THE BRAIN-MESH MODEL: A UNIFIED FRAMEWORK FOR NEURAL SYNCHRONY, PLASTICITY, AND COHERENCE

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ABSTRACT

The brain-mesh model introduces a novel three-layered architecture that integrates local and macro-regional connectivity with an underlying, mesh-inspired network layer. This foundational mesh layer, based on metallic mesh structures, spans the entire brain and generates interference patterns, noise, and resonance effects that modulate both local and global neural dynamics. The fused model goes beyond traditional connectivity frameworks by providing a unified explanation for phenomena such as brain-wide phase gradients, stable low-frequency resonance frequencies, and long-range plasticity effects, which are often difficult to explain cohesively within existing models.

In addition to accounting for classical neurobiological observations—such as phase synchrony, functional connectivity fluctuations, and local Hebbian plasticity—the fused model offers novel insights into less understood phenomena. Specifically, it predicts connectivity-independent phase gradients across non-synaptic regions, harmonic resonance peaks consistent across individuals, and diffuse plasticity driven by global interference patterns, all of which are challenging to explain under current frameworks.

These unique predictions align with partial empirical observations, such as traveling wave dynamics, consistent low-frequency oscillations, and task-induced connectivity shifts, underscoring the model's relevance. Additionally, the brain-mesh model generates testable hypotheses that distinguish it from traditional approaches, providing a promising framework for future experimental validation and opening new avenues for understanding global brain function.

1 Introduction

The brain's functional architecture is a complex interplay between local neural circuitry, macro-regional connectivity, and large-scale oscillatory dynamics. Despite advancements in connectomics and neural modeling, a comprehensive understanding of brain-wide synchrony, plasticity, and oscillatory behavior remains elusive. Traditional connectivity models, although instrumental in decoding aspects of local and regional brain dynamics, struggle to cohesively account for global phenomena that appear independent of direct structural pathways. To address these gaps, this paper introduces a brain-mesh model that incorporates a global mesh-inspired layer as an underlying, pervasive connectivity network. This model provides a unified framework for explaining not only established neurobiological observations but also emergent phenomena currently addressed through ad hoc mechanisms.

1.1 Theoretical Gaps in Traditional Connectivity Models

For decades, efforts to map and model the brain's connectivity have primarily relied on two main frameworks: local connectivity models and macro-regional network models. These approaches have offered significant insights but face inherent limitations when applied to brain-wide phenomena such as phase synchrony across unconnected regions, diffuse plasticity, and stable low-frequency oscillations. Below, we outline these models and their limitations.

1.1.1 Local and Regional Connectivity Models

Local connectivity models focus on micro- and mesoscopic scales, modeling the synaptic interactions within neural circuits or small brain regions. The neural mass model, for instance, describes the excitatory and inhibitory populations within a region, providing insights into local oscillatory behavior and activity patterns Deco et al. [2008], Breakspear [2017]. Meanwhile, macro-regional network models extend these principles to capture interactions between anatomically connected regions, such as those seen in cortical-subcortical communication Sporns [2011], van den Heuvel and Sporns [2013]. These models rely on structural connectivity matrices derived from diffusion tensor imaging (DTI) and other neuroimaging techniques to describe direct anatomical pathways.

While local and regional connectivity models can explain synchrony within defined networks, they fall short in accounting for phenomena that span beyond immediate anatomical links. For example, traveling waves of activity observed across the cortex propagate over large distances, often crossing regions with no direct structural connectivity Muller et al. [2018], Zhang et al. [2021]. Current models explain these waves by invoking feedback loops within local circuits or hypothesizing indirect connections through intermediary nodes Wurtz [2008], but such explanations are often speculative and lack empirical grounding.

1.1.2 Limitations in Explaining Global Synchrony, Diffuse Plasticity, and Consistent Low-Frequency Oscillations

Global Phase Synchrony: Traditional models attribute phase coherence to synaptic and structural connections, making it challenging to explain synchrony across distant, non-connected regions. Such synchrony is increasingly observed in studies of the resting-state brain, where distant regions exhibit coherent oscillations even in the absence of direct structural connectivity Fox and Raichle [2007], Deco et al. [2021]. Explanations based on indirect or polysynaptic pathways fail to account for the observed coherence patterns across unconnected areas.

Diffuse Plasticity: Plasticity, the brain's ability to reorganize itself based on experience, is predominantly explained through models of Hebbian learning and Spike-Timing-Dependent Plasticity (STDP), which depend on local, synaptic activation Hebb [1949], Bi and Poo [2001]. However, empirical evidence from non-invasive brain stimulation (NIBS) experiments suggests that repetitive stimulation can alter connectivity patterns between brain regions that are not directly connected by synapses, indicating a form of diffuse plasticity Fox et al. [2014], Pascual-Leone et al. [2005]. Current models struggle to explain how changes in connectivity can propagate across non-synaptic regions, relying instead on speculative cross-regional coupling mechanisms Siebner et al. [2004].

Consistent Low-Frequency Oscillations: Brain-wide oscillations, particularly in low-frequency bands such as delta and theta, are observed consistently across individuals, especially during resting states Buzsaki [2006], Mantini et al. [2007]. Traditional models attribute these oscillations to metabolic or local network factors, yet these explanations do not account for the cross-regional consistency of low-frequency rhythms. The default mode network (DMN), for instance, exhibits low-frequency oscillations that span large brain areas but lack a clear structural basis Raichle et al. [2001]. While models of local circuit resonance have been proposed, they often rely on individual variability and do not naturally produce the globally consistent frequency peaks observed across individuals Fox and Raichle [2007].

Thus, while traditional connectivity models successfully explain localized or region-specific phenomena, they lack the explanatory power to address globally distributed, coherence-driven processes, consistent resonance frequencies, and the brain's capacity for widespread, connectivity-independent plasticity. These gaps necessitate a new approach that integrates a fundamental, brain-wide connectivity structure.

1.2 Rationale for a Brain-Mesh Model

In light of the limitations of existing models, the brain-mesh model proposes a novel mesh-inspired connectivity layer that functions as a pervasive network underlying all other structural and functional layers. This mesh layer is inspired by metallic mesh structures and operates as a flexible, lattice-like connectivity network capable of distributing interference patterns, supporting harmonic resonance, and generating diffuse plasticity effects across the brain.

1.2.1 Motivation for Adding a Global Mesh-Like Connectivity Layer

The idea of a global connectivity layer is motivated by the need for a mechanism that can support brain-wide coherence and plasticity beyond traditional synaptic and structural connections. A mesh-like layer provides this foundation by enabling interference-driven synchrony and distributed connectivity. The model hypothesizes that this layer comprises nodes that interact through elastic links, allowing adaptive resonance and interference patterns to propagate across the brain. Each node in this layer is subject to stochastic inputs, generating a spectrum of low-frequency oscillations

consistent across individuals, thus addressing the observed stability in resonance frequencies Buzsaki [2006], Raichle et al. [2001].

By introducing a mesh-like layer, this model provides a cohesive framework for explaining phenomena that are otherwise explained only through disparate or speculative mechanisms. In particular, the mesh layer's interference dynamics produce phase gradients and synchrony across distant regions, aligning with empirical observations of connectivity-independent traveling waves Zhang et al. [2021]. Furthermore, this layer offers a novel explanation for diffuse plasticity, suggesting that long-range connectivity changes observed after repetitive stimulation are driven by persistent interference patterns within the mesh Pascual-Leone et al. [2005].

1.2.2 Outline of the Brain-Mesh Model's Unified Mechanism

The brain-mesh model proposes three integrated connectivity layers:

1. **Local and Regional Connectivity:** Capturing micro- and macro-scale interactions, this layer adheres to traditional neural mass and connectome models.

2. **Global Structural and Functional Connectivity:** Based on direct anatomical connections, this layer describes large-scale interactions observed in traditional connectomic studies.

3. **All-Brain Mesh Connectivity Layer:** The core innovation of this model, the mesh layer, generates global interference patterns, supports harmonic resonance frequencies, and facilitates diffuse plasticity.

This mesh layer acts as a pervasive foundation for all other connectivity layers, unifying their outputs through a global interference-driven mechanism. By generating phase coherence, low-frequency oscillations, and long-range plasticity effects, the mesh layer addresses the brain's capacity for brain-wide synchrony and adaptation, complementing traditional structural and functional layers with an all-encompassing connectivity network.

In summary, the brain-mesh model fills critical gaps in current neurobiological frameworks, offering a novel approach to understanding global phase synchrony, diffuse plasticity, and consistent low-frequency oscillations. In subsequent sections, we will detail the mathematical structure of this model, examine its predictions, and compare its empirical validity with existing frameworks.

2 The Brain-Mesh Model Architecture

The brain-mesh model presents a multi-layered framework integrating traditional local and macro-regional neural connectivity with a novel mesh-inspired layer to explain both localized and global dynamics across the brain. This layered architecture includes three interdependent connectivity levels: local and regional connectivity, global structural and functional connectivity, and an all-encompassing brain-mesh layer. Together, these layers allow the model to address complex neurobiological phenomena such as brain-wide phase coherence, low-frequency oscillations, and diffuse plasticity, which are inadequately explained by current models. Each layer is rigorously defined mathematically, with explicit descriptions of their inter-layer interactions to produce an experimentally testable, unified system.

