

CELLULAR MECHANISMS DETERMINING SOMA-DENDRITE CORRELATION

by

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Abstract

Neurons receive synaptic inputs across spatially separated compartments, and the relationship between dendritic processing and somatic output can shape how information is represented in spikes and bursts. However, it remains unclear how compartmentalization within a single neuron interacts with multiplexing, in which multiple input streams are represented simultaneously in a single spike train. This thesis uses a two-compartment neuron model to study how dendritic and somatic dynamics jointly support multiplexed coding. The model separates dendritic integration from somatic spike generation while allowing interaction in both directions through forward dendrite-to-soma drive and spike-triggered soma-to-dendrite feedback. Compartmentalization is quantified operationally using the Pearson correlation between dendritic and somatic membrane voltages, and multiplexing is quantified by the extent to which two output features, event rate and burst fraction, track two compartment-specific input streams. The results indicate that multiplexing is strongest at an intermediate level of compartmentalization, as measured by soma-dendrite voltage correlation: when this correlation is high, event rate and burst fraction become less specific to their corresponding input streams, whereas when this correlation is low, dendritic and somatic activity are less effectively linked. Finally, the thesis introduces a compact metric to quantify how noise, forward dendrite-to-soma drive, and spike-triggered soma-to-dendrite feedback shape the relationship between compartmentalization and multiplexing. This work offers a perspective on the design principles of neural computation and may inform future modeling of signal processing in complex neural structures.

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Glossary

bAPs Back-propagating Action Potentials. [ix](#), [5](#), [11](#), [15](#), [16](#), [19](#), [22](#), [23](#), [24](#), [29](#), [30](#)

BF Burst Fraction. [x](#), [4](#), [6](#), [12](#), [13](#), [15](#), [20](#), [21](#), [29](#), [30](#), [31](#), [32](#), [55](#)

BPM Burst Propagation Model. [x](#), [9](#), [13](#), [14](#), [29](#), [31](#), [32](#), [37](#), [44](#), [51](#), [52](#), [54](#)

ER Event Rate. [x](#), [4](#), [6](#), [12](#), [13](#), [15](#), [19](#), [20](#), [21](#), [29](#), [30](#), [31](#), [32](#), [55](#)

s-d Soma-dendrite. [1](#), [29](#), [43](#), [47](#), [56](#)

s-d correlation Soma-dendrite Correlation. [v](#), [ix](#), [3](#), [4](#), [5](#), [7](#), [9](#), [13](#), [14](#), [22](#), [30](#)

Chapter 1

Introduction

A long-standing view in cellular neuroscience is that dendrites are strongly electrically compartmentalized, allowing different dendritic regions to perform semi-independent computations before the neuron produces a single axonal output. This view supports the influential picture of a neuron as a “network-in-a-neuron” (i.e., a single neuron containing multiple semi-independent dendritic subunits). However, recent *in vivo* functional imaging studies in cortex have often reported substantial coordination between dendritic and somatic activity, suggesting that strong compartmentalization may not always be the dominant operating regime in behaving animals [8].

This apparent tension motivates a re-examination of what dendritic compartmentalization means *in vivo* and how it relates to computation. In particular, Francioni et al. [8] argue that coordinated dendrite–soma activity can itself support key cortical functions, including nonlinear combinations of multiple signals in neural responses, prediction and expectation-related processing, multiplexing, and learning-related credit assignment (linking synaptic changes to the appropriate inputs). The central goal of this thesis is to establish a mechanistic connection between an operational measure of soma-dendrite (s-d) coordination to multiplexed neural coding.

1.1 Morphology and the separation of integration from output

Neurons exhibit a polarized morphology that separates where inputs arrive from where output spikes are generated, while bursts are bouts of high frequency firing. Synaptic inputs are distributed across dendritic arbors, while action potentials are initiated near the axon initial segment and then propagate along the axon to drive synaptic transmission [1,10,25].

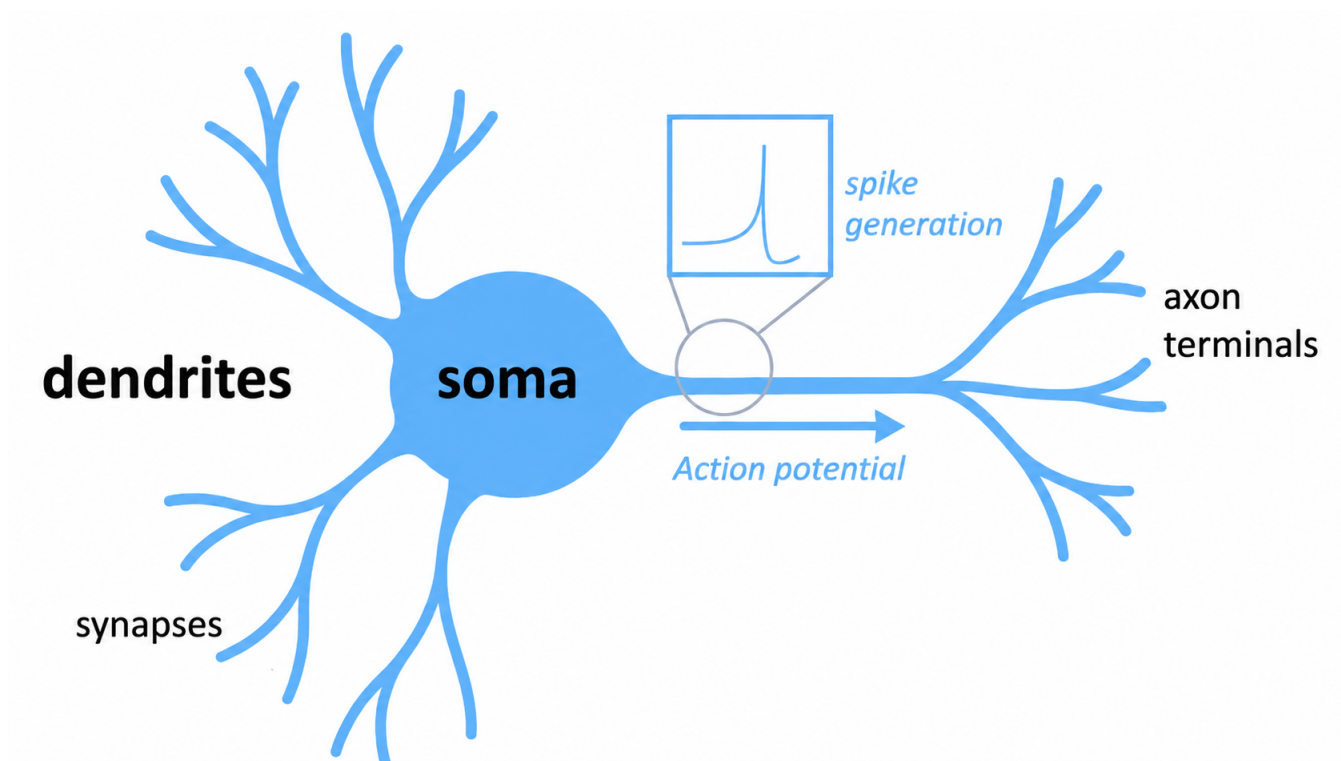


Figure 1.1: Morphological structure of a neuron. Spikes are initiated near the axon initial segment and propagate to axon terminals, where synaptic transmission converts electrical signals into chemical signals [38].

These observations motivate a central question: how does local dendritic processing relate to somatic voltage dynamics and spike–burst output [8,16,17,24]? In some regimes, dendritic activity may remain relatively compartment-specific, preserving a local represen-

tation of dendritic input [16, 17, 24]. In other regimes, dendritic and somatic voltages may co-vary more strongly, so that dendritic activity is more closely reflected in the output of the cell [8, 9]. In this thesis, soma–dendrite voltage correlation is used as an operational read-out of this voltage-level relationship. A high soma–dendrite correlation indicates stronger co-fluctuation between dendritic and somatic membrane voltages, whereas a low correlation indicates greater compartmental separation. Importantly, this correlation does not by itself identify the mechanism producing the relationship; it may arise from intrinsic soma-dendrite interactions, shared synaptic drive, or both [8, 9].

1.2 Rethinking electrical compartmentalization in vivo: why correlation matters

Soma–dendrite correlation (**s-d correlation**) is used here as an operational measure of the voltage-level relationship between dendritic and somatic membrane activity. Specifically, it is quantified as the Pearson correlation coefficient $\text{Corr}(V_s, V_d)$ between dendritic and somatic membrane-potential traces obtained from simultaneous recordings or simulations [2, 9, 37].

In this thesis, this **coordination** is considered at the voltage level: it describes how strongly dendritic and somatic membrane potentials co-fluctuate over time, without by itself specifying the underlying biophysical mechanism.

Importantly, a high or low correlation value does not uniquely identify the source of this relationship: it may reflect intrinsic soma–dendrite interactions, shared synaptic drive in which dendrites and soma are affected by the same input pattern, or state-dependent changes in excitability, such as neuromodulatory or brain-state effects [8].

The review by Francioni et al. [8] provides a concrete computational reason to take correlation seriously. If dendritic and somatic activity are strongly coordinated in vivo, then

correlation is not merely a nuisance that hides compartmentalization; instead, it may be a feature that enables learning and computation at the circuit level. This perspective shifts the question from “Is the neuron compartmentalized?” to “What computations are supported at different coordination regimes?”

In this thesis, the computation of interest is *multiplexing* [20]. Multiplexing refers to encoding multiple input streams within a single spike train by using separable output features. A widely studied example is burst-based multiplexing, where bursts and isolated spikes provide two distinct readouts. To quantify such readouts, two output metrics are used throughout [20, 35]:

- **Burst Fraction (BF)**: the fraction of output *events* that are bursts rather than isolated spikes;
- **Event Rate (ER)**: the rate of events, where an event is either an isolated spike or a burst (timestamped by the first spike of the burst).

These definitions allow a direct test of the idea emphasized by [8]: moderate dendrite–soma dynamics may support multiplexing, but excessive coordination may also blur the separability of output features. A mechanistic model is therefore needed to map how correlation regimes emerge and how they affect multiplexed readouts.

