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Modeling the Biological Mind

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in voce **a.Maxwell**

Abstract

Prologue *I, the Custodian of Inquiry, open this article on Modeling the Biological Mind to orient our readers to the questions ahead.* *May these reflections prepare your discernment for the inq...

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I, the Custodian of Inquiry, open this article on Modeling the Biological Mind to orient our readers to the questions ahead.

May these reflections prepare your discernment for the inquiry that follows.

Introduction

The mind, that most elusive of natural phenomena, has long beckoned the physicist to render its motions in the language of mathematics. From the first attempts to describe muscular contraction as a mechanical oscillation to the present endeavour of mapping whole-brain activity, the quest has been one of unification: to discover a set of equations whose solutions embody thought, perception, and volition. In the tradition of Newton's universal gravitation and my own own electromagnetic theory, we aspire to a *Maxwellian synthesis*—a framework in which the disparate observations of neurobiology are subsumed under a common mathematical edifice.

The present work is motivated by three intertwined imperatives. First, the rapid accumulation of high-resolution electrophysiological data demands models that are not merely descriptive but predictive. Second, the success of statistical-mechanical methods in condensed matter and the triumph of dynamical-systems theory in fluid turbulence suggest that similar tools may illuminate the collective dynamics of neuronal assemblies. Third, the burgeoning field of machine learning, itself built upon artificial neural networks, invites a dialogue between engineered and biological computation.

Historically, the lineage of neural modelling proceeds from the mechanical analogues of Galvani and Helmholtz, through the electrical circuit representations of Kirchhoff, to the biophysical conductance models of Hodgkin and Huxley. Each stage has refined the correspondence between physical law and neural function. Our aim is to chart a path that unites these stages, employing differential equations, variational principles, and rigorously derived approximations in the spirit of my own treatise on the electromagnetic field.

Methodologically we adopt a *physics-first* stance: beginning with Maxwell's equations in the quasi-static approximation, we derive the cable equation governing dendritic conduction, then ascend to continuum field descriptions, and finally descend to stochastic formulations that

capture the inherent noise of synaptic transmission. Throughout we emphasise analytical insight alongside numerical tractability, seeking a balance between depth and accessibility for readers across physics, biology, and engineering.

1. Foundations of Biological Neural Modelling

1.1 Historical Perspective: From Newtonian Mechanics to Neural Dynamics

The earliest attempts to rationalise the excitability of nerves were couched in the language of mechanics. Galvani's frog-leg experiments (1791) suggested that a "muscular electricity" could be likened to a hidden force akin to gravity, while Helmholtz's measurement of nerve conduction velocity (1850) introduced a temporal scale reminiscent of wave propagation in elastic media. By the mid-nineteenth century, the burgeoning field of electrophysiology had embraced the analogy of *circuits*; Kirchhoff's laws (1845) provided a formalism for the conservation of charge and the distribution of potential across resistive and capacitive elements.

These mechanical and electrical metaphors set the stage for the biophysical revolution of the 1950s, when Hodgkin and Huxley, inspired by the circuit approach, constructed a set of differential equations that captured the ionic currents underlying the action potential. Their work demonstrated that the nervous system, far from being a mysterious organ, obeys the same principles of charge conservation and energy balance that govern any electromagnetic system.

1.2 Physical Analogues: Electrical Circuits and Membrane Potentials

A neuron may be idealised as a *leaky capacitor* in parallel with a suite of ion-specific conductances. The membrane capacitance (C_m) stores charge, while the leak resistance (R_{ell}) represents passive ionic flow. Applying Kirchhoff's current law to the soma yields the classic RC equation

$$[C_m \frac{dV}{dt} = -\frac{V - E_{\text{ell}}}{R_{\text{ell}}} + I_{\{\text{ion}\}}(V,t) + I_{\{\text{ext}\}}(t),]$$

where (V) is the transmembrane potential, (E_{ell}) the leak reversal potential, (I_{ion}) the sum of voltage-gated ionic currents, and (I_{ext}) an externally applied stimulus.

From the full set of Maxwell's equations, one may obtain this relation by invoking the quasi-static limit (displacement currents negligible compared with conduction currents) and assuming isotropic, homogeneous intracellular and extracellular media. The derivation proceeds by integrating Faraday's law over a small membrane patch and invoking the constitutive relation ($\mathbf{J} = \sigma \mathbf{E}$) for the ionic currents, thereby recovering the circuit description as a low-frequency approximation of the electromagnetic field.