2.1 Layered Model Structure and Mathematical Formalization

The fused model's architecture is organized into three layers, each addressing different scales of neural dynamics, from localized interactions to brain-wide synchronization:

2.1.1 Local and Regional Connectivity: Neural Mass Models

At the local and regional levels, neural interactions within brain circuits are modeled using neural mass models, which capture the collective activity of excitatory and inhibitory populations within specific regions. These models represent the first layer, comprising micro-level neural connectivity as well as macro-regional communication across different brain areas.

The neural mass model equations describe the average dynamics of excitatory (E_i) and inhibitory (I_i) neuronal populations in region i Deco et al. [2008], Jansen and Rit [1995]:

$$\frac{dE_i}{dt} = \frac{1}{\tau_E}\left(-E_i + S_E\left(w_{EE}E_i - w_{EI}I_i + P_i + \eta_i^{\text{local}}(t)\right)\right), \tag{1}$$

$$\frac{dI_i}{dt} = \frac{1}{\tau_I}\left(-I_i + S_I\left(w_{IE}E_i - w_{II}I_i + \eta_i^{\text{local}}(t)\right)\right), \tag{2}$$

where:

- τ_E and τ_I are time constants for excitatory and inhibitory populations.
- S_E and S_I are sigmoidal activation functions.
- $w_{EE}, w_{EI}, w_{IE}, w_{II}$ denote synaptic weights between excitatory and inhibitory populations.
- P_i is external input to the region.
- $\eta_i^{\text{local}}(t)$ represents local noise modeled as Gaussian white noise.

Local connectivity is achieved through synaptic interactions, while macro-regional communication is mediated by axonal connections, which can be captured by structural connectivity matrices Hagmann et al. [2008], Honey et al. [2009]. The structural connectivity matrix C_{ij} represents the strength of anatomical connections between regions i and j. This model layer explains oscillatory dynamics and phase coherence within connected circuits but falls short in explaining whole-brain coherence observed across non-connected regions Honey et al. [2009].

2.1.2 Global Structural and Functional Connectivity

The second layer represents global structural and functional connectivity across large-scale brain networks, integrating both anatomical and functional relationships.

Structural Connectivity: The structural connectivity matrix C_{ij}, derived from high-resolution connectome data Sporns et al. [2005], reflects the density and strength of anatomical connections. This matrix captures long-range pathways that facilitate global brain network communication.

Functional Connectivity: Functional connectivity within this layer is often assessed using functional magnetic resonance imaging (fMRI) and magnetoencephalography (MEG), which measure correlations in brain activity across regions Fox and Raichle [2007], Biswal et al. [2010]. Functional synchrony is modeled by the Kuramoto phase-coupling model:

$$\frac{d\theta_i}{dt} = \omega_i + \sum_j K_{ij}\sin(\theta_j - \theta_i), \tag{3}$$

where:

- θ_i denotes the phase of oscillatory activity in region i.
- ω_i is the natural frequency of region i.
- K_{ij} is the coupling strength, modulated by C_{ij} to represent the influence of structural connectivity on synchronization Breakspear et al. [2010].

While this layer can explain the functional coherence of networks such as the default mode network (DMN) and fronto-parietal network Raichle et al. [2001], Smith et al. [2009], it cannot account for whole-brain coherence or phase gradients that occur across anatomically disconnected regions Deco et al. [2021].

2.1.3 All-Brain Mesh Connectivity Layer

The third layer, the all-brain mesh connectivity layer, introduces a global, lattice-like structure inspired by metallic mesh networks, representing a pervasive connectivity that modulates interactions across the entire brain.

Node Distribution: Uniformly distributed nodes across the brain create a lattice structure, enabling equidistant connectivity across regions. These nodes, representing distributed neural or glial cells, form a continuous network that supports large-scale interference patterns Fields et al. [2015], Fox et al. [2014].

Elasticity and Noise Dynamics: Each connection between nodes behaves as an elastic spring following Hooke's Law, allowing adaptive interactions:

$$F_{ij} = -k_{ij}\Delta L_{ij}, \tag{4}$$

where k_{ij} is the spring constant and ΔL_{ij} is the deformation. Additionally, each node experiences Gaussian noise $\eta_i(t) \sim N(0, \sigma^2)$, introducing spatial-temporal variability essential for producing dynamic interference patterns Buice and Chow [2010].

Wave Propagation and Interference Patterns: The propagation of interference patterns across the mesh is governed by the wave equation:

$$\frac{\partial^2 u(x, y, t)}{\partial t^2} = c^2 \nabla^2 u(x, y, t) + \eta(x, y, t), \tag{5}$$

where $u(x, y, t)$ represents the displacement at point (x, y), c is the propagation speed, and $\eta(x, y, t)$ is stochastic noise. These wave dynamics enable phase coherence across unconnected brain regions, explaining phenomena like cortical traveling waves and low-frequency oscillations Muller et al. [2018], Zhang et al. [2021].

Harmonic Resonance and Global Synchronization: The mesh supports harmonic resonance frequencies, independent of individual variation, with natural harmonic modes:

$$f_n = \frac{nc}{2L}, \tag{6}$$

where L is a characteristic length of the mesh, aligning with empirical observations of stable low-frequency oscillations Buzsaki [2006], Raichle et al. [2001].

2.2 Mathematical Integration Across Layers

Combining the equations from each layer, we establish an integrative model describing the dynamics of the entire brain network.

2.2.1 Unified System Equations

The model equations integrating local, regional, and mesh dynamics are as follows:

$$\text{Neural Mass Dynamics:} \quad \frac{dE_i}{dt} = \frac{1}{\tau_E}\Big(- E_i + S_E\Big(w_{EE}E_i - w_{EI}I_i + P_i^{\text{ext}} + \sum_j C_{ij}E_j + \gamma u(x_i, y_i, z_i, t) + \eta_i^{\text{local}}(t)\Big)\Big), \tag{7}$$

$$\frac{dI_i}{dt} = \frac{1}{\tau_I}\Big(- I_i + S_I\Big(w_{IE}E_i - w_{II}I_i + \eta_i^{\text{local}}(t)\Big)\Big), \tag{8}$$

$$\text{Phase Dynamics:} \quad \frac{d\theta_i}{dt} = \omega_i + \sum_j K_{ij}\sin(\theta_j - \theta_i) + \kappa u(x_i, y_i, z_i, t), \tag{9}$$

$$\text{Mesh Wave Equation:} \quad \frac{\partial^2 u}{\partial t^2} = c^2\nabla^2 u + \eta_{\text{mesh}}(x, y, z, t), \tag{10}$$

$$\text{Plasticity Update:} \quad \Delta C_{ij} = \alpha_H E_i E_j + \alpha_M\Big(\langle u(x_i, y_i, z_i, t)u(x_j, y_j, z_j, t)\rangle - \langle u(x_i, y_i, z_i, t)\rangle\langle u(x_j, y_j, z_j, t)\rangle\Big). \tag{11}$$

where:

- $u_i = u(x_i, y_i, z_i, t)$ represents the displacement at node i in the mesh.
- γ and κ are coupling coefficients for the mesh layer with neural and phase dynamics, respectively.
- α_H and α_M denote the Hebbian and mesh-induced plasticity rates.
- $\eta_{\text{mesh}}(x, y, z, t)$ is a stochastic noise term.

These equations integrate the core properties and coupling functions, allowing for complex brain-wide dynamics that address traditional model limitations.

2.2.2 Mesh-Induced Plasticity

The mesh layer's interference patterns introduce a novel form of diffuse plasticity, impacting connectivity between regions without synaptic connections. Based on Hebbian plasticity principles and mesh-induced co-activity:

$$\Delta C_{ij} = \alpha_H E_i E_j + \alpha_M\Big(\langle u(x_i, y_i, z_i, t)u(x_j, y_j, z_j, t)\rangle - \langle u(x_i, y_i, z_i, t)\rangle\langle u(x_j, y_j, z_j, t)\rangle\Big), \tag{12}$$

where:

- α_H and α_M represent the rates of Hebbian and mesh-induced plasticity.
- $\langle u(x_i, y_i, z_i, t)u(x_j, y_j, z_j, t)\rangle$ is the time-averaged co-activity between mesh nodes i and j.
- $\langle u(x_i, y_i, z_i, t)\rangle$ is the average displacement at node i.

This mechanism allows the mesh layer to influence plasticity in otherwise unconnected brain regions Pascual-Leone et al. [2005], Fox et al. [2014].

2.3 Interpretation and Empirical Alignment

The brain-mesh model provides a comprehensive framework for understanding brain dynamics by integrating local neural interactions, global phase coherence, and diffuse plasticity effects within a single system. Here, we interpret how

each layer contributes to observed brain functions and align these interpretations with empirical data. Each sublayer in this model addresses phenomena previously unexplained or insufficiently described by conventional neural models. We also present the mathematical mechanisms through which the model captures key neural dynamics, making predictions that can be empirically tested.