1.3 Mechanistic interpretation: local dendritic events and two-way coupling

Building on the *in vivo* perspective emphasized in [8], a key point is that **s-d correlation** is an aggregate measure of dendrite–soma coordination and therefore cannot be mapped uniquely to a single biophysical mechanism. To interpret **s-d correlation** mechanistically, it is therefore useful to separate at least two contributions. First, correlation can reflect

intrinsic coupling between compartments, i.e., how efficiently voltage fluctuations and currents are transmitted between dendrite and soma. Second, correlation can reflect the *structure of synaptic drive*, for example whether dendrite and soma simultaneously receive the same wave of synaptic input, even if intrinsic coupling is unchanged.

Crucially, the rethinking framework also highlights that dendritic nonlinearities can make coordination state- and context-dependent. Active dendritic mechanisms modulate both contributions by changing the *effective* coupling and gain. Local dendritic spikes (mediated by voltage-gated conductances and NMDA receptors) can selectively amplify dendritic inputs and promote their impact on soma output, whereas backpropagating action potentials (bAPs) carry a feedback signal from spike initiation to dendritic locations, shaping coincidence detection and synaptic plasticity [18, 24, 31, 33]. As a result, dendrite–soma coordination is a dynamical outcome rather than a fixed anatomical property.

A key implication is that identical correlation values can correspond to different operating regimes. For example, high correlation may arise from strong intrinsic coupling, from strongly shared input drive, or from regimes in which nonlinear dendritic events effectively increase the impact of dendritic activity on soma output. Disentangling these possibilities motivates a controlled modeling approach in which coupling parameters and input statistics can be varied independently, enabling causal tests of how coordination relates to multiplexed readouts [8].

1.4 Main research questions and thesis approach

To mechanistically link dendrite–soma coordination to multiplexed coding, this thesis uses a reduced two-compartment neuron model that separates dendritic integration from somatic spike generation while retaining two-way dendrite–soma coupling (dendrite-to-soma drive and soma-to-dendrite feedback) [13, 23, 27]. This framework allows systematic control of coupling strength, intrinsic time scales, and input statistics, and thus allows causal tests of how *s-d correlation* relates to burst-based readouts.

The thesis addresses three questions:

1. Which intrinsic and input parameters most strongly shape $\text{Corr}(V_s, V_d)$ in a two-compartment setting?
2. Across coordination regimes, when do [ER](#) and [BF](#) provide separable readouts of distinct input streams (i.e., effective multiplexing)?
3. Can the observed trade-off between compartmentalization and multiplexing be summarized by compact, interpretable metrics?

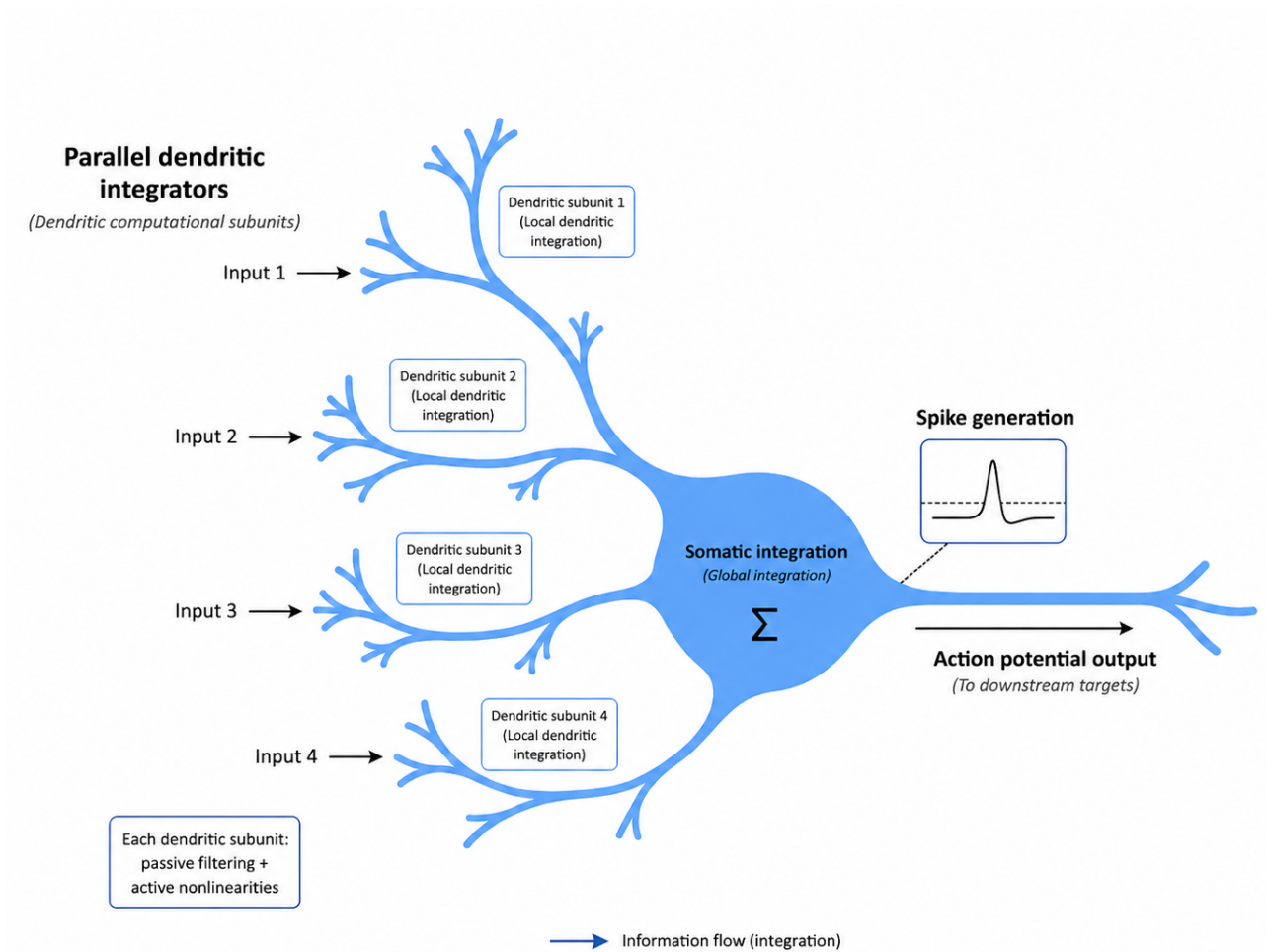


Figure 1.2: This schematic illustrates a conceptual framework in which a neuron operates as a set of parallel integrators [34]. Dendrites act as computational subunits that transform compartment-specific input drive through passive filtering and active nonlinearities, whereas the soma serves as the final integration stage that determines whether an action potential is generated. This division of labor motivates interpreting [s-d correlation](#) as a readout of how strongly dendritic processing is coordinated with somatic output.

Chapter 2

Method and software

This chapter describes the methods and software used to develop and analyze the computational models in this study. Specifically, we implemented two-compartment neuron models in Python to simulate dendritic and somatic interactions. These approaches provide a comprehensive framework for understanding neuronal signal processing and improving predictive accuracy through statistical and computational techniques. Parameter tables are presented in Appendix A.

Code availability. The Python code used for the numerical simulations, parameter sweeps, data analysis, and figure generation in this thesis is publicly available at <https://github.com/lunarAppulse/Soma-Dendrite-Multiplexing-Simulations>. The repository contains the implementations of the Burst Propagation Model and the linear passive two-compartment model used in this study.

2.1 Two-compartment model and quantification of s-d correlation and multiplexing

To investigate how these two phenomena (Multiplexing and Compartmentalization) interact, we implemented a nonlinear Burst Propagation Model (BPM) as a two-compartment system of coupled ordinary differential equations, following the reduced-model framework introduced by Pinsky and Rinzel [23] and adapted in later work [21].

This model incorporates compartment-specific excitability, adaptation, and active dendritic mechanisms, allowing us to study how variations in s-d correlation affect both input integration and the structure of the output spike train. The resulting dynamics are governed by the following system:

Somatic compartment:

$$\dot{V}_s = -\frac{1}{\tau_s}(V_s - E_L) + \frac{1}{C_s}[g_s f(V_d) + \frac{g_c}{p}(V_d - V_s) + I_s + \xi_s(t) - w_s] \quad (2.1)$$

$$\tau_{w_s} \dot{w}_s = -w_s + b\tau_{w_s} S(t) \quad (2.2)$$

Dendritic compartment:

$$\dot{V}_d = -\frac{1}{\tau_d}(V_d - E_L) + \frac{1}{C_d}[g_d f(V_d) + \frac{g_c}{1-p}(V_s - V_d) + c_d(K * S)(t) + I_d + \xi_d(t) - w_d] \quad (2.3)$$

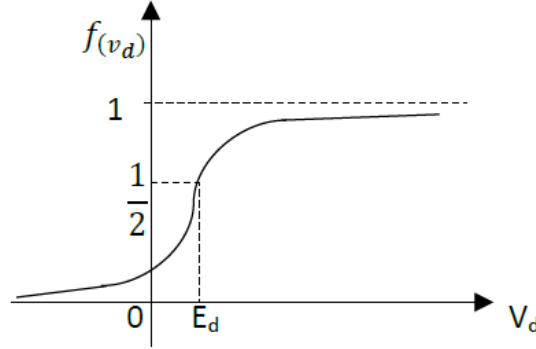
$$\tau_{w_d} \dot{w}_d = -w_d + a_w(V_d - E_L) \quad (2.4)$$

$$f(V_d) = \left(1 + \exp\left[-\frac{(V_d - E_d)}{D_d}\right]\right)^{-1} \quad (2.5)$$

Here, all variables and parameters are defined as follows:

- V_s, V_d (mV): membrane potentials of soma and dendrite.