1.3 Mathematical Formalisms: Differential Equations and Stochastic Processes

The Hodgkin–Huxley formalism expands the current term (I_{ion}) into a sum of conductances ($g_i(V,t)$) multiplied by their respective driving forces ($(V - E_i)$). Each conductance obeys a first-order kinetic scheme

$$\left[\frac{dx}{dt} = \alpha_x(V)(1-x) - \beta_x(V)x, \right]$$

with (x) representing the activation or inactivation variable of a channel. These ordinary differential equations (ODEs) constitute a high-dimensional dynamical system whose trajectories describe the evolution of membrane potential and channel states.

Synaptic transmission, however, is intrinsically stochastic. The arrival of neurotransmitter vesicles, the opening of ligand-gated channels, and the thermal fluctuations of ion channels collectively generate *noise* that can be modelled by Langevin equations

$$\left[\frac{dx}{dt} = f(x,V) + \sqrt{2D}\eta(t), \right]$$

where ($\eta(t)$) is a Gaussian white-noise process and (D) a diffusion coefficient related to the variance of the underlying stochastic events. The associated probability density ($P(x,t)$) obeys a Fokker–Planck equation, furnishing a bridge between microscopic randomness and macroscopic ensemble behaviour [Koch, 1999].

2. Computational Architectures Inspired by Biology

2.1 Hodgkin–Huxley and Conductance-Based Models

The full Hodgkin–Huxley (HH) model comprises four coupled nonlinear ODEs: one for the membrane voltage and three for the gating variables (m), (h), and (n). Its elegance lies in its derivation from experimentally measured voltage-clamp data, yielding parameter values (g_{Na}), (g_{K}), and (g_{ell}) that faithfully reproduce the characteristic spike. Modern computational practice refines these parameters via optimisation algorithms (e.g., Levenberg–Marquardt) applied to high-throughput electrophysiological recordings, thereby extending the HH framework to diverse neuronal phenotypes [Naundorf et al., 2006].

2.2 Integrate-and-Fire and Simplified Neuronal Units

While the HH model is biophysically faithful, its complexity hampers large-scale network simulations. The *integrate-and-fire* (IAF) paradigm reduces the dynamics to a single ODE for (V) with a hard threshold (θ). When (V) reaches (θ), a spike is emitted and (V) is reset. Analytically, the subthreshold trajectory for a constant input current (I) is

$$[V(t) = V_{\text{rest}} + \frac{I R}{\tau_m} \left(1 - e^{-t/\tau_m} \right),]$$

where ($\tau_m = R C$) is the membrane time constant. The IAF model admits closed-form expressions for interspike intervals and firing rates, facilitating the derivation of population-level transfer functions and the investigation of synchrony in coupled oscillator networks [Gerstner & Kistler, 2002].

2.3 Network Topologies: Small-World, Scale-Free, and Connectomics

Real cortical circuits exhibit non-random wiring. Empirical studies of the macaque and human connectome reveal *small-world* clustering (high local connectivity with short global path lengths) and *scale-free* degree distributions, wherein a few hub neurons possess

disproportionately many connections. Graph-theoretic metrics—clustering coefficient (C), characteristic path length (L), and degree exponent (γ)—quantify these properties.

The presence of hubs influences dynamical phenomena such as *synchronisation* and *robustness*. For instance, the Kuramoto order parameter applied to a network of phase-oscillating neurons demonstrates that a modest increase in hub connectivity can precipitate a sudden transition from asynchronous firing to global coherence, reminiscent of phase transitions in statistical physics [Strogatz, 2000].

3. Dynamical Systems and Field Theories of the Mind

3.1 Continuum Descriptions: Neural Field Equations

When neuronal populations are sufficiently dense, a *continuum* description becomes advantageous. Amari's integral equation

$$\frac{\partial u(\mathbf{x}, t)}{\partial t} = -u(\mathbf{x}, t) + \int_{\Omega} w(\mathbf{x}, \mathbf{x}') f(\text{big}(u(\mathbf{x}', t)\text{big}), d\mathbf{x}') + I(\mathbf{x}, t),$$

captures the evolution of the mean firing rate field ($u(\mathbf{x}, t)$) over cortical domain (Ω). The kernel (w) encodes synaptic coupling strength and spatial spread, while (f) is a sigmoidal activation function.

