

Online Resource 5

Alternative dynamical realizations

Article

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

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Author

Andreu Ballús Santacana

Affiliation

Department of Philosophy, Universitat Autònoma de Barcelona, Cerdanyola del Vallès, Spain

Corresponding author

andreu.ballus@uab.cat

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Neuronal, connectomic, damped-field and glia-dependent plasticity formulations and their evidential limits.

S5 Text. Neuronal, field and plasticity realizations of SMM

S5.1 Dynamical members of the SMM class

The SMM causal architecture distinguishes neuronal dynamics and connectivity from a separately parameterized glial spatial-control operator. Candidate members include local neuronal rate and phase reductions, damped effective fields and glia-dependent plasticity laws. These require different dynamical assumptions and cannot be treated as simultaneously calibrated physiological mechanisms. SMM-K is the potassium-mediated realization analyzed quantitatively in the main text. Related conceptual proposals appear in Ballús (2024) and Ballús Santacana (2026).

S5.2 Local neuronal state and anatomical coupling

A local rate-based implementation can use excitatory and inhibitory populations on anatomical nodes i ,

$$\tau_E \dot{E}_i = -E_i + \mathcal{S}_E(w_{EE}E_i - w_{EI}I_i + P_i + \eta_{g,i}(z_i)),$$

$$\tau_I \dot{I}_i = -I_i + \mathcal{S}_I(w_{IE}E_i - w_{II}I_i).$$

An anatomical connectivity operator W_N or weighted matrix C_{ij} may enter the neuronal drive P_i through delayed couplings. Alternative phase reductions introduce connectomic coupling of the form $\sum_j C_{ij} \sin(\theta_j - \theta_i)$ plus an independently specified glial modulation. These are candidate neuronal realizations, not mathematical consequences of glial transport. In the reported SMM-K numerical experiment, the local neuronal block is instead an explicitly transcribed eight-state QIF-derived linearization; the data do not require the phenomenological Wilson–Cowan or Kuramoto alternatives to be fitted simultaneously.

S5.3 A damped-field realization requires additional dynamics

A schematic second-order effective mesh field $u(x, t)$ can be written

$$\partial_t^2 u + 2\gamma \partial_t u + \omega_0^2 u - c_g^2 \Delta_g u = S_g(x, t) + \eta_g(x, t),$$

where Δ_g is the appropriate graph/continuum glial operator, $\gamma > 0$ the damping, S_g an independently specified neuron-to-glia source and ω_0 an optional local restoring parameter. Under ideal homogeneous, stable boundaries, a spatial Laplacian eigenmode with eigenvalue $\lambda_n \geq 0$ has damped roots

$$s_{n,\pm} = -\gamma \pm \sqrt{\gamma^2 - (\omega_0^2 + c_g^2 \lambda_n)}.$$

Only modes satisfying $\omega_0^2 + c_g^2 \lambda_n > \gamma^2$ are underdamped; their angular frequencies obey $\omega_{d,n} = \sqrt{\omega_0^2 + c_g^2 \lambda_n - \gamma^2}$. A mode spectrum alone does not establish corresponding measured neuronal EEG rhythms. In particular, the passive two-compartment potassium realization has real negative ionic poles, so it is incorrect to identify those ionic modes with underdamped traveling wave harmonics. The wave ansatz would need a distinct *active* physical mechanism, coupling law, valid tissue topology and independently calibrated field units.

The early preprint simulation stated $c_g = 0.015 \text{ mm/ms} = 15 \text{ mm/s}$, while parts of its biological discussion invoked much slower calcium-wave speeds of roughly $10\text{--}30 \text{ }\mu\text{m/s}$. These scales differ by factors of approximately 500–1500. This does not prove that no glial signal can propagate quickly through any mechanism, but it prevents the original simulation from being treated as quantitatively calibrated to those calcium-wave measurements. The early 43-person coherence fit and reported median Pearson correlation $r \simeq 0.917$ are retained as historical reports in S2, not reintroduced as independent prospective validation of this physiological realization.

S5.4 Distributed plasticity remains a specific optional extension

An optional glia-mediated plasticity law couples neuronal Hebbian activity to a separately specified glial-state contribution. One candidate member of this model class is

$$\dot{W}_{ij} = \eta_H \mathcal{H}(x_i, x_j) + \eta_g \mathcal{G}(z_i, z_j, x_i, x_j) - \lambda_W W_{ij},$$

with $\eta_H, \eta_g \geq 0$ and a bounded or constrained weight domain. The two components are not interchangeable: $\mathcal{H}$ depends on the local timing/activity variables, whereas $\mathcal{G}$ must depend on specified glial states or on their coupling operator in a way not already determined by the neuronal connectome. Unless $\mathcal{G}$ is defined quantitatively and validated under a selective perturbation, the equation expresses an admissible mechanism, not an established explanation of distributed learning. A plasticity experiment must separate glial transport effects from indirect effects on ordinary neuronal firing.

S5.5 Relation between ionic, synaptic, metabolic, neuromodulatory and wave versions

The four candidate glial channels in the published *Frontiers* precursor are ionic, synaptic/gliotransmitter, metabolic/vascular and neuromodulatory. A consistent abstract SMM model may allow a block state $z = (z_K, z_S, z_M, z_U)$ with graph-dependent glial interactions, but this is an *organizational possibility*, not a numerical claim that all four channels share a universal diffusion parameter or time constant. Different effective channel variables require separately specified driving signals, conservation relations where relevant, and observation functions. The wave-field, plasticity, neuronal rate and ionic formulations share the proposed role of a distinct physiological control geometry, not identical dynamical equations.

S5.6 Evidence boundaries of the alternative models

The following statements describe the model class and its evidence:

1. Neuronal, anatomical-connectomic and glial-control structures are distinct modeling objects.
2. A physiological glial state can influence neuronal gain, threshold, damping or spatial response when explicitly coupled.
3. In one potassium realization, transport changes the modeled neuronal response after focal but not uniform forcing, with a quantified mode sensitivity.
4. An EEG predictor that omits the spatial glial operator cannot test its spatial parameter.
5. The inherited damped-wave and distributed-plasticity ideas are conditional extensions requiring their own physiological calibration and empirical tests.

The five statements distinguish mathematical model properties from conditional biological hypotheses. They do not provide independent experimental validation of the damped-wave or plasticity mechanisms. Preprint computations and their numerical qualifications are documented separately in Online Resource 2.