- C_s, C_d (pF): membrane capacitances determining voltage responsiveness.
- τ_s, τ_d (ms): membrane time constants controlling passive decay rates.
- E_L (mV): leak reversal (resting) potential. When only leak channels are considered, the membrane potential relaxes toward E_L , which sets the resting level of the passive (leak-only) dynamics. It is not the threshold for "discharge or not," but only a reference point for passive dynamics.
- g_c (nS): passive coupling conductance between soma and dendrite, scaled by the geometric ratios p and $1 - p$.
- p (dimensionless): capacitance (area) fraction assigned to the soma, with $1 - p$ assigned to the dendrite. The factors $1/p$ and $1/(1-p)$ in the coupling terms implement an asymmetry in voltage sensitivity: for the same coupling current, the compartment with smaller effective capacitance exhibits a larger voltage change. We therefore use p as a phenomenological weight that controls the impedance asymmetry (mismatch) between the two compartments.
- g_s, g_d (nS or pA/mV): **active gain factors** mediating nonlinear voltage-dependent drive from dendrite to soma and within the dendrite itself, while g_s is active coupling to soma.
- c_d (nS): the active coupling to dendrite.
- $f(V_d)$: sigmoidal activation function modeling the steady-state activation of voltage-gated channels (e.g., NMDA or Ca^{2+} currents); E_d (mV) is the half-activation voltage, D_d (mV) controls how rapidly the function changes with V_d . $f(V_d)$ is an S-shaped curve, it has function value from 0 – 1, reaching 1/2 when $V_d = E_d$.



- $I_s(t)$, $I_d(t)$ (pA): external input currents to soma and dendrite, typically square-wave or oscillatory drives.
- $\xi_s(t)$, $\xi_d(t)$ (pA): independent Ornstein–Uhlenbeck noise processes providing colored stochastic input.
- w_s , w_d (pA): adaptation variables that implement slow, negative feedback. Their time constants τ_{w_s} and τ_{w_d} (ms) govern the recovery rates, while b and a_w control the adaptation strength in soma and dendrite.
- τ_{w_s} , τ_{w_d} (ms): The two are the adaptation time constants, controlling the recovery speed of the adaptation variables w_s , w_d . τ_{w_s} : When a spike occurs, w_s is instantaneously increased by b , and then decays with a time constant of τ_{w_s} (spike-triggered negative feedback). τ_{w_d} : when the dendrite depolarizes, w_d is "slowly pushed up" and then decays with a time constant of τ_{w_d} (subthreshold-driven negative feedback).
- $S(t) = \sum_i \delta(t - t_i)$: soma spike train, where each δ represents an action potential at time t_i . In the code, this is stored as a list of spike times updated when V_s crosses the threshold V_T .
- $(K * S)(t) = \sum_i K(t - t_i)$: convolution of $S(t)$ with a causal kernel $K(t)$ modeling back-propagating action potentials (bAPs) from the soma to the dendrite. In simulation, $K(t)$ is implemented as a rectangular window (0.5–2.5 ms), such that each spike induces a brief step-like current of amplitude c_d into the dendrite.

- V_T (mV): spike threshold used in the event rule for soma. When $V_s(t)$ reach V_T , a spike is emitted, V_s is reset to V_r (mV), and w_s is incremented by b . This threshold mechanism defines the transition between **subthreshold dynamics** (continuous evolution governed by Eqs. 2.1–2.4) and discrete spike events.
- V_r (mV): reset potential following a spike. [6, 10, 12, 13, 16, 23, 24, 27, 33]

Model interpretation. Equations 2.1–2.4 describe a system that evolves continuously while $V_s < V_T$ (the subthreshold regime). When V_s reaches the fixed threshold V_T , a discrete spike event is triggered according to the reset rule above. The bAP term $c_d(K * S)(t)$ provides a short-lived depolarizing feedback from soma to dendrite, facilitating bursts. Together, these equations reproduce nonlinear interactions between somatic and dendritic compartments responsible for burst initiation, propagation, and modulation by noise or coupling.

Quantitative metrics. Compartmentalization was quantified as the correlation coefficient between somatic and dendritic voltages:

$$\text{Compartmentalization metric} = |\text{Corr}(V_s, V_d)|.$$

Multiplexing was quantified by the summed correlation coefficients of each input with its corresponding output feature:

$$\text{Multiplexing metric} = (\text{Corr}(I_s, \text{ER}) + \text{Corr}(I_d, \text{BF}))/2,$$

where **ER** is the somatic event rate and **BF** is the dendritic burst fraction [20, 35].

Correlation coefficient. For two aligned sequences $\{x_t\}_{t=1}^T$ and $\{y_t\}_{t=1}^T$, we define:

$$\text{Corr}(X, Y) = \frac{\sum_{t=1}^T (x_t - \bar{x})(y_t - \bar{y})}{\sqrt{\sum_{t=1}^T (x_t - \bar{x})^2} \sqrt{\sum_{t=1}^T (y_t - \bar{y})^2}}, \quad \bar{x} = \frac{1}{T} \sum_{t=1}^T x_t, \quad \bar{y} = \frac{1}{T} \sum_{t=1}^T y_t. \quad (2.6)$$

All series used in a given correlation were aligned to the *same* T time bins (after any downsampling), and a short edge margin was discarded to avoid boundary effects.

2.2 Linear cable model approximation

The nonlinear [BPM](#) is required to generate spikes, bursts, and event-based readouts ([ER](#) and [BF](#)). To obtain a more interpretable description of how dendrite–soma coordination depends on coupling and input statistics, we also used a linear two-compartment cable model as an analytical approximation [\[6, 13, 27\]](#). This linear model captures passive signal transmission between compartments and provides closed-form intuition for trends in [s-d correlation](#).

Model definition. Following Pinsky and Rinzel’s two-compartment reduction of the Traub CA3 model [\[23\]](#), we introduce a dimensionless parameter p that specifies the fraction of total membrane area (and hence capacitance) associated with the soma. With the total surface area normalized to one, the somatic and dendritic areas are p and $1 - p$, so that $C_s = p C_m$ and $C_d = (1 - p) C_m$, where C_m is the specific membrane capacitance (in $\mu\text{F}/\text{cm}^2$) and C_s, C_d are the effective compartment capacitances (also in $\mu\text{F}/\text{cm}^2$). This construction ensures that all transmembrane currents, expressed as current densities (in $\mu\text{A}/\text{cm}^2$), are correctly converted into compartment-specific current densities. This yields coupling and input terms g_c/p , $g_c/(1 - p)$, I_s/p and $I_d/(1 - p)$ in the current-balance equations, where g_c is a coupling conductance density (in mS/cm^2) and I_s, I_d are current densities (in $\mu\text{A}/\text{cm}^2$). As in [\[23\]](#), we choose $p = 0.5$ as a phenomenological lumped representation of somatic versus dendritic surface area for a CA3 pyramidal cell: for this intermediate value, the dendritic compartment remains large enough ($1 - p \gtrsim 0.5$) to support calcium spikes and bursting, while the overall firing frequencies and bursting regime closely match those of the original multi-compartment Traub model.

The resulting linear dynamics are:

$$\dot{V}_s = -\frac{g_L}{C_m}(V_s - V_L) + \frac{g_c}{pC_m}(V_d - V_s) + \frac{I_s(t) + \xi_s(t)}{pC_m}, \quad (2.7)$$

$$\dot{V}_d = -\frac{g_L}{C_m}(V_d - V_L) + \frac{g_c}{(1 - p)C_m}(V_s - V_d) + \frac{I_d(t) + \xi_d(t)}{(1 - p)C_m}. \quad (2.8)$$

where the overdot denotes a derivative with respect to time t (in ms).

Here V_s and V_d are the somatic and dendritic membrane potentials (in mV), and V_L is the leak reversal potential (in mV). The leak term represents passive membrane conductance that drives each compartment voltage toward the leak reversal potential V_L , with current $I_{\text{leak}} = g_L (V - V_L)$. In the voltage equation it appears as $-\frac{g_L}{C_m} (V - V_L)$. The parameter g_L is the passive leak conductance density (in mS/cm²), g_c is the axial coupling conductance density between soma and dendrite (in mS/cm²), and C_m is the specific membrane capacitance (in $\mu\text{F}/\text{cm}^2$). The input current density $I_s(t)$ is measured in $\mu\text{A}/\text{cm}^2$, and $\xi_1(t)$ denotes a zero-mean stochastic input with the same units as $I_s(t)$ (e.g. Gaussian white noise), so that the ratios $(I_s + \xi_1)/(pC_m)$ and $(I_s + \xi_1)/((1-p)C_m)$ both have units of voltage change per unit time (mV/ms). The parameter p is a dimensionless proportion of total membrane capacitance associated with the soma (so that $C_s = pC_m$ and $C_d = (1-p)C_m$), analogous to p above.

Relation to the nonlinear model. Equations (2.7)–(2.8) can be viewed as a controlled simplification of the [BPM](#) in which nonlinear dendritic activation, adaptation, and spike-triggered feedback are removed (i.e., $f(V_d)$ is omitted, $w_s = w_d = 0$, and the bAP term is absent). This isolates passive coupling and shared input effects, allowing us to attribute changes in $\text{Corr}(V_s, V_d)$ to input statistics and coupling strength rather than to burst-generation mechanisms.

Use in this thesis. The linear cable model was used to (i) validate qualitative trends observed in the nonlinear simulations, (ii) provide an interpretable baseline for how [s-d correlation](#) depends on g_c , p , and the noise statistics, and (iii) guide parameter choices for regimes where passive transmission dominates. Correlation was computed using the same procedure as in Eq. (2.6), after discarding initial transients and aligning all signals to the same time bins.

Chapter 3

Burst Propagation and Compartmentalization in Neuronal Multiplexing

This chapter examines how a two-compartment neuron (soma–dendrite) trades off compartmentalization and multiplexing. We study how the forward, nonlinear drive from dendrite to soma and the [bAPs](#) feedback from soma to dendrite jointly shape the soma–dendrite voltage correlation and the separation of two information streams. Here we quantify compartmentalization using the soma–dendrite voltage correlation, $\text{Corr}(V_s, V_d)$. Conceptually, the event rate ([ER](#)), defined as the rate of events where each isolated spike contributes one event and each burst contributes one event at burst onset, mainly carries somatic-drive information, whereas the burst fraction ([BF](#); bursts normalized by ER) emphasizes dendritic-drive information. Moderate coupling tends to support high multiplexing with acceptable compartmentalization, while overly strong coupling raises soma–dendrite correlation at the cost of mixing the two output channels.