A formal analogy with Maxwell's equations arises by interpreting (u) as a scalar potential and the kernel (w) as a Green's function mediating long-range interaction, much as the electromagnetic vector potential propagates through space. In the limit of short-range coupling, one may expand the integral to obtain a *reaction-diffusion* PDE, akin to the diffusion term in the heat equation, thereby situating neural fields within the broader family of field theories.

3.2 Bifurcation and Chaos in Cognitive Processes

Neural systems are poised near critical points where small parameter variations induce qualitative changes in dynamics. A *Hopf bifurcation* in a cortical column model, for example, can generate rhythmic oscillations that underlie alpha or gamma band activity. Likewise, a *saddle-node* bifurcation may account for perceptual switching in binocular rivalry, where two competing attractors correspond to alternative interpretations of an ambiguous stimulus.

Empirical EEG recordings have revealed low-dimensional chaotic attractors, as evidenced by positive Lyapunov exponents and fractal dimensions on the order of 3–5 [Freeman, 1991]. These findings suggest that the brain exploits deterministic chaos to enhance its repertoire of dynamical states, facilitating rapid transitions between functional modes while preserving a degree of stability.

3.3 Energy Principles and Variational Methods

Just as the electromagnetic field may be derived from a Lagrangian density ($\mathcal{L} = \frac{1}{2}(\epsilon_0 \mathbf{E}^2 - \frac{1}{\mu_0} \mathbf{B}^2)$), one can construct a *neural Lagrangian*

$$[\mathcal{L}_{\text{neu}} = \frac{1}{2} \tau \left(\frac{\partial u}{\partial t} \right)^2 - \frac{1}{2} D |\nabla u|^2 - V(u),]$$

where (τ) is an effective inertial time constant, (D) a diffusion coefficient, and ($V(u)$) a potential encoding local excitability. The Euler–Lagrange equation ($\frac{d}{dt} \frac{\partial \mathcal{L}}{\partial \dot{u}} - \nabla \cdot \frac{\partial \mathcal{L}}{\partial (\nabla u)} + \frac{\partial \mathcal{L}}{\partial u} = 0$) reproduces a damped wave-type neural field equation.

Variational minimisation of an *action functional* ($S = \int \mathcal{L}_{\text{neu}} d\mathbf{x}, dt$) yields the most probable trajectories of activity, offering a principled route to predict pattern formation such as cortical columns or travelling waves. This approach dovetails with the *principle of least action* that underlies all classical field theories, reinforcing the Maxwellian aspiration for a unified description.

4. Statistical Mechanics of Large-Scale Neural Ensembles

4.1 Mean-Field Approaches and the Ising Model

When neuronal states are coarse-grained to binary variables ($s_i = \pm 1$) (spiking versus silent), the collective behaviour may be mapped onto an *Ising spin system* with Hamiltonian

$$[\mathcal{H} = -\frac{1}{2} \sum_{i \neq j} J_{ij} s_i s_j - \sum_i h_i s_i,]$$

where (J_{ij}) encapsulates effective synaptic coupling and (h_i) an external bias. The *mean-field* (Curie–Weiss) approximation assumes uniform coupling ($J_{ij}=J/N$), leading to a self-consistency equation for the magnetisation ($m = \langle s_i \rangle$)

$$[m = \tanh(\beta (J m + h)),]$$

with (β) the inverse temperature, interpreted here as a measure of neuronal noise. This framework has successfully reproduced the pairwise correlation structure observed in retinal ganglion cell populations [Schneidman et al., 2006].

4.2 Information Theory and Entropy in Neural Coding

Shannon’s entropy ($H = -\sum_k p_k \log p_k$) quantifies the uncertainty of a neural ensemble’s state distribution ($\{p_k\}$). Mutual information

$$[I(S;R) = \sum_{s,r} p(s,r) \log \frac{p(s,r)}{p(s)p(r)},]$$

measures the reduction in uncertainty about a stimulus (S) afforded by the neural response (R). Empirical studies have shown that cortical networks operate near the *information-theoretic optimum* where entropy is maximised subject to metabolic constraints, a condition reminiscent of the *maximum entropy* principle of statistical mechanics [Tkacik et al., 2014].

4.3 Thermodynamic Analogues: The Free-Energy Principle

Karl Friston's *free-energy principle* posits that biological systems minimise a variational bound on surprise (negative log evidence) by updating internal models to predict sensory inputs.

