

Online Resource 1

Analytical proofs and modal transport identifiability

Article

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

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Contents

Formal derivations for the passive ionic block, spatial-mode transfer sensitivity, and a prospective intervention contrast.

S1 Text. Mathematical foundation, modal prediction and falsification boundary

All results in this resource are conditional consequences of the specified linearized SMM-K system, not observations of glial tissue or human participants. The six operational properties of the model class were formulated after the 2026 EEG outcome and were not part of the frozen EEG success criterion.

S1.1 The model class and biological interpretation

The Syncytial Mesh Model is not one linear filter. It is a causal family with neuronal state x , neuronal architecture W_N , glial/extracellular control state z , glial operator L_g and a neuronal observation y . The physiological field interpretation requires measurable tissue-level meaning for z and a neuronal state response that changes with the glial coupling geometry for at least one intervention with W_N held fixed. Ionic, calcium/IP3, metabolic and neuromodulatory channels may all instantiate this hypothesis, but the present executable evidence concerns the ionic pathway alone. The local nearest-neighbor glial graph is **not** proposed to be a new long-range axonal connectome, nor is a continuous brain-wide calcium wave assumed.

The existence of a glial transport operator does not guarantee that it is identifiable in a given EEG experiment. A sensor projection that does not depend on L_g cannot recover L_g regardless of sample size. Conversely, exact spectral similarity does not disprove an unobserved glial process. These are statements about model maps, not positive evidence that glia are involved in a given dataset.

S1.2 Dissipativity of the isolated K subsystem

Under $J_N = 0$, $\phi_e, \phi_a, \kappa_e, \kappa_a, \kappa_N > 0$, $D_e, D_g \geq 0$ and symmetric positive semidefinite L_e, L_g , the two-component ionic equations are

$$\begin{bmatrix} \phi_e I & 0 \\ 0 & \phi_a I \end{bmatrix} \begin{bmatrix} \dot{k} \\ \dot{a} \end{bmatrix} = \begin{bmatrix} -(\kappa_e + \kappa_N)I - D_e L_e & \kappa_a I \\ \kappa_e I & -\kappa_a I - D_g L_g \end{bmatrix} \begin{bmatrix} k \\ a \end{bmatrix}.$$

The energy $V = (\phi_e \kappa_e \|k\|^2 + \phi_a \kappa_a \|a\|^2)/2$ has derivative

$$\dot{V} = -\|\kappa_e k - \kappa_a a\|^2 - \kappa_e \kappa_N \|k\|^2 - \kappa_e D_e k^T L_e k - \kappa_a D_g a^T L_g a.$$

It is strictly negative for nonzero states and induces a global exponential bound on the source-free linear dynamics. The two transport operators need not commute, because each diffusion contribution enters as an independently nonnegative form. The stability claim excludes the feedback-modified neuronal equations and cannot establish global stability of the full nonlinear model. For a shared spatial eigenvector, the ionic 2×2 matrix has a discriminant of $(\alpha_\mu - \beta_\mu)^2 + 4\kappa_e \kappa_a / (\phi_e \phi_a) > 0$, hence real roots; under the sign assumptions both are negative. The passive ionic subsystem is not a delta/theta oscillator.

S1.3 Mode-specific transfer and identifiability

Assume for this derivation that L_e and L_g share a spatial eigenbasis; the seven-patch experiment deliberately uses a single path graph for both operators. Let the spatial eigenvalues be μ_e, μ_g (not necessarily equal in a more general commuting pair). With extracellular neuronal drive J , the Laplace-domain ionic equations are

$$\begin{aligned} (s + \alpha_\mu)K - \frac{\kappa_a}{\phi_e}A &= \frac{J}{\phi_e}, \\ -\frac{\kappa_e}{\phi_a}K + (s + \beta_\mu)A &= 0. \end{aligned}$$

Direct inversion gives the source-to-K response

$$H_K(s; \mu_e, \mu_g, D_g) = \frac{s + \beta_\mu}{\phi_e[(s + \alpha_\mu)(s + \beta_\mu) - \chi]}, \quad \alpha_\mu = \frac{\kappa_e + \kappa_N + D_e \mu_e}{\phi_e}, \quad \beta_\mu = \frac{\kappa_a + D_g \mu_g}{\phi_a}, \quad \chi = \frac{\kappa_a \kappa_e}{\phi_e \phi_a}.$$