3.1 Theory

Model view. The neuron consists of a dendritic and a somatic compartment. Dendrites receive I_d and noise and exhibit a local nonlinearity $f(V_d)$; the soma receives I_s and noise and is responsible for spike generation and reset. Two active pathways are central: (i) a forward drive $g_s f(V_d)$ that makes the soma more excitable, and (ii) a bAP term that transiently depolarizes V_d following somatic spikes (scaled by c_d). In some baseline runs, c_d was set to zero to isolate forward dendrite-to-soma coupling. In the parameter-sweep experiments reported below, c_d was varied systematically, as described in Sec. 3.2.

Mechanistic intuition. A somatic spike raises dendritic voltage for a short window (bAPs). When spikes cluster (bursts), overlapping bAPs keep V_d elevated longer, which enhances the forward drive to the soma and favors subsequent spiking—a short, positive feedback loop. In contrast, somatic reset and spike-triggered adaptation create a brief anti-phase segment (high V_d , low V_s immediately after a spike), reducing instantaneous correlation. The observed soma–dendrite correlation is therefore the net effect of these synchronizing (forward drive and bAP) and desynchronizing (reset/adaptation) forces.

Readouts and metrics (concept). We quantify compartmentalization via the correlation coefficient between V_s and V_d . Multiplexing is quantified by combining two input–output correlations: $\text{Corr}(I_s, \text{ER})$ for the event channel and $\text{Corr}(I_d, \text{BF})$ for the burst channel. In reporting, we use the absolute-value convention and average the two terms. This definition is used consistently throughout the numerical analysis below. Operational definitions of ER/BF, alignment and all implementation details are given in Sec. 3.2.

3.2 Numerical implementation and discrete-time simulation

The neuronal dynamics in Eqs. 2.1–2.4 were integrated with a fixed-step Euler–Maruyama scheme. All state variables were updated on a uniform grid with time step $\Delta t = 0.1$ ms for a total duration $T = 600$ ms ($N = T/\Delta t = 6000$ steps). This step is $\approx 70\times$ smaller than the fastest membrane time constant ($\tau_d = 7$ ms), providing stable integration and sufficient temporal resolution for spike detection.

Ornstein–Uhlenbeck (OU) noise. Each compartment received an independent OU process

$$\frac{d\xi}{dt} = -\alpha \xi + \sigma \eta(t),$$

with $\eta(t)$ Gaussian white noise. We used the Euler–Maruyama update

$$\xi_{n+1} = \xi_n - \alpha \xi_n \Delta t + \sigma \sqrt{\Delta t} z_n, \quad z_n \sim \mathcal{N}(0, 1),$$

which matches the implementation `noise[i+1] = noise[i] - alpha*noise[i]*dt + z[i]*np.sqrt(dt)`.

Discrete-time membrane updates. The neuronal dynamics were integrated with a fixed-step Euler–Maruyama scheme. At each time step, the dendritic voltage was updated according to

$$V_d^{(n+1)} = V_d^{(n)} + \left[-\frac{V_d^{(n)} - E_L}{\tau_d} + \frac{1}{C_d} \left(g_d f(V_d^{(n)}) + \frac{g_c}{1-p} (V_s^{(n)} - V_d^{(n)}) \right. \right. \\ \left. \left. + I_d^{(n)} + \xi_d^{(n)} - w_d^{(n)} + c_d (K * S)^{(n)} \right) \right] \Delta t.$$

The somatic voltage was updated in a piecewise manner. If the somatic voltage at the

previous step remained below threshold, it evolved according to

$$V_s^{(n+1)} = V_s^{(n)} + \left[-\frac{V_s^{(n)} - E_L}{\tau_s} + \frac{1}{C_s} \left(g_s f(V_d^{(n)}) + \frac{g_c}{p} (V_d^{(n)} - V_s^{(n)}) + I_s^{(n)} + \xi_s^{(n)} - w_s^{(n)} \right) \right] \Delta t, \quad \text{if } V_s^{(n)} < V_T.$$

Thus, the passive soma–dendrite coupling terms

$$\frac{g_c}{p} (V_d - V_s), \quad \frac{g_c}{1-p} (V_s - V_d)$$

are part of the full discrete-time update equations. In parameter regimes where $g_c = 0$, these passive coupling contributions automatically vanish, so the dynamics reduce to the active dendrite-to-soma drive $g_s f(V_d)$ together with the spike-triggered soma-to-dendrite feedback $c_d(K * S)$.

Spike generation and adaptation. A somatic spike was registered whenever $V_s^{(n)} \geq V_T$. In that case, the somatic voltage was not updated by the subthreshold Euler step; instead, a spike time t_i was recorded and the somatic voltage was reset according to

$$V_s^{(n+1)} = V_r.$$

The adaptation variables were updated consistently with the code implementation. The dendritic adaptation variable followed

$$w_d^{(n+1)} = w_d^{(n)} + \left(-\frac{w_d^{(n)}}{\tau_{w_d}} + a_w (V_d^{(n)} - E_L) \right) \Delta t.$$

The somatic adaptation variable was updated piecewise:

$$w_s^{(n+1)} = \begin{cases} w_s^{(n)} + \left(-\frac{w_s^{(n)}}{\tau_{w_s}} \right) \Delta t + b, & \text{if } V_s^{(n)} \geq V_T, \\ w_s^{(n)} + \left(-\frac{w_s^{(n)}}{\tau_{w_s}} \right) \Delta t, & \text{if } V_s^{(n)} < V_T. \end{cases}$$

This corresponds directly to the code logic: `if Vs[i-1] >= VT: Vs[i] = Vr; ws[i] = (-ws[i-1]/tau_ws)*dt + b + ws[i-1]`, and otherwise `Vs[i] = ... ; ws[i] = (-ws[i-1]/tau_ws) + ws[i-1]`. Therefore, in the numerical implementation, spike emission, reset, and the spike-triggered increment of w_s are handled explicitly as a threshold-triggered branch rather than through a single unified subthreshold update rule.

Back-propagating action potential (bAPs) term. At each time t , we look back over the last 0.5–2.5 ms and count how many somatic spikes occurred in that window, $(K * S)(t) = \sum_i K(t - t_i)$ is the number of spike times t_i with $[0.5, 2.5]$ ms, this count is the bAP drive to the dendrite (scaled by c_d). Numerically, we do not rescan the entire spike list at every step; instead we maintain an event-driven sliding count of spikes in the moving window $(t - 2.5, t - 0.5]$ using two pointers that update the count by +1 when the right boundary crosses a spike time and by -1 when the left boundary crosses it. The resulting integer $(K * S)^{(n)}$ enters the dendritic update as $+c_d (K * S)^{(n)} / C_d$:

$$(K * S)(t) = |\{i : t - 2.5 \text{ ms} < t_i \leq t - 0.5 \text{ ms}\}| = \underbrace{|\{i : t_i \leq t - 0.5 \text{ ms}\}|}_{\text{Right boundary accumulation}} - \underbrace{|\{i : t_i \leq t - 2.5 \text{ ms}\}|}_{\text{Left boundary accumulation}} .$$

Event/burst extraction. We used an inter-spike-interval (ISI) threshold of $\theta_B = 15$ ms to separate *bursts* from *spikes not belonging to any burst*. Two consecutive spikes with $\text{ISI} \leq 15$ ms are assigned to the same burst; otherwise the spike is treated as non-burst.

Time was divided into non-overlapping 1 ms bins using a left-closed, right-open convention,

$$[0, 1), [1, 2), \dots$$

so that an event occurring exactly at the right endpoint was assigned to the next bin. Within each 1 ms bin, we defined: (i) **FR** (firing rate), the total number of spikes in the bin; (ii) **BR** (burst rate), the number of bursts in the bin, counting each burst once at its onset; and (iii) **ER** (event rate), the number of non-burst spikes plus the number of bursts.

Equivalently, this counting rule can be represented at the spike level by an event indicator

e_k :

$$e_k = \begin{cases} 1, & \text{if } t_i \text{ is a non-burst spike or the first spike of a burst,} \\ 0, & \text{if } t_i \text{ is a subsequent spike within a burst.} \end{cases}$$

Let E denote the set of event times, i.e., the set of isolated-spike times together with burst-onset times. Then

$$\text{ER}[n] = |\{t \in E \mid n \leq t < n+1\}|.$$

In the code, this is implemented by assigning each event time t to bin $\lfloor t \rfloor$ and incrementing `eventRate[math.floor(t)]`.

The burst fraction is computed per bin as

$$\text{BF} = \begin{cases} \frac{\text{BR}}{\text{ER}}, & \text{ER} > 0, \\ 0, & \text{ER} = 0. \end{cases}$$

as in the code lines `if eventRate[i] != 0: burstFraction[i] = burstRate[i] / eventRate[i]`

Since averaging across a fixed number of neurons differs from summation only by a constant scaling factor, the Pearson correlation is unchanged. The above procedure follows standard practice in burst-ensemble multiplexing analyses [20].

Alignment and correlations (code-consistent). Spike-derived readouts (FR/BR/ER/BF) are computed on non-overlapping 1 ms bins using a left-closed, right-open convention $[m, m+1)$ ms. Input currents (I_s, I_d) are simulated at 0.1 ms; for correlations with the 1 ms readouts we downsample by taking every 10th sample starting at $t = 100.5$ ms so that each input sample aligns with the midpoint of its corresponding 1 ms bin (implemented as `Is[1005:-1000:10]` and `Id[1005:-1000:10]`).

