# Dendritic morphology affects the neuronal excitability type and the network state

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**Abstract** Neurons can produce a number of qualitatively different spiking dynamics, determined by external conditions like temperature and internal properties such as ion channel composition. The dynamical excitability type can impact the activity of the network, particularly its (de)synchronisation state. Neurons with an onset of spiking via a saddle homoclinic orbit (HOM) bifurcation tend to synchronise inhibitory networks while they organise the spiking activity of excitatory networks into a splay state, where firing phases differ across neurons such that they are regularly distributed in time. Based on mathematical neuron models with dendrites, we here demonstrate that the extent of dendritic arborisation can switch the excitability type to a HOM spiking onset and hence can induce (de)synchronised network states. Our analysis is applicable to a general class of spike-generating neuron models (from ball-and-stick models to anatomically reconstructed neuron models) and highlights the importance of neuronal morphology for the susceptibility of neural tissue to synchronisation in health and disease.

## Introduction

Due to the variety of biophysical quantities which can affect spike generation, neurons can produce a number of qualitatively different spiking dynamics. The spiking dynamics is not merely limited to the spike shape as captured by the voltage trace but rather encompasses the whole set of time-varying quantities responsible for spike generation — including ion channel (in)activation. For regularly spiking neurons, the dynamical spiking type at the rheobase current (in terms of the underlying spike onset bifurcation) is referred to as the excitability type. It is related to the excitability classes as observed by Hodgkin (***Hodgkin, 1948***), as bifurcations differ in their firing frequency at spike onset (arbitrarily low versus distinctly non-zero).

Given the considerable heterogeneity between dendritic arbours — even in neurons of the same class and layer (***Larkman and Mason, 1990***; ***Mohan et al., 2015***; ***Van Aerde and Feldmeyer, 2015***) — the extent to which dendritic arborisation affects the excitability type, and hence the resulting network response, is a functionally relevant consideration. It is known that even a passive dendritic arbour can play an important role in processing inputs (***Eyal et al., 2014***; ***Tiroshi and Goldberg, 2019***; ***Beniaguev et al., 2022***). Furthermore, background synaptic activity targeting the dendritic arbour affects integrative properties of the neuron (***Bindman et al., 1988***; ***Holmes and Woody, 1989***; ***Häusser and Clark, 1997***; ***Paré et al., 1998***; ***Destexhe and Paré, 1999***; ***Rudolph and Destexhe, 2003***). In this study we therefore explore how the dynamic excitability type can be switched by changing the extent and properties of the dendritic arbour.

According to the different excitability types, regular spiking can be classified into at least three qualitatively different spike onset bifurcations. The main two of interest here are the saddle-node on an invariant cycle (SNIC) and saddle homoclinic orbit (HOM) bifurcations. This is firstly because switching from SNIC to HOM onset yields strong changes in spike timing properties. Furthermore, since these two excitability types neighbour each other in parameter space, a small change in neuronal parameters can induce large changes in the network state. For example, neurons with SNIC onset have almost no tendency to synchronise weakly-coupled networks (***Izhikevich, 2007b***). In contrast, we recently showed that neurons with HOM onset tend to synchronise networks of inhibitory neurons, and arrange the spiking events of excitatory networks into a splay state (***Hesse et al., 2017***, ***2022***; ***Hürkey et al., 2022***), where neurons fire regularly with distinct phases that are temporally distributed across neurons.

Experimental studies have found that a neuron's $f$–$I$ curve can be switched from class I (zero onset frequency) to class II (non-zero onset frequency) via cholinergic modulation (***Stiefel et al., 2008***), in vivo-like stimulation (***Prescott et al., 2008b***), and GABAergic stimulation (***Kirst et al., 2017***). Similarly, a neuron's excitability type can be switched via a change in external conditions or intrinsic properties. Thus far, examples of the SNIC to HOM switch are primarily found in modelling studies (though see ***Hesse et al.*** (***2022***)). Previous research has shown this switch can be induced via changes to the membrane capacitance (***Hesse et al., 2017***), extracellular potassium concentration (***Contreras et al., 2021***), increasing the leak conductance (***Kirst et al., 2017***), increasing the M-current conductance (***Al-Darabsah and Campbell, 2021***), and increasing the temperature (***Hesse et al., 2022***).

Research on excitability type switching typically uses conductance-based neuron models inspired by the Hodgkin-Huxley model (***Hodgkin and Huxley, 1952***). However, these models usually consider an isopotential neuron and neglect the extensive dendritic arborisation of neurons. Previous dendritic modelling studies of conductance-based models include: the effect of dendritic load on the firing frequency (***Schwemmer and Lewis, 2011***) and burst firing (***Mainen and Sejnowski, 1996***), the effect of dendritic perturbations on spike timing (***Crook et al., 1998***; ***Goldberg et al., 2007***), and the effect of dendritic coupling between spike-generating zones in the same neuron (***Remme et al., 2009***). However, an analysis of how dendrites alter the excitability type and ultimately the (de)synchronisation state of the network has not yet been conducted.

In this article we methodically demonstrate that the input impedance provided by a dendritic arbour can be tuned to promote (de)synchronisation of network activity. Using a neuron model of an active conductance-based soma attached to a passive dendrite, and with the neuronal input conductance and external input current as bifurcation parameters, we first show how to calculate the local bifurcation structure when neuronal morphology is included. Of particular interest is the Bogdanov-Takens (BT) bifurcation, which acts as an organising centre of different spiking onset types (***Pereira et al., 2015***; ***Kirst et al., 2017***). In the second step, we show that the change in onset bifurcation structure from dendritic load is reflected in the spike timing response near the onset bifurcation. Thirdly, we verify our results in a detailed, anatomical model, demonstrating that our results from the simplified dendrite-and-soma model are widely applicable. Finally, we demonstrate the effect of dendritic arborisation on the network synchronisation state by simulating a group of neurons at input conductances on either side of the SNIC to HOM excitability switch,

underlining the relevance of cellular morphology for the dynamical state of neuronal networks.

## Results

### Passive Impedance Properties

Given that changing the speed at which the neuron's membrane potential changes can induce all the dynamical excitability switches for regular spiking (***Kirst et al., 2017***; ***Hesse et al., 2017***), we start our investigation of how neuronal morphology affects (de)synchronisation state by analysing how dendrites filter changes in the neuron's membrane potential. To differentiate the voltage filtering effects caused by a dendritic arbour versus those captured by an isopotential point neuron, we first calculate the passive impedance properties of the single-compartment (S) and dendrite-and-soma models (DS). While the passive input impedance of an S model neuron is a first-order low-pass filter, spatial neuron models yield qualitatively different impedance profiles that depend on the dendritic properties (***Rall, 1960***; ***Tuckwell, 1988***).

As we show in ***Figure 1***, the DS model has an active soma attached to a passive dendrite which represents the equivalent cable of a branched dendritic arbour (***Rall and Rinzel, 1973***; ***Rall, 2011***). If the active conductances in the soma have the same valued parameters and equations as the S model, then the two models differ only in their passive properties. The passive properties of the dendrite can be parametrised in terms of its length $L$, electrotonic length constant $\lambda$ passive time constant $\tau_\delta$, and dendritic dominance factor $\rho$ (***Rall, 2011***) (the ratio between the characteristic dendritic conductance and the somatic leak conductance).

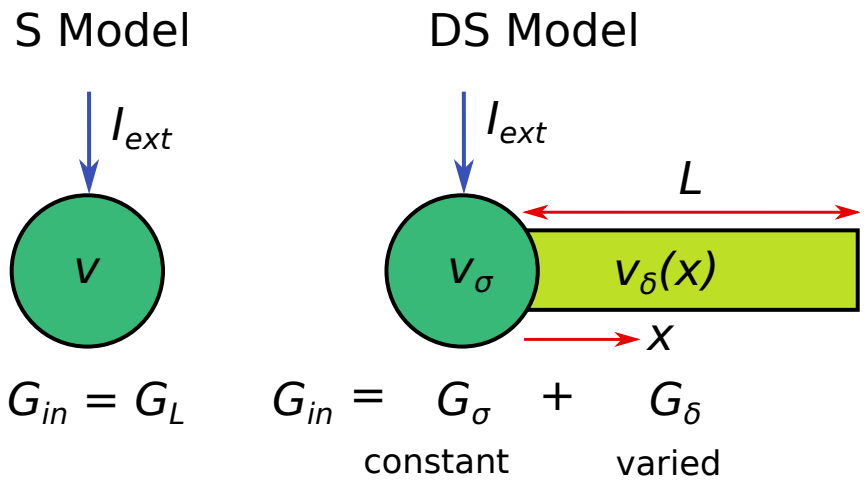


**Figure 1.** A comparison of the single-compartment (S) model with a dendrite-and-soma (DS) model with equivalent passive input conductance $G_{\text{in}}$. In the DS model, the constant input current $I_{\text{ext}}$ is applied to the somatic compartment. In the S model, $G_{\text{in}}$ is equivalent to the lumped leak conductance $G_L$, while for the DS model, $G_{\text{in}}$ is the sum of the passive somatic ($G_\sigma$) and dendritic ($G_\delta$) contributions. When we increase $G_{\text{in}}$ in the DS model, $G_\sigma$ is kept constant while $G_\delta$ is increased.

In this study, we vary the neuron's arborisation as captured by the dendritic contribution to the passive input conductance ($G_\delta$). A neuron with more dendrites radiating from the soma will have a thicker equivalent cable and thus contribute more to the total passive input conductance $G_{\text{in}}$. The passive input conductance is given by the constant level of external input current $I_{\text{ext}}$ required to change the voltage from its steady state value (when all active conductances are zero) by an amount $\Delta v$

$$I_{\text{ext}} = G_{\text{in}} \Delta v. \tag{1}$$

In the DS model, we apply $I_{\text{ext}}$ to the soma and use the voltage change $\Delta v$ at the soma for $G_{\text{in}}$. In each case $G_{\text{in}}$ is the sum of its somatic ($G_\sigma$) and dendritic ($G_\delta$) contributions. For a dendrite of effective length $\ell = L/\lambda$

$$G_{\text{in}} = G_\sigma + G_\delta = G_\sigma(1 + \rho \tanh \ell). \tag{2}$$

When $\ell \gg 1$, we can treat the dendrite as semi-infinite and the passive input conductance of the DS model becomes

$$G_{\text{in}} = G_\sigma + G_\delta = G_\sigma(1 + \rho). \tag{3}$$

For the S model $G_{\text{in}} = G_L$, the total leak conductance, allowing for a straightforward comparison between the two morphologies. While it is possible to adjust the dendritic parameters $(\rho, \lambda, L)$ to

match $G_{\text{in}}$ for the S and DS models, this is not possible for the passive input admittance $Y_{\text{in}}$ (the reciprocal of the input impedance, $Y_{\text{in}} \equiv Z_{\text{in}}^{-1}$). Denoting the angular frequency of the input signal as $\omega$, for the S model $Y_{\text{in}}$ is simply given as a first-order filter

$$Y_{\text{in}}^{\text{S}}(\omega) = G_L + i\omega C_{\text{m}}, \tag{4}$$

while for the semi-infinite and finite DS models we have

$$Y_{\text{in}}^{\text{F}}(\omega) = G_\sigma + i\omega C_\sigma + \rho G_\sigma \gamma(\omega) \tanh(\ell \gamma(\omega)), \tag{5}$$

$$Y_{\text{in}}^{\infty}(\omega) = G_\sigma + i\omega C_\sigma + \rho G_\sigma \gamma(\omega), \tag{6}$$

where $\gamma(\omega)$ is defined as

$$\gamma(\omega) = \sqrt{1 + i\omega\tau_\delta}. \tag{7}$$

This mismatch in the passive input admittance encapsulates the difference between the S and DS models, and we will explore the implications that it has on the neuronal excitability type. For the semi-infinite DS model, $Y_{\text{in}}$ can be fully parametrised by $G_{\text{in}}$ and $\tau_\delta$. From the form of $Y_{\text{in}}$, we see that $\tau_\delta$ is a key parameter for comparing the DS and S models. When $\tau_\delta = 0$, $\gamma = 1$ at all frequencies and hence $Y_{\text{in}}^F$ and $Y_{\text{in}}^\infty$ become equal to $Y_{\text{in}}^{\text{S}}$ in this limit for $G_{\text{in}} = G_L$. Thus the voltage dynamics of $v_\sigma$ in the DS model become identical to the dynamics of $v$ in the S model when $\tau_\delta = 0$.

We illustrate the differences between the passive input admittances of the S and DS models via its more commonly measured reciprocal, the input impedance $Z_{\text{in}}$. Figure ***Figure 2***A shows that for any given $G_{\text{in}}$, $|Z_{\text{in}}|$ is higher in the S model than the semi-infinite DS model at all non-zero frequencies. In Figure ***Figure 2***B, we see that $|Z_{\text{in}}|$ decreases at all non-zero frequencies when we increase $\tau_\delta$ in the semi-infinite DS model. In the finite DS model, Figure ***Figure 2***C shows that if we hold $G_{\text{in}}$ constant while decreasing $\ell$, then $|Z_{\text{in}}|$ decreases at all non-zero frequencies.

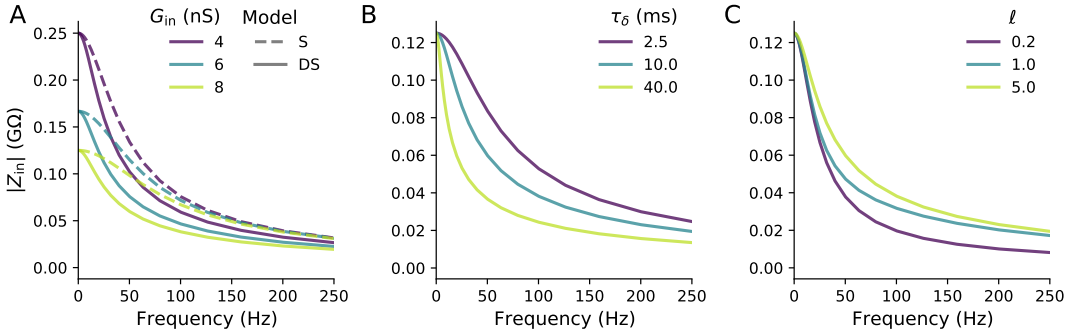


**Figure 2.** (**A**) When one fixes the input conductance $G_{in}$ between the S model and a semi-infinite DS model, the magnitude of the input impedance $Z_{in}$ will be higher for the S model for non-zero frequencies. $\tau_\delta = 10$ ms. (**B**) Increasing $\tau_\delta$ of the DS model decreases $|Z_{in}|$ at all non-zero frequencies. $G_{in} = 8$ nS. (**C**) Decreasing the effective dendritic length $\ell$ while maintaining the same $G_{in}$ also decreases $|Z_{in}|$ at all non-zero frequencies. $\tau_\delta = 10$ ms.

In our analyses of the DS model, we will vary directly $G_{in}$ and $\tau_\delta$ independently of each other rather than the underlying electrophysiological parameters they are defined in terms of (Equation 12). However, these parameter changes have various important biophysical interpretations. Firstly, increasing the dendritic diameter $d$ increases $G_{in}$ without affecting $\tau_\delta$. Additionally, $\tau_\delta$ can be increased without altering $G_{in}$ by increasing the dendritic membrane capacitance per unit area $c_\delta$. Variations in the effective dendrite diameter between neurons can be significant and neuronal membrane capacitance per area may vary between mammalian species, though there are conflicting findings on this (***Eyal et al., 2016***; ***Beaulieu-Laroche et al., 2018***).

While the two biophysical changes discussed so far are typically set once the neuron has developed and will not vary over short time scales, it is important to recognise that $Y_{in}$ can be changed dynamically. One example of this is that increased synaptic bombardment distributed across the dendrite will increase the per-area leak conductance of the dendrite $g_\delta$ . This in turn will increase both $G_{in}$ and decrease $\tau_\delta$ (***Bindman et al., 1988***; ***Paré et al., 1998***; ***Destexhe and Paré, 1999***). Increasing $d$ or decreasing $g_\delta$ also increases the length constant $\lambda$ (Equation 12). For short dendrites, this is an important consideration, however for long dendrites with inputs applied to the soma, $\lambda$ does not appear in $Y_{in}$ or in any of the calculations of the local bifurcations as we show in the Methods section.

## Bifurcation Structure

We now make the DS model active by giving the soma the dynamics of the Morris-Lecar (ML) model (***Morris and Lecar, 1981***) while keeping the dendrite passive. The ML model was chosen because it has a single time-dependent gating variable, making it one of the simpler conductance-based neuron models, and also because it has been extensively studied for its ability to change excitability type upon parameter variation (***Ermentrout, 1996***; ***Tsumoto et al., 2006***; ***Prescott et al., 2008a***; ***Liu et al., 2014***; ***Kirst et al., 2017***). We chose the default class I set of parameters of the ML model with $G_\sigma = 2$ nS, where details of the model's dynamics and parameters are given in the Appendix.

Given that the DS model differs from the S model in terms of its passive input impedance, and that the input impedance can be fully described in terms of $(G_{\text{in}}, \tau_\delta)$ for the semi-infinite dendrite, it naturally follows that we should choose $(G_{\text{in}}, \tau_\delta)$ as bifurcation parameters along with $I_{\text{ext}}$ in assessing the effect of morphology on the excitability type. Since the passive leak conductance in the soma $G_\sigma = 2$ nS, values of $G_{\text{in}}$ above 2 nS denote the conductance load added to the neuron by the dendrite.

In ***Figure 3***, we show several two-dimensional bifurcation diagrams in terms of $(I_{\text{ext}}, G_{\text{in}})$ plotted for various values of the third bifurcation parameter $\tau_\delta$. As discussed earlier in for the analysis of the input impedance, the case of $\tau_\delta = 0$ is equivalent to the S model where $G_{\text{in}} = G_L$. There are two saddle-node (SN) bifurcations at lower and higher $I_{\text{ext}}$, which we will hereafter refer to as "lower" and "higher" respectively. These two SN bifurcations converge at the cusp as $G_{\text{in}}$ increases. Three fixed points exist for $I_{\text{ext}}^{\text{SN,low}} < I_{\text{ext}} < I_{\text{ext}}^{\text{SN,high}}$, one stable and two unstable. When spiking onset is caused by a SNIC bifurcation, this occurs on the higher SN bifurcation. These bifurcations are shown in black because they do not vary with $\tau_\delta$, and hence occur at exactly the same values of $(I_{\text{ext}}, G_{\text{in}})$ in the S and DS models.

