

Traveling fronts in a generalized neural field model that couples to the extracellular space

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Extracellular ion concentrations directly influence neuronal excitability, synaptic transmission, and the generation of electrical signals. Capturing the ionic dynamics is essential for modeling both healthy and pathophysiological brain function. Here we explore how variations in ion concentration in the extracellular space affect the generation and propagation of traveling fronts of activity in cortical tissue. To this end, we develop a neural field model in which neuronal excitability is modulated by the local extracellular ionic concentration. Using an interface dynamics approach, we derive an analytic expression for the speed of propagating activity fronts. We demonstrate that increasing the ionic diffusion coefficient leads to a reduction in front speed. In addition, analysis of stationary bump solutions reveals that increases in the diffusion rate reduces the size of a region of high activity required to initiate a propagating front.

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I. INTRODUCTION

In the cortex, neuronal activity is controlled by the exchange of ions with the extracellular space. The role of the extracellular space has often been overlooked due to limitations in experimental techniques and a focus on intracellular research [1]. However, it plays a key role in both the supply of ions necessary for the activity of neurons and the modulation of membrane potentials by local agents such as potassium and calcium [2]. It also facilitates signaling between neurons and glia mediated by chemical transmitters such as glutamate and ATP [3].

There is a large separation of time scales between the slow diffusion of ions in the extracellular space and fast long-range action potential communication along axons. The slow rate of diffusion is a consequence of the complex geometry and low volume fraction of the extracellular space [4].

Existing models coupling extracellular ion dynamics to neural activity have often focused on local models of ions in the extracellular space, neglecting diffusion [5–9]. Here we advocate for an approach that couples neural field models with the dynamics from the extracellular space [10,11]. Neural fields are integrodifferential equations well suited for modeling the spatiotemporal dynamics of activity across the cortical surface [12]. For example, a recent neural field model for seizure propagation includes the diffusion of potassium in the extracellular space and couples the local concentrations so as to modify model parameters such as excitability and gap-junction coupling [10]. Extracellular ion dynamics can

also give rise to slowly propagating pathological states such as cortical spreading depression (CSD) [13]. CSD is present in diseases such as migraine and epilepsy, in which waves of highly perturbed ion concentrations spread across the cortex. Numerical studies in models of CSD have shown that the nonlocal interactions present in the brain play an important role in the control of its propagation [14].

Recently, neural field models have been developed to capture the propagation dynamics of CSD in which potassium dynamics are coupled to neural activity [11]. The propagation speed is shown to be modulated by the neural connectivity strength and the local production rate of potassium. While the model effectively captures key physiological mechanisms and fits to experimental data, its complexity—including necessary nonlinearities and coupling across multiple neuronal populations—poses challenges for analytical treatment.

Neural field models are known to support a wide range of spatiotemporal patterns including bumps, periodic waves, and traveling fronts [15]. By approximating the firing rate function of the neural model to be a Heaviside step function, the dynamics of these models reduce to a simplified low-dimensional description of dynamics on the interface between sub- and superthreshold activity. This approach has been applied to the case of models with static [16,17] and stochastic [18] thresholds, and planar models [19]. See Chap. 9 in [20] for a review.

Here we propose a tractable model to couple neural activity to extracellular ion dynamics. The model captures some of the behavior of the more complex models while still allowing for analysis via the interface dynamics approach for a Heaviside firing rate by extending it to include dynamics in the threshold.