To avoid transients and boundary effects, we discard the first/last 100 ms: for voltage correlations on the 0.1 ms sampling we use `Vs[1000:-1000]` and `Vd[1000:-1000]`; on

the 1 ms bins we use `eventRate[100:-100]` and `burstFraction[100:-100]`. Counts are pooled across neurons (sum over cells) before computing zero-lag Pearson correlations, which are reported in absolute value. All arrays are serialized to `.pkl` for plotting and statistics.

In Fig. 3.1 we illustrate the two-compartment structure used throughout this thesis. The soma compartment is driven by an external current $I_s(t)$ plus a noise term, and its membrane voltage is denoted $V_s(t)$. The dendrite compartment is driven by $I_d(t)$ plus an independent noise term, with voltage $V_d(t)$. The two voltages interact through an axial coupling conductance g_c : whenever $V_s \neq V_d$, the voltage difference $\Delta V = V_d - V_s$ generates a coupling current that tends to reduce this difference. In the nonlinear model, spikes are initiated at the axon initial segment and can produce a spike-triggered feedback signal that propagates from soma toward dendrite (a bAP-like effect). Together, this schematic summarizes how compartment-specific inputs and noise shape V_s and V_d , and how coupling (and, when included, spike-triggered feedback) determines the degree of dendrite-soma coordination quantified by $\text{Corr}(V_s, V_d)$.

Fig. 3.2 further clarifies the operational criteria for distinguishing **spikes** from **bursts** in this modeling framework. A single spike is defined by the depolarization of the membrane voltage beyond a threshold $V_{threshold}$, followed by a reset to V_{reset} . A **burst** is defined operationally using the ISI criterion described above: consecutive spikes separated by ISIs ≤ 15 ms are assigned to the same burst, suggesting a temporally compressed firing event that can carry distinct coding significance.

This figure provides a qualitative “target pattern” for what successful multiplexing looks like in our model: two compartment-specific input streams (I_s and I_d ; top) are reflected in two distinct output features. The event rate (ER) tracks the alternation of the somatic input, whereas burst-related readouts (BF and BR) increase when dendritic drive is stronger and when soma and dendrite are co-activated. The simulation experiments in the remainder of this chapter systematically sweep parameters (e.g., coupling and noise) to identify the regime in which this qualitative multiplexing signature is most robust.

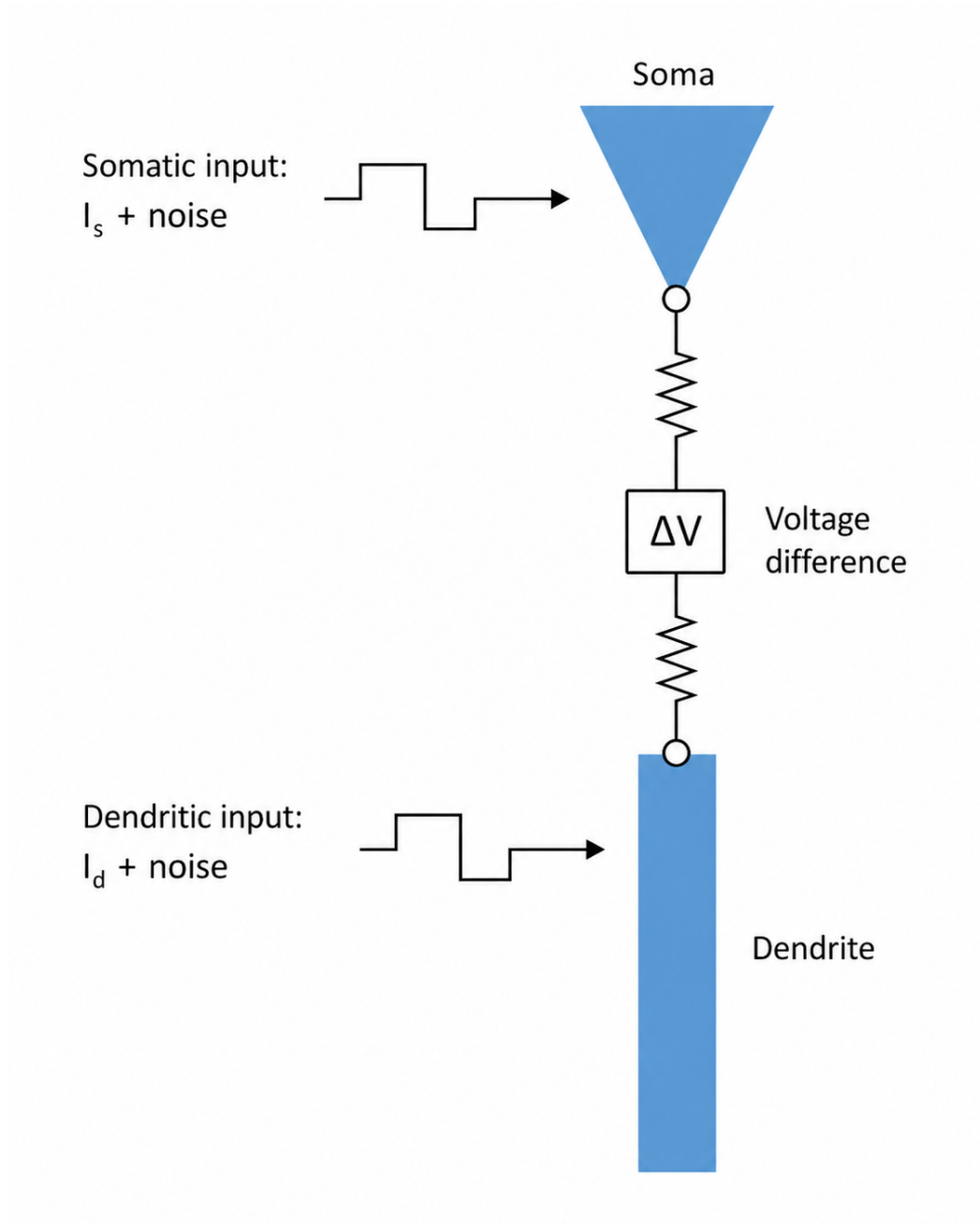


Figure 3.1: Modeling of [s-d correlation](#), schematic of the network. External currents and noise affecting each compartment. Dendritic compartment (square) receives a current I_d and noise, while the somatic compartment receives a current I_s and distinct noise. [bAPs](#) originate from soma and propagate into the dendrite. Voltage interactions between the two compartments occur via coupling conductance g_c .

3.3 Simulation experiment and data collection

In addition to the passive (resistive) soma–dendrite coupling implemented by the ohmic term $g_c(V_d - V_s)$, two parameters of particular interest in this context are g_s and c_d . The parameter g_s scales an additional active dendrite-to-soma coupling term $g_s f(V_d)$, which is nonlinear in the dendritic voltage and becomes prominent during strong dendritic depolarizations, such as those produced by clustered synaptic input or local dendritic spikes. In this way, $g_s f(V_d)$ does not replace the passive coupling $g_c(V_d - V_s)$ but controls the extra somatic drive generated by regenerative dendritic activity on top of the passive resistive coupling. This active interaction is particularly relevant for complex firing patterns observed in cortical neurons, where dendritic nonlinearities strongly influence action potential initiation and synaptic integration [8]. In contrast, c_d scales the spike-triggered feedback from soma to dendrite, implemented as the convolution $(K * S)(t)$ of the soma spike train with a causal kernel [4]. A systematic analysis of these parameters is necessary for quantifying their influence on excitability, firing thresholds, and gain modulation. Such analysis not only facilitates the calibration of neuron models against empirical data but also enables the exploration of biologically plausible regimes of dendritic processing and synaptic integration [14, 22]. Consequently, characterizing the effects of g_s and c_d provides critical insight into the computational capabilities of neurons and the biophysical constraints that underlie signal propagation in dendrite–soma systems.

c_d represents the **bAPs** intensity. It models the influence of somatic spikes on dendritic signals. The term $g_s f(V_d)$ represents an active and nonlinear influence from dendrite to soma, and scales how strongly dendritic nonlinear processing affects the soma.

In Fig. 3.4, as the **bAPs** feedback strength (c_d) initially increases, the multiplexing metric also increases, reaching a maximum at different c_d values for each curve. This suggests that there is an optimal range of c_d for multiplexing efficiency.

After the peak, the multiplexing metric declines as c_d continues to increase, indicating a degradation in performance due to oversaturation or interference at higher c_d levels.

We now explain the mathematical basis of the simulations and link the equations to the figures.

In the short-time regime, where membrane capacitance dominates the voltage response, a compartment with smaller capacitance shows a larger voltage change for the same axial current and can therefore be viewed as having a higher effective impedance. Since p sets how the total capacitance is divided between soma and dendrite, it controls this impedance mismatch: smaller p makes the soma effectively more impedance-dominant (more sensitive to coupling currents), whereas larger p makes the soma less sensitive. Thus, p serves as a phenomenological impedance-mismatch weight between soma and dendrite.

At low **bAPs** feedback strength (c_d), the dendrite operates more independently because the soma-to-dendrite influence (via c_d) is minimal. Multiplexing is weak because coordination between soma and dendrite is limited.

At moderate **bAPs** feedback strength (c_d), a moderate **bAPs** intensity balances dendrite-to-soma and soma-to-dendrite interactions. This supports optimal multiplexing, where soma and dendrite can coordinate their signals without losing independence.

At high **bAPs** feedback strength (c_d), excessive **bAPs** disrupt the independence of the dendrite. The dendrite becomes dominated by somatic signals, reducing its ability to process independent inputs and thereby weakening multiplexing.

In Fig. 3.5, our results suggest that the optimal subthreshold coupling to the soma, g_s , for multiplexing depends significantly on the impedance mismatch p . Lower values of p (for example, $p = 0.2$) achieve optimal multiplexing at lower conductance values, possibly because less overall synaptic drive requires less conductance to reach effective levels of neuronal activation. Conversely, a higher capacitance fraction (for example, $p = 0.8$) requires higher conductance values, likely because stronger conductance is needed to balance the increased synaptic input while still avoiding saturation of the synaptic response.