For $\tau_\delta = 0$, the BT point is located on the higher SN bifurcation and has a subcritical Hopf bifurcation emerging from it. This Hopf bifurcation permits class II $f$–$I$ curves. Thus the BT point has often been used heuristically as separating class I and class II $f$–$I$ curve relationships. Increasing $\tau_\delta$ from zero initially shifts the BT bifurcation to higher $G_{\text{in}}$ until it reaches the cusp at $\tau_\delta^{\text{BTC}} = 12.9$ ms (hereafter denoted as the BTC point). Increasing $\tau_\delta$ beyond $\tau_\delta^{\text{BTC}}$ moves the BT point onto the lower SN bifurcation curve and $G_{\text{in}}^{\text{BT}}$ now decreases as $\tau_\delta$ increases. As the BT point passes the cusp as $\tau_\delta$ increases, the criticality of the emerging Hopf bifurcation switches from subcritical to supercritical

at the BTC point.

When the Hopf bifurcation that emerges from the BT point is subcritical, it can be switched to supercritical by increasing $G_{\text{in}}$. This criticality switch moves to lower $G_{\text{in}}$ as $\tau_\delta$ increases towards $\tau_\delta^{\text{BTC}}$. At all $\tau_\delta$, there is a fold of Hopf bifurcations when $G_{\text{in}}$ becomes sufficiently higher, and spiking is no longer possible at any applied current. The value of $G_{\text{in}}$ for the fold of Hopf bifurcations decreases as $\tau_\delta$ increases. The changes to the BT and Hopf bifurcations with $\tau_\delta$ mean that we would expect the transition between class I and class II $f$–$I$ curves to occur at higher $G_{\text{in}}$ in the DS model for $\tau_\delta < \tau_\delta^{\text{BTC}}$.

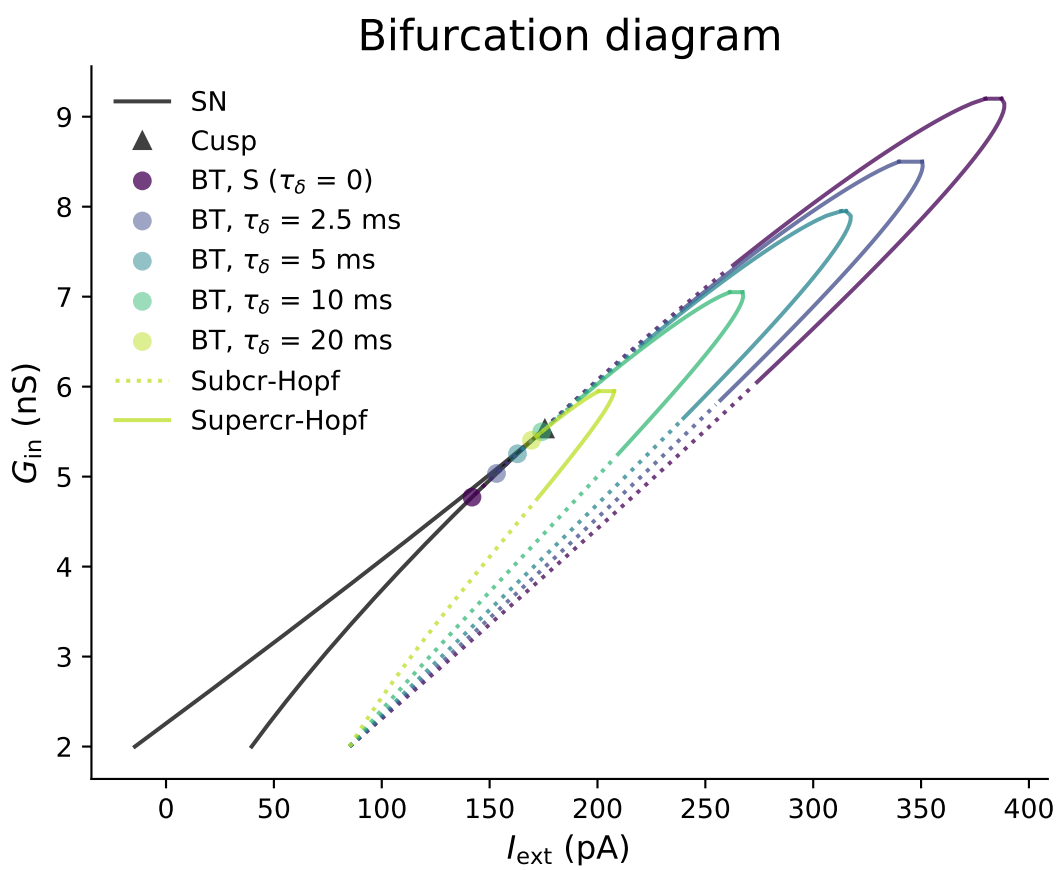


**Figure 3.** Two-parameter local bifurcation diagram in terms of $(I_{\text{ext}}, G_{\text{in}})$ of the ML DS model for various dendritic time constants $\tau_\delta$. At $\tau_\delta = 0$, the DS model is equivalent to an S model with a leak conductance of $G_{\text{in}}$. Increasing $\tau_\delta$ shifts the BT bifurcation and shrinks the Hopf bifurcation curve. Initially increasing $\tau_\delta$ moves the BT point to higher $G_{\text{in}}$ until it reaches the cusp at the BTC point. Increasing $\tau_\delta$ beyond $\tau_\delta^{\text{BTC}}$ moves the BT point to the lower SN bifurcation curve and thus decreases $G_{\text{in}}^{\text{BT}}$. The Hopf bifurcation emerging from the BT point also switches from subcritical to supercritical when $\tau_\delta$ passes the BTC point. The saddle-node and cusp bifurcations are shown in black because they are unaffected by changes to $\tau_\delta$.

To identify the input conductance for the SNIC to HOM switch, we must look at the saddle-node-loop (SNL) bifurcation (***Schecter, 1987***; ***Guckenheimer and Labouriau, 1993***; ***Izhikevich, 2007a***; ***Hesse et al., 2017***). In ***Figure 4***, we show the bifurcation types responsible for excitability switching as a function $(\tau_\delta, G_{\text{in}})$. For all $\tau_\delta$, $G_{\text{in}}^{\text{SNL}} < G_{\text{in}}^{\text{BT}}$, with SNIC onset for $G_{\text{in}} < G_{\text{in}}^{\text{SNL}}$. The limit cycle formed by the HOM bifurcation in this case contains all three fixed points of the system (big-HOM). Since the SNL bifurcation converges to the BTC point, it therefore makes sense that $G_{\text{in}}^{\text{SNL}}$ also increases with $\tau_\delta$ when $\tau_\delta < \tau_\delta^{\text{BTC}}$. Indeed, this is what we see in ***Figure 4***, with not only $G_{\text{in}}^{\text{SNL}}$ increasing with $\tau_\delta$ but also the conductance difference between the BT and SNL points decreasing before eventually converging at $\tau_\delta^{\text{BTC}}$. Thus both the SNIC → HOM switch and the $G_{\text{in}}$ range for homoclinic onset are affected by the dendritic time constant. For $\tau_\delta > \tau_\delta^{\text{BTC}}$, we do not find an SNL bifurcation until $\tau_\delta$ becomes large (not shown).

Strictly speaking there can exist another global bifurcation in the region $G_{\text{in}}^{\text{SNL}} < G_{\text{in}} < G_{\text{in}}^{\text{BT}}$ for which a fold of limit cycles (FLC) appears with a bifurcation current $I_{\text{ext}}^{\text{FLC}} < I_{\text{ext}}^{\text{HOM}}$, thus making the FLC the onset bifurcation (***De Maesschalck and Wechselberger, 2015***; ***Kirst et al., 2017***). While this bifurcation can determine the switch between class I and class II $f$–$I$ curves, we neglect to show it for the following reasons: (1) the spike timing perturbation response for FLC onset in this region is extremely similar to HOM onset; (2) though HOM onset in theory has class I $f$–$I$ curve, it is extremely steep at $I_{\text{ext}}^{\text{HOM}}$ making it hard to distinguish from a class II $f$–$I$ curve; and (3) the difference between $I_{\text{ext}}^{\text{FLC}}$ and $I_{\text{ext}}^{\text{HOM}}$ is typically extremely small (***Kirst, 2011***) and thus $I_{\text{ext}}^{\text{HOM}}$ gives an accurate approximation of the onset current in this region. Thus due to simplicity and the high degree of functional similarity, we refer to the onset type for the whole of $G_{\text{in}}^{\text{SNL}} < G_{\text{in}} < G_{\text{in}}^{\text{BT}}$ as being HOM.

We see that some bifurcations are affected by $\tau_\delta$ while others are not. Both the SN and cusp bifurcations are calculated from the existence of fixed points alone, and so the timescale of the dynamics (such as from $\tau_\delta$) will not affect them. On the other hand, the Hopf and BT bifurcations involve the stability switch of fixed points, for which knowledge of the timescale of the dynamics is necessary. For the SNL bifurcation, one can extend the reasoning in ***Hesse et al.*** (***2017***) to show that changing the timescale of the dynamics will break any existing homoclinic orbits at a given $G_{\text{in}}$. As a result, one would expect that changing $\tau_\delta$ changes the value of $G_{\text{in}}^{\text{SNL}}$.

Our bifurcation analysis indicates that switching between excitability types can be induced by increasing the dendritic arborisation as parametrised by $G_{\text{in}}$. The switching between excitability

types is qualitatively similar to increasing $G_L$ in the single-compartment model, however the $G_{\text{in}}$ values for the SNIC → HOM switch and subcritical Hopf onset increase with $\tau_\delta$, whilst the value of $G_{\text{in}}$ for supercritical Hopf onset decreases with $\tau_\delta$.

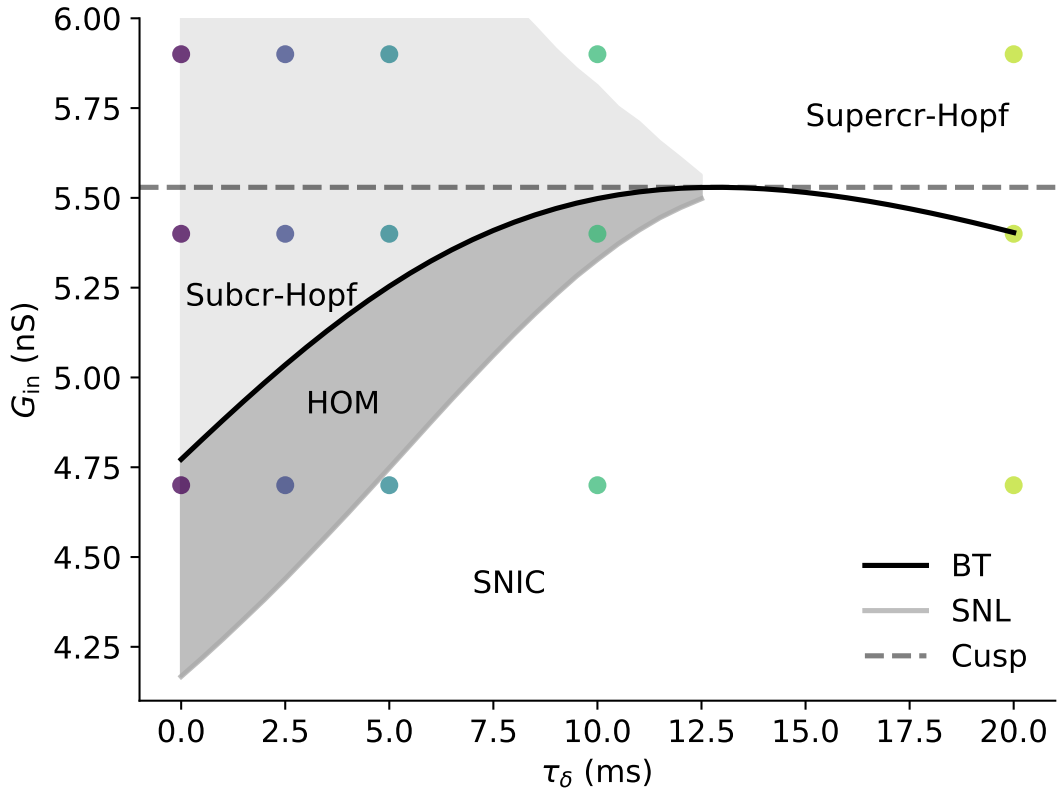


**Figure 4.** Bifurcation diagram of the ML DS model in terms of $(\tau_\delta, G_{\text{in}})$ focussing on excitability switches. Here we have taken $I_{\text{ext}}$ as the onset current for every value of $(\tau_\delta, G_{\text{in}})$. Points indicate values of $(\tau_\delta, G_{\text{in}})$ we later use for the spike timing response. At the saddle-node loop (SNL) bifurcation, the excitability type switches from SNIC to HOM, while Hopf onset becomes possible at the BT bifurcation. Increasing $\tau_\delta$ above zero increases both $G_{\text{in}}^{\text{SNL}}$ and $G_{\text{in}}^{\text{BT}}$ until the BT and SNL bifurcations meet the cusp at the BTC point at $\tau_\delta^{\text{BTC}}$. The difference between $G_{\text{in}}^{\text{BT}}$ and $G_{\text{in}}^{\text{SNL}}$ decreases as $\tau_\delta$ increases, meaning that the range of $G_{\text{in}}$ for which HOM excitability exists becomes smaller. For $\tau_\delta > \tau_\delta^{\text{BTC}}$, the SNL bifurcation no longer exists and $G_{\text{in}}^{\text{BT}}$ decreases. Furthermore, the Hopf onset for $G_{\text{in}} > G_{\text{in}}^{\text{BT}}$ switches from subcritical to supercritical. $G_{\text{in}}$ for subcritical Hopf onset increases with $\tau_\delta$ until $\tau_\delta^{\text{BTC}}$, while $G_{\text{in}}$ for supercritical Hopf onset decreases with $\tau_\delta$ throughout the whole range.

## Phase-Response Curves

The dynamics of a neuronal spike affects how the neuron responds to external perturbations, which can come from chemical synapses, gap junction coupling, local field potentials, or externally applied currents. This has been found in both experimental (***Phoka et al., 2010***; ***Wang et al., 2013***)

and in modelling studies (***Hansel et al., 1995***; ***Ermentrout, 1996***). The change in spike time caused by a perturbation to a tonically spiking neuron is described by the phase-response curve (PRC). In many cases, such as when neurons are weakly coupled or subject to weak correlated inputs, one can use an individual neuron's PRC to infer synchronisation conditions and the overall network state (***Ermentrout, 1996***; ***Teramae and Tanaka, 2004***; ***Ko and Ermentrout, 2009***).

To see how the different excitability types affect the neuron's spiking response, we calculated the phase-response curves (PRCs) for the S and DS models for a range of $(G_{\text{in}}, \tau_\delta)$. We chose the $(G_{\text{in}}, \tau_\delta)$ values to be in the neighbourhood of the SNL, BT, and cusp bifurcations, as shown by the coloured points in ***Figure 4***.

At $G_{\text{in}} = 3$ nS, the ML neuron has SNIC excitability type for all $\tau_\delta$. Hence ***Figure 5***A shows symmetric positive-valued at this input conductance. At $G_{\text{in}} = 4.7$ nS (***Figure 5***B), the neuron is operating in the HOM regime for lower $\tau_\delta$ and thus we see asymmetric positively-skewed PRCs associated with HOM excitability (***Brown et al., 2004***; ***Hesse et al., 2017***; ***Schleimer and Schreiber, 2018***), while for higher $\tau_\delta$ the SNL bifurcation has not yet been reached and we still have symmetric SNIC PRCs. Further increasing $G_{\text{in}}$ (***Figure 5***C) causes the neuron with $\tau_\delta = 10$ ms to pass its SNL point, inducing an asymmetric HOM PRC, while the PRC for $\tau_\delta = 20$ ms has negative regions after passing its BT bifurcation and adopting supercritical Hopf onset. Finally when $G_{\text{in}}$ is above the cusp value (***Figure 5***D), all the neurons have Hopf onset with negative regions in their PRCs. However, the neuron with $\tau_\delta > 20$ ms has a far more sinusoidal PRC with a greater negative region due to its onset being via a supercritical rather than subcritical Hopf bifurcation.

Thus the procession of PRCs of the DS model as $G_{\text{in}}$ is similar to the S model when $\tau_\delta < \tau_\delta^{\text{BTC}}$. For $\tau_\delta > \tau_\delta^{\text{BTC}}$, the asymmetric positive-valued PRC associated with HOM onset is not found, as predicted from the disappearance of the SNL bifurcation in the previous section. The $G_{\text{in}}$ bifurcation values found earlier are thus useful predictors of the neuron's spiking response in the dendrite-and-soma model.