II. MODEL

To model the interaction between neural activity and the extracellular space, we consider a generalization of a scalar neural field model in which the local excitability is modulated

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by the availability of ions in the extracellular space. The ion concentration is modeled by a reaction-diffusion equation in which ions are produced as a result of the neural activity and regulated by homeostatic processes such as the reuptake by neurons and glial cells. These ions are free to diffuse through the extracellular space. The full model takes the form of a one-dimensional scalar neural field equation for the activity $u(x, t)$ where $x \in \mathbb{R}, t \geq 0$ coupled to a reaction-diffusion equation for ion concentrations $h(x, t)$ given by

$$\begin{aligned} \frac{\partial u}{\partial t} &= -u + \int_{-\infty}^{\infty} w(|x - y|) f(u(y, t) - h(y, t); \beta) dy, \\ \tau \frac{\partial h}{\partial t} &= -(h - h_0) + \gamma u + D \frac{\partial^2 h}{\partial x^2}. \end{aligned} \quad (1)$$

Here w is a normalized symmetric kernel, τ is the characteristic time scale for the ion dynamics relative to the neural time scale, h_0 is the resting ion concentration, γ is the ion release rate due to neural activity, D is the diffusion coefficient, and β parametrizes the steepness of the sigmoidal firing rate function $f(x; \beta) = (1 + e^{-\beta x})^{-1}$.

The dynamics of h are inspired by that of ions moving between the intracellular and extracellular spaces. The first term captures the homeostatic pressure from the reuptake by neurons and glial cells driving the ion concentration to an equilibrium value h_0 . The second term describes the transfer of ions into the extracellular space due to the metabolic activity of the neurons. When a neuron spikes, ions are rapidly transferred across the cell membrane. While some ions such as sodium are transferred into the cell, we consider only the dynamics of ions which are transported outside of the cell during a spike. In the model, only the resulting excitability is modeled directly. We then assume that the concentration of ions remains in an approximately fixed proportion such that the effect on the neural dynamics can be captured by a single variable. Since spiking causes the release of ions, here the rate of production is modeled as proportional to the local activity u . The final term represents the diffusion of ions across the cortex. In the cortex, the complex geometry of the extracellular space will restrict ion transport to within the regions between cells. This may be thought of as diffusion through a porous medium which, at large spatial scales, reduces back to a simple diffusion equation across the surface with a reduced effective diffusion coefficient D which depends on the geometry.

Changes in the concentration of extracellular ions modifies the local neural excitability by altering the concentration gradient across the cell membrane. This effect is modeled by a shift in the firing rate function f representing a change in the excitability of the neural population. Consider a decrease in h , the firing rate for a given level of activity u increases, and hence the neural activity also increases via Eq. (1).

It is worth mentioning that the use of dynamic thresholds has previously been considered in neural field models as in [21]. Here a nonlinear threshold dynamics (in the absence of diffusion) was shown to lead to interesting interfacial dynamics for the description of traveling waves and breathers. Similar behavior can also be seen in models with fixed thresholds and spike frequency adaptation [22].

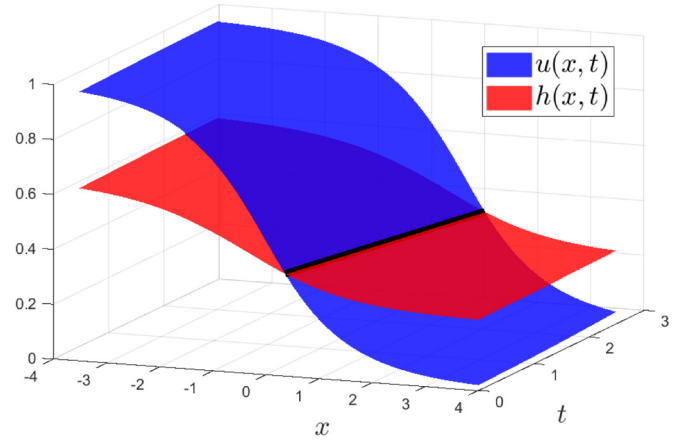


FIG. 1. Direct numerical simulation of a traveling front solution for the model given by Eq. (1) with parameter values $D = 0.4$, $\gamma = 0.4$, $\tau = 1$, $\sigma = 1$, $\beta = 50$, and $h_0 = 1/4$. In this case there is a single crossing point between the threshold h (red) and the neural field u (blue) defining a unique interface $x_0(t)$ (black line).