In parallel, the membrane time constants τ_s and τ_d define the temporal response character-

istics of the soma and dendrite, respectively [13, 19]. While τ_d governs dendritic integration over time, allowing dendrites to act as low-pass filters or coincidence detectors, in this study only τ_s is systematically varied, not τ_d . The parameter τ_s governs the temporal integration of synaptic and dendritic inputs in the soma [13]. A larger τ_s means a slower response, whereas a smaller τ_s means faster membrane-potential changes [10].

This behavior confirms that τ_s modulates the temporal filtering characteristics of the soma. Therefore, analyzing τ_s is essential for understanding how the neuron’s signal-multiplexing capacity, that is, its ability to simultaneously encode and propagate different types of signals, varies with its intrinsic temporal filtering properties [8, 15].

To interpret the role of p in the simulation results, in particular its influence on multiplexing in Figs. 3.5–3.6, we now revisit the definition of p in our two-compartment model. Returning to the p introduced in Chapter 1, we interpret it as a phenomenological impedance-mismatch weight between soma and dendrite [7, 32, 36]. According to Equations 2.1–2.4, the axial coupling contributions to the voltage dynamics can be written as

$$\dot{V}_s \Big|_{\text{axial}} = \frac{1}{C_s} \frac{g_c}{p} (V_d - V_s), \quad \dot{V}_d \Big|_{\text{axial}} = \frac{1}{C_d} \frac{g_c}{1-p} (V_s - V_d),$$

where $0 < p < 1$ and C_s, C_d denote the effective somatic and dendritic capacitances. We choose

$$p = \frac{C_s}{C_s + C_d}, \quad 1 - p = \frac{C_d}{C_s + C_d},$$

so that p and $1 - p$ are complementary weights, with $p + (1 - p) = 1$, associated with the two compartments. With this convention, the same axial current produces different voltage changes on the two sides, scaled by the factors $1/p$ and $1/(1 - p)$.

The units of the axial term in the somatic equation are consistent with a voltage change per unit time:

$$\frac{1}{C_s} \left(\frac{g_c}{p} (V_d - V_s) \right) : \quad \frac{1}{\text{pF}} \times \frac{\text{pA}}{\text{mV}} \times \text{mV} = \frac{\text{pA}}{\text{pF}} = \frac{\text{mV}}{\text{ms}}.$$

In morphology-based cable models such as the Tripod model of Quaresima et al. [26], the

axial current between soma and dendrite is written as

$$I_{\text{ax}} = g_{\text{ax}}(V_d - V_s),$$

where g_{ax} (in nS) is determined by dendritic geometry and resistivity and therefore links morphology to coupling strength. In our reduced two-compartment model, g_c plays the role of an effective soma-dendrite coupling parameter. For a symmetric choice $p = 0.5$, we choose g_c such that the magnitude of the axial voltage update in our equations is comparable to that produced by a morphology-based axial conductance g_{ax} . For other values of p , we keep g_c fixed and interpret changes in p as phenomenological adjustments of the impedance mismatch, rather than as exact mappings from a specific morphology [11, 28, 29].

If the membrane behavior is approximately capacitively dominant, or if the leakage conductance on both sides is equal, the effective membrane impedance approximately satisfies

$$\text{impedance mismatch ratio} \approx \frac{C_s}{C_d} \approx \frac{p}{1-p}.$$

This indicates that the same axial current is asymmetrically scaled in the voltage equations by the two **complementary weights** $1/p$ and $1/(1-p)$:

- **Small** p (C_s small) $\Rightarrow \frac{g_c}{p}$ is large $\Rightarrow$ the soma side is more “sensitive” to coupling;
- **Large** p (C_s large) $\Rightarrow \frac{g_c}{p}$ is small $\Rightarrow$ the soma side is more “dull” (less sensitive).

We follow standard compartmental practice and set membrane capacitance from area,

$$C = c_m A,$$

and use the impedance mismatch

$$p = \frac{C_s}{C_s + C_d}$$

as an impedance-mismatch weight for soma versus dendrite [5, 10]. The axial coupling follows the classical two-compartment reduction [23], while the morphology-to-coupling link is expressed via g_{ax} [13, 26].

In Fig. 3.6, different colors represent the curves obtained under different τ_s values. Here $\tau_{s0} = 16$ ms and $\tau_{d0} = 7$ ms denote the baseline membrane time constants of the soma and dendrite, respectively. In all simulations, the variable time constants τ_s and τ_d are chosen as relative changes around these baseline values, so that their numerical values reflect realistic variations in membrane time scales rather than arbitrary units. In this figure, τ_d is fixed at τ_{d0} and only τ_s is varied, as indicated by the different colors.

To further understand these simulation results, we now analyze the correlation from the structure of the voltage equations rather than relying only on physiological intuition. However, it is noteworthy that in the region where $g_s < 2000$ pA, certain simulation outcomes deviate from the trends predicted by the previous analysis.

In our two-compartment formulation, following the classical Rall-type cable reduction [27], the term $g_c(V_d - V_s)$ represents the passive (resistive) soma-dendrite coupling. This resistive coupling is always present and linearly transmits voltage differences between the two compartments. In addition, we introduce an active dendrite-to-soma coupling term $g_s f(V_d)$, which describes how regenerative dendritic activity adds extra drive to the somatic voltage beyond the passive resistive coupling.

Therefore, the larger the value of g_s , the larger the correlation coefficient $\rho(V_s, V_d)$ tends to be in the two-compartment model, as illustrated in Fig. 3.6 by the entire purple curve, the red, green, and orange curves, and the second half of the blue curve.

On the other hand, τ_s in Eq. (2.1) is the somatic membrane time constant. It determines how quickly the somatic voltage V_s returns to the leak potential E_L when the inputs and coupling terms are fixed. A smaller τ_s means that V_s relaxes back to E_L more rapidly. In this case, any depolarization produced by the active coupling term $g_s f(V_d)$, the passive coupling term $g_c(V_d - V_s)$, or the somatic input current I_s is quickly cancelled by the leak current, so their net contribution to the somatic response is reduced [27].

Because the soma-to-dendrite term with coefficient c_d is triggered by somatic spikes, somatic spiking becomes important for the voltage correlation when g_s is large. When τ_s is

small, the somatic voltage V_s returns quickly to the leak potential E_L , so it is harder for V_s to reach the threshold V_T and generate spikes. As a result, the effective soma-to-dendrite coupling mediated by c_d is weakened, even if g_s and g_c are large. This reduction in effective coupling explains why, in our simulations with $g_s > 2000$ pA, the two-compartment correlation $\rho(V_s, V_d)$ decreases as τ_s decreases.

However, in the region where $g_s < 2000$ pA, the simulated curves deviate from the simple trends predicted by the above qualitative arguments. To understand this, we first consider the blue and orange curves, which correspond to larger values of τ_s . When τ_s is large, the somatic voltage V_s returns only slowly to the leak potential E_L . In this case, voltage changes produced by the internal coupling terms g_s , g_c , and c_d , as well as by the external input currents I_s and I_d , are less strongly damped and can accumulate over time, so all of these mechanisms have a substantial impact on the observed correlation.

Notably, the intrinsic correlation between I_s and I_d also contributes to the observed correlation between V_s and V_d . In the simulations, we quantify this input correlation as the Pearson correlation coefficient between the time series $I_s(t)$ and $I_d(t)$, which are generated from phase-shifted deterministic square-wave inputs, as illustrated in Fig. 3.1. In addition to this input-driven component, $\rho(V_s, V_d)$ receives a further contribution from the soma-dendrite coupling terms g_s , g_c , and c_d . These two sources of correlation do not simply add linearly: depending on the parameter values, the coupling-induced correlations can either reinforce or partially cancel the correlations arising from the shared inputs, which may account for the non-monotonic behavior observed for $g_s < 2000$ pA.

Under this hypothesis, when g_s is very small ($g_s < 300$ pA), the correlation $\rho(V_s, V_d)$ is dominated by the shared external inputs: most of the voltage correlation simply reflects the correlation between $I_s(t)$ and $I_d(t)$. As g_s increases from zero, the internal dendrite-to-soma coupling term $g_s f(V_d)$ starts to contribute to the voltages. In this parameter range, the correlation component generated by the intrinsic coupling interacts with the input-driven component, and their combined effect leads to a decrease in the total correlation, producing the initial dip seen in the red and green curves. When g_s is increased further, the contribution from internal soma-dendrite coupling becomes dominant, while the statistics

of the external inputs I_s and I_d are kept fixed, so $\rho(V_s, V_d)$ rises again with g_s .

For the purple curve, which represents a very small τ_s , the membrane voltage recovers quickly toward the leak potential E_L . This strong recovery effect suppresses voltage fluctuations, making it harder for the somatic and dendritic voltages V_s and V_d to become correlated. As a result, when g_s increases, its influence on the voltage correlation is initially limited. The correlation $\rho(V_s, V_d)$ rises slowly at first because g_s must overcome the effect of this fast recovery. However, once g_s becomes large enough to overcome the effect of recovery, the internal coupling between compartments strengthens and begins to dominate. At that point, this internal interaction contributes more to the voltage correlation than the shared input signals do, leading to a more rapid increase in the correlation between soma and dendrite, quantified by $\rho(V_s, V_d)$.

3.4 Conclusion from the burst propagation model

The [BPM](#) describes how [s-d](#) coupling shapes a neuron’s ability to multiplex inputs. In this model, two coupling parameters control the interaction between soma and dendrite. The parameter g_s represents active dendrite-to-soma coupling and captures the influence of dendritic depolarization on somatic voltage. The parameter c_d represents active soma-to-dendrite coupling and captures the effect of back-propagating action potentials ([bAPs](#)) on dendritic voltage. Together, these couplings determine how somatic and dendritic inputs are converted into isolated spikes and bursts in the output spike train, and thus how event rate ([ER](#)) and burst fraction ([BF](#)) can carry different information.