2.3.1 Local Dynamics and Regional Interactions

In the model's local and regional layers, dynamics are primarily governed by neural mass models, capturing the excitatory (E) and inhibitory (I) interactions within each brain region. The neural mass models employ the following differential equations to describe the population-level behavior within each region i:

$$\frac{dE_i}{dt} = \frac{1}{\tau_E}\Big(- E_i + S_E\Big(w_{EE}E_i - w_{EI}I_i + P_i + \eta_i^{\text{local}}(t)\Big)\Big), \tag{13}$$

$$\frac{dI_i}{dt} = \frac{1}{\tau_I}\Big(- I_i + S_I\Big(w_{IE}E_i - w_{II}I_i + \eta_i^{\text{local}}(t)\Big)\Big), \tag{14}$$

where:

- τ_E and τ_I are time constants for excitatory and inhibitory populations,
- S_E and S_I are sigmoidal activation functions,
- $w_{EE}, w_{EI}, w_{IE}, w_{II}$ are synaptic weights between populations,
- P_i is an external input to region i,
- $\eta_i^{\text{local}}(t)$ represents Gaussian noise, capturing the stochastic effects observed at the local level.

This local dynamic model is well-supported by empirical evidence from neurophysiology studies showing specific excitatory-inhibitory balance as fundamental for the stability of regional neural activity Deco et al. [2008]. Neural mass models have also been shown to simulate brain oscillations, such as those occurring in the thalamocortical loop during sleep and wake states Steriade [2006].

2.3.2 Global Synchronization and Phase Coherence

A novel aspect of the brain-mesh model is its ability to generate global synchronization and phase coherence across the brain, even in regions without direct structural connections. This is achieved by integrating a metallic mesh-inspired layer that introduces interference patterns across the brain. Synchronization is governed by the following phase dynamics, incorporating both structural connectivity and mesh-induced coupling:

$$\frac{d\theta_i}{dt} = \omega_i + \sum_j K_{ij}\sin(\theta_j - \theta_i) + \kappa u(x_i, y_i, z_i, t), \tag{15}$$

where:

- ω_i represents the natural frequency of region i,
- K_{ij} is the coupling strength between regions i and j based on structural connectivity,
- κ is the coupling coefficient of the mesh-induced interference $u(x_i, y_i, z_i, t)$.

The mesh layer's contribution, $u(x_i, y_i, z_i, t)$, is derived from the elastodynamic wave equation, which allows for wave propagation independent of anatomical connectivity:

$$\frac{\partial^2 u}{\partial t^2} = c^2\nabla^2 u + \eta_{\text{mesh}}(x, y, z, t), \tag{16}$$

where:

- c is the speed of wave propagation across the mesh,

- $\eta_{\text{mesh}}(x, y, z, t)$ represents stochastic fluctuations within the mesh layer.

The wave-like interference patterns allow for phase coherence across distant brain regions, aligning with empirical observations of traveling cortical waves, as shown in studies by Muller et al. [2018], Zhang et al. [2021]. This mechanism effectively explains the presence of large-scale synchrony and phase gradients in cortical activity, phenomena that traditional connectivity models fail to capture without introducing complex, ad hoc assumptions about indirect or cascading connectivity.

2.3.3 Diffuse Plasticity Across Non-Local Connections

The mesh layer enables a form of diffuse plasticity that extends beyond the synaptic-level constraints typically assumed in local models of Hebbian learning. In the fused model, connectivity between regions i and j can change based on prolonged mesh-induced co-activity, even in the absence of direct synaptic connections. This form of plasticity is governed by the following equation:

$$\Delta C_{ij} = \alpha_H \big(E_i E_j\big) + \alpha_M F\Big(\langle u(x_i, y_i, z_i, t) u(x_j, y_j, z_j, t)\rangle - \langle u(x_i, y_i, z_i, t)\rangle \langle u(x_j, y_j, z_j, t)\rangle\Big), \tag{17}$$

where:

- ΔC_{ij}: Represents the change in effective connectivity between regions i and j,
- α_H: The rate of traditional Hebbian plasticity, driven by co-activation of neurons in regions i and j,
- $E_i E_j$: Represents the Hebbian term, reflecting the product of activity levels in regions i and j,
- α_M: The scaling factor for mesh-induced plasticity, determining the overall strength of the mesh's influence on synaptic change,
- $F(...)$: A function that captures the non-linear relationship between mesh-induced co-activity and synaptic change. This function could be sigmoidal or take other forms to reflect threshold effects or saturation in plasticity,
- $\langle u(x_i, y_i, z_i, t) u(x_j, y_j, z_j, t)\rangle$: Represents the time-averaged co-activity between mesh nodes at locations i and j, induced by mesh interference patterns,
- $\langle u(x_i, y_i, z_i, t)\rangle \langle u(x_j, y_j, z_j, t)\rangle$: Represents the product of the time - averaged mesh activity at locations i and j, serving as a baseline to measure the co-activity against.

This equation clarifies that the mesh-induced term modulates the overall synaptic change, scaling the impact of the Hebbian term. The function $F(...)$ captures the non-linear relationship between mesh-induced co-activity and synaptic change, allowing for various plasticity rules depending on the specific form of $F(...)$.

Synaptic Tagging and Capture: The mesh-induced plasticity could be further elaborated by connecting it to the concept of "synaptic tagging and capture." In this framework, the mesh's influence could be seen as modulating the tagging process, where specific patterns of mesh activity could tag synapses for subsequent plasticity. Alternatively, the mesh could influence the availability of plasticity-related proteins (PRPs), which are necessary for long-term potentiation (LTP) and long-term depression (LTD). By modulating the availability of PRPs, the mesh could indirectly regulate the strength and persistence of synaptic changes induced by Hebbian mechanisms. This connection to synaptic tagging and capture provides a more concrete biological mechanism for how the mesh layer could influence synaptic plasticity at a molecular level.

This diffuse plasticity mechanism aligns with findings from non-invasive brain stimulation studies showing that connectivity changes can occur between brain regions without direct synaptic connections, as documented by Pascual-Leone et al. [2005], Fox et al. [2014]. This suggests that the model's mesh-induced plasticity provides a more comprehensive explanation for observed functional reorganization effects, challenging traditional views that restrict plasticity to synaptically coupled regions.

3 Comparative Analysis and Predictive Power of the Brain-Mesh Model

In this section, we examine how the brain-mesh model not only replicates the explanatory power of traditional models for well-known neurobiological phenomena but also enhances explanatory coherence by reducing the need for

supplementary assumptions. By integrating a pervasive mesh layer with conventional local and regional connectivity frameworks, the fused model supports phase synchrony, stable low-frequency oscillations, diffuse plasticity, and state-dependent functional connectivity modulations in a unified manner. Each subsection provides a comparative analysis of these capabilities, elucidating how the fused model extends beyond traditional models in its predictive scope.

3.1 Phase Synchrony and Regional Oscillatory Dynamics

Traditional Models

Current models of phase synchrony rely heavily on structural connectivity matrices and oscillatory dynamics governed by local neural circuits and long-range anatomical pathways. Synchrony between brain regions is often modeled using coupled oscillator frameworks, such as the Kuramoto model:

$$\frac{d\theta_i}{dt} = \omega_i + \sum_j K_{ij} \sin(\theta_j - \theta_i), \tag{18}$$

where:

- θ_i is the phase of the oscillator in region i,
- ω_i represents the natural frequency of each region,
- K_{ij} is the coupling strength based on structural connectivity.

These models successfully explain phase coherence across directly connected or indirectly connected regions, aligning with the connectome structure Honey et al. [2009], Breakspear et al. [2010]. However, this framework is limited in accounting for coherence across regions without structural pathways, as it lacks an inherent mechanism for global synchrony.

Fused Model Prediction

In the fused model, the mesh layer enables widespread phase coherence without requiring strong direct connectivity, as oscillatory synchrony is achieved through mesh-based interference patterns. This effect is mathematically described by extending the Kuramoto phase model to incorporate mesh interference:

$$\frac{d\theta_i}{dt} = \omega_i + \sum_j K_{ij} \sin(\theta_j - \theta_i) + \kappa u(x_i, y_i, z_i, t), \tag{19}$$

where $u(x_i, y_i, z_i, t)$ represents mesh interference at region i, and κ is the coupling strength between phase dynamics and the mesh layer. The inclusion of the mesh interference term allows for phase coherence across anatomically disconnected regions, aligning with observed whole-brain coherence in resting-state networks Muller et al. [2018], Zhang et al. [2021]. This capacity for coherence is driven by wave dynamics within the mesh, supporting phase synchrony across distant regions, which traditional models cannot explain without additional indirect pathways or hypothetical coupling mechanisms.

3.2 Consistent Low-Frequency Oscillations

Traditional Models

Low-frequency oscillations (LFOs) in resting-state networks are typically attributed to intrinsic circuit properties and metabolic constraints within specific brain regions Raichle et al. [2001], Deco and Jirsa [2012]. Oscillations are often modeled as emergent properties of local excitatory-inhibitory balance and feedback loops, with an assumption that local circuits drive these rhythms:

$$f_i = \frac{1}{2\pi}\sqrt{\frac{1}{\tau_E \tau_I}}, \tag{20}$$

where f_i is the resonance frequency of the circuit in region i, dependent on excitatory (τ_E) and inhibitory (τ_I) time constants. However, these circuit-based explanations cannot fully account for the consistency of low-frequency peaks observed across individuals, nor can they explain stable oscillations that span multiple regions beyond local connectivity.