III. TRAVELING FRONTS

In the cortex, regions of high activity can propagate to regions of low activity creating a traveling front [23]. The propagation speed is important for understanding pathological brain dynamics; for example, in epilepsy seizures may begin as localized regions of high activity which spread across the brain. The propagation of a front involves a combination of nonlocal interactions in which activity is communicated along the long range axonal connections and the local movement of ions diffusing in the extracellular space.

Due to the sigmoidal nonlinearity f in the nonlocal convolution it is not possible to construct analytically traveling front solutions to the model. Here we take the limit as the sigmoidal firing rate f becomes infinitely steep and reduces to a Heaviside step function. In this Heaviside limit we use the interface approach of Amari [24] to reduce the dynamics of front solutions in one spatial dimension to the dynamics of a single interfacial point. Here, we only study the model in a single spatial dimension although the interface approach can be extended to planar dynamics [19].

We assume there is a single point in which the threshold h is crossed at position $x = x_0(t)$ as shown in Fig. 1 (this approach can be extended to multiple crossings). At threshold we have $u(x_0(t), t) = h(x_0(t), t)$ which may be differentiated to yield the speed of the interface as

$$\frac{dx_0}{dt} = - \frac{u_t - h_t}{u_x - h_x} \Big|_{x=x_0(t)}. \quad (2)$$

We now seek solutions for the case of a traveling front moving with a constant speed $x_0(t) = ct$, where we assume $x_0(0) = 0$ without loss of generality.

For a front with $f = 1$ for $x < x_0$ and $f = 0$ for $x > 0$, the neural field may be reduced to the form

$$u(x, t) = \int_{-\infty}^t e^{-(t-s)} \int_{cs}^{\infty} w(|x - y|) dy ds. \quad (3)$$

We now make an explicit choice for the connectivity kernel $w = e^{-|x|/\sigma}/2\sigma$. For this choice of kernel and restricting attention to $c > 0$, the integral in Eq. (3) can be computed analytically to give

$$u(x, t) = \begin{cases} \frac{1}{2} \frac{1}{1+c/\sigma} e^{-\frac{x-ct}{\sigma}} & x > ct, \\ 1 - \frac{1}{2} \frac{1}{1-c/\sigma} e^{\frac{x-ct}{\sigma}} - \frac{c^2}{c^2 - \sigma^2} e^{\frac{x-ct}{c}} & x \leq ct, \end{cases} \quad (4)$$

where the neural activity profile u is given in terms of the unknown front speed c , with implicit dependence on h .

To find the solution for the front in closed form we require an expression for the speed c . Here we compute an algebraic expression for each term in Eq. (2).

Letting $z = u_x$ and then differentiating the neural field equation in Eq. (1) with respect to x yields

$$z_t = -z - w(x - x_0(t)). \quad (5)$$

This equation is straightforwardly integrated (neglecting transients) and restricting to a uniformly moving front $x_0(t) = ct$ yields

$$z(x_0(t), t) = -\frac{1}{c} \int_0^\infty e^{-\zeta/c} w(\zeta) d\zeta, \quad (6)$$

which may be represented simply by $z(x_0(t), t) = -\tilde{w}(1/c)/c$, where $\tilde{w}(\lambda)$ denotes the Laplace transform of w with spectral parameter λ defined by $\tilde{w}(\lambda) = \int_0^\infty w(t) e^{-\lambda t} dt$.

We also require u_t evaluated at the interface which can be found from the model definition given in Eq. (1),

$$u_t|_{x=x_0(t)} = -h(x_0, t) + \tilde{w}(0). \quad (7)$$

For the exponential kernel, the Laplace transform takes the form $\tilde{w}(\lambda) = (\lambda + 1/\sigma)^{-1}/2\sigma$.

To find the remaining terms in Eq. (2), we need an expression for h_t and h_x in terms of the front speed c . To construct these we first obtain an analytic expression for h by solving the diffusion equation in Eq. (1).