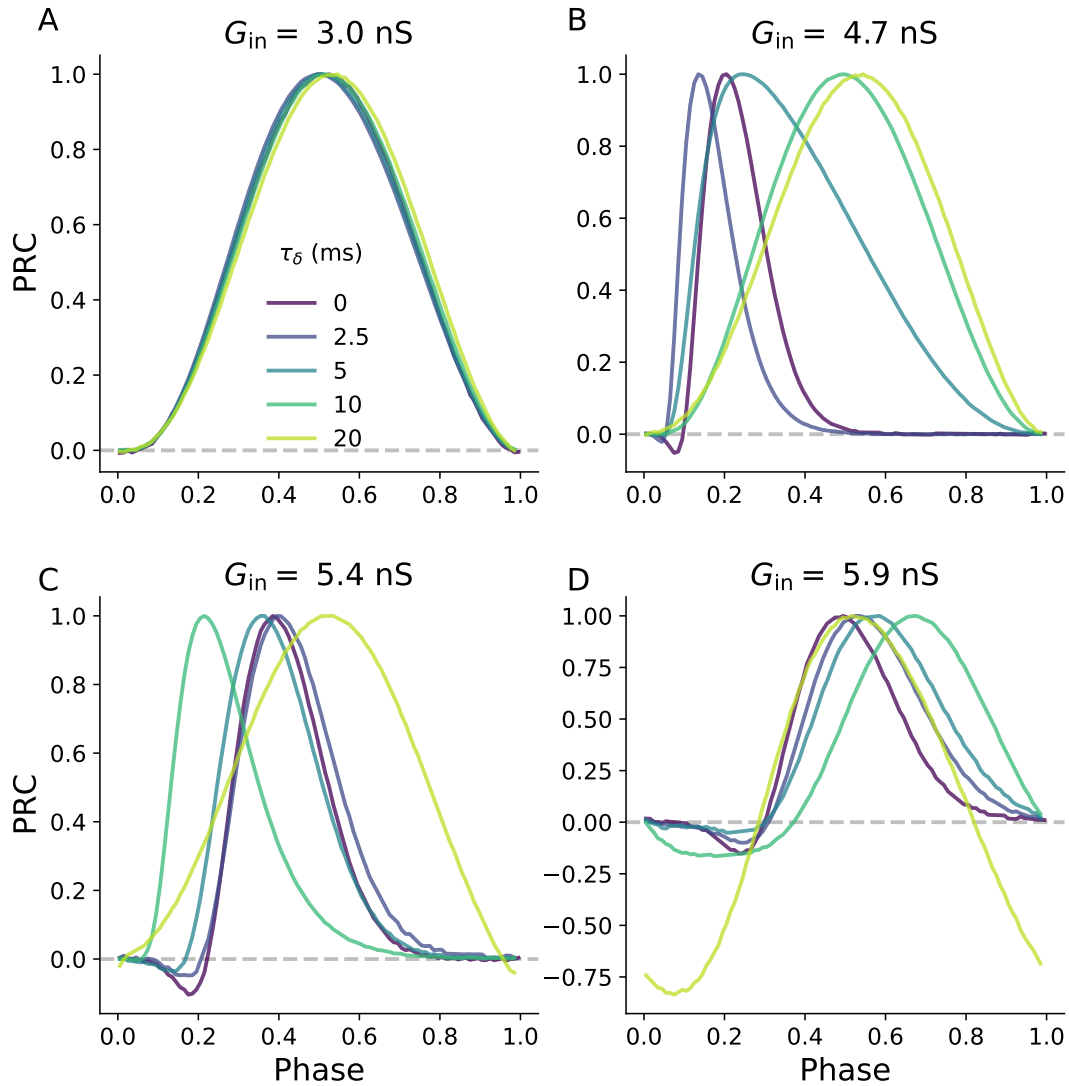


**Figure 5.** Phase-response curves (PRCs) of the ML DS model for various $\tau_\delta$ and $G_{in}$. Values of $G_{in}$ and $\tau_\delta$ have been chosen to be around the excitability switches in the ML DS model. For example at $\tau_\delta = 0$, $G_{in}^{BT} = 4.77$ nS and for all $\tau_\delta$ the cusp bifurcation lies at $G_{in}^{C} = 5.53$ nS. When $\tau_\delta < \tau_\delta^{BTC}$ ($\tau_\delta^{BTC} = 12.9$ ms), increasing $G_{in}$ switches the onset PRC shape first from a symmetric SNIC PRC (**A**) to an asymmetric HOM PRC (**B-C**), and later to a Hopf-like PRC (**D**). Increasing $\tau_\delta$ increases the value of $G_{in}$ at which the SNIC → HOM transition occurs and also decreases the $G_{in}$ value of the HOM → Hopf transition. For $\tau_\delta > \tau_\delta^{BTC}$, the PRC transitions straight from SNIC to Hopf-like. DS parameters used $\ell = 5$.

## Full Morphology Test

To demonstrate that switches in excitability from increased input conductance are applicable to more complex and realistic neuronal morphologies, we calculated the PRCs from simulations of reconstructed dendritic arbours. In this case, we used the reconstructed dendritic arbour of a real

Purkinje cell from NeuroMorpho.Org (***Vetter et al., 2001***; ***Ascoli et al., 2007***). We kept the somatic properties the same as the DS model investigated earlier and set all the dendritic compartments to have passive dynamics with $\tau_\delta = 2.5$ ms.

Starting with only the soma (representing the S model with default class I parameters), we increased $G_{\text{in}}$ by adding dendritic compartments to reconstruct the full dendritic arbour in stages, as shown in the top row of ***Figure 6***. At each stage of arborisation, we found the somatic onset current and measured the PRC. If we choose the $G_{\text{in}}$ of the full dendritic arbour to be what would be in the subcritical Hopf regime of the DS model, then we can find different PRCs representing their respective excitability types by tuning the extent of dendritic arborisation. For instance, at the small arborisation in ***Figure 6***A, $G_{\text{in}}$ is low and we have a symmetric PRC indicative of SNIC onset. Increasing $G_{\text{in}}$ first gives rise to an asymmetric HOM-like PRC (***Figure 6***B-C) before eventually yielding a PRC with substantial negative regions representing Hopf onset (***Figure 6***D).

We then compared the detailed-morphology PRCs with PRCs obtained from an equivalent DS model. For this DS model, the dendritic length $L$ and length constant $\lambda$ were extracted for each stage of the Purkinje cell by reducing the arbour to an equivalent cable (***Rall and Rinzel, 1973***; ***Amsalem et al., 2020***). Due to the different number of compartments between the detailed arbours and their DS equivalent models, we normalised the PRCs when comparing them by setting each PRC peak value to one. ***Figure 6*** shows that the relative PRCs of the equivalent DS model in each stage agree very closely with the PRCs obtained from the full morphology, with small deviations of PRC peak location in the HOM region being the only noticeable difference.

While we have not attempted to accurately capture the complex active dynamics of Purkinje cells (whether in the soma or in the dendrites), we have used its highly branched and complex dendritic arbour to demonstrate that our modelling of a single equivalent dendrite and soma is adequate to approximately capture the affect on neuronal excitability of realistic dendritic arbours. Our analysis is thus not limited to the equivalent dendrite morphology in the DS model, but is applicable to realistic morphologies with highly bifurcated arbours with tapering dendrites.

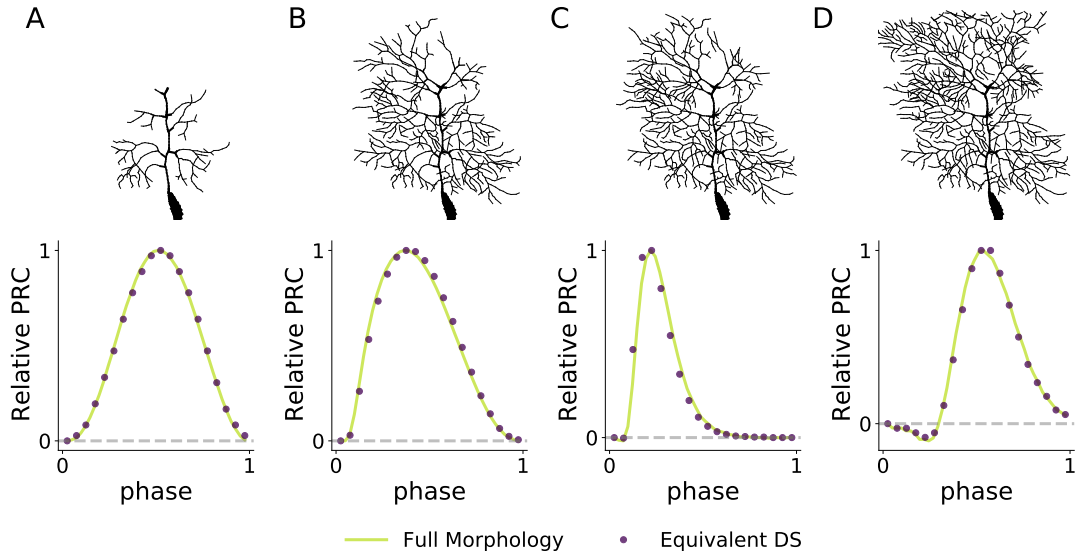


**Figure 6.** The PRCs obtained by simulating detailed multicompartment Purnkinje cell neuron show that the results of the simplified DS morphology are applicable to complicated and realistic dendritic arbours. Here we increase the input conductance of the multicompartment model by "growing" the dendritic arbour. We can see this switches the PRC shape from SNIC (**A**) to HOM (**B-C**) to Hopf (**D**) as in the DS model earlier. PRCs obtained from the equivalent DS model closely agree with the full morphology at each of the morphological stages.

## Network Simulations

We now illustrate how the dendritic conductance load can affect the network synchronisation states of a small population of these neurons via a switch in excitability type. For a group of neurons where each member has a similar spiking frequency, we measure the synchronicity of the network by the scaled standard deviation of the phase differences $\psi_i$ between the $N$ neurons

$$R = \sqrt{\frac{N}{N-1}\left(\sum_{i=1}^{N}\psi_i^2 - \langle\psi\rangle\right)}. \tag{8}$$

For $N$ neurons all synchronised in-phase, $R = 1$, while in the splayed-out state with $\psi_i = 1/N$ for all $i$, $R = 0$.

We simulated two separate networks each with 5 neurons with all-to-all connectivity of excitatory delta synapses. All synapses were connected to the soma of each neuron with zero transmission delay. In both networks the neurons had identical properties with a common excitability type: the first consisted of neurons with SNIC excitability and the second of neurons with HOM excitability. The synaptic strengths were chosen such that the maximum phase advance from a single synaptic input as predicted by the PRC is $\sim 0.1$, as shown in ***Figure 7***D. The firing rate of each

uncoupled neuron in both networks was set at 1 Hz. Thus the two networks differ in their spiking dynamical type and not the spiking frequency or the effective synaptic strength.

The first network consisted of neurons all with low dendritic load ($G_{\text{in}} = 3.45$ nS), placing them in the region of SNIC excitability. We see in ***Figure 7***A that the phase relations between neurons approaches a synchronous state but Figure ***Figure 7***C shows that this network does not settle to stable in-phase synchrony. The lack of a strongly attractive stable network spiking state for this network arises from the near-perfect symmetry of the SNIC PRC (***Izhikevich, 2007b***).

By contrast, the second network consists of neurons all with higher dendritic load in the region of HOM excitability ($G_{\text{in}} = 4.53$ nS). ***Figure 7***B shows that this network converges to a splayed-out network spiking state which is approached rapidly as shown in ***Figure 7***C. This splay state is made stable by the fact that the HOM PRC is asymmetric with a peak at a phase less than $1/N = 0.2$ (***Ashwin et al., 2016***).

Thus we have demonstrated how changes to the neuronal dynamics conferred by a more extensive neuronal morphology can affect the network behaviour. While these network states will be altered further by synaptic dynamics, transmission delays, and heterogeneities in neuronal properties, in the weak-coupling regime the neuron's PRC will remain an essential component in determining the stable network states.

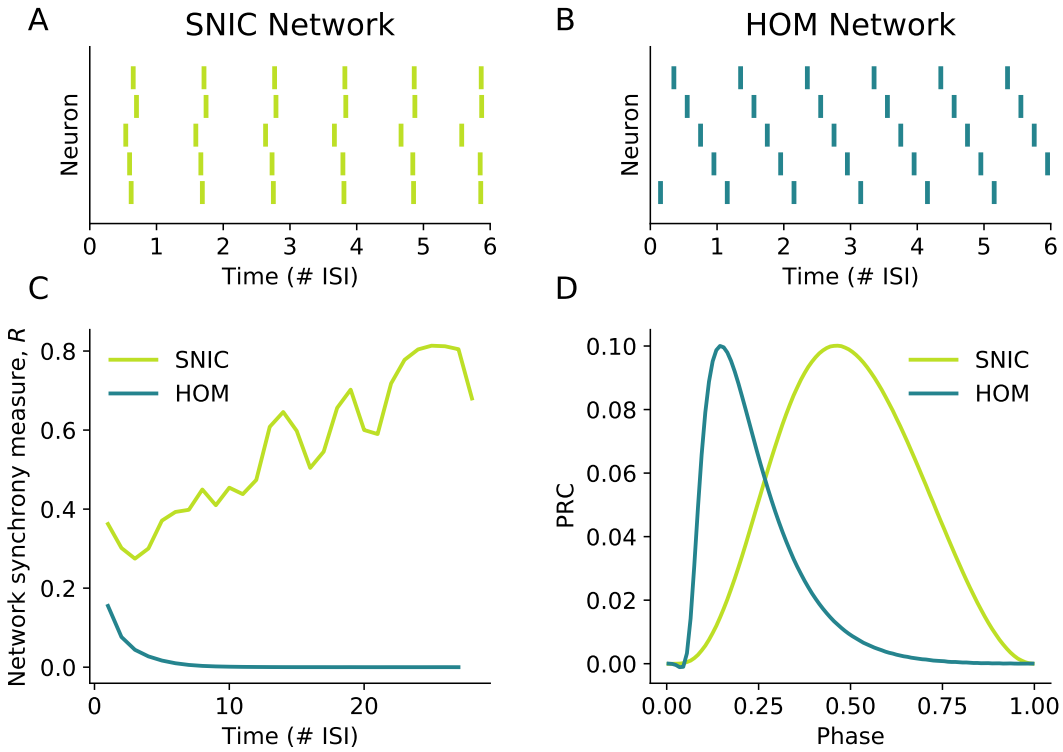


**Figure 7.** A comparison of two all-to-all networks with excitatory coupling between 5 neurons, one in which all the neurons have SNIC onset and the other in which all the neurons have HOM onset. Both networks had the same initial phase relations in the initial state with the final network states shown in (**A-B**). (**A**) The SNIC network almost achieves a synchronous network state, though this is weakly stable due to the near-symmetry of the PRC. (**B**) The HOM network achieves a splay state in which the neurons are phase-locked in which neuron $i$ has a constant phase difference of $1/5$ with neuron $i+1$. (**C**) The synchrony measure over time shows that the SNIC network gradually and non-monotonically approaches the synchronous state while the HOM network converges to the splay state far more rapidly. In panels (**A**-**C**), time is measured in terms of the number of interspike-intervals (ISIs) of the network spiking rate. (**D**) The PRCs of each neuron in the SNIC and HOM networks.

## Discussion

In this article we have shown that not only can passive dendrites be included in the analysis of conductance-based neuron models, but also that the addition of a dendrite switches the onset excitability type of the neuron. In particular, the reduction in input impedance caused by the dendrite can induce the SNIC → HOM excitability switch, changing the onset PRC from symmetric to asymmetric. This excitability switch means that altering the dendritic arborisation allows the network dynamics to achieve stable (de)synchronisation states.

From a model consisting of an active soma attached to a spatially continuous passive dendrite, we calculated important local bifurcations of the system, with the steps shown in the Methods section. This approach is not restricted to the Morris-Lecar model examined in this article, but to any conductance-based neuron model with independent voltage-activated ion channels (e.g. the Wang-Buzsáki model (***Wang and Buzsáki, 1996***)). The calculation of the BT and BTC bifurcations allowed us to predict how the dendrite affects the onset type, as these bifurcations act as organising centres for different onset types and different switches between onset types respectively.

Using the external input current, passive input conductance and dendritic time constant as bifurcation parameters, we found that the dendrite differentially affects the local bifurcations of the system. The saddle-node and cusp bifurcations are unaffected by the dendritic time constant of the system, meaning that all dendrite-and-soma models have the same saddle-node bifurcation locations as a one-compartment model with equivalent leak conductance. In contrast, the BT bifurcation moves in an "anticlockwise" direction about the cusp as the dendritic time constant increases, with the emerging Hopf bifurcation switching from subcritical to supercritcal at the BTC bifurcation. This change in the BT bifurcation with the dendritic time constant demonstrated that the effect of passive dendritic load on excitability type cannot be fully replicated with a one-compartment model with an increased leak conductance.

The "anticlockwise" shift in the BT bifurcation with increasing $\tau_\delta$ meant that the switch between class I and class II $f$–$I$ relationships occurs at higher input conductances for $\tau_\delta < \tau_\delta^{\mathrm{BTC}}$. Furthermore, it means that the switch between SNIC and HOM onset at the SNL bifurcation occurs at higher input conductance, and that the input conductance region of HOM onset is smaller. Examining the PRCs confirmed the predicted spiking onset types from the bifurcation analysis: the SNIC → HOM switch is shifted to higher $G_{\mathrm{in}}$ when $\tau_\delta$ is increased in the ML model. When $\tau_\delta$ is above the BTC value, the HOM PRC region was eliminated.

The agreement between the PRCs obtained from the morphologically detailed Purkinje cell reconstruction and its DS model demonstrated the validity of reducing the dendritic arbour to a single equivalent cylinder in our earlier analysis. This shows that one can predict the dynamical excitability type of a morphologically detailed neuron with passive dendrites by knowing just its equivalent dendrite reduction and its active spike-generating currents. Furthermore, the equivalent dendrite reduction implies that an arbour that has more dendrites branching off the soma and thicker dendrites will have a greater impact on the neuron's dynamical excitability type.

Finally, our network simulations showed that neurons with greater dendritic load could achieve splay states for excitatory coupling due to their HOM onset. These splay states could not be achieved by neurons with lower dendritic load which had SNIC onset. Thus one can achieve different network (de)synchronisation states by tuning passive dendritic properties common to all neurons.

The differential effect on the local bifurcations and PRCs when comparing the single-compartment and dendrite-and-soma models has utility for reducing the number of compartments in neuronal models for larger network simulations. If the input conductance is far from that of the BT point in the SNIC onset, then a single-compartment model with leak conductance equal to the input conductance of the morphology is appropriate. However, if $G_{\mathrm{in}}$ is close to $G_{\mathrm{in}}^{\mathrm{BT}}$, then one must use more sophisticated approximations of the input impedance (for example ***Major et al.*** (***1993***); ***Wybo et al.*** (***2013***, 2021)). Our method showing how to calculate $G_{\mathrm{in}}^{\mathrm{BT}}$ in spatially extended models thus informs one when these more sophisticated approximations are required.

Our results also prompt the hypothesis that neuronal morphology may be one crucial factor in explaining a differential susceptibility of brain areas to pathological network states like epileptiform activity or spreading depolarisation (***Jiruska et al., 2013***; ***Wei et al., 2014***). This could occur in two related ways. Firstly, pathological spiking patterns have been produced in modelling studies of single cells by changing the spiking dynamics (***Wei et al., 2014***; ***Depannemaecker et al., 2022***). Therefore, since we have shown that the dendritic impedance alters spiking dynamics, changing the dendritic arborisation could therefore move the dynamical state of the neuron to a pathological region. Second, pathological behaviour is often associated with either surplus or insufficient synchronous activity (***Bergman and Deuschl, 2002***; ***Uhlhaas and Singer, 2010***; ***Jiruska et al., 2013***). Since we have shown that the excitability switches resulting from added dendritic leak affect (de)synchronisation states, it follows that dendritic arborisation may move the degree of network synchrony to or from a pathological state.

