}$$

Where:

$\overline{\boldsymbol{g_k}}$ is the maximum potassium conductance. The behavior of n is described by the differential equation:

$$\frac{dn}{dt} = \alpha_n (V_m)(1 - \mathbf{n}) - \beta_n (V_m)\mathbf{n}$$

Where:

$\boldsymbol{\alpha_n}$ and $\boldsymbol{\beta_n}$ are the transfer rates of the activation particles, which depend on the membrane potential. Similar differential equations apply to the m and h particles for sodium channels. (Hodgkin & Huxley, 1952, pp. 500–544).

By integrating the membrane potential equation with the equations for n, m, and h, we derive a system of four nonlinear ordinary differential equations, collectively referred to as the Hodgkin-Huxley equations:

$$I = C_m \frac{dV_m}{dt} + \overline{g_{\square}}\, n^{\square}(V_m - V_K) + \overline{g_{Na}}\, m^{\square\square}(V_m - V_{Na}) + g_l(V_m - V_l)$$

$$\frac{dn}{dt} = \alpha_n(V_m)(1 - n) - \beta_n(V_m)n$$

$$\frac{dm}{dt} = \alpha_m(V_m)(1 - m) - \beta_m(V_m)m$$

$$\frac{dh}{dt} = \alpha_h(V_m)(1 - h) - \beta_h(V_m)h$$

7. Linearization of the Hodgkin–Huxley Model

The linearization of the Hodgkin–Huxley model is performed around the equilibrium point corresponding to the resting membrane potential (V_0, m_0, h_0, n_0). The procedure begins by introducing perturbation variables representing small deviations from the equilibrium values of the membrane voltage and gating variables.

After defining the perturbation variables, the nonlinear ionic current equations are expanded around the equilibrium point using a first-order Taylor series expansion. During this process, only first-order terms are retained, while higher-order nonlinear terms are neglected because they are assumed to be sufficiently small near the equilibrium point (Khalil, 2002, pp. 120–130; Strogatz, 2018, pp. 135–145).

The linearization procedure is then applied separately to the sodium, potassium, and leakage currents. Partial derivatives with respect to the membrane potential and gating variables are evaluated at the equilibrium point to obtain the linearized current variations. These linearized current expressions are subsequently substituted into the membrane equation to derive the linearized voltage dynamics.

Similarly, the gating-variable equations are linearized by expanding the nonlinear activation and inactivation functions around the equilibrium point. This results in a set of coupled linear differential equations describing the local dynamics of the membrane voltage and gating variables under small perturbations (Gerstner et al., 2014, pp. 120–130; Brette & Destexhe, 2012, pp. 210–215).

The resulting linearized system preserves the essential local behavior of the original Hodgkin–Huxley model while providing a mathematically tractable framework for analytical investigation of neuronal dynamics (Poznanski, 2020, pp. 1–6).

7-1. Equilibrium Point

Let the state vector be defined as:

$$x = [V_m \quad m \quad h \quad n]^T$$

The equilibrium point is denoted by:

$$x_0 = [V_0 \quad m_0 \quad h_0 \quad n_0]^T$$

At equilibrium, all state derivatives are equal to zero:

$$\frac{dV_m}{dt} = \frac{dm}{dt} = \frac{dh}{dt} = \frac{dn}{dt} = 0$$

Therefore, the equilibrium input current satisfies:

$$I_0 = g_{Na}^{max} m_0^3 h_0 (V_0 - V_{Na}) + g_K^{max} n_0^4 (V_0 - V_K) + g_L (V_0 - V_L)$$

This condition represents the resting operating point of the neuron, where the applied current balances the sodium, potassium, and leakage currents. (Kandel et al., 2021, pp. 150–155).

7-2. Perturbation Variables

Small deviations from the equilibrium point are introduced as follows:

$$\delta V = V_m - V_0, \qquad \delta m = m - m_0, \qquad \delta h = h - h_0, \qquad \delta n = n - n_0$$

$$\delta I = I - I_0$$

Equivalently, each variable can be written as the sum of its equilibrium value and a small perturbation:

$$V_m = V_0 + \delta V, \qquad m = m_0 + \delta m, \qquad h = h_0 + \delta h, \qquad n = n_0 + \delta n$$

This transformation shifts the analysis from the absolute variables to the local deviations around the equilibrium point.

7-3. First-Order Taylor Expansion

For a nonlinear system written as $dx/dt = f(x, I)$, the first-order Taylor expansion around the equilibrium point is:

$$f(x, I) \approx f(x_0, I_0) + \left.\frac{\partial f}{\partial x}\right|_{x_0, I_0} (x - x_0) + \left.\frac{\partial f}{\partial I}\right|_{x_0, I_0} (I - I_0)$$

Since $f(x_0, I_0) = 0$ at equilibrium, the linearized model becomes:

$$\delta \dot{x} = A \delta x + B \delta I$$

Where:

$$A = \left.\frac{\partial f}{\partial x}\right|_{x_0, I_0}, \qquad B = \left.\frac{\partial f}{\partial I}\right|_{x_0, I_0}$$

The matrix A is the Jacobian matrix evaluated at the equilibrium point, while B describes how the external current perturbation enters the system.