Shifting to a traveling wave frame by introducing the wave coordinate $\xi = x - ct$, we seek solutions of the form $h(x, t) = q(\xi)$ which satisfy

$$Qq = \gamma u + h_0, \quad (8)$$

where $Q = 1 - \tau c \frac{d}{d\xi} - D \frac{d^2}{d\xi^2}$. The solution to Eq. (8) is given by

$$q(\xi) = \int_{-\infty}^\infty \eta(\xi - \zeta) [\gamma u(\zeta) + h_0] d\zeta, \quad (9)$$

where η is the Green's function of Q defined by $Q\eta = \delta$, δ denotes the Dirac delta, η is given by

$$\eta(\xi) = \frac{1}{\sqrt{\tau^2 c^2 + 4D}} \begin{cases} e^{-\omega_+ \xi}, & \xi \geq 0, \\ e^{-\omega_- \xi}, & \xi < 0, \end{cases} \quad (10)$$

and

$$\omega_\pm = \frac{\tau c \pm \sqrt{\tau^2 c^2 + 4D}}{2D}. \quad (11)$$

Differentiating $q(\xi)$ yields

$$q_\xi(\xi) = \gamma \int_{-\infty}^\infty \eta_\xi(\xi - \zeta) u(\zeta) d\zeta, \quad (12)$$

which by applying the chain rule gives the spatial and temporal derivatives of h in terms of q_ξ as $h_t = -cq_\xi$ and $h_x = q_\xi$.

Evaluating the integrals at the interface $\xi = 0$ yields

$$q(0) = h_0 + \frac{\gamma}{\sqrt{\tau^2 c^2 + 4D}} \left[\frac{1}{\omega_+} + \frac{1}{2} \frac{1}{1+c/\sigma} \frac{1}{\omega_- - 1/\sigma} - \frac{1}{2} \frac{1}{1-c/\sigma} \frac{1}{\omega_+ + 1/\sigma} - \frac{c^2}{c^2 - \sigma^2} \frac{1}{\omega_+ + 1/c} \right], \quad (13)$$

and

$$q_\xi(0) = \frac{\gamma}{\sqrt{\tau^2 c^2 + 4D}} \left[1 + \frac{1}{2} \frac{1}{1+c/\sigma} \frac{\omega_-}{\omega_- - 1/\sigma} - \frac{1}{2} \frac{1}{1-c/\sigma} \frac{\omega_+}{\omega_+ + 1/\sigma} - \frac{c^2}{c^2 - \sigma^2} \frac{\omega_+}{\omega_+ + 1/c} \right]. \quad (14)$$

Finally, substituting these results into the expression for the front speed Eq. (2) yields an algebraic expression which implicitly defines the front speed c in terms of the model parameters γ, D, σ and h_0 :

$$c = \frac{-q(0) + \tilde{w}(0) + cq_\xi(0)}{\tilde{w}(1/c)/c + q_\xi(0)}. \quad (15)$$

For a receding front, the same process can be followed to derive an equation for $c < 0$ given by

$$c = -\frac{-q(0) + \tilde{w}(0) + cq_\xi(0)}{\tilde{w}(-1/c)/c - q_\xi(0)}. \quad (16)$$

In general, c is only defined implicitly. However, we can recover explicit expressions for the front speed in the limit of small and large diffusion. For $c > 0$ we find

$$\begin{aligned} \lim_{D \rightarrow 0} c &= \frac{\sigma}{4h_0\tau} \left(-2h_0\tau - 2h_0 + \tau + \left[4 \left(-\frac{1}{2} + h_0 \right)^2 \tau^2 - 8 \left(\gamma + h_0 - \frac{1}{2} \right) h_0\tau + 4h_0^2 \right]^{\frac{1}{2}} \right), \\ \lim_{D \rightarrow \infty} c &= \sigma \frac{1 - 2h_0 - \gamma}{2h_0 + \gamma}. \end{aligned} \quad (17)$$

The black curve in Fig. 2(a) shows the front speed as the coupling γ varies, with all other parameters fixed. The front speed decreases monotonically as γ increases. At $\gamma = 0.5$ the front is stationary with further increase leading to a retreating front with $c < 0$.