Presently there is much research interest in how dendrites contribute to neuronal computation. This has largely focused on how either the nonlinear active dendritic channels (***Behabadi et al., 2012***; ***Tzilivaki et al., 2019***; ***Gidon et al., 2020***) or nonlinear dendritic synapses in conjunction with passive dendritic compartments (***Stöckel and Eliasmith, 2022***) affect the voltage or firing rate at the soma (i.e. voltage or rate coding). In showing how the passive dendritic contribution changes the excitability type generated by the spiking compartment, we have shown one way how dendrites

can affect the temporal encoding of neuronal networks.

This work provides a framework from which other influences of the dendritic arbour on a neuron's spiking dynamics can be explored. As a first example, it allows us to explore the effect of inputs applied to the dendrites. For a steady external current applied at an arbitrary location, our approach to calculating the local bifurcation structure can still be applied, as detailed in the Appendix. Meanwhile for transient dendritic perturbations, the PRCs from dendritic input have been simulated and observed experimentally in previous studies (***Tiroshi and Goldberg, 2019***; ***Crook et al., 1998***; ***Goldberg et al., 2007***). In these cases, it is found that the amplitude of the PRC decreases with the distance of the perturbation from the soma. On the other hand, the PRC shape is unchanged for positive-valued PRCs and is shifted for PRCs with negative-valued regions.

The effect of some dendritic active currents can also, in principle, be included in our modelling approach. If the currents are linearisable under the quasi-active approximation (***Koch, 1984***), then the effect of the active currents can included in the filtering properties of the dendrite (***Coombes et al., 2007***; ***Remme et al., 2009***; ***Remme and Rinzel, 2011***; ***Zhuchkova et al., 2013***). Weakly active dendritic currents, such as $I_{\mathrm{h}}$ and small-conductance $Ca^{2+}$-activated potassium current $I_{\mathrm{SK}}$, have been found to change the PRCs from being positive-valued everywhere to having negative regions (***Goldberg et al., 2007***; ***Schultheiss et al., 2010***), and thus may change the network synchronisation state. If the active currents are strong enough to elicit dendritic spikes (***Larkum et al., 2022***), then this falls outside of the framework described in our work, firstly because there are now multiple spiking compartments, and second because the interaction between strong dendritic nonlinearities (e.g. spikes, plateau potentials) and axosomatic action potentials can induce bursting behaviour outside of the regular spiking regime (***Larkum et al., 1999***; ***Williams and Stuart, 1999***; ***Grienberger et al., 2014***). However, some aspects of our work may still be used if one considers the strongly active dendritic region to be another oscillator coupled dendritically to the axosomatic compartment (***Remme et al., 2009***; ***Yi et al., 2017***).

Lastly, there has been much interest in how the geometry of the axonal initial segment (AIS) affects spike threshold and spike shape (***Kole and Stuart, 2012***; ***Hamada et al., 2016***; ***Goethals and Brette, 2020***) and encoding of time-varying input (***Eyal et al., 2014***; ***Zhang et al., 2022***). Extending our framework to include features of the axon, such as the spatial separation between the soma and the spike-initiation point on the AIS, would allow exploration of how the geometry of the system affects the excitability type. Changes in excitability type would in turn give further insight into

how axonal geometry affects the neuron's temporal encoding of input.

In summary, we demonstrate that neuronal morphology can significantly influence the state of neuronal networks. Moreover, our results describe a method by which one can directly assess the impact of a dendritic arbour on neuronal excitability. Our approach is flexible in allowing for any conductance-based neuron model and external input currents applied to any location. Thus, the proposed approach enables further exploration of how dendritic arbour impacts the tuning of temporal encoding via changes to the network (de)synchronisation state. Our work highlights neuronal morphology as a contributor to neural function via changes to network synchrony. Neuronal morphology is therefore likely to be relevant for the differential sensitivity of neuronal tissue to synchronisation in health and disease.

## Model and Methods

### Active Soma and Passive Dendrite Model

A single-compartment (S) conductance-based neuron model consists of a leak current, $B$ voltage-activated currents $I_{a,j}$ and an externally applied current $I_{\text{ext}}$

$$C_{\text{m}}\frac{\text{d}v}{\text{d}t} = G_L(E_L - v) + \sum_{j=1}^{B} I_{\text{a},j}(\boldsymbol{a}_j, v) + I_{\text{ext}} = C_{\text{m}} f_{\text{S}}(\boldsymbol{a}, v), \tag{9}$$

where each voltage-activated current depends on a set of activation/inactivation gating variables (hereafter termed "active variables"), $\boldsymbol{a}_j = (a_{j1}, ..., a_{jb_j})$. Altogether this gives $K$ active variables, and we will hereafter index all the active variables $a_i$ from $i = 1, ..., K$ for simplicity. As in prior research regarding conductance-based models (***Kirst et al., 2017***; ***Schleimer and Schreiber, 2018***; ***Al-Darabsah and Campbell, 2021***), we will assume that the active variables are independent of each other and that their steady state distributions $a_{i,\infty}(v)$ saturate as $v \to \pm\infty$. Each active current has a reversal potential $E_j$, a maximal conductance $G_j$, and depends on the product of its active variables

$$I_{\text{a},j}(\boldsymbol{a}_j, v) = G_j(E_j - v)\prod_{i\in\boldsymbol{a}_j} a_i(v)^{p_i}, \tag{10}$$

where $p_i$ is the gating exponent associated with active variable $a_i$. Finally, the each active variable evolves in time with a voltage-dependent time constant $\tau_i(v)$

$$\frac{\text{d}a_i}{\text{d}t} = \frac{a_{i,\infty}(v) - a_i}{\tau_i(v)} = f_{a,i}(a_i, v). \tag{11}$$

Using Rall's principle of an equivalent cylinder, a passive dendritic arbour emanating from the soma can be simplified to a single equivalent dendrite (***Rall and Rinzel, 1973***; ***Rall, 2011***). We therefore modify the above model to include the dendritic morphology by attaching a passive equivalent dendrite to a conductance-based active soma. The dendrite is spatially continuous, with the coordinate $x$ denoting the distance away from the soma and $v_\delta(x)$ denoting the dendritic potential at location $x$.

The dendrite is parametrised by its electrotonic length constant $\lambda$, its passive time constant $\tau_\delta$ and the dendritic dominance factor $\rho$ (the ratio between the characteristic dendritic conductance and the somatic leak conductance) (***Rall, 2011***). These are each defined in terms of electrophysiological parameters

$$\tau_\delta = \frac{c_\delta}{g_\delta}, \quad \lambda = \sqrt{\frac{d}{4r_a g_\delta}}, \quad \rho = \frac{\pi d g_\delta \lambda}{G_\sigma}, \tag{12}$$

where $c_\delta$ is the dendritic membrane capacitance per unit area, $g_\delta$ is the leak membrane conductance per unit area, $r_a$ is the axial resistivity, $d$ is the dendritic diameter and $G_\sigma$ is the leak conductance of the soma.

Recalling that we denoted $\mathrm{d}v/\mathrm{d}t$ in (9) as $f_\mathrm{S}$, this means that the somatic voltage $v_\sigma$ evolves as

$$\frac{\mathrm{d}v_\sigma}{\mathrm{d}t} = f_\mathrm{S}(\boldsymbol{a}, v_\sigma) + \frac{\rho G_\sigma \lambda}{C_\sigma} \left.\frac{\partial v_\delta}{\partial x}\right|_{x=0}, \tag{13}$$

where the last term represents the axial current flowing from the dendrite to the soma. Note also that we have replaced the membrane capacitance of the one-compartment model, $C_\mathrm{m}$, with the capacitance of the soma, $C_\sigma$.

The dendritic potential obeys the passive cable equation

$$\tau_\delta \frac{\partial v_\delta}{\partial t} = E_L - v_\sigma + \lambda^2 \frac{\partial^2 v_\delta}{\partial x^2}, \quad 0 < x < L, \tag{14}$$

where for simplicity we have assumed that the leak reversal potential is the same in the dendrite as the soma. The dendritic potential is subject to continuity of potential at $x = 0$ so $v_\delta(x = 0) = v_\sigma$, and a sealed-end boundary condition at $x = L$

$$\left.\frac{\partial v_\delta}{\partial x}\right|_{x=L} = 0. \tag{15}$$

Defining the electrotonically normalised length as $\ell = L/\lambda$, when $\ell \gg 1$, the distal dendritic end is too far away to be influenced by somatic activity, and we can simplify the model by making the dendrite semi-infinite in extent.

## Calculation of Local Bifurcations

Here we will describe how the local bifurcations of the DS system can be calculated for any conductance-based soma model with $(I_{\text{ext}}, G_{\text{in}}, \tau_\delta)$ as the bifurcation parameters. Although we primarily focus on the semi-infinite dendrite in this article, the method we outline here can be adapted for the finite dendrite, and equations to solve for the finite dendrite are given in the Appendix. The equations we ultimately derive for each bifurcation are applicable to a spatially continuous dendrite rather than for a specific finite number of dendritic compartments.

### Fixed Points

Since local bifurcations are defined by the properties of a fixed point, we must first outline how to calculate fixed points of the DS system. The fixed points are defined as the values $(\boldsymbol{a}^*, \boldsymbol{v}^*)$ for which all time derivatives of the system (11, 13, 14) are equal to zero. From (11), each of the active variables satisfies $a_i^* = a_{i,\infty}(v_\sigma)$ at equilibrium. The cable equation of (14) at equilibrium becomes the second-order ODE

$$0 = E_L - v_\delta^* + \lambda^2 \frac{\mathrm{d}^2 v_\delta^*}{\mathrm{d}x^2}, \quad v_\delta^*(0) = v_\sigma^*, \quad \left.\frac{\mathrm{d}v_\delta^*}{\mathrm{d}x}\right|_{x=L} = 0. \tag{16}$$

This has the following solution in terms of $v_\sigma$

$$v_\delta^*(x) = E_L + (v_\sigma^* - E_L)\frac{\cosh[(L-x)/\lambda]}{\cosh(L/\lambda)}, \tag{17}$$

which in the semi-infinite limit reduces to

$$v_\delta^*(x) = E_L + (v_\sigma^* - E_L)e^{-x/\lambda}. \tag{18}$$

Thus all the active variables and the dendritic potential for all $x$ at equilibrium are given in terms of the somatic resting potential $v_\sigma^*$. Defining the steady-state current as

$$I_\infty(v_\sigma^*) = C_\sigma f_{\text{S}}(\boldsymbol{a}_\infty(v_\sigma^*), v_\sigma^*) + \rho g_\sigma \lambda \left.\frac{\mathrm{d}v_\delta^*}{\mathrm{d}x}\right|_{x=0}, \tag{19}$$

we can substitute in the dendritic voltage derivative at $x = 0$ to give in the semi-infinite case

$$I_\infty(v_\sigma^*) = C_\sigma f_{\text{S}}(\boldsymbol{a}_\infty(v_\sigma^*), v_\sigma^*) + \rho G_\sigma (E_L - v_\sigma^*). \tag{20}$$

The somatic equilibrium potential $v_\sigma^*$ is obtained by numerically solving $I_\infty = 0$. We note that the steady-state current equation (20) is the same as what we would find for the S model if $G_L = G_\sigma(1+\rho)$ as $\tau_\delta$ is not present.

## Saddle-Node (SN) Bifurcation

In conductance-based neuron models, class I spiking onset often (though not always) occurs at a saddle-node bifurcation, where class I here means that the onset spiking frequency is zero. At a saddle-node bifurcation, a saddle and node fixed-point meet. Therefore we have a repeated root $v_\sigma^{\mathrm{SN}}$ of $I_\infty = 0$, which means at the saddle-node

$$\frac{\mathrm{d}I_\infty}{\mathrm{d}v_\sigma}(v_\sigma^{\mathrm{SN}}) = 0. \tag{21}$$

We can also derive this condition from the fact that the Jacobian of the system evaluated at a saddle-node equilibrium has one zero eigenvalue. For codimension one bifurcations such as the saddle-node bifurcation, we choose $I_{\mathrm{ext}}$ as the bifurcation parameter as we are interested in the onset current for spiking. Hence we can numerically solve (21) to obtain $v_\sigma^{\mathrm{SN}}$ as it does not contain $I_{\mathrm{ext}}$. $I_{\mathrm{ext}}^{\mathrm{SN}}$ is obtained by rearranging $I_\infty = 0$. Note that for the same $G_{\mathrm{in}}$, (21) is also identical for both S and DS models.

## Cusp Bifurcation

At the cusp, two saddle-node bifurcations meet. Thus, the cusp bifurcation satisfies the condition

$$\frac{\mathrm{d}^2 I_\infty}{\mathrm{d}v_\sigma^2}(v_\sigma^{\mathrm{C}}) = 0. \tag{22}$$

For codimension-two bifurcations such as the cusp, we will use $(I_{\mathrm{ext}}, G_{\mathrm{in}})$ as our bifurcation parameters. This is analogous to the prior use of $(I_{\mathrm{ext}}, g_L)$ in point-neuron models (***Kirst et al., 2017***). Since $I''_{\mathrm{ext}}(v_\sigma)$ does not depend on $G_{\mathrm{in}}$ or $I_{\mathrm{ext}}$, we numerically solve (22) to obtain $v_\sigma^{\mathrm{C}}$. $G_{\mathrm{in}}^{\mathrm{C}}$ and $I_{\mathrm{ext}}^{\mathrm{C}}$ are obtained from rearranging (21) and (20) respectively. As with the saddle-node bifurcation, the cusp bifurcation condition does not depend on $\tau_\delta$, thus for the same $G_{\mathrm{in}}$ the cusp bifurcation values will be the same for the S and DS models.

## The Method of Lines

For finite-dimensional dynamical systems, such as conductance-based point-neuron models, the Jacobian of the system has a finite number of eigenvalues. This gives a clear, but by no means trivial, approach to calculating the Hopf, BT and BTC bifurcations (***Hesse et al., 2017***; ***Kirst et al., 2017***; ***Al-Darabsah and Campbell, 2021***). However, for a spatially continuous system, the number of dimensions is not finite. Fortunately, by using the method lines, we can derive equations for the bifurcations in an analogous fashion.

The method of lines discretises a spatially continuous system by dividing the cable into $M$ compartments with index $k = 1, .., M$ (***Verwer and Sanz-Serna, 1984***). In this case, we will use a constant spatial step size $\Delta x$ such that $v_\delta(k\Delta x, t) = v_k(t)$ and use the following discretised approximations of the first and second-order spatial derivatives

$$\frac{\partial^2 v_k}{\partial x^2} \approx \frac{v_{k+1} - 2v_k + v_{k-1}}{\Delta x^2}, \quad \frac{\partial v_k}{\partial x} \approx \frac{v_{k+1} - v_k}{\Delta x}. \tag{23}$$

Note that $v_{k=0} = v_\sigma$ and that we must incorporate the sealed-end boundary condition, which changes the second derivative approximation at the distal dendritic end to

$$\frac{\partial^2 v_M}{\partial x^2} \approx \frac{2v_{M-1} - 2v_M}{\Delta x^2}. \tag{24}$$

This discretisation means that we now have a $M + K + 1$ dimensional dynamical system, where we recall that $K$ is the number of active variables. The active variable equations remain unchanged from (11), while the voltage equations in the discretised system are

$$\begin{aligned} \frac{\mathrm{d}v_\sigma}{\mathrm{d}t} &= f_S(\boldsymbol{a}, v_\sigma) + \frac{\rho G_\sigma \lambda}{C_\sigma \Delta x}(v_1 - v_\sigma), \\ \frac{\mathrm{d}v_k}{\mathrm{d}t} &= \frac{E_L - v_k}{\tau_\delta} + \lambda^2 \frac{v_{k+1} - 2v_k + v_{k-1}}{\Delta x^2 \tau_\delta}, \quad k = 1, ..., M-1 \\ \frac{\mathrm{d}v_M}{\mathrm{d}t} &= \frac{E_L - v_M}{\tau_\delta} + \lambda^2 \frac{2v_{M-1} - 2v_M}{\Delta x^2 \tau_\delta}. \end{aligned} \tag{25}$$

From (11) and (25) we can obtain the Jacobian $\boldsymbol{J}$ for the discretised system, allowing us to approach the problem of calculating the local bifurcations in a similar manner to the point-neuron model. Although the Jacobian now has $(M + K + 1) \times (M + K + 1)$ elements, most of these are zero, and those associated with the dendritic equations do not depend on the state variables. Further details of this approach are given in the Appendix, but it is sufficient here to say that the method of lines allows local bifurcations to be calculated for the *discretised* dendrite-and-soma model. With the equations from the discretised dendrite, we can go further and take the continuum limit ($\Delta x \to 0$) to yield equations for the spatially *continuous* DS model. It is these spatially continuous bifurcation values which we show in the bifurcation diagrams in this article.

With the method of lines established, we can now describe how to calculate the Hopf, BT and BTC bifurcations in a spatially continuous DS model.

## Hopf Bifurcation

At a Hopf bifurcation, a fixed-point changes stability and a limit cycle appears. The criticality of the Hopf bifurcation determines the stability of the limit cycle involved; at a subcritical Hopf bifurca-

tion we have the transition: stable FP + unstable LC → unstable FP, whilst at a supercritical Hopf bifurcation we have: stable FP → unstable FP + stable LC.

Spiking initiated by a Hopf bifurcation has a class II $f$–$I$ curve, which means that the onset frequency is non-zero. However note that the existence of a Hopf bifurcation is neither a necessary nor sufficient condition for class II spiking onset. Hopf bifurcations also feature when a neuron stops spiking when the input current becomes too high. The offset of spiking at excitation block in conductance-based neuron models typically occurs via a supercritical Hopf bifurcation or a fold of limit cycles preceded by a subcritical Hopf bifurcation.