Simulations show that multiplexing is supported only in an intermediate range of c_d . When c_d is very small, [bAPs](#) have little influence on the dendrite. Somatic spikes then mainly reflect somatic input, and they do not reliably indicate joint activation of soma and dendrite. As c_d increases from this weakly coupled regime, bursts become more strongly associated with combined somatic and dendritic drive. In this regime, bursts tend to occur when both somatic and dendritic inputs are strong, whereas isolated spikes are still driven mainly by

somatic input. Varying somatic input strength produces a clear change in the rate of isolated spikes, while varying dendritic input has only a weak effect on them. Thus, the part of **ER** formed by isolated spikes primarily reflects somatic drive, whereas **BF** depends more strongly on dendritic input and on both somatic and dendritic inputs being strong at the same time. The multiplexing metric therefore increases because **ER** and **BF** carry more distinct information about the two inputs. For large c_d , however, **bAPs** strongly affect dendritic voltage, reduce dendritic independence, and make the distinction between somatic-driven spikes and dendritic-driven bursts less clear.

A similar pattern appears when varying g_s . For very small g_s , dendritic nonlinear events have little effect on somatic voltage and rarely trigger bursts. The output then behaves almost like a simple rate code for somatic input. When g_s is increased, strong dendritic depolarizations more easily produce bursts at the soma. Dendritic input is then better reflected in **BF**, and multiplexing improves. If g_s is too large, dendritic activity dominates somatic dynamics and the soma and dendrite behave almost as a single compartment. In this strongly coupled regime, the neuron can no longer represent soma-specific and dendrite-specific information in separable features of the spike train.

These effects are also visible in the **s-d correlation**, quantified as $\text{Corr}(V_s, V_d)$. We use this correlation as a proxy for compartmentalization. When g_s and c_d are small, $\text{Corr}(V_s, V_d)$ is low, which indicates strong compartmentalization but weak interaction and coupling, and therefore poor multiplexing. As coupling increases, $\text{Corr}(V_s, V_d)$ grows and multiplexing improves, reaching its maximum at moderate correlation. For very strong coupling, $\text{Corr}(V_s, V_d)$ becomes high and multiplexing breaks down because the two compartments become too similar in their voltage fluctuations. Across all simulated parameter sets, higher $\text{Corr}(V_s, V_d)$ systematically corresponds to lower multiplexing performance, indicating a clear negative relationship between **s-d correlation** and the multiplexing metric.

The parameter p , introduced in Section 3.3, allows us to interpret these results in terms of impedance mismatch between soma and dendrite. In our two-compartment formulation, the axial current is

$$I_{\text{ax}} = g_{\text{ax}}(V_d - V_s).$$

The coupling terms in the voltage equations can be written as

$$\dot{V}_s \supset \frac{g_c}{p}(V_d - V_s), \quad \dot{V}_d \supset \frac{g_c}{1-p}(V_s - V_d),$$

with $0 < p < 1$ and $p + (1 - p) = 1$.

We choose

$$p = \frac{C_s}{C_s + C_d}, \quad 1 - p = \frac{C_d}{C_s + C_d}.$$

With this choice, the coupling terms can be written as $(g_c/p)(V_d - V_s)$ and $(g_c/(1-p))(V_s - V_d)$. The same coupling conductance g_c then appears with two different coefficients, g_c/p and $g_c/(1-p)$, so that a given voltage difference between soma and dendrite changes V_s and V_d by different amounts. In a short-time, capacitively dominated regime, the voltage change produced by the soma-dendrite coupling term is inversely proportional to the compartmental capacitance, so that $\Delta V \propto 1/C$. The effective membrane impedance is therefore inversely proportional to capacitance, and the impedance ratio between soma and dendrite satisfies

$$\frac{C_d}{C_s} = \frac{1-p}{p}.$$

A small value of p thus corresponds to a larger effective impedance at the soma, whereas a large value of p corresponds to a smaller somatic impedance.

In this view, the coupling strengths that optimize multiplexing correspond to a moderate impedance mismatch between soma and dendrite, neither very small nor very large. If the mismatch is very strong, one compartment dominates and the other has little influence on the output. If the mismatch is very weak, soma and dendrite behave almost identically and cannot support two distinct channels. For moderate values of p and moderate coupling strengths (g_s, c_d) , the neuron retains enough compartmentalization to keep [ER](#) and [BF](#) partially independent, while the coupling is still strong enough for dendritic input to exert a substantial influence on burst generation at the soma.

Under these conditions, the [BPM](#) demonstrates that a single neuron can efficiently multiplex two input streams within one spike train.

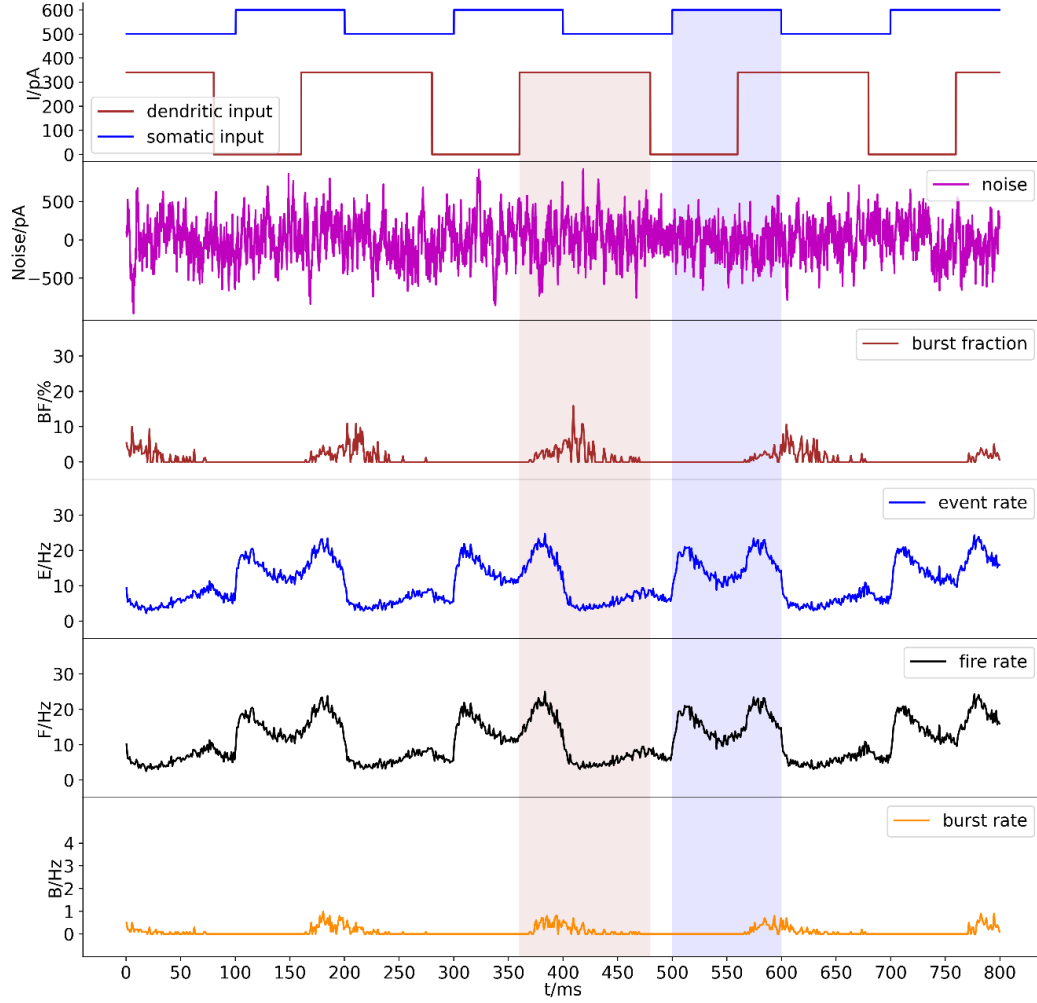


Figure 3.2: Example time series illustrating multiplexing in the BPM. Top: alternating square-wave inputs to the soma (I_s) and dendrite (I_d) with independent noise. Bottom: population readouts computed from an ensemble of 10,000 two-compartment neurons, including burst fraction (BF), event rate (ER), firing rate (FR), and burst rate (BR). The BF reflects the alternating dendritic input. The ensemble ER reflects the alternating somatic input.