Similarly, the black curve in Fig. 2(b) shows the front speed as a function of D with all other parameters fixed. Perhaps surprisingly, increasing the diffusion coefficient causes the front speed to decrease monotonically. To understand this we consider the effect of D on the front shape: increased diffusion causes h to spread leading to a front with a shallower profile, resulting in a higher value of h at the interface.

IV. NUMERICAL SIMULATIONS FOR SIGMOIDAL FIRING RATES

The analytical results shown rely on the choice of a Heaviside firing rate to reduce the model to the dynamics on the

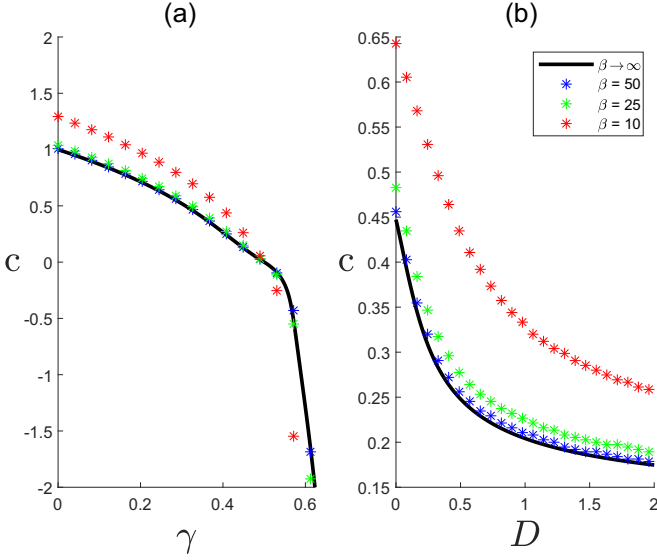


FIG. 2. Variation of the speed of a traveling front solution of Eq. (1) as (a) the coupling γ varies with $D = 0.4$ fixed; (b) the diffusion coefficient D varies with $\gamma = 0.4$. Other parameters are $\tau = 1$, $\sigma = 1$, and $h_0 = 1/4$. The value of β is indicated in the legend. The theoretical prediction for $\beta \rightarrow \infty$ (solid black curve) shows good agreement for sigmoidal steepness $\beta \geq 25$; however, the qualitative shape of the curve is still preserved (up to a shift in front speed) at $\beta = 10$. At the indicated parameter, values the limiting cases in Eq. (17) provide $c = 1/\sqrt{5}$ and $c = 1/9$, respectively, in accordance with Eq. (15), though we truncate D before the large- D limit.

interface. To explore the extent to which the results here extend to a smooth sigmoidal firing rate, we compare the results from the Heaviside theory to numerical simulations with finite steepness β .

We solve the model numerically by approximating the infinite domain by a large periodic domain $[-L, L]$, where $L = 100$. For the spatial components, the PDE for h is discretized by a central difference scheme into $N = 25\,000$ mesh points while the convolution term in the neural activity dynamics is computed using fast Fourier transforms (FFT) and by making use of the convolutional structure of the neural field equation. This results in a set of ODEs which are then integrated using a fifth order Runge-Kutta scheme.

To simulate the propagation of a front in the neural field, initial data is chosen as step functions for the activity $u(x, 0) = \Theta(x)$ and the threshold $h(x, 0) = h_0 + (2\Theta(x) - 1)\Delta h$, where $\Theta(x)$ denotes the Heaviside step function. For all numerical simulations we choose $\Delta h = 0.1$. The initial data and the size of the periodic domain were chosen such that multiple front interactions did not arise. An example simulation result is shown in Fig. 1.