The Jacobian evaluated at a Hopf bifurcation has two purely imaginary conjugate eigenvalues $\pm i\omega_{\mathrm{H}}$. Denoting the corresponding right-eigenvector as $\boldsymbol{q}$ and its complex conjugate as $\overline{\boldsymbol{q}}$, this means that we have

$$\boldsymbol{J}\boldsymbol{q} = i\omega_{\mathrm{H}}\boldsymbol{q}, \quad \boldsymbol{J}\overline{\boldsymbol{q}} = -i\omega_{\mathrm{H}}\overline{\boldsymbol{q}}. \tag{26}$$

From this pair of equations, we can derive two simultaneous nonlinear equations in terms of $(v_\sigma^{\mathrm{H}}, \omega_{\mathrm{H}})$ for the discretised system. Then we take the continuum limit to obtain two nonlinear equations for the continuous dendrite (see appendix for further detail). For the semi-infinite dendrite these equations are

$$\begin{aligned}
&\sum_{i=1}^{K} \frac{1}{1+\omega_{\mathrm{H}}^2\tau_i^2}\frac{\partial f_{\mathrm{S}}}{\partial a_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\rho G_\sigma}{2C_\sigma}s_\gamma(\omega_{\mathrm{H}}) = 0,\\
&\sum_{i=1}^{K} \frac{\tau_i}{1+\omega_{\mathrm{H}}^2\tau_i^2}\frac{\partial f_{\mathrm{S}}}{\partial a_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\rho G_\sigma}{2C_\sigma}\frac{d_\gamma(\omega_{\mathrm{H}})}{\omega_{\mathrm{H}}} + 1 = 0,
\end{aligned} \tag{27}$$

where

$$\begin{aligned}
s_\gamma(\omega_{\mathrm{H}}) &= \sqrt{2+2\sqrt{1+\omega_{\mathrm{H}}^2\tau_\delta^2}},\\
d_\gamma(\omega_{\mathrm{H}}) &= \mathrm{sgn}(\omega_{\mathrm{H}})\sqrt{-2+2\sqrt{1+\omega_{\mathrm{H}}^2\tau_\delta^2}}.
\end{aligned} \tag{28}$$

As in the saddle-node bifurcation, for the codimension one Hopf bifurcation we will use $I_{\mathrm{ext}}$ as our bifurcation parameter. Therefore for Hopf bifurcations, we first obtain $v_\sigma^{\mathrm{H}}$ from (27) and then obtain $I_{\mathrm{ext}}^{\mathrm{H}}$ from $I_\infty = 0$. Unlike all the previous bifurcations listed, the Hopf bifurcation does depend on $\tau_\delta$. Therefore, we should expect the bifurcation values of the Hopf bifurcation to differ between the S and DS models.

The criticality of the Hopf bifurcation can be calculated using the approach outlined in ***Kuznetsov*** (***2013***). In addition to the right-eigenvectors $(\boldsymbol{q}, \overline{\boldsymbol{q}})$ described earlier, this approach requires the left-

eigenvectors of $\boldsymbol{J}$,

$$\boldsymbol{p}\boldsymbol{J} = -i\omega_{\mathrm{H}}\boldsymbol{p}, \quad \overline{\boldsymbol{p}}\boldsymbol{J} = i\omega_{\mathrm{H}}\overline{\boldsymbol{p}}. \tag{29}$$

The Hopf criticality is present in our bifurcation diagrams, but since the equations involved are highly verbose, we direct the interested reader to the appendix for their explicit form.

## Bogdanov-Takens (BT) Bifurcation

At the BT point, a saddle-node, Hopf and saddle-node homoclinic orbit (HOM) bifurcations meet. Since the global HOM bifurcation can also determine the spiking onset type of the neuron (***Izhikevich, 2007a***; ***Pereira et al., 2015***; ***Hesse et al., 2017***; ***Kirst et al., 2017***), the BT point acts as an organising centre in two parameter dimensions for different spiking onset types. Varying the bifurcation parameters $(I_{\mathrm{ext}}, G_{\mathrm{in}})$ in the vicinity of the BT point can therefore induce spiking onsets associated with the three codimension one bifurcations that meet here.

From the coexistence of saddle-node and Hopf bifurcations, the Jacobian of the corresponding dynamical system will have two zero eigenvalues at the BT equilibrium. This means that $\boldsymbol{J}$ will have orthogonal left, $\boldsymbol{l}$, and right, $\boldsymbol{r}$, eigenvectors satisfying

$$\boldsymbol{l}\boldsymbol{J} = \boldsymbol{0}, \quad \boldsymbol{J}\boldsymbol{r} = \boldsymbol{0}, \quad \boldsymbol{l}\boldsymbol{r} = \boldsymbol{0}. \tag{30}$$

These equations can be reduced to a single equation

$$\sum_{i=1}^{K} \left(\alpha_0\tau_\delta + \tau_i\right) \frac{\partial f_{\mathrm{S}}}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + 1 + \alpha_0\tau_\delta \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} = 0, \tag{31}$$

where for the semi-infinite dendrite $\alpha_0 = 1/2$. Since (31) has no dependence on our bifurcation parameters $(I_{\mathrm{ext}}, G_{\mathrm{in}})$, we can numerically solve it to find the equilibrium potential $v_\sigma^{\mathrm{BT}}$. $G_{\mathrm{in}}^{\mathrm{BT}}$ can then be obtained by rearranging the saddle-node condition (21) and $I_{\mathrm{ext}}^{\mathrm{BT}}$ from the equilibrium condition $I_\infty = 0$. We again see that the BT bifurcation depends on $\tau_\delta$ and thus we should expect the location of the BT bifurcation to change with $\tau_\delta$.

## Bogdanov-Takens-Cusp (BTC) Bifurcation

Finally, we outline how to find the BTC point. The BTC point is of particular interest because bifurcations associated with spike onset transitions coincide at this point. At a spiking onset transition, the spiking changes from one type to another. We should distinguish this from the lower codimension BT point by stating that the BT point organises bifurcations responsible for spiking onsets,

while the BTC point organises bifurcations responsible for *transitions* of spiking onsets. The bifurcations that meet at the BTC point include the Bautin (where a Hopf bifurcation changes stability), neutral saddle-node (where a homoclinic bifurcation and a fold of limit cycles meet), saddle-node loop (where saddle-node and homoclinic bifurcations meet), and BT bifurcations (***Kuznetsov, 2013***; ***Pereira et al., 2015***; ***Kirst et al., 2017***).

Since the BTC point acts as an organising centre for spiking onset transitions, deviations in the bifurcation parameters around it will lead to variations in the spiking onset structure in two parameter dimensions. The BTC point also has the advantage of being easier to calculate than the global bifurcations which coalesce at it, as it is calculable from the properties of fixed-points.

The BTC point has codimension 3, meaning that its location must be expressed in terms of three bifurcation parameters. In the work of Kirst et al ***Kirst et al.*** (***2017***), an equation for the BTC point for point-neuron conductance-based models is given with $(I_{\text{ext}}, g_L, C_{\text{m}})$ as the bifurcation parameters, while in (***Al-Darabsah and Campbell, 2021***), Al-Darabsah and Campbell use the M-current conductance instead of $C_{\text{m}}$. Here we use the bifurcation parameters $(I_{\text{ext}}, \rho, \tau_\delta)$ as they are common to all conductance-based neuron models and it includes two dendritic parameters. Furthermore, $\tau_\delta$ behaves similarly to $C_{\text{m}}$, in that increasing it slows the response of the dendrite to somatic activity.

As the BTC point is where a cusp and BT bifurcation coincide, one can use the cusp condition (22) to obtain the equilibrium voltage $v_\sigma^{\text{BTC}}$, the saddle-node condition (21) to give $G_{\text{in}}^{\text{BTC}}$, and the BT condition (31) to yield $\tau_\delta^{\text{BTC}}$.

## Estimation of Global Bifurcations

Global bifurcations govern many spiking onset types and spiking onset transitions, but cannot be directly calculated from the properties of fixed points. Therefore one must use numerical continuation methods (***Doedel et al., 2007***; ***Allgower and Georg, 2012***) and/or numerical simulation in order to estimate the locations of global bifurcations. For simplicity of implementation and to be understood by a wider audience, here we outline how simulation can be used to estimate the location of two global bifurcations of interest.

### Homoclinic Bifurcation

At a homoclinic (HOM) bifurcation, a homoclinic orbit is formed at a saddle. If this orbit is stable, then it forms a spiking cycle. HOM onset is class I but its $f$–$I$ curve typically grows very rapidly in

comparison to SNIC onset (***Izhikevich, 2007a***). Since the HOM bifurcation does not involve changes to the stability of fixed points, a stable homoclinic orbit typically coexists with a stable fixed point. This means that HOM onset allows bistability between quiescence and regular spiking. If the homoclinic orbit contains a single fixed point (other than the saddle), then it is termed a small-HOM (sHOM) bifurcation, while if orbit contains all three fixed points, it is a big-HOM (bHOM) bifurcation.

Using $I_{\text{ext}}$ as a bifurcation parameter, if a stable HOM bifurcation exists then it often precedes a SN bifurcation, $I_{\text{ext}}^{\text{HOM}} < I_{\text{ext}}^{\text{SN}}$. Thus we can find the HOM bifurcation as follows:

1. At a given $G_{\text{in}} < G_{\text{in}}^{\text{BT}}$, find the onset current $I_{\text{ext}}^{\text{on}}$ for regular spiking.
2. If $I_{\text{ext}}^{\text{on}} > I_{\text{ext}}^{\text{SN}}$ there is no bistability, and spiking onset occurs via a SNIC bifurcation.
3. If $I_{\text{ext}}^{\text{on}} < I_{\text{ext}}^{\text{SN}}$, bistability exists and a HOM bifurcation is a candidate for the onset type.
4. Repeat this process at various $G_{\text{in}}$ to obtain an estimate of the HOM bifurcation for the input conductance range of interest.

We specify $G_{\text{in}} < G_{\text{in}}^{\text{BT}}$ in step (1) here to avoid the presence of Hopf bifurcations. In step (3), we can only conclude that a HOM bifurcation is a candidate for the onset type because the true onset bifurcation may be a fold of limit cycles (FLC) that shortly precedes a subcritical HOM bifurcation (***De Maesschalck and Wechselberger, 2015***; ***Kirst et al., 2017***). Nevertheless, this procedure forms the basis of finding the switch between SNIC and HOM onset.

### Saddle-Node-Loop (SNL) Bifurcation

A saddle-node-loop (SNL) bifurcation is where a saddle-node and HOM bifurcation meet and has codimension two. With $(G_{\text{in}}, I_{\text{ext}})$ as bifurcation parameters, we can therefore switch between SNIC and HOM onset by varying $G_{\text{in}}$ around this bifurcation. The SNL bifurcation can be estimated via the following bisection procedure:

1. Find input conductances $G_{\text{in}}^{\text{SNIC}}$ and $G_{\text{in}}^{\text{HOM}}$ for which spiking onset occurs via SNIC and (candidate) HOM bifurcations respectively.
2. Choose a new input conductance between these two points $G_{\text{in}}^{\text{test}} = \frac{1}{2}(G_{\text{in}}^{\text{SNIC}} + G_{\text{in}}^{\text{HOM}})$ and find its onset current $I_{\text{ext}}^{\text{test}}$.
3. Using the procedure for evaluating whether the onset is due to a HOM bifurcation, evaluate the candidate bifurcation type for onset at $G_{\text{in}}^{\text{test}}$.
4. Repeat step (1) with $G_{\text{in}}^{\text{test}}$ replacing the bound of its candidate bifurcation type.

5. Stop after a given number of steps or after $G_{\text{in}}^{\text{test}}$ converges. The final value of $G_{\text{in}}^{\text{test}}$ gives an estimate of $G_{\text{in}}^{\text{SNL}}$.

### Simulation Details

To simulate the DS models, a cable of length $L = 1000$ μm was discretised into $M = 50$ evenly spaced spatial compartments with step size $\Delta x = 20$ μm. The soma occupied a separate compartment at $x = 0$. All simulations utilised the DifferentialEquations.jl package (***Rackauckas and Nie, 2017***) in the Julia programming language (***Bezanson et al., 2017***), from which the Tsitouras 5/4 Runge-Kutta method was used.

For the SNL bifurcation and PRCs, we required an estimate of the onset current at which stable spiking begins. Here we used the value of $I_{\text{ext}}$ which produced regular spiking at the lowest possible frequency above 1 Hz. This means that for class I onset the spiking frequency was approximately 1 Hz, while for class II onset this was typically greater.

At this onset current, PRCs were obtained by increasing the somatic potential by an amount $\Delta v$ at a single time $t_k$ corresponding to a phase $\theta_k$. To ensure that the phase shift is approximately in the linear regime, $\Delta v$ was chosen for each neuron such that the PRC maximum was never greater than 0.1. This procedure was repeated for input phases corresponding to $\theta_k = 0.01k - 0.005$, $k = 1, ..., 100$.

Code used to run all simulations, along with that used to calculate the local and global bifurcations, will be made available in the supplementary data.

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## Appendix

### Equations and Parameters of the Morris-Lecar Model

The single-compartment Morris-Lecar voltage equation is

$$f_{\S}(n, v_\sigma) = \frac{1}{C_\sigma}\left[G_\sigma(E_L - v_\sigma) + m_\infty(v_\sigma)G_{\text{Ca}}(E_{\text{Ca}} - v_\sigma) + nG_{\text{K}}(E_K - v_\sigma) + I_{\text{ext}}\right], \tag{32}$$

while the dynamics of the recovery variable $n$ are given by

$$\frac{\mathrm{d}n}{\mathrm{d}t} = \frac{n_\infty(v_\sigma) - n}{\tau_n(v_\sigma)}, \quad \tau_n(v_\sigma) = \left[\phi \cosh\left(\frac{v_\sigma - A_\mathrm{K}}{4\Delta_\mathrm{K}}\right)\right]^{-1}. \tag{33}$$

Steady-state values of the activation variables are given in terms of sigmoidal functions of $v_\sigma$

$$m_\infty(v_\sigma) = \left[1 + \exp\left(-\frac{v_\sigma - A_\mathrm{Ca}}{\Delta_\mathrm{Ca}}\right)\right]^{-1}, \quad n_\infty(v_\sigma) = \left[1 + \exp\left(-\frac{v_\sigma - A_\mathrm{K}}{\Delta_\mathrm{K}}\right)\right]^{-1} \tag{34}$$

| Parameter | Value | Units |
|---|---|---|
| $G_\sigma$ | 2 | nS |
| $C_\sigma$ | 20 | pF |
| $E_L$ | -60 | mV |
| $G_\mathrm{Ca}$ | 4 | nS |
| $E_\mathrm{Ca}$ | 120 | mV |
| $A_\mathrm{Ca}$ | -1.2 | mV |
| $\Delta_\mathrm{Ca}$ | 9 | mV |
| $G_\mathrm{K}$ | 8 | nS |
| $E_\mathrm{K}$ | -80 | mV |
| $A_\mathrm{K}$ | 12 | mV |
| $\Delta_\mathrm{K}$ | 8.7 | mV |
| $\phi$ | 1/15 | $\mathrm{ms}^{-1}$ |

**Table 1.** Default parameters used in this paper for the Morris-Lecar model, taken from ***Morris and Lecar*** (***1981***).

## Derivation of the Local Bifurcation Equations

### Discretised Jacobian Matrix

Upon discretising the dendrite into $M$ compartments each of width $\Delta x$, we have the following $M + K + 1$ differential equations for the system

$$\frac{\mathrm{d}a_i}{\mathrm{d}t} = f_{ai}(a_i, v_\sigma) = \frac{a_{i,\infty}(v_\sigma) - a_i}{\tau_i(v_\sigma)}, \quad i = 1, ..., K, \tag{35}$$

$$\frac{\mathrm{d}v_\sigma}{\mathrm{d}t} = f_\sigma(\boldsymbol{a}, \boldsymbol{v}) = f_\mathrm{S}(\boldsymbol{a}, v_\sigma) + \frac{\lambda\rho G_\sigma}{\Delta x C_\sigma}(v_1 - v_\sigma), \tag{36}$$

$$\frac{\mathrm{d}v_k}{\mathrm{d}t} = f_k(\boldsymbol{v}) = \frac{E_L - v_k}{\tau_\delta} + \frac{\lambda^2}{\Delta x^2 \tau_\delta}(v_{k-1} - 2v_k + v_{k+1}), \quad k = 1, ..., M - 1 \tag{37}$$

$$\frac{\mathrm{d}v_M}{\mathrm{d}t} = f_M(\boldsymbol{v}) = \frac{E_L - v_M}{\tau_\delta} + \frac{\lambda^2}{\Delta x^2 \tau_\delta}(2v_{M-1} - 2v_M). \tag{38}$$

This allows us to write the Jacobian matrix as

$$\boldsymbol{J} = \begin{pmatrix} \frac{\partial f_{a1}}{\partial a_1} & \dots & \frac{\partial f_{a1}}{\partial a_K} & \frac{\partial f_{a1}}{\partial v_\sigma} & \frac{\partial f_{a1}}{\partial v_1} & \dots & \frac{\partial f_{a1}}{\partial v_M} \\ \dots & \dots & \dots & \dots & \dots & \dots & \dots \\ \frac{\partial f_\sigma}{\partial a_1} & \dots & \frac{\partial f_\sigma}{\partial a_K} & \frac{\partial f_\sigma}{\partial v_\sigma} & \frac{\partial f_\sigma}{\partial v_1} & \dots & \frac{\partial f_\sigma}{\partial v_M} \\ \frac{\partial f_1}{\partial a_1} & \dots & \frac{\partial f_1}{\partial a_K} & \frac{\partial f_1}{\partial v_\sigma} & \frac{\partial f_1}{\partial v_1} & \dots & \frac{\partial f_1}{\partial f_M} \\ \dots & \dots & \dots & \dots & \dots & \dots & \dots \\ \frac{\partial f_M}{\partial a_1} & \dots & \frac{\partial f_M}{\partial a_K} & \frac{\partial f_M}{\partial v_\sigma} & \frac{\partial f_M}{\partial f_1} & \dots & \frac{\partial f_M}{\partial v_M} \end{pmatrix}, \tag{39}$$

which we will split into four block matrices

$$\boldsymbol{J} = \begin{pmatrix} \mathcal{A} & \mathcal{B} \\ \mathcal{C} & \mathcal{D} \end{pmatrix}, \quad \mathcal{A}_{ij} = \frac{\partial f_{ai}}{\partial a_j}, \quad i, j = 1, ..., K, \quad \mathcal{B}_{ij} = \frac{\partial f_{ai}}{\partial v_j}, \quad i = 1, ..., K,\ j = \sigma, 1, ..., M,$$

$$\mathcal{C}_{ij} = \frac{\partial f_i}{\partial a_j}, \quad i = \sigma, 1, ..., M,\ j = 1, ..., K, \quad \mathcal{D}_{ij} = \frac{\partial f_i}{\partial v_j}, \quad i = \sigma, 1, ..., M,\ j = \sigma, 1, ..., M. \tag{40}$$