Fused Model Prediction

The fused model predicts low-frequency oscillations as an emergent resonance of the mesh layer rather than as localized circuit phenomena. These oscillations arise from wave interference patterns within the mesh, governed by the wave equation:

$$\frac{\partial^2 u}{\partial t^2} = c^2 \nabla^2 u + \eta_{\text{mesh}}(x, y, z, t), \tag{21}$$

where c is the wave propagation speed, and $\eta_{\text{mesh}}(x, y, z, t)$ represents Gaussian noise in the mesh. The harmonic structure of the mesh layer leads to stable low-frequency oscillations (e.g., delta and theta bands) that remain consistent across individuals. These oscillations are characterized by resonance frequencies:

$$f_n = \frac{nc}{2L}, \tag{22}$$

where f_n is the n-th harmonic frequency, L is a characteristic length of the mesh, and c is the wave speed. This harmonic structure aligns with empirical observations of consistent low-frequency oscillations in resting-state networks Buzsaki [2006], Raichle et al. [2001]. Unlike circuit-based models, the mesh-derived LFOs exhibit minimal individual variability, as the resonance properties of the mesh are less influenced by local circuit changes, providing a unified explanation for stable LFOs across individuals.

3.3 Local and Macro-Regional Plasticity

Traditional Models

Plasticity in conventional models is typically explained by Hebbian learning and spike-timing-dependent plasticity (STDP), where synaptic weights are adjusted based on the co-activation of connected neurons. The change in synaptic strength Δw_{ij} between neurons i and j follows:

$$\Delta w_{ij} = \alpha \left(E_i E_j \right), \tag{23}$$

where α is the learning rate, and E_i, E_j represent pre- and post-synaptic activation levels. This model, while effective for explaining local learning, cannot accommodate plasticity across non-synaptically connected regions. To address long-range plasticity, current models require additional assumptions about indirect pathways or synchronized co-activation Pascual-Leone et al. [2005].

Fused Model Prediction

In the fused model, plasticity arises not only from local interactions but also from diffuse plasticity mediated by the mesh layer. This non-local plasticity is governed by a mesh-induced plasticity rule, where connectivity between regions i and j can be modified based on co-activity in the mesh layer:

$$\begin{aligned}\Delta C_{ij} = \alpha_H E_i E_j + \alpha_M \Big(&\langle u(x_i, y_i, z_i, t) u(x_j, y_j, z_j, t) \rangle \\ &- \langle u(x_i, y_i, z_i, t) \rangle \langle u(x_j, y_j, z_j, t) \rangle \Big),\end{aligned} \tag{24}$$

where:

- α_H: The traditional Hebbian plasticity rate,
- α_M: The mesh-induced plasticity rate,
- $\langle \cdot \rangle$: Time-averaged co-activity induced by mesh interference.

This mechanism enables plasticity to occur across distant, non-synaptic connections, aligning with empirical findings of non-local plasticity in response to brain stimulation Fox et al. [2014]. By supporting adaptive connectivity changes driven by mesh co-activity, the fused model can explain both local learning and long-range plasticity effects, including those observed in brain stimulation studies that cannot be readily accounted for by traditional, synapse-limited plasticity rules.

3.4 Functional Connectivity Modulation by Task vs. Resting States

Traditional Models

Current models explain changes in functional connectivity between task and resting states by suggesting that task engagement enhances the stability of functional connections within task-relevant networks, while resting states exhibit more variable, flexible connectivity Smith et al. [2009]. However, traditional models often lack a clear mechanism for modulating connectivity dynamics as a function of cognitive state without assuming external modulators.

Brain-Mesh Model Prediction

The fused model provides a mechanism for task-dependent modulation by incorporating task-induced resistance to mesh interference. During task engagement, task-relevant regions exhibit increased coherence due to resistance against the diffuse effects of mesh-driven synchrony. This can be formalized by modulating the coupling strength κ between task-related regions and the mesh layer:

$$\kappa_{\text{task}} = \kappa_{\text{baseline}} - \beta, \tag{25}$$

where β is a task-related modulation parameter, reducing the influence of mesh interference on task-active regions. During resting states, where β is minimized, functional connectivity reflects variable interference patterns from the mesh, leading to the dynamic, flexible connectivity observed in resting-state networks.

Task-induced modulation of mesh layer input aligns with findings on task-state stability versus resting-state variability in functional connectivity Raichle et al. [2001], providing a coherent mechanism for state-dependent functional modulation that traditional models do not capture directly.

4 Novel Contributions: Predictions Unique to the Brain-Mesh Model

This section examines the unique predictions of the brain-mesh model, focusing on phenomena that traditional neural connectivity frameworks do not predict. These novel contributions emphasize the model's potential to explain brain dynamics through connectivity-independent mechanisms, phase gradients, harmonic resonances, and mesh-driven plasticity. Each prediction is rooted in rigorous mathematical formalism, with reference to existing empirical data, highlighting the model's capacity to address longstanding gaps in neurobiological theory.

4.1 Prediction 1: Connectivity-Independent Phase Gradients Across Non-Connected Regions

Mathematical Basis

The brain-mesh model introduces phase gradients across neural regions that are not structurally connected, challenging traditional phase coherence models, which typically depend on synaptic connectivity or indirect relay pathways. By incorporating a mesh layer with wave-propagation dynamics, the model enables phase gradients to form and persist independently of anatomical connections.

To rigorously derive the relationship between the mesh's physical properties and observed phase gradients, we start with the wave equation describing phase dynamics within the mesh:

$$\nabla^2 \phi(\mathbf{r}, t) - \frac{1}{c^2} \frac{\partial^2 \phi(\mathbf{r}, t)}{\partial t^2} = 0, \tag{26}$$

where $\phi(\mathbf{r}, t)$ is the phase at position $\mathbf{r}$ and time t, and c is the speed of wave propagation within the mesh layer. This wave equation emerges from the interaction between the mesh's elasticity—allowing for the spatial propagation of phase disturbances—and the distributed oscillators (nodes) within the mesh, which act as coupled, phase-synchronous oscillators.

In this model, the mesh's elasticity is represented as a lattice-like network of springs connecting adjacent nodes, with a spring constant k representing the stiffness of these connections. The distributed nodes themselves are modeled as Kuramoto oscillators, each with an intrinsic frequency ω_i and influenced by neighboring nodes through the elastic connections of the mesh. Thus, phase dynamics in the mesh layer arise from a balance between the natural frequencies of the oscillators and the phase-coupling mediated by the mesh elasticity.

Combining these structural elements, we derive a phase-dynamic wave equation where elasticity, represented by k, determines the wave propagation speed c. Meanwhile, the spatial distribution and interaction strength of oscillators within the mesh dictate the evolution of $\phi(\mathbf{r}, t)$, producing a model of phase gradients that does not rely on structural connectivity.

Wave Interference and Phase Gradients

Wave interference within the mesh layer is a fundamental mechanism for generating phase gradients. As waves propagate across the mesh, their interactions result in constructive and destructive interference patterns, creating regions with consistent phase differences, which manifest as phase gradients.

The elasticity of the mesh facilitates the transmission of these waves across nodes, while the spatial configuration of the nodes influences the resultant interference patterns. These interference patterns lead to the formation of either stable or dynamic phase gradients, depending on the mesh's physical parameters and external influences. When external inputs, such as sensory stimuli, interact with the mesh, phase gradients can shift, but their formation remains connectivity-independent—a unique property enabled by the wave-propagation capacity of the mesh.

Empirical Expectations

The brain-mesh model predicts that phase gradients will be observable across non-connected regions in resting-state conditions, revealing smooth phase shifts that do not align with known anatomical connectivity. Electroencephalography (EEG) and magnetoencephalography (MEG) should detect such gradients, particularly as continuous phase transitions across regions, showing connectivity-independent coherence that extends beyond structural boundaries.

Existing Partial Validation

Empirical studies on cortical traveling waves provide preliminary support for connectivity-independent phase gradients, a phenomenon not fully explained by traditional models. For example, cortical waves have been observed to propagate across large regions of the brain in ways that do not correspond to anatomical connectivity Muller et al. [2018], Zhang et al. [2021]. The mesh layer in the brain-mesh model naturally facilitates this traveling-wave phenomenon, offering a coherent basis for these observations without requiring direct or indirect structural pathways.

Research has shown that phase coherence can extend across spatially separated cortical areas in ways that defy direct structural mapping Breakspear [2017]. These findings align with the brain-mesh model's prediction that an underlying interference-driven mechanism—like the mesh layer—could sustain coherence across regions regardless of direct connectivity.

4.2 Prediction 2: Fixed Harmonic Resonance Peaks with Consistency Across Individuals

Mathematical Basis

The mesh layer's physical constraints predict resonance frequencies that are stable across individuals. These harmonic resonance peaks arise from the mesh's lattice structure, which supports a consistent series of natural frequencies. The resonant frequencies f_n for a harmonic series in the mesh layer are given by:

$$f_n = \frac{nc}{2L}, \tag{27}$$

where:

- f_n is the n-th harmonic frequency,
- c represents wave propagation speed within the mesh,
- L is a characteristic length related to the mesh structure.

This series predicts stable low-frequency oscillations that align with observed resting-state frequencies, as the mesh enforces a harmonic structure with minimal inter-individual variability.

Empirical Expectations

Low-frequency oscillations measured via fMRI, EEG, and MEG should exhibit stable peaks that remain consistent across individuals and recording sessions. These resonance frequencies, such as those observed in the delta and

theta bands, are expected to be more uniform than circuit-based oscillations, which tend to show individual-specific variability.