We compare the theoretical predictions for front speed in the Heaviside limit against those obtained for finite β found by direct simulation in Fig. 2(a). For large values of β and across a range of choices for γ we observe excellent agreement between theory and simulations. As β decreases the sigmoid becomes increasingly flat and a larger deviation is seen from the theoretical prediction; however, the general monotonically

decreasing trend is preserved. The point at which the front transitions from progressing to retreating occurs for decreasing values of γ as β decreases. In addition, we compare simulations as D varies in Fig. 2(b); in this case there is again good agreement for large β and the mismatch increases as β decreases.

V. INITIATION OF PROPAGATING SOLUTIONS

In considering the construction of the traveling front solutions above, we assume that we have an initial globally separated state with regions of high and low activity and a single interface between the two. An important consideration is how this configuration can form within the cortex. One mechanism is for a spatially localized state such as a stationary bump to grow. Initially a small region of high activity may grow and leave behind a region of high activity with fronts which propagate into the low activity background. In a scalar neural field with a constant threshold value h , initial bumps will grow as long as the bump half-width is larger than the critical half-width $\ell = \rho^{-1}(h_0)/2$ [17]. Here ρ^{-1} denotes the inverse of the integral of the kernel w ,

$$\rho(x) = \int_0^x w(y)dy. \quad (18)$$

For the choice of the exponential kernel, this gives

$$\ell = -\frac{\sigma}{2} \ln(1 - 2h_0). \quad (19)$$

With $h_0 = 0.25$ fixed, we find that the critical bump width is $2\ell = \sigma \ln(2)$. This size dependence for the onset of traveling solutions has also been seen for the generation and propagation of cortical spreading depression in brain slices [25].

In this section we extend such an analysis to include the diffusive threshold model Eq. (1). We seek a stationary bump solution $(u_b(x), h_b(x))$ in which there are two crossing points $x = \pm\ell$ defined by $u_b(\pm\ell) = h_b(\pm\ell)$. Under the Heaviside choice this defines the active region as $x \in [-\ell, \ell]$. Then we have that $u_b(x)$ is defined in terms of ℓ as

$$u_b(x) = \int_{-\ell}^{\ell} w(x-y)dy = \rho(x+\ell) - \rho(x-\ell). \quad (20)$$

Substituting a stationary bump into the equation for h yields a linear ODE of the same form as Eq. (8) with $Q = 1 - D \frac{d^2}{dx^2}$. The solution can be written as a convolution of u with the Green's function of Q yielding

$$h_b(x) = h_0 + \frac{\gamma}{2\sqrt{D}} \int_{-\infty}^{\infty} \exp\left(-\frac{|x-y|}{\sqrt{D}}\right) u_b(y)dy. \quad (21)$$

The critical half-width is then implicitly defined by $h_b(\ell) = u_b(\ell)$. For the exponential kernel, we obtain

$$\begin{aligned} u_b(\ell) &= \frac{1}{2} \left[1 - \exp\left(-\frac{2\ell}{\sigma}\right) \right], \\ h_b(\ell) &= h_0 + \frac{\gamma}{2} \left[1 - \frac{\sigma^2}{\sigma^2 - D} \exp\left(-\frac{2\ell}{\sigma}\right) \right. \\ &\quad \left. + \frac{D}{\sigma^2 - D} \exp\left(-\frac{2\ell}{\sqrt{D}}\right) \right]. \end{aligned} \quad (22)$$