For the partial derivatives of $f_{ai}$ we have

$$\frac{\partial f_{ai}}{\partial a_p} = \begin{cases} -\frac{1}{\tau_i(v)}, & i = p \\ 0, & i \neq p \end{cases}, \tag{41}$$

$$\frac{\partial f_{ai}}{\partial v_\sigma} = \frac{1}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + (a_{i,\infty} - a_i)\frac{\partial}{\partial v_\sigma}\left(\frac{1}{\tau_i}\right), \tag{42}$$

where $\partial f_{ai}/\partial v_\sigma = \frac{1}{\tau_i}\mathrm{d}a_{i,\infty}/\mathrm{d}v_\sigma$ when evaluated at equilibrium. Since the active variables depend only on the somatic voltage, $\partial f_{ai}/\partial v_k = 0$. In terms of the block matrices, this means that $\mathcal{A}$ is a diagonal matrix with

$$\mathcal{A}_{ii} = -\frac{1}{\tau_i}, \quad i = 1, ..., K, \tag{43}$$

while the matrix $\mathcal{B}$ only has one non-zero column

$$\mathcal{B}_{i\sigma} = \frac{1}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}, \quad B_{ij} = 0,\ j \neq \sigma. \tag{44}$$

For the somatic voltage equation, we define $\Lambda = \lambda^2/\Delta x^2$ and write the voltage derivatives as

$$\frac{\partial f_\sigma}{\partial v_\sigma} = \frac{\partial f_\mathrm{S}}{\partial v_\sigma} - \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}, \quad \frac{\partial f_\sigma}{\partial v_1} = \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}, \quad \frac{\partial f_\sigma}{\partial v_k} = 0, \quad k > 1. \tag{45}$$

Turning to the dendritic equations, since the dendrite is passive $\partial f_k/\partial a_i = 0$ for all $i, k$. For the voltage derivatives

$$\frac{\partial f_1}{\partial v_\sigma} = \frac{\Lambda}{\tau_\delta}, \quad \frac{\partial f_k}{\partial v_\sigma} = 0, \quad k \neq 1, \quad \frac{\partial f_k}{\partial v_j} = \begin{cases} \Lambda/\tau_\delta, & j = k \pm 1,\ k \neq M \\ -(1+2\Lambda)/\tau_\delta, & j = k \\ 0, & \text{otherwise} \end{cases}, \quad \frac{\partial f_M}{\partial v_{M-1}} = 2\Lambda/\tau_\delta, \tag{46}$$

which means that the matrix $\mathcal{C}$ only has one non-zero row

$$\mathcal{C}_{\sigma j} = \frac{\partial f_\sigma}{\partial a_j}, \quad \mathcal{C}_{ij} = 0, \quad i \neq \sigma, \tag{47}$$

while $\mathcal{D}$ is a tridiagonal matrix

$$\begin{gathered} \mathcal{D}_{\sigma\sigma} = \frac{\partial f_S}{\partial v_\sigma} - \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}, \quad \mathcal{D}_{\sigma 1} = \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}, \\ \mathcal{D}_{i,i\pm1} = \frac{\Lambda}{\tau_\delta}, \quad \mathcal{D}_{ii} = -\frac{(1+2\Lambda)}{\tau_\delta}, \quad i = 1, ..., M-1, \quad \mathcal{D}_{M,M-1} = \frac{2\Lambda}{\tau_\delta}, \quad \mathcal{D}_{MM} = -\frac{(1+2\Lambda)}{\tau_\delta}. \end{gathered} \tag{48}$$

## Bogdanov-Takens Bifurcation

At the Bogdanov-Takens bifurcation, the Jacobian has two zero eigenvalues. This means that $\boldsymbol{J}$ has left- and right-eigenvectors which not only satisfy $\boldsymbol{l}\boldsymbol{J} = \boldsymbol{0}$ and $\boldsymbol{J}\boldsymbol{r} = \boldsymbol{0}$ but also the orthogonality condition $\boldsymbol{l}\boldsymbol{r} = \boldsymbol{0}$. Denoting th elements of the right-eigenvector as $\boldsymbol{r} = (r_{a1}, ..., r_{aK}, r_\sigma, r_1, ..., r_M)$, the right-eigenvector equations at the BT bifurcation are

$$\begin{gathered} -\frac{r_{ai}}{\tau_i} + \frac{r_\sigma}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} = 0, \\ \sum_{i=1}^{K} r_{ai}\frac{\partial f_\sigma}{\partial a_i} + r_\sigma\left(\frac{\partial f_S}{\partial v_\sigma} - \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}\right) + r_1\frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma} = 0, \\ r_{k-1}\frac{\Lambda}{\tau_\delta} - r_k\frac{1+2\Lambda}{\tau_\delta} + r_{k+1}\frac{\Lambda}{\tau_\delta} = 0, \quad k = 1, ..., M-1, \\ 2r_{M-1}\frac{\Lambda}{\tau_\delta} - r_M\frac{1+2\Lambda}{\tau_\delta} = 0. \end{gathered} \tag{49}$$

The aim here is to rewrite all the elements of the eigenvector $\boldsymbol{r}$ in terms of $r_\sigma$, and we can immediately see that $r_{ai} = r_\sigma \mathrm{d}a_{i,\infty}/\mathrm{d}v_\sigma$. The dendritic elements meanwhile form a discrete difference equation (DDE)

$$r_{k-1}\Lambda - r_k(1+2\Lambda) + r_{k+1}\Lambda = 0, \quad r_0 = r_\sigma, \quad 2r_{M-1}\Lambda - r_M(1+2\Lambda) = 0, \tag{50}$$

which has the general solution

$$r_k = c_+\mu_+^k + c_-\mu_-^k, \quad \mu_\pm = \frac{1+2\Lambda \pm \sqrt{1+4\Lambda}}{2\Lambda}. \tag{51}$$

Utilising the relations $\mu_+ + \mu_- = (1+2\Lambda)/\Lambda$ and $\mu_+\mu_- = 1$, we can substitute in the boundary conditions to give the specific solution as

$$r_k = r_\sigma \frac{\mu_-^k \mu_+^{M-1}(1-\mu_+^2) - \mu_+^k \mu_-^{M-1}(1-\mu_-^2)}{\mu_+^{M-1}(1-\mu_+^2) - \mu_-^{M-1}(1-\mu_-^2)}, \tag{52}$$

and hence we have shown that all the right-eigenvector elements can be written in terms of $r_\sigma$. Meanwhile the left-eigenvector equations are

$$\begin{gathered}
-\frac{l_{ai}}{\tau_i} + l_\sigma \frac{\partial f_\sigma}{\partial a_i} = 0, \\
\sum_{i=1}^{K} \frac{l_{ai}}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + l_\sigma\left(\frac{\partial f_\mathrm{S}}{\partial v_\sigma} - \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}\right) + l_1\frac{\partial f_1}{\partial v_\sigma} = 0, \quad l_\sigma \frac{\rho G_\sigma\sqrt{\Lambda}}{C_\mathrm{m}} - l_1\frac{1+2\Lambda}{\tau_\delta} + l_2\frac{\Lambda}{\tau_\delta} = 0, \\
l_{k-1}\frac{\Lambda}{\tau_\delta} - l_k\frac{1+2\Lambda}{\tau_\delta} + l_{k+1}\frac{\Lambda}{\tau_\delta} = 0, \quad k = 2, ..., M-2, \\
l_{M-2}\frac{\Lambda}{\tau_\delta} - l_{M-1}\frac{1+2\Lambda}{\tau_\delta} + l_M\frac{2\Lambda}{\tau_\delta} = 0, \quad l_{M-1}\frac{\Lambda}{\tau_\delta} - l_M\frac{1+2\Lambda}{\tau_\delta} = 0,
\end{gathered} \tag{53}$$

where we can see that $l_{ai} = l_\sigma \tau_i \partial f_\sigma / \partial a_i$. The dendritic elements of $\boldsymbol{l}$ follow the same DDE as before, but with different boundary conditions. This has the solution in terms of $l_1$

$$l_k = l_1 \frac{\mu_-^{k-1}\mu_+^{M-2}(1-\mu_+^2) - \mu_+^{k-1}\mu_-^{M-2}(1-\mu_-^2)}{\mu_+^{M-2}(1-\mu_+^2) - \mu_-^{M-2}(1-\mu_-^2)}, \quad k = 1, ..., M-1, \tag{54}$$

where $\mu_\pm$ is given by (51) and $l_M = l_{M-1}\Lambda/(1+2\Lambda)$. The dendritic elements can be written in terms of $l_\sigma$ via

$$l_\sigma \frac{\tau_\delta \rho G_\sigma \sqrt{\Lambda}}{C_\sigma} = l_1(1+2\Lambda-\Lambda S), \quad S = l_{k=2}/l_1, \tag{55}$$

however $\rho$ is intrinsically linked to $g_\mathrm{in}$, our bifurcation parameter. We can write $\rho$ in terms of the somatic fixed-point voltage $v_\sigma^*$ from the somatic right-eigenvector equation

$$\begin{gathered}
\sum_{i=1}^{K} r_{ai}\frac{\partial f_\sigma}{\partial a_i} + r_\sigma \frac{\partial f_\mathrm{S}}{\partial v_\sigma} - \frac{\rho G_\sigma\sqrt{\Lambda}}{C_\sigma}(r_\sigma - r_1) = 0 \\
\rho(v_\sigma^*) = \frac{C_\sigma r_\sigma}{G_\sigma\sqrt{\Lambda}(r_\sigma - r_1)}\left(\frac{\partial f_\mathrm{S}}{\partial v_\sigma} + \sum_{i=1}^{K}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}\frac{\partial f_\sigma}{\partial a_i}\right).
\end{gathered} \tag{56}$$

This means that each term in our eigenvector product is proportional to $l_\sigma r_\sigma$

$$\begin{aligned}
\boldsymbol{lr} &= \sum_{i=1}^{K} l_{ai} r_{ai} + l_\sigma r_\sigma + \sum_{k=1}^{M} l_k r_k = 0 \\
&= 1 + \sum_{i=1}^{K}\left(\tau_i + \alpha_0\tau_\delta\right)\frac{\partial f_\sigma}{\partial a_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \alpha_0\tau_\delta\frac{\partial f_\mathrm{S}}{\partial v_\sigma},
\end{aligned} \tag{57}$$

$$\alpha_0 = \frac{r_\sigma \Sigma_\delta}{(r_\sigma - r_1)(1+2\Lambda-\Lambda S)}, \tag{58}$$

where $\Sigma_\delta$ represents the summation of the dendritic terms,

$$\Sigma_\delta = \sum_{k=1}^{M-1} \frac{\mu_-^{k-1}\mu_+^{M-2}(1-\mu_+^2) - \mu_+^{k-1}\mu_-^{M-2}(1-\mu_-^2)}{\mu_+^{M-2}(1-\mu_+^2) - \mu_-^{M-2}(1-\mu_-^2)} \times \frac{\mu_-^{k}\mu_+^{M-1}(1-\mu_+^2) - \mu_+^{k}\mu_-^{M-1}(1-\mu_-^2)}{\mu_+^{M-1}(1-\mu_+^2) - \mu_-^{M-1}(1-\mu_-^2)}$$
$$+ \frac{\Lambda}{1+2\Lambda} \frac{(1-\mu_+^2) - (1-\mu_-^2)}{\mu_+^{M-2}(1-\mu_+^2) - \mu_-^{M-2}(1-\mu_-^2)} \times \frac{\mu_-(1-\mu_+^2) - \mu_+(1-\mu_-^2)}{\mu_+^{M-1}(1-\mu_+^2) - \mu_-^{M-1}(1-\mu_-^2)}. \tag{59}$$

Hence for the *discrete* system, one can numerically solve the eigenvector product equation (57) to obtain $v_\sigma^{\mathrm{BT}}$ and then obtain $G_{\mathrm{in}}^{\mathrm{BT}}$ and $I_{\mathrm{ext}}^{\mathrm{BT}}$ from $I'_\infty = 0$ and $I_\infty = 0$ respectively. However, we can also take the continuum limit to obtain a BT equation for a spatially *continuous* system.

A semi-infinite cable attached to a soma can be analysed by taking the limit $M \to \infty$. Note that we will first consider the spatial step size to be still non-zero, $\Delta x > 0$, and later we will take the continuum limit $\Delta x \to 0$. With an infinite number of compartments, the DDEs for $r_k$ and $l_k$ simply greatly as the new distal boundary conditions are

$$\lim_{k\to\infty} |r_k| < \infty, \quad \lim_{k\to\infty} |l_k| < \infty. \tag{60}$$

From our earlier definition of $\mu_\pm$, for $\Lambda > 0$ $|\mu_+| > 1$ and $|\mu_-| < 1$. This for both $l_k$ and $r_k$ the solutions are

$$r_k = r_\sigma \mu_-^k, \quad l_k = l_1 \mu_-^{k-1}, \quad k = 1, 2, \ldots. \tag{61}$$

This means that the summation of dendritic terms is

$$\Sigma_\delta = \sum_{k=1}^{\infty} \mu_-^{2k-1} = \frac{\mu_-}{1-\mu_-^2}, \tag{62}$$

and hence the factor $\alpha_0$ is

$$\alpha_0 = \frac{\mu_-}{(1-\mu_-)(1+2\Lambda-\Lambda\mu_-)(1-\mu_-^2)}. \tag{63}$$

Now we take the continuum limit $\Delta x \to 0$ which in this case is equivalent to $\Lambda \to \infty$. $\alpha_0$ is the only constant that depends on $\Lambda$, and has the limit

$$\lim_{\Lambda\to\infty} \alpha_0 = \frac{1}{2}. \tag{64}$$

Substituting $\alpha_0$ into the BT condition thus gives

$$\sum_{i=1}^{K} \left(\frac{\tau_\delta}{2} + \tau_i\right) \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + 1 + \frac{\tau_\delta}{2} \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} = 0. \tag{65}$$

For the finite cable, after resolving the summation (59), we define $\Delta z = \Delta x/\lambda$. This allows $M$ to be rewritten as $M = \ell/\Delta z$ and $\Lambda = \Delta z^{-2}$. After much algebra, taking the limit $\Delta z \to 0$ gives for $\alpha_0$

$$\lim_{\Delta z\to 0} \alpha_0 = \frac{1}{2} + \frac{\ell}{2\cosh\ell\,\sinh\ell}. \tag{66}$$

## Hopf Bifurcation

At the Hopf bifurcation, there exist right-eigenvectors of the Jacobian $\boldsymbol{J}$ which satisfy $\boldsymbol{J}\boldsymbol{q} = i\omega_{\mathrm{H}}\boldsymbol{q}$ and $\boldsymbol{J}\overline{\boldsymbol{q}} = -i\omega_{\mathrm{H}}\overline{\boldsymbol{q}}$. We can use the discretised Jacobian outlined earlier to obtain an expressing for the somatic voltage at the Hopf bifurcation, $v_\sigma^{\mathrm{H}}$. For the right eigenvectors we will use the indexing $\boldsymbol{q} = (q_{a1}, ..., q_{aK}, q_\sigma, q_1, ..., q_M)$, and hence our right-eigenvector equations are

$$-\frac{q_{ai}}{\tau_i} + \frac{q_\sigma}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} = i\omega_{\mathrm{H}} q_{ai}, \quad \sum_{i=1}^{K} q_{ai}\frac{\partial f_\sigma}{\partial a_i} + q_\sigma\left(\frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\sqrt{\Lambda}\rho G_\sigma}{C_\sigma}\right) + q_1\frac{\partial f_\sigma}{\partial v_1} = i\omega_{\mathrm{H}} q_\sigma,$$

$$q_{k-1}\frac{\Lambda}{\tau_\delta} - q_k\frac{1+2\Lambda}{\tau_\delta} + q_{k+1}\frac{\Lambda}{\tau_\delta} = i\omega_{\mathrm{H}} q_k, \quad k = 1, ..., M-1, \quad 2q_{M-1}\frac{\Lambda}{\tau_\delta} - q_M\frac{1+2\Lambda}{\tau_\delta} = i\omega q_M. \tag{67}$$

We see that the dendritic elements follow a DDE equation similar to the one for the BT bifurcation. We rewrite the DDE and its boundary conditions as

$$q_{k-1}\Lambda - q_k(1 + 2\Lambda + i\omega_{\mathrm{H}}\tau_\delta) + q_{k+1}\Lambda = 0, \quad q_0 = q_\sigma,$$

$$2q_{M-1}\Lambda - q_M(1 + 2\Lambda + i\omega_{\mathrm{H}}\tau_\delta) = 0, \tag{68}$$

where the DDE has a general solution in terms of the bases $\eta_\pm$, a complex analogue of $\mu_\pm$

$$q_k = c_+\eta_+^k + c_-\eta_-^k, \quad \eta_\pm = \frac{1 + 2\Lambda + i\omega_{\mathrm{H}}\tau_\delta \pm \sqrt{(1 + 2\Lambda + i\omega_{\mathrm{H}}\tau_\delta)^2 - 4\Lambda^2}}{2\Lambda}. \tag{69}$$

We use the boundary conditions to find $c_\pm$ and hence the specific solution to $q_k$

$$q_k = q_\sigma\frac{\eta_-^k\eta_+^{M-1}(1-\eta_+^2) - \eta_+^k\eta_-^{M-1}(1-\eta_-^2)}{\eta_+^{M-1}(1-\eta_+^2) - \eta_-^{M-1}(1-\eta_-^2)}. \tag{70}$$

Defining $Q(n) = \eta_+^n(1-\eta_+^2) - \eta_-^n(1-\eta_-^2)$, we can substitute $q_1$ and $q_{ai}$ into the equation for $q_\sigma$ to yield

$$\sum_{i=1}^{K}\frac{1 - i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2}\frac{\partial f_\sigma}{\partial a_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \frac{\rho G_\sigma\sqrt{\Lambda}}{C_\sigma}\left[\frac{Q(M-2)}{Q(M-1)} - 1\right] - i\omega_{\mathrm{H}} = 0. \tag{71}$$

Turning back to the eigenvector $\overline{\boldsymbol{q}}$, we obtain the same form of specific solution for the dendritic eigenvector elements, but this time in terms of $\overline{\eta}_\pm$

$$\overline{\eta}_\pm = \frac{1 + 2\Lambda - i\omega_{\mathrm{H}}\tau_\delta \pm \sqrt{(1 + 2\Lambda - i\omega_{\mathrm{H}}\tau_\delta)^2 - 4\Lambda^2}}{2\Lambda}. \tag{72}$$

This means that the same substitutions into the equation for $\overline{q}_\sigma$ gives

$$\sum_{i=1}^{K}\frac{1 + i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2}\frac{\partial f_\sigma}{\partial a_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \frac{\rho G_\sigma\sqrt{\Lambda}}{C_\sigma}\left[\frac{\overline{Q}(M-2)}{\overline{Q}(M-1)} - 1\right] + i\omega_{\mathrm{H}} = 0, \tag{73}$$

where $\overline{Q}(n)$ has the same definition as $Q$, but in terms of $\overline{\eta}_\pm$.