Existing Partial Validation

Empirical data has shown consistent low-frequency oscillations across subjects, with peaks often aligning with delta and theta bands Buzsaki [2006], Raichle et al. [2001]. Traditional models, which rely on metabolic or circuit-specific explanations, struggle to account for this consistency across individuals. In contrast, the fused brain-mesh model explains these stable frequencies as intrinsic harmonics of the mesh layer, offering a robust alternative to circuit-based explanations.

4.3 Prediction 3: Diffuse, Mesh-Driven Plasticity Across Non-Synaptically Connected Regions

Mathematical Basis

The brain-mesh model proposes a form of plasticity driven by mesh interference patterns, allowing for diffuse plasticity effects that span non-synaptic, spatially distributed regions. This diffuse plasticity is modeled as a function of persistent co-activation patterns within the mesh layer, altering connectivity strengths over time. A mesh-based plasticity rule can be expressed as:

$$\Delta A_{ij} = \alpha\Big(\langle I_i(t)I_j(t)\rangle_{\text{mesh}} - \langle I_i(t)I_j(t)\rangle_{\text{baseline}}\Big), \tag{28}$$

where:

- ΔA_{ij} represents the change in effective connectivity between regions i and j,
- α is a learning rate constant,
- $\langle I_i(t)I_j(t)\rangle_{\text{mesh}}$ represents co-activity induced by mesh interference,
- $\langle I_i(t)I_j(t)\rangle_{\text{baseline}}$ represents baseline co-activity.

This plasticity mechanism allows for adaptive changes across both synaptically and non-synaptically connected regions, accommodating long-range, non-local adaptive plasticity.

Empirical Expectations

Following repetitive stimulation, resting-state functional connectivity should reveal connectivity changes in non-connected regions. Such changes are anticipated to persist beyond stimulation periods, indicating mesh-driven plasticity that is independent of direct anatomical connections.

Existing Partial Validation

Empirical findings in non-invasive stimulation studies have shown functional connectivity changes that extend beyond the targeted stimulation sites Pascual-Leone et al. [2005], Fox et al. [2014]. These findings suggest a form of non-local plasticity that aligns with the mesh model's non-synaptic plasticity prediction, as traditional models lack mechanisms to explain these diffuse changes.

4.4 Prediction 4: Task vs. Rest Differential Response to Mesh Interference

Mathematical Basis

The mesh model posits that task states modulate the influence of mesh layer interference on neural connectivity, reducing variability during tasks compared to rest. This differential response to mesh interference is based on the hypothesis that task engagement introduces stabilizing inputs that counteract mesh-generated variability. The effect of task state can be modeled by a modulation of mesh input, such that:

$$P_i^{\text{task}} = \gamma_{\text{task}} u(x_i, y_i, z_i, t), \tag{29}$$

where:

- P_i^{task} represents mesh-influenced input during tasks,
- $\gamma_{\text{task}} < \gamma_{\text{rest}}$, indicating reduced mesh influence during task engagement,
- $u(x_i, y_i, z_i, t)$ is the mesh displacement field.

This task-modulated interference is expected to reduce variability in task-state connectivity, as observed in empirical studies contrasting resting-state and task-state connectivity.

Empirical Expectations

Connectivity measures (such as those derived from fMRI or MEG) should show higher coherence stability during tasks and greater fluctuation in resting-state data. The mesh model predicts lower task-induced variability due to reduced mesh influence, aligning with stable connectivity observed in task-focused neural states.

Existing Partial Validation

Empirical studies indicate increased variability in resting-state connectivity compared to task-induced states, where connectivity is more stable Raichle et al. [2001], Mantini et al. [2007]. This aligns with the mesh model's prediction that task engagement buffers neural connectivity from mesh-driven fluctuations, providing a unified explanation for differences in task vs. resting-state coherence that traditional models cannot fully explain.

Summary

These predictions highlight the unique explanatory power of the fused brain-mesh model, addressing complex neural phenomena with novel mechanisms that complement and extend beyond existing models. Each prediction, supported by preliminary empirical evidence, sets a foundation for further experimental validation, suggesting that the mesh model offers a rigorous and innovative approach to understanding brain-wide dynamics.

5 Empirical Strategy for Validation

This section proposes a rigorous empirical framework for testing the predictions unique to the fused brain-mesh model and provides comparative strategies to evaluate its distinctiveness relative to existing neural connectivity models. The goal is to implement a multi-layered empirical approach involving cross-modal testing, targeted interference disruption, longitudinal observation of plasticity, and direct comparative analysis. This empirical strategy is designed to substantiate the fused model's predictions, test their robustness across methods, and critically compare outcomes against traditional models.

5.1 Cross-Modal Empirical Testing

To empirically validate the fused brain-mesh model's predictions, a cross-modal approach using fMRI, EEG, and MEG will be essential for measuring neural activity under different conditions. This multi-modal strategy allows for comprehensive testing of key predictions, particularly regarding phase gradients, harmonic resonance, and diffuse plasticity effects, which can be assessed at the levels of spatial resolution (fMRI) and temporal precision (EEG/MEG).

5.1.1 Resting-State Testing

One of the core predictions of the fused model is the existence of connectivity-independent phase gradients, which should manifest in resting-state activity as continuous phase shifts across both connected and non-connected brain regions. Using MEG, we can capture real-time oscillatory phase activity, enabling the detection of traveling waves or phase gradients independent of known structural pathways Muller et al. [2018], Zhang et al. [2021]. Additionally, resting-state fMRI can be used to detect stable phase coherence across regions where direct connectivity is absent, confirming the spatial consistency of these gradients.

5.1.2 Task and Post-Stimulation Conditions

Testing predictions of reduced variability in task versus rest conditions requires EEG/MEG measurements during cognitive tasks with well-documented patterns of neural activity, such as working memory or attentional tasks. Reduced task-state variability should correlate with the model's prediction that mesh interference has less influence on task-driven activity. In post-stimulation conditions, EEG/MEG can also verify if harmonic resonance peaks—predicted to be stable across subjects—are retained or altered.

The cross-modal approach will allow for correlating oscillatory phase and coherence findings in resting-state versus task conditions and confirm that these phenomena are mesh-layer-specific, offering robust comparisons to existing models, which often predict connectivity based primarily on anatomical pathways Honey et al. [2009], Deco et al. [2021].

5.2 Targeted Interference Disruption Experiments

To empirically test the robustness of phase coherence, resonance, and diffuse plasticity as predicted by the fused brain-mesh model, we propose targeted interference disruption experiments using transcranial magnetic stimulation (TMS) and transcranial alternating current stimulation (tACS). These experiments are designed to manipulate interference patterns generated by the mesh layer within specific brain regions and to observe the resultant effects on connectivity and oscillatory dynamics. The approach highlights the model's predictions of connectivity-independent coherence and non-local plasticity effects, distinguishing it from traditional connectivity models.

5.2.1 TMS Disruption of Phase Coherence

In this experiment, TMS will be used to deliver brief, focal disruptions to brain areas associated with stable phase coherence within resting-state networks, particularly the default mode network (DMN) Raichle et al. [2001]. According to the fused brain-mesh model, interference-driven phase coherence should extend beyond direct anatomical connections, such that disrupting coherence locally should produce immediate, observable impacts on distal, non-connected regions. This prediction diverges significantly from models that limit coherence effects to anatomically connected networks Breakspear et al. [2010], Sporns [2011].

Experimental Setup:

- **Target Region:** The posterior cingulate cortex (PCC), a central hub of the DMN, will be targeted for disruption, leveraging its known role in resting-state coherence.
- **Stimulation Parameters:** TMS will be administered with a figure-eight coil, delivering biphasic pulses at 1 Hz and 110% of the resting motor threshold to maximize efficacy without overstimulation.
- **Data Acquisition:** EEG recordings will be collected from 64 scalp electrodes, focusing on theta band coherence (4–8 Hz) between the PCC and other DMN regions, such as the medial prefrontal cortex (mPFC), as well as non-DMN regions like the dorsolateral prefrontal cortex (dlPFC).

Hypotheses and Expected Results: We hypothesize that TMS-induced disruption in the PCC will reduce theta coherence not only in the mPFC but also in the dlPFC, despite the absence of direct anatomical connections between the PCC and dlPFC. If observed, such coherence reduction would lend support to the brain-mesh model's prediction of connectivity-independent phase gradients, reinforcing the notion that global coherence is mediated by an underlying mesh rather than solely by anatomical pathways.

5.2.2 tACS Modulation of Resonance Peaks

The brain-mesh model posits that harmonic resonance peaks within low-frequency oscillations arise from the mesh layer's intrinsic properties rather than from local circuit dynamics. To validate this hypothesis, we will apply tACS to induce or disrupt oscillatory activity at frequencies predicted by the mesh model's harmonic series, with the aim of observing shifts in power and coherence across the brain. Such shifts, if consistent across individuals, would support the model's prediction of resonance driven by the mesh layer, independent of individual variability in local circuits Buzsaki [2006].

Experimental Setup:

- **Target Region:** Bilateral stimulation will be applied to the prefrontal cortex using electrodes placed at F3 and F4, following the 10–20 EEG electrode placement system.
- **Stimulation Parameters:** A sinusoidal current of 10 Hz (alpha band) and 1 mA intensity will be applied, targeting resonance effects within the alpha frequency range.
- **Data Acquisition:** EEG recordings from 64 electrodes will measure changes in alpha power and coherence across frontal, parietal, and occipital regions.

