

Online Resource 2

Relation to prior publications and preprints

Article

The Syncytial Mesh Model: glial spatial control, neuronal response geometry, and the limits of local EEG reduction

Journal

Cognitive Neurodynamics

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Contents

Publication overlap, preprint history, prior model claims and their respective evidential status.

S2 Text. Preprint lineage, supersession and publication overlap

Purpose. This resource records the relationship between the present SMM-K study, the technical SMM preprints and the separately published conceptual article. Preprint revisions are not independent experiments or confirmations. The table identifies supported results, conditional alternative mechanisms and claims retained solely for provenance.

Lineage

- November 2024: *The Brain-Mesh Model: A Unified Framework for Neural Synchrony, Plasticity, and Coherence*, arXiv:2412.12106, with related bioRxiv posting doi:10.1101/2024.11.22.624908. Earlier drafts proposed three layers, phenomenological field waves, phase gradients, distributed plasticity and exploratory EEG/MEG comparisons. Later revisions used the title *The Syncytial Mesh Model*.
- August 2026: Ballus Santacana, *Glial syncytial control fields and the low-frequency resonance of the human brain*, *Frontiers in Human Neuroscience* 20:1896291, doi:10.3389/fnhum.2026.1896291. This is a published conceptual/theoretical precursor. It explicitly anticipated a separate technical SMM treatment. It cannot be reclassified retrospectively as unpublished preprint text.
- 2026 SMM technical V5–V9 research development: a more explicit SMM causal class, a potassium realization, linear stability checks, local QIF reconstruction, spatial sensitivity results, frozen observation audit, and an amended negative human EEG comparison. Earlier source and verification packages were preserved in full rather than silently erased.
- The present definitive article unites that program in a single *scientifically bounded technical presentation*. It does not manufacture a second independent article out of the unfavorable EEG audit or imply that the original preprints independently replicate the new calculation.

What the earlier preprints asserted and what survives

Earlier component	Definitive disposition	Evidential reason
Three layers: local neural population, anatomical neuronal coupling, mesh control	Retained as organizing biological hypothesis	Expressed with separate neuronal and glial operators and intervention criteria
Astrocytic syncytium as possible slow regulatory substrate	Retained with attribution and physiological bounds	Glial regulation and coupling are established biological antecedents, but SMM identity is not experimentally measured here
Mesoscopic slow fields modulating neuronal gain, damping and coherence	Retained as class-level proposal	Developed already in published <i>Frontiers</i> ; new paper operationalizes rather than claiming conceptual priority
Damped traveling-wave PDE with prescribed speed	Reclassified as optional effective phenomenology	Passive K subsystem has real negative poles; no demonstration that a literal calcium wave generates human delta/theta rhythms
Earlier grid simulation’s phase gradients and harmonic matches	Archival exploratory work, not reused as biological validation	Original input and physical-unit calibration do not warrant inferential conclusions in the present audited package
Two-mode coherence fit to 43 subjects and reported median spectral correlation about 0.917	Recorded as historical report only, not a result of the definitive article	It is not an independent preregistered validation and has not been freshly reproduced to modern verification standards
Diffuse mesh-mediated plasticity laws	Retained as a candidate mechanistic extension	No verified plasticity experiment or parameterized causal comparison supplied in the current evidence
Potassium ionic, extracellular and astroglial states	Developed quantitatively as SMM-K	Equations and limited independent numerical checks available

Earlier component	Definitive disposition	Evidential reason
Passive ionic Lyapunov result	Retained as conditional lemma	Mathematically verified, but not a general neuronal–glial stability theorem
Seven-domain neuronal response to glial transport	Promoted to core conditional positive prediction	Reproduced with fixed neuronal/extracellular parameters and explicit focal vs uniform comparison
Frozen whole-brain EEG-M3 likelihood	Retained as restricted local projection	Executable omits $D_g L_g$ despite retaining a slow K filter
Human M3 vs M2 outcome	Retained unfavorable, with exact provenance	Original primary NOT EVALUABLE; separate POST_OPEN_AMENDED 535-subject analysis negative
Full nonlinear R1/R2 FEniCS numerical equivalence	Not claimed	Reference high-fidelity solver not independently executed

Overlap with published *Frontiers* (2026)

The *Frontiers* paper introduced the hypothesis of glial syncytial control fields, the proposed relationship to the low-frequency resonance and a taxonomy of four physiological channels. These items are cited, not claimed as new. The definitive technical article provides a new conditional dynamical model, formal result, seven-patch neuronal intervention, transport-dependent modal transfer, source-code-level identifiability diagnosis, and an independently assembled account of the negative EEG results. Published *Frontiers* prose and figures are not republished as new material.

The preprints can remain publicly visible as dated research history. The recommended route is to update the **same** arXiv paper identifier (and the related bioRxiv record if its platform permits revisions) with this version and to point each prior record to the eventual journal DOI. ‘Supersedes’ means a scientific redefinition of the current manuscript, not erasure of permanent preprint history. The publication submission should disclose both preprint records and the previously published theory article in the cover letter.

Evidence hierarchy and safeguards

All numerical assertions in the definitive main text can be traced to (a) independently rerun analytical/numerical verifications; (b) stored original fixed-code identity or archived evaluation output; or (c) clearly delimited source-level exploratory reconstructions. Evidence of source numerical reproduction is not substituted for a measured biological effect. Earlier reports of empirical agreement are not pooled with the 535-participant EEG analysis. The final article contains a single empirical comparison result: a nonconfirmatory, post-open amended failure of the local M3 comparator against M2. The paper’s forward-looking positive claim is a **conditional prediction**, not a positive human validation.