7-4. Linearization of Ionic Currents

The ionic currents are linearized by retaining only first-order perturbation terms and neglecting higher-order products such as $\delta V \delta n$ or $\delta m \delta h$.

i. Potassium Current

The potassium current is

$$I_K = g_K^{max} n^4 (V_m - V_K)$$

The first-order variation is:

$$\delta I_K = \left.\frac{\partial I_K}{\partial V_m}\right|_0 \delta V + \left.\frac{\partial I_K}{\partial n}\right|_0 \delta n$$

Where:

$$\left.\frac{\partial I_K}{\partial V_m}\right|_0 = g_K^{max} n_0^4$$

and

$$\left.\frac{\partial I_K}{\partial n}\right|_0 = 4 g_K^{max} n_0^3 (V_0 - V_K)$$

Thus,

$$\delta I_K = g_K^{max} n_0^4 \delta V + 4 g_K^{max} n_0^3 (V_0 - V_K) \delta n$$

ii. Sodium Current

The sodium current is

$$I_{Na} = g_{Na}^{max} m^3 h (V_m - V_{Na})$$

The first-order variation is:

$$\delta I_{Na} = \left.\frac{\partial I_{Na}}{\partial V_m}\right|_0 \delta V + \left.\frac{\partial I_{Na}}{\partial m}\right|_0 \delta m + \left.\frac{\partial I_{Na}}{\partial h}\right|_0 \delta h$$

Where:

$$\left.\frac{\partial I_{Na}}{\partial V_m}\right|_0 = g_{Na}^{max} m_0^3 h_0$$

$$\left.\frac{\partial I_{Na}}{\partial m}\right|_0 = 3 g_{Na}^{max} m_0^2 h_0 (V_0 - V_{Na})$$

$$\left.\frac{\partial I_{Na}}{\partial h}\right|_0 = g_{Na}^{max} m_0^3 (V_0 - V_{Na})$$

Therefore,

$$\delta I_{Na} = g_{Na}^{max} m_0^3 h_0 \delta V + 3 g_{Na}^{max} m_0^2 h_0 (V_0 - V_{Na}) \delta m + g_{Na}^{max} m_0^3 (V_0 - V_{Na}) \delta h$$

iii. Leakage Current

The leakage current is

$$I_L = g_L (V_m - V_L)$$

Since it is linear in V_m, its perturbation is simply

$$\delta I_L = g_L \delta V$$

7-5. Linearized Voltage Equation

Substituting the linearized sodium, potassium, and leakage currents into the membrane equation gives the linearized voltage equation:

$$C_m \frac{d(\delta V)}{dt} = \delta I - k_V \delta V - k_m \delta m - k_h \delta h - k_n \delta n$$

Where:

$$k_V = g_{Na}^{max} m_0^3 h_0 + g_K^{max} n_0^4 + g_L$$

$$k_m = 3 g_{Na}^{max} m_0^2 h_0 (V_0 - V_{Na}), \qquad k_h = g_{Na}^{max} m_0^3 (V_0 - V_{Na})$$

$$k_n = 4 g_K^{max} n_0^3 (V_0 - V_K)$$

This equation shows how small changes in the membrane potential and gating variables affect the local voltage dynamics.

7-6. Linearization of Gating Variables

For the activation variable m, the nonlinear function is:

$$f_m(V_m, m) = \alpha_m(V_m)(1 - m) - \beta_m(V_m) m$$

The first-order perturbation equation is:

$$\delta \dot{m} = [\alpha_m'(V_0)(1 - m_0) - \beta_m'(V_0) m_0] \delta V - [\alpha_m(V_0) + \beta_m(V_0)] \delta m$$

Similarly, for the sodium inactivation variable h and the potassium activation variable n:

$$\delta \dot{h} = [\alpha_h'(V_0)(1 - h_0) - \beta_h'(V_0) h_0] \delta V - [\alpha_h(V_0) + \beta_h(V_0)] \delta h$$

$$\delta \dot{n} = [\alpha_n'(V_0)(1 - n_0) - \beta_n'(V_0) n_0] \delta V - [\alpha_n(V_0) + \beta_n(V_0)] \delta n$$

7-7. Complete Linearized Hodgkin–Huxley Model

By combining the linearized membrane equation with the linearized gating-variable dynamics, the Hodgkin–Huxley model around the equilibrium point (V_0, m_0, h_0, n_0 is given by the following system of linear differential equations:

- $C_m \frac{d(\delta V)}{dt} = \delta I - (g_{Na}^{max} m_0^3 h_0 + g_K^{max} n_0^4 + g_L)\delta V - (3g_{Na}^{max} m_0^2 h_0 (V_0 - V_{Na}))\delta m - (g_{Na}^{max} m_0^3 (V_0 - V_{Na}))\delta h - (4g_K^{max} n_0^3 (V_0 - V_K))\delta n$
- $\delta \dot{m} = [\alpha_m{}'(V_0)(1 - m_0) - \beta_m{}'(V_0) m_0]\delta V - [\alpha_m(V_0) + \beta_m(V_0)]\delta m$
- $\delta \dot{h} = [\alpha_h{}'(V_0)(1 - h_0) - \beta_h{}'(V_0) h_0]\delta V - [\alpha_h(V_0) + \beta_h(V_0)]\delta h$
- $\delta \dot{n} = [\alpha_n{}'(V_0)(1 - n_0) - \beta_n{}'(V_0) n_0]\delta V - [\alpha_n(V_0) + \beta_n(V_0)]\delta n$

7-8. Interpretation of the Linearized Model

The resulting linear model describes how small deviations from the resting state evolve over time. If the eigenvalues of *A* have negative real parts, small perturbations decay and the equilibrium is locally stable. If at least one eigenvalue has a positive real part, perturbations grow and the equilibrium is locally unstable. Therefore, the linearized system provides a mathematical basis for studying local neuronal stability, sensitivity, and response to small current inputs (Khalil, 2002, pp. 120–130; Strogatz, 2018, pp. 135–145).

8. Results and Interpretation

The linearization of the Hodgkin–Huxley model produced a simplified mathematical representation of neuronal dynamics around the resting equilibrium point. By applying first-order Taylor series expansion, the original nonlinear system was transformed into a system of coupled linear differential equations describing small perturbations in the membrane potential and gating variables.

The resulting linearized model preserves the essential local behavior of the original Hodgkin–Huxley equations while significantly reducing mathematical complexity. The derived equations demonstrate that variations in membrane voltage are influenced not only by external current perturbations but also by changes in the sodium activation variable m, sodium inactivation variable h, and potassium activation variable n. This confirms the strong coupling between membrane potential and ion channel dynamics in neuronal systems.

The study also showed that the coefficients of the linearized equations depend on the equilibrium values of the gating variables and ionic conductances. Consequently, the neuronal response near equilibrium is strongly influenced by the operating point of the system. This provides a mathematical interpretation of how small perturbations evolve around the resting state of the neuron.

Furthermore, the linearized formulation enables the application of analytical tools from linear systems theory, including stability analysis, eigenvalue analysis, transient response analysis, and transfer function derivation. Such analyses are difficult to perform directly on the original nonlinear Hodgkin–Huxley equations because of their mathematical complexity (Khalil, 2002, pp. 120–130; Strogatz, 2018, pp. 135–145).

From a physiological perspective, the model demonstrates that neuronal stability depends strongly on the interaction between membrane voltage and ion channel kinetics. Small variations in sodium and potassium conductances can significantly influence neuronal excitability and local dynamic behavior. Therefore, the linearized model provides valuable insight into the mechanisms governing neuronal response under small disturbances.

Overall, the study demonstrates that linearization provides an effective local approximation of the Hodgkin–Huxley model and establishes a mathematically tractable framework for investigating neuronal stability and dynamic behavior in computational neuroscience and biomedical system analysis.

9. Conclusion

The Hodgkin–Huxley model provides a fundamental mathematical description of neuronal membrane dynamics and action potential generation. Despite its physiological accuracy, the nonlinear nature of the model introduces significant mathematical complexity that limits direct analytical investigation. In this study, the model was successfully linearized around the resting equilibrium point using first-order Taylor series expansion.

The linearization process involved defining perturbation variables, deriving the equilibrium conditions, and approximating the nonlinear ionic currents and gating-variable equations using first-order terms. The resulting system of coupled linear differential equations preserved the essential local behavior of the original nonlinear model while significantly simplifying the mathematical analysis.

The derived linearized model provides a useful framework for studying local neuronal dynamics, stability, and response to small perturbations. Furthermore, it enables the application of analytical techniques from linear systems theory, which are difficult to apply directly to the nonlinear Hodgkin–Huxley equations. Therefore, the study demonstrates the effectiveness of linearization as a mathematical tool for simplifying

complex neuronal systems and supporting further investigations in computational neuroscience and biomedical system analysis.

10. Recommendations

- Extend the present study by performing a complete stability analysis of the linearized system using eigenvalues and phase-plane analysis methods.
- Derive transfer functions for the linearized Hodgkin–Huxley model to investigate neuronal response characteristics under external stimulation.
- Compare the behavior of the linearized model with numerical simulations of the original nonlinear system to evaluate the accuracy of the approximation.
- Apply the linearization methodology to more advanced neuronal models and larger neural networks.
- Investigate the biomedical applications of linearized neuronal models in neurological disorder analysis, neuroprosthetics, and computational neuroscience research.

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