Formally, the *variational free energy*

$$[F = \int q(\mathbf{x}) \log \frac{q(\mathbf{x})}{p(\mathbf{y}, \mathbf{x})}, d\mathbf{x},]$$

where $q(\mathbf{x})$ is an approximate posterior over hidden states $(\mathbf{x})$ and $(p(\mathbf{y}, \mathbf{x}))$ the generative model of observations $(\mathbf{y})$, plays the role of a thermodynamic potential. Gradient descent on (F) yields *predictive coding* dynamics, which can be cast as a set of coupled differential equations analogous to dissipative field equations. This synthesis of Bayesian inference and statistical physics offers a compelling bridge between cognition and the laws governing matter.

5. Towards a Unified Computational Theory

5.1 Multi-Scale Modelling: From Ion Channels to Behaviour

A truly unifying theory must respect the hierarchy of scales that characterise the nervous system. At the microscopic level, stochastic Markov models of ion-channel gating capture the discrete transitions that give rise to macroscopic conductances. At the mesoscopic level, conductance-based compartmental models integrate these currents across dendritic trees, producing the spatiotemporal voltage fields described by cable theory. At the macroscopic level, neural field equations and statistical-mechanical ensembles encapsulate the collective dynamics that manifest as perception, decision, and movement.

Coupling across scales can be achieved through *coarse-graining* procedures that preserve essential conserved quantities (e.g., charge, energy) while systematically eliminating fast variables, much as the derivation of the Navier–Stokes equations proceeds from the Boltzmann

kinetic equation. Recent work employing *hierarchical Bayesian inference* has demonstrated the feasibility of fitting such multi-scale models to simultaneous intracellular, extracellular, and behavioural datasets [Kappel et al., 2021].

5.2 Integration with Machine Learning: Deep Networks and Biophysical Constraints

Artificial neural networks (ANNs) have achieved remarkable feats, yet they remain abstracted from the biophysical realities of their biological counterparts. Embedding conductance-based dynamics within ANNs yields *physics-informed neural networks* (PINNs) that respect conservation laws and can be trained on sparse experimental data. For example, a deep recurrent architecture whose hidden state obeys a discretised Hodgkin–Huxley update rule can learn to reproduce spike-timing patterns while maintaining physiological plausibility [Rudy et al., 2023].

Conversely, insights from statistical mechanics—such as the role of criticality and phase transitions—inform the design of *adaptive* learning algorithms that modulate network connectivity to operate near optimal computational regimes. The synergy between biophysical fidelity and machine-learning flexibility promises a new generation of models capable of both explanatory depth and predictive power.

5.3 Prospects for Predictive Modelling and Experimental Validation

The ultimate test of any theoretical edifice lies in its capacity to predict the outcome of *novel* experiments. Optogenetic perturbations, wherein specific neuronal populations are driven with millisecond precision, provide a stringent benchmark: a unified model should anticipate the spatiotemporal spread of evoked activity, the resultant behavioural change, and the accompanying shifts in statistical measures such as entropy and mutual information.

A proposed roadmap involves iterative cycles of *model-driven hypothesis generation* → *targeted experimentation* → *parameter refinement*, echoing the methodological spirit of my own investigations into electromagnetic phenomena. Open-source simulators that integrate

conductance-based compartments, neural field solvers, and statistical-mechanical inference engines will be essential infrastructure for this endeavour.

Conclusion

In the spirit of the unifying ambition that guided my own work on the electromagnetic field, we have charted a *Maxwellian synthesis* of neural physics. Beginning with the circuit analogues of the membrane and progressing through stochastic differential equations, continuum field theories, and statistical-mechanical models, we have assembled a coherent mathematical portrait of the biological mind. The synthesis respects the hierarchy of scales, embraces the richness of network topology, and incorporates the variational principles that underlie both classical fields and modern inference.

Nevertheless, gaps remain. Plasticity—both synaptic and structural—introduces time-dependent modification of the very parameters that our equations presume fixed. Neuromodulatory systems, with their diffuse chemical signalling, demand extensions beyond purely electrical descriptions. Moreover, the integration of metabolic constraints and glial contributions awaits a fuller treatment.

Future research, guided by the principles articulated herein, will strive to close these lacunae, forging a truly unified computational theory that can illuminate cognition, inspire engineered intelligence, and deepen our appreciation of the mind as a physical system governed by the same elegant laws that bind light, electricity, and the cosmos.

Epilogue

I, the Custodian of Inquiry, conclude this article on Modeling the Biological Mind with gratitude for your sustained attention.

Carry its insights into your own circles of inquiry and return with what you discover.

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