For the semi-infinite dendrite first we take $M \to \infty$, which has the effect of simplifying the specific solution of the DDEs to

$$q_k = q_\sigma\eta_-^k, \quad \overline{q}_k = q_\sigma\overline{\eta}_-^k. \tag{74}$$

The equations for $q_\sigma$ and $\overline{q}_\sigma$ are hence simplified to

$$\sum_{i=1}^{K} \frac{1 - i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \frac{\rho G_\sigma \sqrt{\Lambda}}{C_\sigma}(\eta_- - 1) - i\omega_{\mathrm{H}} = 0, \tag{75}$$

$$\sum_{i=1}^{K} \frac{1 + i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \frac{\rho G_\sigma \sqrt{\Lambda}}{C_\sigma}(\overline{\eta}_- - 1) + i\omega_{\mathrm{H}} = 0. \tag{76}$$

Now we take the continuum limit ($\Lambda \to \infty$) of the only term that varies with $\Lambda$

$$\lim_{\Lambda\to\infty} \sqrt{\Lambda}(\eta_- - 1) = -\frac{\sqrt{4\Lambda + 4i\omega_{\mathrm{H}}\tau_\delta\Lambda}}{2\sqrt{\Lambda}} = -\sqrt{1 + i\omega_{\mathrm{H}}\tau_\delta} = -\gamma(\omega_{\mathrm{H}}). \tag{77}$$

We similarly find that

$$\lim_{\Lambda\to\infty} \sqrt{\Lambda}(\overline{\eta} - 1) = -\sqrt{1 - i\omega_{\mathrm{H}}\tau_\delta} = -\gamma(-\omega_{\mathrm{H}}). \tag{78}$$

It can be shown that $\gamma(\omega_{\mathrm{H}})$ and $\gamma(-\omega_{\mathrm{H}})$ are complex conjugates with a sum and difference given by

$$\gamma(\omega_{\mathrm{H}}) + \gamma(-\omega_{\mathrm{H}}) := s_\gamma(\omega_{\mathrm{H}}) = \sqrt{2 + 2\sqrt{1 + \omega_{\mathrm{H}}^2\tau_\delta^2}}, \tag{79}$$

$$\gamma(\omega_{\mathrm{H}}) - \gamma(-\omega_{\mathrm{H}}) := i d_\gamma(\omega_{\mathrm{H}}) = i\,\mathrm{sgn}(\omega_{\mathrm{H}})\sqrt{-2 + 2\sqrt{1 + \omega_{\mathrm{H}}^2\tau_\delta^2}}. \tag{80}$$

This means that summing (75) and (76) in the continuum limit gives the real-valued equation

$$\sum_{i=1}^{K} \frac{1}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\rho G_\sigma}{2C_\sigma} s_\gamma(\omega_{\mathrm{H}}) = 0, \tag{81}$$

while subtracting (75) from (76) gives an equation in terms of the imaginary part

$$\sum_{i=1}^{K} \frac{\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\rho G_\sigma}{2C_\sigma} \frac{d_\gamma(\omega_{\mathrm{H}})}{\omega_{\mathrm{H}}} + 1 = 0. \tag{82}$$

These nonlinear equations can be solved simultaneously to find $\omega_{\mathrm{H}}$ and $v_\sigma^{\mathrm{H}}$.

To find the finite continuum limit solution for the Hopf bifurcation, we first find the continuum limits of $\sqrt{\Lambda}[Q(M-2)/Q(M-1) - 1]$ and $\sqrt{\Lambda}[\overline{Q}(M-2)/\overline{Q}(M-1) - 1]$ in (71) and (73) by substituting $\Lambda = \Delta z^{-2}$ and $M = l/\Delta z$ as in the BT section. After doing so, the first Hopf equation (71) becomes

$$\sum_{i=1}^{K} \frac{1 - i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\rho G_\sigma}{C_\sigma}\gamma(\omega_{\mathrm{H}})\tanh[l\gamma(\omega_{\mathrm{H}})] - i\omega_{\mathrm{H}} = 0, \tag{83}$$

and the continuum-limit of the second Hopf equation (73) is

$$\sum_{i=1}^{K} \frac{1 + i\omega_{\mathrm{H}}\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\rho G_\sigma}{C_\sigma}\gamma(-\omega_{\mathrm{H}})\tanh[l\gamma(-\omega_{\mathrm{H}})] + i\omega_{\mathrm{H}} = 0. \tag{84}$$

Now when we sum and difference (83) and (84) we get equations for the real and imaginary parts

$$\sum_{i=1}^{K} \frac{1}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\partial v_\sigma} + \frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} - \frac{\rho G_\sigma}{2C_\sigma} \frac{s_\gamma \sinh(l s_\gamma) - d_\gamma \sin(l d_\gamma)}{\cosh(l s_\gamma) + \cos(l d_\gamma)} = 0, \tag{85}$$

$$\sum_{i=1}^{K} \frac{\tau_i}{1 + \omega_{\mathrm{H}}^2\tau_i^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + \frac{\rho G_\sigma}{2C_\sigma} \frac{d_\gamma \sinh(l s_\gamma) + s_\gamma \sin(l d_\gamma)}{\omega_{\mathrm{H}}[\cosh(l s_\gamma) + \cos(l d_\gamma)]} + 1 = 0. \tag{86}$$

These two nonlinear equations must be solved simultaneously. In this case, we used a trust-region method implemented by the NLsolve.jl package ***NLs*** (***2020***).

## Criticality of the Hopf Bifurcation

The criticality of the Hopf bifurcation can be obtained from the first Lyapunov coefficient of the equilibrium that undergoes the Hopf bifurcation

$$\mathcal{L}_1 = \frac{1}{2\omega_{\mathrm{H}}} \mathrm{Re}\left\{\overline{\boldsymbol{p}}\boldsymbol{C}(\boldsymbol{q},\boldsymbol{q},\overline{\boldsymbol{q}}) - 2\overline{\boldsymbol{p}}\boldsymbol{B}[\boldsymbol{q},\boldsymbol{J}^{-1}\boldsymbol{B}(\boldsymbol{q},\overline{\boldsymbol{q}})] + \overline{\boldsymbol{p}}\boldsymbol{B}[\overline{\boldsymbol{q}},(2i\omega_{\mathrm{H}}\boldsymbol{I}-\boldsymbol{J})^{-1}\boldsymbol{B}(\boldsymbol{q},\boldsymbol{q})]\right\}, \tag{87}$$

where $\boldsymbol{q}$ is the right-eigenvector of $\boldsymbol{J}$ at the Hopf bifurcation found earlier, $\boldsymbol{p}$ is the left-eigenvector of $\boldsymbol{J}$, and $\boldsymbol{B}$ and $\boldsymbol{C}$ are the second and third order tensors of the system. When $\mathcal{L}_1 < 0$, the Hopf bifurcation is supercritical, while when $\mathcal{L}_1 > 0$ it is subcritical. The left-eigenvectors satisfy $\boldsymbol{p}\boldsymbol{J} = -i\omega_{\mathrm{H}}\boldsymbol{p}$ and $\overline{\boldsymbol{p}}\boldsymbol{J} = i\omega_{\mathrm{H}}\overline{\boldsymbol{p}}$ while the $i$th element of each evaluated tensor is

$$B_i(\boldsymbol{x},\boldsymbol{y}) = \sum_{j,k} \frac{\partial^2 f_i}{\partial\xi_j\partial\xi_k} x_j y_k, \quad C_i(\boldsymbol{x},\boldsymbol{y},\boldsymbol{z}) = \sum_{j,k,l} \frac{\partial^3 f_i}{\partial\xi_j\partial\xi_k\partial\xi_l} x_j y_k z_l. \tag{88}$$

Due to the linearity of the passive dendrite, the dendritic terms of $B_i$ and $C_i$ will be zero. Thus, we only need calculate the $K+1$ terms for each active value and the somatic voltage, which use the eigenvector elements $q_i, ..., q_K, q_\sigma$. Using the normalisation $\overline{\boldsymbol{p}}\boldsymbol{q} = 1$, we can write all the eigenvector elements of $\boldsymbol{p}$ and $\boldsymbol{q}$ in terms of $q_\sigma$ in the continuum limit

$$\overline{p}_\sigma = \frac{q_\sigma}{\kappa}, \quad \kappa = \sum_{i=1}^{K} \frac{\tau_i}{(1+i\omega_{\mathrm{H}}\tau_i)^2} \frac{\partial f_\sigma}{\partial a_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma} + 1 + \frac{\tau_\delta \rho g_\sigma}{C_\sigma} \frac{(e^{2\ell\gamma} - e^{-2\ell\gamma} + 4\ell\gamma)}{2\gamma(e^{\ell\gamma} + e^{-\ell\gamma})^2}, \tag{89}$$

$$\overline{p}_{ai} = \frac{\overline{p}_\sigma \tau_i}{1+i\omega_{\mathrm{H}}\tau_i} \frac{\partial f_\sigma}{\partial a_i}, \quad q_{ai} = \frac{q_\sigma}{1+i\omega_{\mathrm{H}}\tau_i} \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}. \tag{90}$$

For the terms involving inverse matrices, elements required for calculation of $\boldsymbol{B}$ also have continuum limits. Denoting $\boldsymbol{u} = \boldsymbol{J}^{-1}\boldsymbol{B}(\boldsymbol{q},\overline{\boldsymbol{q}})$, we require the elements $u_{a1}, ..., u_{aK}, u_\sigma$. Since only the first $K+1$ elements of $\boldsymbol{B}$ are non-zero, this means only the calculation of $(K+1)\times(K+1)$ elements of $\boldsymbol{J}^{-1}$ is necessary. The block structure of $\boldsymbol{J}$ defined earlier (39) means that its inverse elements can be found in terms of the blocks

$$\boldsymbol{J} = \begin{pmatrix} \mathcal{A} & \mathcal{B} \\ \mathcal{C} & \mathcal{D} \end{pmatrix}, \quad \boldsymbol{J}^{-1} = \begin{pmatrix} \mathcal{A}^{-1} + \mathcal{A}^{-1}\mathcal{B}\mathcal{S}^{-1}\mathcal{C}\mathcal{A}^{-1} & -\mathcal{A}^{-1}\mathcal{B}\mathcal{S}^{-1} \\ -\mathcal{S}^{-1}\mathcal{C}\mathcal{A}^{-1} & \mathcal{S}^{-1} \end{pmatrix}. \tag{91}$$

Here $\mathcal{S} = \mathcal{D} - \mathcal{C}\mathcal{A}^{-1}\mathcal{B}$ is the Schur complement. Using this approach, the upper-left $(K+1)\times(K+1)$ quadrant of $\boldsymbol{J}^{-1}$ can be found as

$$[\boldsymbol{J}^{-1}]_{ij} = \begin{cases} -\tau_i + [\mathcal{S}^{-1}]_{11}\tau_i \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}\frac{\partial f_\sigma}{\partial a_i}, & i = j, \quad i,j \neq \sigma \\ [\mathcal{S}^{-1}]_{11}\tau_j \frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}\frac{\partial f_\sigma}{\partial a_j}, & i \neq j, \quad i,j \neq \sigma \\ [\mathcal{S}^{-1}]_{11}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}, & i \neq \sigma, \quad j = \sigma \\ [\mathcal{S}^{-1}]_{11}\tau_j \frac{\partial f_\sigma}{\partial a_j}, & i = \sigma, \quad j \neq \sigma \\ [\mathcal{S}^{-1}]_{11}, & i = j = \sigma \end{cases}, \tag{92}$$

where in the continuum limit $[\mathcal{S}^{-1}]_{11}$ is the inverse of the total derivative of the somatic voltage equation with respect to $v_\sigma$

$$[\mathcal{S}^{-1}]_{11} = \left(\frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \sum_{i=1}^{K}\tau_i \frac{\partial f_{ai}}{\partial v_\sigma}\frac{\partial f_\sigma}{\partial a_i} - \frac{\rho G_\sigma}{C_\sigma}\tanh\ell\right)^{-1}. \tag{93}$$

The $(K+1)\times(K+1)$ upper-left quadrant can be calculated for the inverse of $\tilde{\boldsymbol{J}} = \boldsymbol{J} - 2i\omega_{\mathrm{H}}\boldsymbol{I}$ in a similar manner, yielding

$$[\tilde{\boldsymbol{J}}^{-1}]_{ij} = \begin{cases} -\tilde{\tau}_i + [\tilde{\mathcal{S}}^{-1}]_{11}\frac{\tilde{\tau}_i^2}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}\frac{\partial f_\sigma}{\partial a_i}, & i = j, \quad i,j \neq \sigma \\ [\tilde{\mathcal{S}}^{-1}]_{11}\frac{\tilde{\tau}_i\tilde{\tau}_j}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}\frac{\partial f_\sigma}{\partial a_j}, & i \neq j, \quad i,j \neq \sigma \\ [\tilde{\mathcal{S}}^{-1}]_{11}\frac{\tilde{\tau}_i}{\tau_i}\frac{\mathrm{d}a_{i,\infty}}{\mathrm{d}v_\sigma}, & i \neq \sigma, \quad j = \sigma \\ [\tilde{\mathcal{S}}^{-1}]_{11}\tilde{\tau}_j\frac{\partial f_\sigma}{\partial a_j}, & i = \sigma, \quad j \neq \sigma \\ [\tilde{\mathcal{S}}^{-1}]_{11}, & i = j = \sigma \end{cases}, \tag{94}$$

where $\tilde{\tau}_i = \tau_i/(1 + 2i\omega_{\mathrm{H}}\tau_i)$ and

$$[\tilde{\mathcal{S}}^{-1}]_{11} = \left\{\frac{\partial f_{\mathrm{S}}}{\partial v_\sigma} + \sum_{i=1}^{K}\tilde{\tau}_i\frac{\partial f_\sigma}{\partial a_i}\frac{\partial f_{ai}}{\partial v_\sigma} - 2i\omega_{\mathrm{H}} - \frac{\rho G_\sigma}{C_\sigma}\gamma(2\omega_{\mathrm{H}})\tanh[\ell\gamma(2\omega_{\mathrm{H}})]\right\}^{-1}, \tag{95}$$

with $\gamma$ having the same form as before. This provides us with all the information required to calculate the criticality of a Hopf bifurcation in the continuum limit.

## Dendritic Current Input

Moving the external current input to an arbitrary dendritic location $x_{\mathrm{in}}$ changes the cable equation to

$$\tau_\delta\frac{\partial v_\delta}{\partial t} = E_L - v_\delta + \lambda^2\frac{\partial^2 v_\delta}{\partial x^2} + \frac{\lambda I_{\mathrm{ext}}}{\rho G_\sigma}\delta(x - x_{\mathrm{in}}), \tag{96}$$

while the somatic equation remains as in (13) with $I_{\text{ext}} = 0$ at the soma. Setting all time derivatives to zero, the dendritic equilibrium voltage for the semi-infinite dendrite satisfies

$$v_\delta^*(x) = E_L + (v_\sigma^* - E_L)e^{-x/\lambda} + [e^{-|x-x_{\text{in}}|/\lambda} - e^{-|x-x_{\text{in}}|/\lambda}], \tag{97}$$

which means that the somatic fixed point potential is found by solving

$$I_\infty(v_\sigma) = C_\sigma f_{\text{S}}(\boldsymbol{a}_\infty(v_\sigma), v_\sigma) + \rho G_\sigma(E_L - v_\sigma) + I_{\text{ext}} e^{-x_{\text{in}}/\lambda} = 0. \tag{98}$$

Meanwhile for finite dendrite, the dendritic equilibrium voltage with $z = x/\lambda$ obeys

$$v_\delta^*(x) = E_L + (v_\sigma - E_L)\cosh z + \left\{ \frac{I_{\text{ext}}}{\rho G_\sigma} \frac{\cosh(\ell - z)}{\cosh \ell} - (v_\sigma - E_L)\tanh \ell \right\} \sinh z - \theta(z - z_{\text{in}}) \frac{I_{\text{ext}}}{\rho G_\sigma} \sinh(z - z_{\text{in}}), \tag{99}$$

which when substituted into the somatic boundary condition yields

$$I_\infty(v_\sigma) = C_\sigma f_{\text{S}}(\boldsymbol{a}_\infty(v_\sigma), v_\sigma) + \rho G_\sigma \tanh \ell (E_L - v_\sigma) + I_{\text{ext}} \frac{\cosh(\ell - z_{\text{in}})}{\cosh \ell} = 0. \tag{100}$$

In both the semi-infinite and finite models, the only term of the steady-state current equations affected by the spatial separation between the soma at point of current injection, $x_{\text{in}}$, is the external current term. The steady-state current equations converge to the somatically driven case when $x_{\text{in}} = 0$ as expected.

This means that for the saddle-node and cusp bifurcations, differentiating (98) or (100) with respect to $v_\sigma$ removes all dependence on the location of synaptic input. Therefore, for the same values of $\tau_\delta$ and $\rho$, $v_\sigma^{\text{SN}}$ does not vary with $x_{\text{in}}$ and has the same value as when external current is applied at the soma. Similarly, for the same $\tau_\delta$, $\rho^{\text{C}}$ and $v^{\text{C}}$ do not depend on $x_{\text{in}}$. $I_{\text{ext}}^{\text{SN}}$ and $I_{\text{ext}}^{\text{C}}$ are the only parameters altered by the spatial location.