Hypotheses and Expected Results: We hypothesize that applying 10 Hz tACS to the prefrontal cortex will disrupt the mesh model's predicted harmonic resonance peak in the alpha band, reducing coherence and power in the alpha range across brain regions. Such a disruption would suggest that resonance properties are mesh-driven and extend beyond the influence of local circuits, aligning with the model's predictions.

6 Empirical Strategy for Validation and Comparison with Existing Models

This section proposes a rigorous empirical framework for testing the predictions unique to the fused brain-mesh model and provides comparative strategies to evaluate its distinctiveness relative to existing neural connectivity models. The goal is to implement a multi-layered empirical approach involving cross-modal testing, targeted interference disruption, longitudinal observation of plasticity, and direct comparative analysis. This empirical strategy is designed to substantiate the fused model's predictions, test their robustness across methods, and critically compare outcomes against traditional models.

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6.2.3 Distinguishing the Brain-Mesh Model from Traditional Connectivity Models

By conducting these targeted interference disruption experiments, we aim to generate empirical evidence that can differentiate the brain-mesh model from conventional anatomical connectivity models. Specific expectations are as follows:

Phase Coherence Across Non-Connected Regions: If disrupting phase coherence in one region (e.g., PCC) alters coherence in non-connected regions (e.g., dlPFC), this would support the brain-mesh model's prediction of connectivity-independent phase coherence, challenging traditional models that restrict coherence effects to directly connected regions.

Mesh-Driven Resonance Peaks: If tACS-induced disruption of resonance peaks alters low-frequency coherence across the brain, independent of individual variability in local circuitry, it would indicate that the mesh layer underpins global resonance. This result would highlight a mechanism for consistent, brain-wide oscillatory synchrony that existing models, focused on localized circuits, cannot fully explain.

By carefully analyzing these predicted alterations in phase coherence and oscillatory resonance, we can gather evidence supporting the brain-mesh model's unique contributions to global brain dynamics. These findings are expected to challenge the spatial constraints of traditional connectivity models, which predict that TMS/tACS effects will be more spatially localized, with minimal influence on non-connected regions.

Experimental Specificity and Scientific Rigor: This experimental design includes precise specifications to enhance scientific rigor:

- **Targeted Brain Regions:** Specific selection of the PCC for TMS and prefrontal cortex for tACS, both central to coherent network dynamics.
- **Stimulation Parameters:** Defined TMS pulse frequency, coil type, tACS frequency, and current intensity to control for variability and ensure reproducibility.
- **Data Collection and Analysis:** EEG monitoring with high electrode density to capture phase coherence and power shifts in specific frequency bands.

By articulating the distinct expected outcomes for the brain-mesh model and comparing them to traditional connectivity model predictions, these interference disruption experiments strengthen the model's scientific foundation and potential impact in the field. Such empirical validation could open new avenues for understanding brain dynamics and the mechanisms underlying coherence and plasticity on a global scale.

6.3 Longitudinal Studies on Plasticity

To validate the prediction of diffuse plasticity arising from mesh interference patterns, longitudinal studies will measure resting-state connectivity shifts over time following stimulation. Longitudinal fMRI will track connectivity changes in non-synaptically connected regions to determine whether mesh-driven plasticity creates sustained changes in network structure, particularly in areas outside traditional anatomical pathways.

6.3.1 Post-Stimulation Connectivity Shifts

In the initial phase, repetitive TMS will be administered over several sessions to brain regions with well-defined, anatomically constrained connectivity. Resting-state fMRI and MEG recordings will then monitor changes in connectivity across both directly connected and remote, non-connected regions over several months. According to the fused model, mesh interference should drive plasticity effects detectable as gradual, mesh-driven shifts in resting-state connectivity even in regions without structural connections to the stimulated site.

6.3.2 Persistence of Mesh-Induced Changes

A critical measure of mesh-driven plasticity is the persistence of connectivity changes. Traditional models might predict temporary connectivity shifts based on Hebbian principles Bi and Poo [2001], but the mesh model suggests that persistent non-synaptic connectivity adjustments should remain detectable for prolonged periods. If confirmed, this would underscore a unique plasticity mechanism distinct from purely synaptic models, supporting the mesh layer's role in generating long-lasting, non-local plasticity Fox et al. [2014], Pascual-Leone et al. [2005].

6.4 Comparative Analysis

To further establish the distinctive predictive capabilities of the fused model, a rigorous comparative analysis will contrast empirical data derived from these experiments with existing explanations from alternative models. This analysis will underscore how the fused model avoids ad hoc assumptions commonly needed in conventional frameworks and provides a unified mechanism for the observed phenomena.

6.4.1 Contrasting Phase Coherence Mechanisms

Existing models attribute phase coherence largely to structural connectivity, with phase synchrony limited to anatomically connected networks Deco and Jirsa [2012]. In contrast, the mesh model predicts that phase gradients and coherence can propagate without direct structural links, explaining traveling waves and cortical synchronization without presupposing connectivity-based constraints Muller et al. [2018], Zhang et al. [2021].

The comparative analysis will involve quantitative contrasts of coherence data gathered under the fused model's predictions versus structural connectivity-based predictions. Such analysis will be conducted on resting-state EEG/MEG data to assess whether the coherence patterns align with mesh-based interference rather than connectivity-driven synchrony, marking a clear divergence from traditional models.

6.4.2 Evaluating Harmonic Resonance Explanations

Current models account for low-frequency oscillations primarily as emergent properties of metabolic or circuit constraints, but these explanations often lack consistency across individuals Buzsaki [2006]. The fused model, however,

predicts that low-frequency peaks correspond to fixed harmonic resonances in the mesh, expected to exhibit consistency across subjects. Comparative analysis of EEG/MEG data will verify whether resonance frequencies align with this predicted consistency, supporting the fused model's resonance-based approach over circuit-limited theories.

6.4.3 Testing Plasticity Mechanisms

Traditional plasticity models, such as spike-timing-dependent plasticity (STDP) and Hebbian mechanisms, predict connectivity changes based on synaptic co-activation Bi and Poo [2001]. These models fall short in accounting for non-local plasticity effects. By systematically comparing the connectivity patterns observed in post-stimulation fMRI studies, the analysis will evaluate whether the mesh model's diffuse plasticity prediction captures empirically observed changes more comprehensively, validating the mesh layer's ability to support plasticity across non-synaptic regions.

6.5 Summary

The empirical strategy outlined here integrates cross-modal testing, targeted interference, longitudinal plasticity observation, and comparative analysis, providing a robust validation pathway for the fused brain-mesh model. This multi-dimensional approach will not only rigorously test the model's unique predictions but also establish its advantages over traditional connectivity frameworks, particularly in its ability to offer a coherent, unified explanation for diverse neurophysiological phenomena without resorting to ad hoc assumptions.

7 Arguments for the Brain-Mesh Model as an Integrative Framework

The fused brain-mesh model, with its addition of an all-brain mesh layer, offers a framework for examining large-scale brain dynamics that builds on and expands existing approaches. By providing a unified mechanism with notable explanatory power, increased predictive specificity, and alignment with empirical findings, this model addresses several gaps in our understanding of brain connectivity. Here, we explore the key reasons the fused model may serve as an effective framework for advancing neurobiological research.

7.1 A Unified Mechanism for Diverse Phenomena

The all-brain mesh layer enables a unified approach to several complex phenomena in neural dynamics, including synchrony, plasticity, and resonance. Unlike traditional models, which may require separate explanations for each phenomenon, the mesh-based structure allows for a cohesive explanation through interference dynamics.

Mathematical Basis: The unified framework is grounded in the physics of wave interference, described by the elastodynamic wave equation:

$$\rho \frac{\partial^2 u}{\partial t^2} = \nabla \cdot (C : \nabla u) + \eta_{\text{mesh}}(x, y, z, t), \tag{30}$$

where $u(x, y, z, t)$ represents mesh displacement, and C is the elasticity tensor. This equation governs wave propagation across the mesh layer, supporting mechanisms for synchrony, coherence, and oscillatory stability over large brain areas without dependence on direct connectivity.

Empirical Implications: This unified mechanism implies that synchrony, plasticity, and resonance may manifest in a coordinated manner across broad networks. This aligns with observations of coherence in resting-state networks and distributed plastic changes following non-invasive stimulation Fox and Raichle [2007], Pascual-Leone et al. [2005]. Traditional models, which often attribute each phenomenon to separate processes, provide limited frameworks for these co-occurrences.

The mesh-based interference mechanism thus inherently supports phase synchrony and plasticity across networks, appearing as continuous effects under the mesh layer, contrasting with the segmented processes in conventional neural mass or connectome-based models Deco et al. [2021], Muller et al. [2018].

7.2 Enhanced Explanatory Power for Cross-Regional Coupling and Network Reorganization

In existing models, cross-regional synchrony and network reorganization are typically addressed with speculative mechanisms or considerable assumptions. The fused brain-mesh model interprets these phenomena through wave interference dynamics, which are physically grounded and avoid ad hoc constructs.

Limitations of Ad Hoc Explanations: Current frameworks often introduce concepts like “functional hubs” or "cross-regional coupling" to explain synchrony across distant regions Raichle et al. [2001], van den Heuvel and Sporns [2013]. These explanations are descriptive and lack robust physical underpinnings. In contrast, the mesh-based interference mechanism allows cross-regional synchrony as an inherent property of the brain’s wave dynamics.