For fixed α_μ , let $Q = (s + \alpha_\mu)(s + \beta_\mu) - \chi$. Since $\partial\beta_\mu/\partial D_g = \mu_g/\phi_a$,

$$\frac{\partial H_K}{\partial D_g} = \frac{\mu_g}{\phi_e \phi_a} \frac{Q - (s + \beta_\mu)(s + \alpha_\mu)}{Q^2} = -\frac{\mu_g \chi}{\phi_e \phi_a Q^2}.$$

Thus the uniform glial mode is exactly insensitive to D_g , whereas a nonuniform mode is sensitive for nonzero exchange and nonsingular frequency. This result is *not* valid without qualification for an arbitrary noncommuting pair, nonlinear heterogeneous tissue, or an observation operator that eliminates the corresponding modes. For general $A(D_g)$, $T(s; D_g) = C(sI - A(D_g))^{-1}B$, and

$$\frac{\partial T}{\partial D_g} = C(sI - A)^{-1} \frac{\partial A}{\partial D_g} (sI - A)^{-1} B,$$

assuming input and observation operators do not depend on D_g . The resolvents make explicit when the sensitivity vanishes because of excitation or measurement nullspaces.

S1.4 What is and is not distinguishable from a local comparator

Suppose the benchmark local filter has identical parameters at every patch and no independent glial transport operator. Holding the neuronal W_N , extracellular $D_e L_e$, intervention map and neuronal observation fixed, this restriction cannot generally reproduce transport-induced changes between a common homogeneous mode and nonhomogeneous glial modes with the *same* local filter. This is a conditional model-class discrimination statement. It is not a claim that any arbitrary state-space model is incapable of reproducing the observed data. A flexible competitor with free spatial latent couplings could emulate an SMM-K spatial transfer. Biological identification therefore additionally demands orthogonal physiological measurement or selective intervention.

For the 7-patch D_g sweep, $\mathcal{R}(20\text{ s})$ is 0.116273, 0.159035, 0.195930, 0.256265 and 0.342327 at $D_g/D_g^* = 0, 0.25, 0.5, 1$ and 2 . The $D_g = 0$ to $D_g = D_g^*$ contrast is a factor 2.204, obtained while retaining extracellular D_e and all neuronal parameters. The response has arbitrary input normalization and is not yet assigned an experimental scale. Under exactly uniform forcing the difference is numerical roundoff, as expected from $L_g \mathbf{1} = 0$.

The supplied script `spatial_prediction.py` computes the spatial responses and checks the transfer derivative by finite differences at 0–40 Hz. Its 10–4 relative/error-floor tolerance accommodates numerical cancellation at high frequencies. This verifies the implemented linear reduction, not its physiological parameterization.

S1.5 A finite-noise falsifiability bound

Let P_c be a finite observed-data law indexed by a nonnegative glial-to-neuronal gain c , and P_0 the glia-free comparator. If $P_c \rightarrow P_0$ in total variation as $c \downarrow 0$, a test with type-I error at most α under P_0 cannot have power uniformly bounded well above α for every $c > 0$. Indeed, for any rejection event R , $P_c(R) \leq \alpha + \|P_c - P_0\|_{TV}$, whose right-hand side approaches α . Consequently, an unrestricted model class allowing arbitrarily weak glial coupling is not uniformly falsifiable by any finite noisy experiment. Falsifiable biological predictions require independently justified parameter bounds, predeclared intervention protocols and a specified minimum detectable effect. This is a limit on claims made for the entire class, not a defense of a particular failed parameter set.

S1.6 An explicit prospective contrast

A suitable primary prospective endpoint in a controlled preparation would be the glial-intervention-by-stimulus-pattern interaction in a prechosen neuronal source-space response statistic. Record under both a spatially focal and a broad approximately uniform input, while recording potassium/astroglial state and estimating independent neuronal coupling. The null or comparator should reproduce the observed baseline neuronal dynamics and extracellular diffusion but omit independently variable astroglial spatial transport. Precommit an acceptable parameter region, minimum effect and error-control criterion in a new protocol. A null effect would disfavor that constrained SMM-K realization under the relevant conditions. A positive effect alone would not prove astroglial causality unless alternative spatial control channels have been constrained. None of this prospective test has been conducted in the data reported here.