For the BT and Hopf bifurcations, we must discretise the cable equation and perform the method of lines. However, since the external dendritic drive does not depend on any of the system variables, the discretised Jacobian $\boldsymbol{J}$ will not depend $x_{\text{in}}$ and will thus be identical to the somatically driven scenario. Since the bifurcation parameters for the BT and Hopf bifurcations are derived from the eigenvectors of $\boldsymbol{J}$, this means that they have values which do not vary with $x_{\text{in}}$. Only $I_{\text{ext}}$ varies with $x_{\text{in}}$, as was the case for the saddle-node and cusp bifurcations.

## References

(2020). Julianlsolvers/nlsolve.jl: v4.5.1 (v4.5.1).

Al-Darabsah, I. and Campbell, S. A. (2021). M-current induced bogdanov–takens bifurcation and switching of neuron excitability class. *The Journal of Mathematical Neuroscience*, 11(1):1–26.

Allgower, E. L. and Georg, K. (2012). *Numerical continuation methods: an introduction*, volume 13. Springer Science & Business Media.

Amsalem, O., Eyal, G., Rogozinski, N., Gevaert, M., Kumbhar, P., Schürmann, F., and Segev, I. (2020). An efficient analytical reduction of detailed nonlinear neuron models. *Nature communications*, 11(1):1–13.

Ascoli, G. A., Donohue, D. E., and Halavi, M. (2007). Neuromorpho. org: a central resource for neuronal morphologies. *Journal of Neuroscience*, 27(35):9247–9251.

Ashwin, P., Bick, C., and Burylko, O. (2016). Identical phase oscillator networks: Bifurcations, symmetry and reversibility for generalized coupling. *Frontiers in Applied Mathematics and Statistics*, 2:7.

Beaulieu-Laroche, L., Toloza, E. H., Van der Goes, M.-S., Lafourcade, M., Barnagian, D., Williams, Z. M., Eskandar, E. N., Frosch, M. P., Cash, S. S., and Harnett, M. T. (2018). Enhanced dendritic compartmentalization in human cortical neurons. *Cell*, 175(3):643–651.

Behabadi, B. F., Polsky, A., Jadi, M., Schiller, J., and Mel, B. W. (2012). Location-dependent excitatory synaptic interactions in pyramidal neuron dendrites. *PLoS computational biology*, 8(7):e1002599.

Beniaguev, D., Shapira, S., Segev, I., and London, M. (2022). Multiple synaptic contacts combined with dendritic filtering enhance spatio-temporal pattern recognition capabilities of single neurons. *bioRxiv*.

Bergman, H. and Deuschl, G. (2002). Pathophysiology of Parkinson's disease: From clinical neurology to basic neuroscience and back. *Movement Disorders*, 17(S3):S28–S40.

Bezanson, J., Edelman, A., Karpinski, S., and Shah, V. B. (2017). Julia: A fresh approach to numerical computing. *SIAM review*, 59(1):65–98.

Bindman, L., Meyer, T., and Prince, C. (1988). Comparison of the electrical properties of neocortical neurones in slices in vitro and in the anaesthetized rat. *Experimental brain research*, 69(3):489–496.

Brown, E., Moehlis, J., and Holmes, P. (2004). On the phase reduction and response dynamics of neural oscillator populations. *Neural computation*, 16(4):673–715.

Contreras, S. A., Schleimer, J.-H., Gulledge, A. T., and Schreiber, S. (2021). Activity-mediated accumulation of potassium induces a switch in firing pattern and neuronal excitability type. *PLoS Computational Biology*, 17(5):e1008510.

Coombes, S., Timofeeva, Y., Svensson, C.-M., Lord, G. J., Josić, K., Cox, S. J., and Colbert, C. M. (2007). Branching dendrites with resonant membrane: a "sum-over-trips" approach. *Biological Cybernetics*, 97(2):137–149.

Crook, S. M., Ermentrout, G. B., and Bower, J. M. (1998). Dendritic and synaptic effects in systems of coupled cortical oscillators. *Journal of computational neuroscience*, 5(3):315–329.

De Maesschalck, P. and Wechselberger, M. (2015). Neural excitability and singular bifurcations. *The Journal of Mathematical Neuroscience (JMN)*, 5(1):1–32.

Depannemaecker, D., Ivanov, A., Lillo, D., Spek, L., Bernard, C., and Jirsa, V. (2022). A unified physiological framework of transitions between seizures, sustained ictal activity and depolarization block at the single neuron level. *Journal of computational neuroscience*, 50(1):33–49.

Destexhe, A. and Paré, D. (1999). Impact of network activity on the integrative properties of neocortical pyramidal neurons in vivo. *Journal of neurophysiology*, 81(4):1531–1547.

Doedel, E. J., Champneys, A. R., Fairgrieve, T., Kuznetsov, Y., Oldeman, B., Paffenroth, R., Sandstede, B., Wang, X., and Zhang, C. (2007). Auto-07p: Continuation and bifurcation software for ordinary differential equations (2007). *Available for download from http://indy. cs. concordia. ca/auto*.

Ermentrout, B. (1996). Type i membranes, phase resetting curves, and synchrony. *Neural computation*, 8(5):979–1001.

Eyal, G., Mansvelder, H. D., de Kock, C. P., and Segev, I. (2014). Dendrites impact the encoding capabilities of the axon. *Journal of Neuroscience*, 34(24):8063–8071.

Eyal, G., Verhoog, M. B., Testa-Silva, G., Deitcher, Y., Lodder, J. C., Benavides-Piccione, R., Morales, J., DeFelipe, J., de Kock, C. P., Mansvelder, H. D., et al. (2016). Unique membrane properties and enhanced signal processing in human neocortical neurons. *Elife*, 5:e16553.

Gidon, A., Zolnik, T. A., Fidzinski, P., Bolduan, F., Papoutsi, A., Poirazi, P., Holtkamp, M., Vida, I., and Larkum, M. E. (2020). Dendritic action potentials and computation in human layer 2/3 cortical neurons. *Science*, 367(6473):83–87.

Goethals, S. and Brette, R. (2020). Theoretical relation between axon initial segment geometry and excitability. *Elife*, 9:e53432.

Goldberg, J. A., Deister, C. A., and Wilson, C. J. (2007). Response Properties and Synchronization of Rhythmically Firing Dendritic Neurons. *Journal of Neurophysiology*, 97(1):208–219.

Grienberger, C., Chen, X., and Konnerth, A. (2014). Nmda receptor-dependent multidendrite ca2+ spikes required for hippocampal burst firing in vivo. *Neuron*, 81(6):1274–1281.

Guckenheimer, J. and Labouriau, J. (1993). Bifurcation of the hodgkin and huxley equations: a new twist. *Bulletin of Mathematical Biology*, 55(5):937–952.

Hamada, M. S., Goethals, S., Vries, S. I. D., Brette, R., and Kole, M. H. P. (2016). Covariation of axon initial segment location and dendritic tree normalizes the somatic action potential. *PNAS*, 113(51).

Hansel, D., Mato, G., and Meunier, C. (1995). Synchrony in excitatory neural networks. *Neural computation*, 7(2):307–337.

Häusser, M. and Clark, B. A. (1997). Tonic synaptic inhibition modulates neuronal output pattern and spatiotemporal synaptic integration. *Neuron*, 19(3):665–678.

Hesse, J., Schleimer, J.-H., Maier, N., Schmitz, D., and Schreiber, S. (2022). Temperature elevations can induce switches to homoclinic action potentials that alter neural encoding and synchronization. *Nature Communications*, 13(1):1–12.

Hesse, J., Schleimer, J.-H., and Schreiber, S. (2017). Qualitative changes in phase-response curve and synchronization at the saddle-node-loop bifurcation. *Physical Review E*, 95(5):052203.

Hodgkin, A. L. (1948). The local electric changes associated with repetitive action in a non-medullated axon. *The Journal of physiology*, 107(2):165.

Hodgkin, A. L. and Huxley, A. F. (1952). A quantitative description of membrane current and its application to conduction and excitation in nerve. *The Journal of Physiology*, 117(4):500–544.

Holmes, W. R. and Woody, C. D. (1989). Effects of uniform and non-uniform synaptic 'activation-distributions' on the cable properties of modeled cortical pyramidal neurons. *Brain research*, 505(1):12–22.

Hürkey, S., Niemeyer, N., Schleimer, J.-H., Ryglewski, S., Schreiber, S., and Duch, C. (2022). Insect asynchronous flight requires neural circuit de-synchronization by electrical synapses. *bioRxiv*.

Izhikevich, E. M. (2007a). *Bifurcations*. MIT press.

Izhikevich, E. M. (2007b). Synchronization. In *Dynamical systems in neuroscience*, chapter 10, pages 443–505. MIT press.

Jiruska, P., De Curtis, M., Jefferys, J. G., Schevon, C. A., Schiff, S. J., and Schindler, K. (2013). Synchronization and desynchronization in epilepsy: controversies and hypotheses. *The Journal of physiology*, 591(4):787–797.

Kirst, C. (2011). Synchronization, neuronal excitability, and information flow in networks of neuronal oscillators. chapter 10. Georg-August-Universität Göttingen.

Kirst, C., Ammer, J., Felmy, F., Herz, A., and Stemmler, M. (2017). Gaba regulates resonance and spike rate encoding via a universal mechanism that underlies the modulation of action potential generation. *bioRxiv*, page 206581.

Ko, T.-W. and Ermentrout, G. B. (2009). Phase-response curves of coupled oscillators. *Physical Review E*, 79(1):016211.

Koch, C. (1984). Cable theory in neurons with active, linearized membranes. *Biological Cybernetics*, 50(1):15–33.

Kole, M. H. P. and Stuart, G. J. (2012). Signal Processing in the Axon Initial Segment. *Neuron*, 73(2):235–247.

Kuznetsov, Y. A. (2013). Elements of applied bifurcation theory. volume 112, chapter 8, pages 293–392. Springer Science & Business Media.

Larkman, A. and Mason, A. (1990). Correlations between morphology and electrophysiology of pyramidal neurons in slices of rat visual cortex. i. establishment of cell classes. *Journal of Neuroscience*, 10(5):1407–1414.

Larkum, M. E., Wu, J., Duverdin, S. A., and Gidon, A. (2022). The guide to dendritic spikes of the mammalian cortex in vitro and in vivo. *Neuroscience*.

Larkum, M. E., Zhu, J. J., and Sakmann, B. (1999). A new cellular mechanism for coupling inputs arriving at different cortical layers. *Nature*, 398(6725):338–341.

Liu, C., Liu, X., and Liu, S. (2014). Bifurcation analysis of a morris–lecar neuron model. *Biological cybernetics*, 108(1):75–84.

Mainen, Z. F. and Sejnowski, T. J. (1996). Influence of dendritic structure on firing pattern in model neocortical neurons. *Nature*, 382(6589):363–366.

Major, G., Evans, J. D., and Jack, J. (1993). Solutions for transients in arbitrarily branching cables: I. voltage recording with a somatic shunt. *Biophysical journal*, 65(1):423–449.

Mohan, H., Verhoog, M. B., Doreswamy, K. K., Eyal, G., Aardse, R., Lodder, B. N., Goriounova, N. A., Asamoah, B., B. Brakspear, A. C., Groot, C., et al. (2015). Dendritic and axonal architecture of individual pyramidal neurons across layers of adult human neocortex. *Cerebral Cortex*, 25(12):4839–4853.

Morris, C. and Lecar, H. (1981). Voltage oscillations in the barnacle giant muscle fiber. *Biophysical journal*, 35(1):193–213.

Paré, D., Shink, E., Gaudreau, H., Destexhe, A., and Lang, E. J. (1998). Impact of spontaneous synaptic activity on the resting properties of cat neocortical pyramidal neurons in vivo. *Journal of neurophysiology*, 79(3):1450–1460.

Pereira, U., Coullet, P., and Tirapegui, E. (2015). The bogdanov–takens normal form: a minimal model for single neuron dynamics. *Entropy*, 17(12):7859–7874.

Phoka, E., Cuntz, H., Roth, A., and Häusser, M. (2010). A new approach for determining phase response curves reveals that purkinje cells can act as perfect integrators. *PLoS computational biology*, 6(4):e1000768.

Prescott, S. A., De Koninck, Y., and Sejnowski, T. J. (2008a). Biophysical basis for three distinct dynamical mechanisms of action potential initiation. *PLoS computational biology*, 4(10):e1000198.

Prescott, S. A., Ratté, S., De Koninck, Y., and Sejnowski, T. J. (2008b). Pyramidal neurons switch from integrators in vitro to resonators under in vivo-like conditions. *Journal of neurophysiology*, 100(6):3030–3042.

Rackauckas, C. and Nie, Q. (2017). Differentialequations.jl–a performant and feature-rich ecosystem for solving differential equations in julia. *Journal of Open Research Software*, 5(1).

Rall, W. (1960). Membrane potential transients and membrane time constant of motoneurons. *Experimental neurology*, 2(5):503–532.

Rall, W. (2011). Core conductor theory and cable properties of neurons. In *Comprehensive Physiology*, chapter 3, pages 39–97. Wiley Online Library.

Rall, W. and Rinzel, J. (1973). Branch input resistance and steady attenuation for input to one branch of a dendritic neuron model. *Biophysical journal*, 13(7):648–688.

Remme, M. W., Lengyel, M., and Gutkin, B. S. (2009). The role of ongoing dendritic oscillations in single-neuron dynamics. *PLoS Comput Biol*, 5(9):e1000493.

Remme, M. W. and Rinzel, J. (2011). Role of active dendritic conductances in subthreshold input integration. *Journal of computational neuroscience*, 31(1):13–30.

Rudolph, M. and Destexhe, A. (2003). A fast-conducting, stochastic integrative mode for neocortical neurons invivo. *Journal of Neuroscience*, 23(6):2466–2476.

Schecter, S. (1987). The saddle-node separatrix-loop bifurcation. *SIAM journal on mathematical analysis*, 18(4):1142–1156.

Schleimer, J.-H. and Schreiber, S. (2018). Phase-response curves of ion channel gating kinetics. *Mathematical Methods in the Applied Sciences*, 41(18):8844–8858.

Schultheiss, N. W., Edgerton, J. R., and Jaeger, D. (2010). Phase response curve analysis of a full morphological globus pallidus neuron model reveals distinct perisomatic and dendritic modes of synaptic integration. *Journal of Neuroscience*, 30(7):2767–2782.

Schwemmer, M. A. and Lewis, T. J. (2011). Effects of dendritic load on the firing frequency of oscillating neurons. *Physical Review E*, 83(3):031906.

Stiefel, K. M., Gutkin, B. S., and Sejnowski, T. J. (2008). Cholinergic neuromodulation changes phase response curve shape and type in cortical pyramidal neurons. *PloS one*, 3(12):e3947.

Stöckel, A. and Eliasmith, C. (2022). Computational properties of multi-compartment lif neurons with passive dendrites. *Neuromorphic Computing and Engineering*.

Teramae, J.-n. and Tanaka, D. (2004). Robustness of the noise-induced phase synchronization in a general class of limit cycle oscillators. *Physical review letters*, 93(20):204103.

Tiroshi, L. and Goldberg, J. A. (2019). Population dynamics and entrainment of basal ganglia pacemakers are shaped by their dendritic arbors. *PLOS Computational Biology*, 15(2):e1006782.

Tsumoto, K., Kitajima, H., Yoshinaga, T., Aihara, K., and Kawakami, H. (2006). Bifurcations in morris–lecar neuron model. *Neurocomputing*, 69(4-6):293–316.

Tuckwell, H. C. (1988). *Introduction to theoretical neurobiology: linear cable theory and dendritic structure*, volume 1. Cambridge University Press.

Tzilivaki, A., Kastellakis, G., and Poirazi, P. (2019). Challenging the point neuron dogma: Fs basket cells as 2-stage nonlinear integrators. *Nature communications*, 10(1):1–14.

Uhlhaas, P. J. and Singer, W. (2010). Abnormal neural oscillations and synchrony in schizophrenia. *Nature Reviews Neuroscience*, 11(2):100–113.

Van Aerde, K. I. and Feldmeyer, D. (2015). Morphological and physiological characterization of pyramidal neuron subtypes in rat medial prefrontal cortex. *Cerebral cortex*, 25(3):788–805.

Verwer, J. G. and Sanz-Serna, J. M. (1984). Convergence of method of lines approximations to partial differential equations. *Computing*, 33(3-4):297–313.

Vetter, P., Roth, A., and Häusser, M. (2001). Propagation of action potentials in dendrites depends on dendritic morphology. *Journal of neurophysiology*, 85(2):926–937.

Wang, S., Musharoff, M. M., Canavier, C. C., and Gasparini, S. (2013). Hippocampal ca1 pyramidal neurons exhibit type 1 phase-response curves and type 1 excitability. *Journal of neurophysiology*, 109(11):2757–2766.

Wang, X.-J. and Buzsáki, G. (1996). Gamma oscillation by synaptic inhibition in a hippocampal interneuronal network model. *Journal of neuroscience*, 16(20):6402–6413.

Wei, Y., Ullah, G., and Schiff, S. J. (2014). Unification of neuronal spikes, seizures, and spreading depression. *Journal of Neuroscience*, 34(35):11733–11743.

Williams, S. R. and Stuart, G. J. (1999). Mechanisms and consequences of action potential burst firing in rat neocortical pyramidal neurons. *The Journal of physiology*, 521(Pt 2):467.

Wybo, W. A., Jordan, J., Ellenberger, B., Mengual, U. M., Nevian, T., and Senn, W. (2021). Data-driven reduction of dendritic morphologies with preserved dendro-somatic responses. *Elife*, 10.

Wybo, W. A., Stiefel, K. M., and Torben-Nielsen, B. (2013). The green's function formalism as a bridge between single-and multi-compartmental modeling. *Biological cybernetics*, 107(6):685–694.

Yi, G., Wang, J., Wei, X., and Deng, B. (2017). Action potential initiation in a two-compartment model of pyramidal neuron mediated by dendritic ca2+ spike. *Scientific Reports*, 7(1):1–16.

Zhang, C., Hofmann, D., Neef, A., and Wolf, F. (2022). Ultrafast population coding and axo-somatic compartmentalization. *PLoS computational biology*, 18(1):e1009775.

Zhuchkova, E., Remme, M. W., and Schreiber, S. (2013). Somatic versus dendritic resonance: differential filtering of inputs through non-uniform distributions of active conductances. *PLoS One*, 8(11):e78908.