Dynamic Reorganization Without Structural Changes: The mesh layer facilitates dynamic reconfiguration of synchrony without structural updates. Interference patterns adjust based on task demands or resting-state fluctuations, providing flexibility in network reorganization Deco et al. [2008], Deco and Jirsa [2012]. This property simplifies explanations for task-state modulation and variability in network configurations.

7.3 Specific, Testable Predictions that Extend Beyond Existing Models

The mesh layer adds distinctive features to the fused model, enabling specific and testable predictions. These include connectivity-independent phase gradients, harmonic frequencies, and task-modulated interference.

Phase Gradients Independent of Connectivity: The model predicts phase gradients observable across the cortex without direct structural connectivity. These gradients emerge as interference-driven phase shifts:

$$\theta(x, y, t) = \theta_0 + \frac{d\theta}{dx}x + \frac{d\theta}{dy}y. \tag{31}$$

This suggests that neuroimaging data (e.g., EEG or MEG) should reveal continuous phase gradients not explained by connectivity matrices Honey et al. [2009].

Consistent Harmonic Resonance: Resonance peaks predicted by the mesh layer are expected to display consistency across individuals, derived from its wave-based harmonic properties:

$$f_n = \frac{nc}{2L}, \tag{32}$$

where f_n is the n-th harmonic frequency, c is the wave propagation speed, and L is the characteristic mesh length. This contrasts with traditional models that attribute low-frequency oscillations to circuit properties or metabolic factors Buzsaki [2006], Mantini et al. [2007].

Task-Related Modulation of Interference: The mesh model predicts that task demands reduce interference and enhance coherence stability. This aligns with findings of increased stability during tasks compared to resting-state variability Smith et al. [2009], Fox et al. [2014].

7.4 Alignment with Empirical Data and Model Parsimony

The mesh model aligns with empirical findings while maintaining parsimony by avoiding the need for multiple, isolated mechanisms.

Cortical Traveling Waves: Traveling waves observed in neurophysiological studies naturally arise from the mesh layer’s wave propagation dynamics Muller et al. [2018], Zhang et al. [2021]. Traditional models require additional assumptions to explain these phenomena.

Low-Frequency Oscillations: Harmonic resonance in the mesh provides a parsimonious explanation for consistent low-frequency oscillations across individuals, which traditional models attribute to metabolic or circuit constraints Buzsaki [2006], Raichle et al. [2001].

Stimulation-Induced Plasticity: Non-invasive stimulation studies demonstrate functional connectivity changes extending beyond synaptic connections, suggesting diffuse plasticity effects Pascual-Leone et al. [2005], Fox et al. [2014]. The mesh model supports such findings through its interference-driven connectivity adjustments.

7.5 Summary

In summary, the fused brain-mesh model advances neurobiological frameworks by unifying diverse phenomena under a wave-interference mechanism. It improves explanatory power for cross-regional interactions, offers specific predictions, and aligns with empirical data in a parsimonious manner. These characteristics make it a promising framework for future exploration of large-scale brain dynamics.

8 Astrocytic Networks as Candidates for a Biological Correlate

Astrocytic networks, with their complex gap-junction connectivity, calcium wave dynamics, and influence on synaptic modulation, provide a robust biological foundation for the proposed mesh-like layer in the brain-mesh model. While astrocytic syncytia do not form a metallic lattice, their structural and functional characteristics align closely with the model's core requirements, supporting low-frequency phase coherence, interference-driven resonance, and distributed plasticity. This section rigorously establishes that astrocytic networks can meet the predictions of the mesh model through their statistical and functional properties, with a known limitation in high-frequency coherence. By analyzing the mathematical and physiological principles that govern astrocytic activity, we demonstrate how these networks form a biologically viable substrate for brain-wide synchrony, phase coherence, and harmonic resonance—fundamental elements of the brain-mesh model.

8.1 Structural Compatibility: Network Connectivity and Small-World Properties

Astrocytes form an extensive syncytium through gap junctions that connect astrocytic domains across cortical and subcortical regions. This network forms a small-world topology characterized by high clustering coefficients and short average path lengths between nodes, facilitating near-global signal propagation Fields et al. [2015], Bullmore and Sporns [2012]. In terms of connectivity, astrocytic networks approximate the mesh-like architecture necessary for the model: while irregular, their density and distribution across brain regions support quasi-lattice properties, enabling efficient communication and coherence across distances.

Mathematically, this network's small-world structure can be modeled with parameters describing connectivity density and path length. For N astrocytes and an average connectivity degree $\langle k \rangle$, the average path length L follows:

$$L \approx \frac{\ln(N)}{\ln(\langle k \rangle)}, \tag{33}$$

confirming that, while astrocytic networks lack strict regularity, their small-world topology allows coherence to propagate widely with minimal loss. This aligns with the brain-mesh model's requirements for brain-wide phase synchrony.

8.2 Wave Dynamics and Resonance: Compatibility with Mesh Model Equations

The propagation of calcium waves within astrocytic networks provides a foundation for interference patterns akin to those predicted by the mesh model. Calcium waves travel via gap junctions at relatively slow speeds (10–30 μm/s), but their propagation follows a wave equation directly comparable to that used in metallic meshes. Adjusted for slower astrocytic signal transmission, the equation is:

$$\frac{\partial^2 C(x,t)}{\partial t^2} = v^2 \nabla^2 C(x,t) - \gamma \frac{\partial C(x,t)}{\partial t} + S(x,t), \tag{34}$$

where $C(x,t)$ represents calcium concentration, v the propagation speed, γ the damping coefficient, and $S(x,t)$ the external excitation from neuronal signaling. Despite the slower speed, calcium waves enable phase coherence and interference patterns essential for synchrony in the low-frequency range Fox and Raichle [2007], Buzsaki [2006].

This alignment with the wave equation structure allows astrocytic networks to create interference-driven coherence that operates effectively below approximately 12 Hz—a range sufficient for most large-scale brain dynamics, including resting-state synchrony.

8.3 Harmonic Resonance and Frequency Stability

The brain-mesh model predicts consistent resonance frequencies across individuals, typically in the low-frequency delta, theta, and alpha bands. Astrocytic networks support this prediction through the intrinsic properties of calcium wave dynamics, which generate harmonic resonance in a way that can be mathematically described similarly to resonance in metallic systems.

For a quasi-continuous network of interconnected astrocytes, the natural frequency f_n of harmonics is given by:

$$f_n = \frac{nv}{2L}, \tag{35}$$

where L represents the effective length of the astrocytic syncytium and n an integer for each harmonic mode. This equation demonstrates that resonance peaks align with a quasi-regular harmonic structure, supported by astrocytes' stable wave propagation speed v.

As observed in neuroimaging studies, low-frequency oscillatory peaks in fMRI and EEG data remain consistent across individuals, supporting the astrocytic network's ability to produce the brain-mesh model's harmonic resonance Mantini et al. [2007].

8.4 Summary and Implications

Astrocytic networks, with their small-world topology, calcium wave dynamics, and harmonic properties, provide a biologically plausible substrate for the mesh layer in the brain-mesh model. Their ability to support phase coherence, resonance, and distributed plasticity aligns with the model's predictions, emphasizing the relevance of glial dynamics in large-scale brain function. By bridging mathematical models with astrocytic physiology, this section establishes the viability of astrocytic networks as an integrative framework for understanding brain-wide dynamics.

8.5 Plasticity and Network-Wide Connectivity Modulation

Astrocytes play an active role in synaptic plasticity through gliotransmitter release and calcium signaling, impacting both local synaptic strength and broader network coherence. The brain-mesh model's prediction of diffuse, mesh-driven plasticity aligns well with astrocytic influence, as astrocytes modulate neuronal connectivity across both synaptic and non-synaptic domains. This enables a form of plasticity that extends beyond direct synaptic connections, shaping long-range connectivity changes through distributed astrocytic signaling.

This plasticity can be mathematically modeled by defining connectivity changes w_{ij} between neurons i and j as follows:

$$\Delta w_{ij} \propto \langle A(x_i)A(x_j)\rangle - \langle A(x_i)\rangle\langle A(x_j)\rangle, \tag{36}$$

where $A(x)$ denotes astrocytic activity near neurons i and j. This framework enables long-range, non-synaptic plasticity through astrocytic interactions, allowing phase coherence and plastic changes across disparate brain regions. Such distributed modulation reflects the mesh model's hypothesized long-range plasticity effects, making astrocytic networks a fitting biological correlate.

8.6 Developmental Implications: Organizing Principles and Sensorimotor Scaffolding

Astrocytic networks emerge early in brain development, creating a scaffold that interacts with sensorimotor inputs to shape neural architecture. This role aligns with the mesh model's developmental implications, providing a basis for consistent structural and functional patterns across individuals. Even in cases of unilateral hemispherectomy, astrocytic networks support compensatory reorganization, promoting topological equivalence in the remaining hemisphere Pascual-Leone et al. [2005], Fields et al. [2015].

The brain-mesh model suggests that a stable, early-developing network layer guides neuronal development and connectivity. Astrocytes, with their early onset and pervasive influence on synaptic organization, provide a suitable substrate for this role. Their quasi-lattice structure, distributed signaling capabilities, and interaction with sensory circuits allow them to adaptively scaffold neural development, preserving core structural patterns that persist across individual variations and environmental interactions.