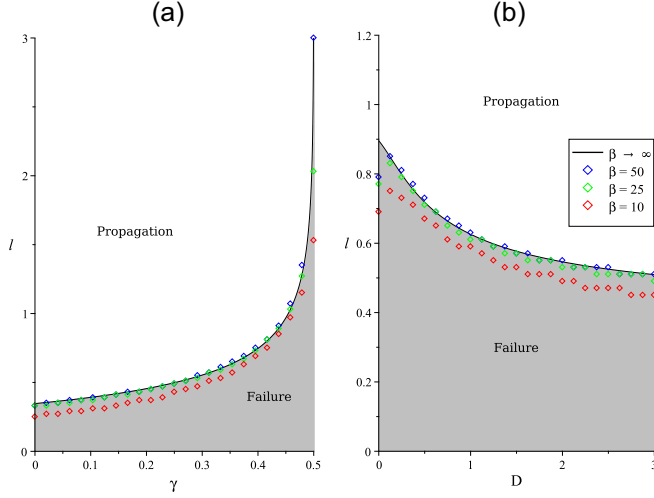


FIG. 3. The critical bump half-width for the propagation of an initial bump, defined by $u_b(\ell) = h_b(\ell)$ from Eq. (22), (a) as the local coupling to the extracellular space γ increases with the diffusion coefficient $D = 0.4$, (b) as the diffusion coefficient D increases with coupling $\gamma = 0.4$ for $\tau = 1$, $\sigma = 1$, and $h_0 = 1/4$. As seen in Fig. 2, theoretical prediction for $\beta \rightarrow \infty$ (solid black curve) shows good qualitative agreement for sigmoidal steepness $\beta \geq 25$ (colored markers).

Again we can examine the two limiting cases of the diffusion coefficient D to yield

$$\lim_{D \rightarrow 0} \ell = -\frac{\sigma}{2} \ln \left(1 - \frac{2h_0}{1 - \gamma} \right),$$

$$\lim_{D \rightarrow \infty} \ell = -\frac{\sigma}{2} \ln(1 - 2h_0). \quad (23)$$

Note that the limit $D \rightarrow \infty$ coincides exactly with the choice of $\gamma = 0$.

Equating the expressions in Eq. (22) yields an implicit equation defining the critical half-width ℓ in terms of the model parameters. Figure 3(a) shows the set of solutions to $h_b(\ell) = u_b(\ell)$ under variation of the coupling coefficient γ . When $\gamma = 0$ the expression matches the known constant threshold case $\ell = \sigma \ln(2)/2$ [17]. As γ increases, the bump width required to initiate a propagating solution increases monotonically up to a critical coupling value, beyond which the model no longer supports stationary bumps in which h and u intersect. Similarly, Fig. 3(b) shows the set of solutions to $h_b(\ell) = u_b(\ell)$ under variation of the diffusion coefficient D . As D increases, the bump width required to initiate a propagating solution decreases monotonically. Figure 3 also shows a comparison with

direct numerical simulations, indicating excellent agreement. We use the same numerical scheme given in Sec. IV. The initial conditions are $h(x, 0) = h_0$, and $u(x, 0)$ is a square bump of width 2ℓ . The threshold of propagation is then found via interval bisection.

The critical bump width determines how large a bump stimulus needs to be to induce a traveling front of activity which spreads across the cortex. A reduced critical bump width increases the likelihood of random dynamics in the brain leading to the onset of a spreading region of high activity (for example a seizure).

VI. SUMMARY

We have introduced a simple model to capture the interaction between neural activity and ions in the extracellular space. By considering a Heaviside firing rate function, we have derived an algebraic equation implicitly defining the speed of traveling fronts. We highlight the strong modulation of wave speeds caused by coupling to the extracellular space. In particular, there is a monotonic decrease in front speed with increasing extracellular ionic diffusion. Direct simulations confirm these trends for smoother sigmoidal firing rate functions.

In addition, analysis of stationary bump solutions in the model shows how the thresholds for front propagation are modified by the dynamics of the extracellular space. The feedback with the extracellular space increases the required bump size in order for propagation to occur. Furthermore, for fixed feedback strength, increasing the diffusion coefficient results in a decrease in the threshold for propagation.

The results presented here emphasize the importance of incorporating the dynamics of the extracellular space when considering traveling waves and bumps in the cortex. We suggest that the phenomenological model presented here is well suited to guiding future, more detailed biophysical studies that aim to incorporate extracellular ion dynamics within models of cortical activity.

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DATA AVAILABILITY

There are no publicly available research data or software supporting this manuscript. Requests for further information or data should be sent to the authors.

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