8.7 Mathematical Compatibility with Metallic Mesh Models

To demonstrate the mathematical fidelity of astrocytic networks to the brain-mesh model, we can directly map their signaling dynamics to the core equations of metallic mesh models. While astrocytic networks operate at a slower propagation speed, the equations retain the same fundamental structure when applied to astrocytic calcium waves, phase coherence, and network plasticity.

8.7.1 Wave Equation Structure

In both metallic and astrocytic meshes, the fundamental wave equation structure holds, with propagation speed adjusted to reflect the slower diffusion-based transmission in astrocytic networks. The wave equation is expressed as:

$$\frac{\partial^2 C(x,t)}{\partial t^2} = v^2\nabla^2 C(x,t) - \gamma\frac{\partial C(x,t)}{\partial t} + S(x,t), \tag{37}$$

where $C(x,t)$ represents calcium concentration, v the wave speed, γ the damping coefficient, and $S(x,t)$ external excitation. This slower transmission, while limiting high-frequency coherence, supports the low-frequency synchronization central to the brain-mesh model.

8.7.2 Elasticity and Resonance

In metallic meshes, elasticity follows Hooke's Law for springs. Astrocytic networks exhibit a comparable "elasticity" through their flexible, gap-junction-based connectivity, allowing local perturbations to propagate throughout the network. This elasticity enables stable low-frequency oscillations and resonance peaks that resemble harmonic structures, even without strict spatial regularity.

8.7.3 Stochastic Interference

Biological noise within astrocytic signaling aligns with the noise term $\eta(x, t)$ in metallic mesh equations. This stochastic component contributes to diffuse coherence across the network, facilitating resilient, mesh-like interference patterns under variable biological conditions.

8.7.4 Plasticity Mechanics

Astrocytes influence synaptic weights through gliotransmitter-mediated modulation, akin to structural adjustments in metallic meshes. The connectivity adjustment equation:

$$\Delta A_{ij} \propto \langle I_i I_j \rangle, \tag{38}$$

enables astrocytic networks to sustain mesh-like plasticity, facilitating distributed plastic changes across brain regions.

8.7.5 Phase Synchronization

Astrocytic networks' phase coherence across distances can be described by a modified Kuramoto model, adjusted for the slower, diffusion-based propagation of calcium waves. This provides a framework for phase alignment across the network, capturing the mesh model's predictions for synchronized, low-frequency dynamics.

8.8 Conclusion: Astrocytic Networks as a Statistically and Functionally Equivalent Mesh-Like Layer

Astrocytic networks meet the brain-mesh model's requirements for low-frequency coherence, resonance, and diffuse plasticity through their quasi-lattice structure and calcium-based wave propagation. While these networks lack the rigid regularity of metallic lattices, their small-world connectivity, adaptable signaling dynamics, and robust interference capabilities provide a biologically feasible substrate for mesh-like brain-wide synchrony.

By supporting a mesh model structure, astrocytic networks enable the brain-mesh model's predictions for global coherence, plasticity, and structural organization, making them an optimal biological correlate for the brain-mesh model's theoretical framework. Further empirical validation, particularly using high-resolution imaging and dynamical modeling, could solidify astrocytic networks as the substrate underlying brain-wide coherence and adaptability, offering new insights into the mechanistic underpinnings of large-scale brain function.

9 Discussion

The Discussion section contextualizes the brain-mesh model within contemporary neurobiology and theoretical neuroscience, exploring its implications, limitations, and potential as a framework for advancing our understanding of brain dynamics. We address the model's contributions and its capacity to interface with empirical research, while acknowledging areas that require further investigation and proposing directions for future work. This section ends with broader reflections on how the fused model aligns with theoretical approaches in network neuroscience and suggests possible applications in neuroengineering and AI.

9.1 Implications for Neurobiology and Cognitive Neuroscience

The brain-mesh model introduces a novel framework for understanding large-scale brain dynamics by integrating local, regional, and global connectivity within a coherent mathematical structure. This multi-layered approach offers a systematic basis for investigating brain function at multiple levels, from synaptic interactions to whole-brain oscillations, which could illuminate mechanisms underlying complex cognitive phenomena.

The model's capacity to generate phase coherence, low-frequency oscillations, and diffuse plasticity across the entire brain suggests it could provide valuable insights into phenomena typically explored in cognitive neuroscience, such as attention, memory, and consciousness. For instance, the model's predictions about connectivity-independent phase gradients resonate with studies indicating that large-scale phase synchronization is crucial for attentional focus and may

modulate task-specific networks Friston [2011], Muller et al. [2018]. By introducing a wave-propagating mesh layer capable of sustaining phase coherence independent of direct connectivity, the model allows a mechanistic exploration of how attentional states might involve widespread coordination across non-connected regions Mantini et al. [2007].

The brain-mesh model could also extend understanding in areas like sleep and neurodevelopment. Sleep is characterized by distinct oscillatory dynamics (e.g., slow-wave sleep, REM) that may be influenced by a brain-wide interference-driven layer, with implications for synaptic consolidation and memory Buzsaki [2006]. During neurodevelopment, structural connectivity and functional synchrony evolve concurrently; the mesh layer might support hypotheses about non-synaptic, diffuse mechanisms contributing to global network maturation and coherence, particularly in the development of resting-state networks Raichle et al. [2001].

9.2 Limitations and Future Directions

Despite its potential, the brain-mesh model presents limitations that must be addressed to refine its theoretical and empirical robustness. The primary challenge lies in its empirical testing; as a high-level model integrating hypothetical wave-propagation mechanics, it requires advanced imaging techniques that can capture real-time, whole-brain oscillations and phase shifts across unconnected regions. Current methods such as fMRI, EEG, and MEG provide only partial alignment with the model's predictions, often limited by spatial or temporal resolution Honey et al. [2009], Zhang et al. [2021].

Future research should focus on computational simulations of the mesh layer's dynamics to explore its parameter space and optimize the model for empirical alignment with known neural phenomena. Computationally modeling the elasticity and wave-interference properties of the mesh layer will allow for the generation of precise predictions and facilitate comparisons with neural data. Additionally, high-resolution imaging technologies, such as 7-Tesla MRI or optogenetic recording techniques, could offer empirical support by revealing oscillatory dynamics and coherence patterns consistent with the model's predictions Fox et al. [2014], Siebner et al. [2004].

We also recommend integrating this model with other connectivity frameworks, such as graph-theoretical approaches, to build a more comprehensive understanding of how the mesh dynamics might modulate traditional network properties, such as hub connectivity and small-world architecture Sporns [2011]. Such integrations could enhance the model's applicability, particularly in studying disorders characterized by dysregulated synchrony or connectivity, such as schizophrenia and autism spectrum disorders.

9.3 Broader Implications for Theoretical Neuroscience

Beyond its specific neurobiological applications, the fused brain-mesh model introduces a theoretical paradigm with broad implications for network theory, neuroengineering, and brain-inspired AI. By combining principles from elastodynamics, phase synchronization, and network connectivity, the model provides a blueprint for creating multi-layered systems capable of dynamic, coherence-driven behavior—a promising direction for neuromorphic engineering Breakspear et al. [2010].

In network theory, the model's use of a mesh layer for coherence and synchronization across non-connected nodes could inspire new approaches in distributed networks, offering solutions for coherence-driven networks that bypass the need for direct connections. This property could have applications in decentralized computing systems and parallel processing algorithms, which seek to maintain coordinated states across large arrays of processors without direct interconnections Kuramoto [1984].

The brain-mesh model also holds promise for brain-inspired AI. Traditional deep learning architectures rely on hierarchical connectivity and fail to capture the coherence seen in biological systems. Integrating mesh-like interference layers that promote global coherence across distributed elements could enable AI systems to achieve task-specific coherence, potentially enhancing the system's capacity for generalization and complex pattern recognition. In this way, the brain-mesh model can contribute to designing novel AI architectures that mirror the brain's functional advantages of coherence and adaptability Deco et al. [2021], van den Heuvel and Sporns [2013].

10 Conclusion

In sum, the brain-mesh model aims at integrating local, regional, and global connectivity mechanisms within a unified, mathematically rigorous framework. This model accounts for diverse neural phenomena, such as phase coherence, low-frequency oscillations, and diffuse plasticity, which are critical for understanding cognition and consciousness. By leveraging a mesh-like interference-driven layer, the model transcends the limitations of traditional connectivity

frameworks, introducing new avenues for explaining brain-wide coherence across disconnected regions and predicting unique patterns of oscillatory behavior.

The brain-mesh model's predictions regarding connectivity-independent phase gradients, harmonic resonance frequencies, and non-local plasticity underscore its potential for empirical validation. Future research utilizing advanced imaging techniques and computational simulations is necessary to substantiate these predictions and refine the model's parameters. If validated, the brain-mesh model could offer a transformative framework in neurobiological research, influencing approaches in cognitive neuroscience, neurodevelopment, and neuroengineering.

In closing, we encourage further experimental testing and theoretical exploration to substantiate the brain-mesh model's predictions and assess its broader implications for neurobiology, network theory, and artificial intelligence. The model's novel approach to brain connectivity represents an exciting frontier in theoretical neuroscience, with the potential to reshape our understanding of the brain's dynamic, coherence-driven nature.

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