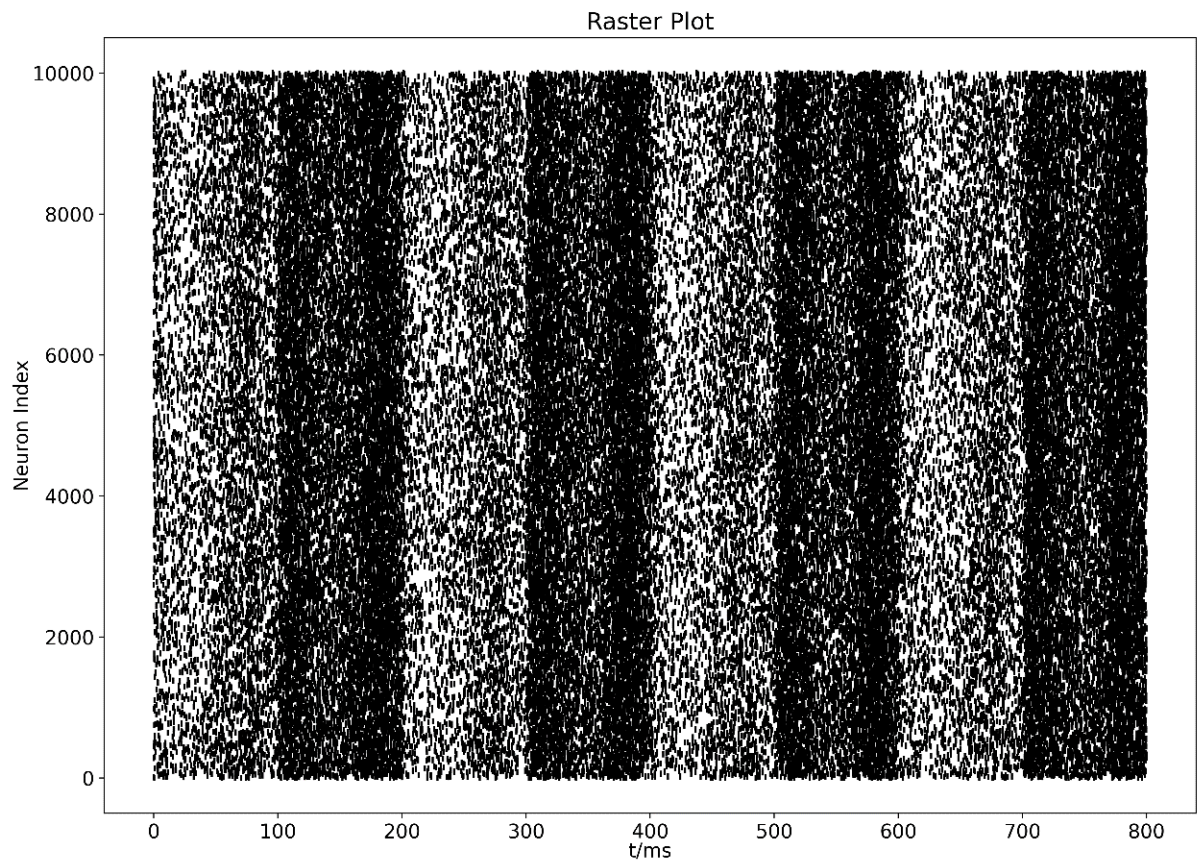


Figure 3.3: A raster plot represents the density of spikes and bursts. The y-axis is the neuron number. Each spike is recorded as a point. The regions with dense points are more likely to contain bursts, and regions with sparse points are more likely to correspond to isolated spikes.

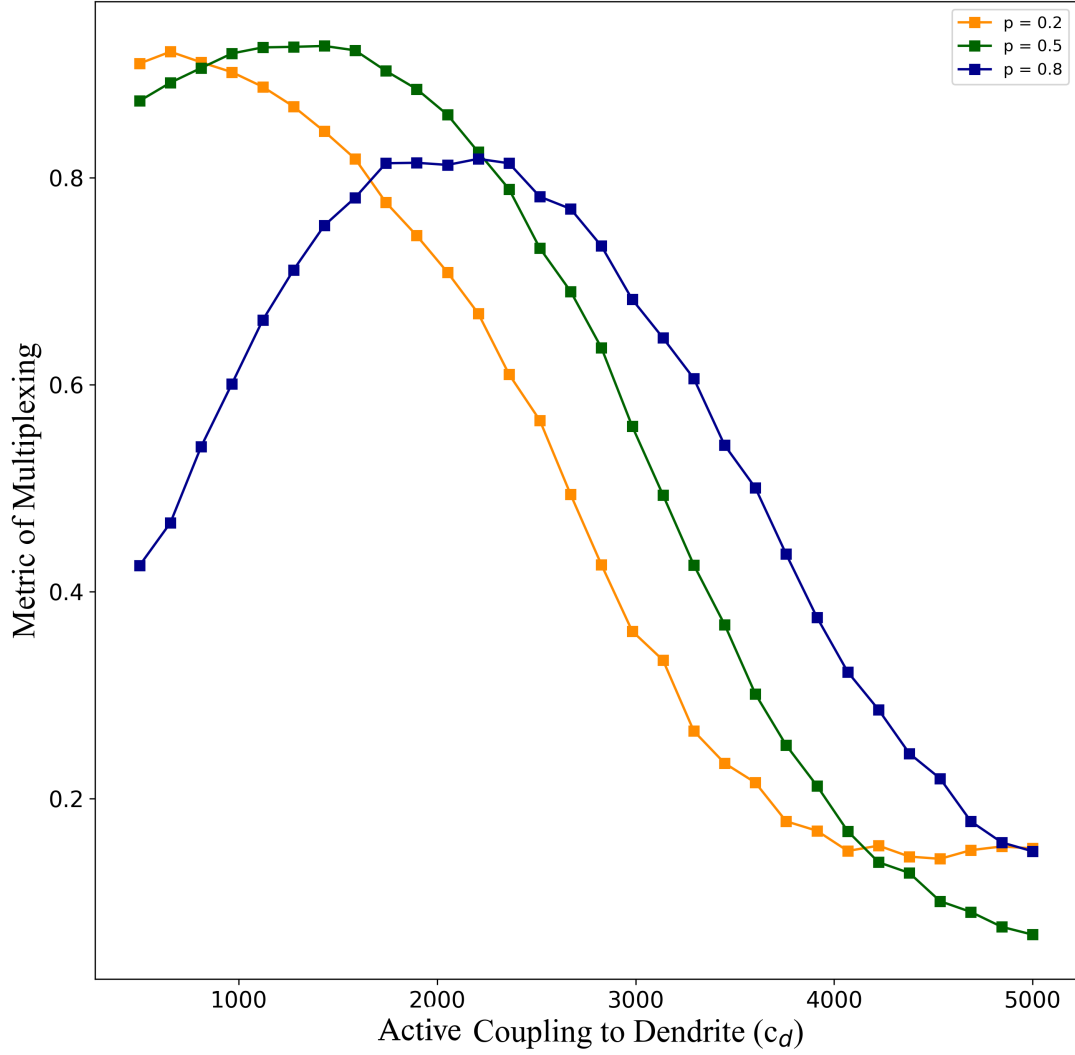


Figure 3.4: Relationship between multiplexing and active coupling to dendrite. A computer simulation experiment illustrates the metric of multiplexing against c_d in pA. The figure displays three data curves, each corresponding to different values of p , where p denotes the impedance mismatch. Specifically, the values $p=0.2$, 0.5 , and 0.8 are represented. The colors (orange for $p=0.2$, blue for $p=0.5$, and green for $p=0.8$) distinguish between the respective data series.

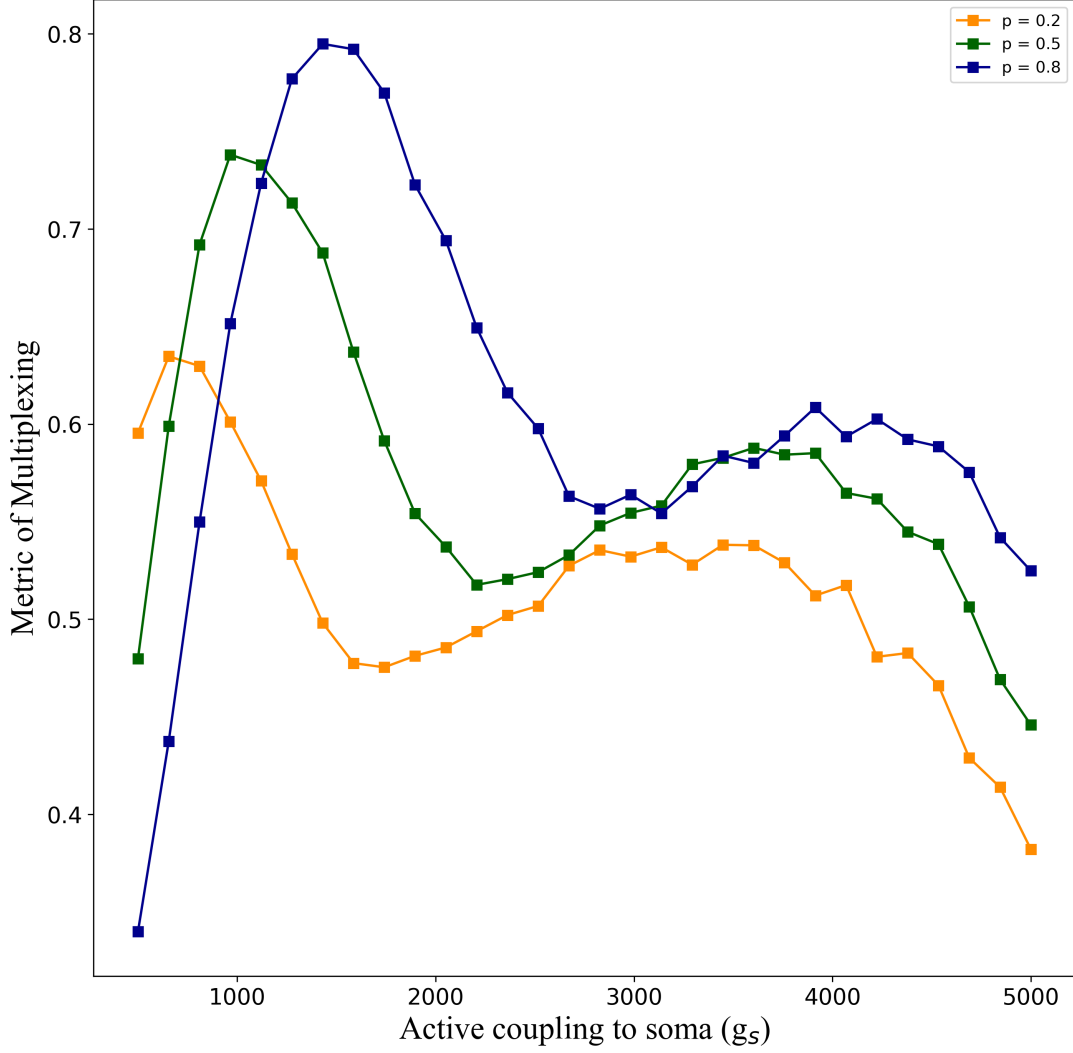


Figure 3.5: Relationship between multiplexing and active coupling to soma. A computer simulation experiment illustrates the multiplexing metric as a function of g_s (pA). There are three curves representing three different ratios of membrane areas ($p = 0.2, 0.5$, and 0.8). Each curve shows a distinct peak, suggesting an optimal g_s value for each p at which the multiplexing metric is maximized. The multiplexing metric exhibits a broad maximum at intermediate g_s , indicating that multiplexing is optimized at moderate active coupling rather than at very weak or very strong g_s . After reaching the peak, the metric decreases with increasing g_s , which may indicate oversaturation or reduced multiplexing efficiency at higher g_s levels.

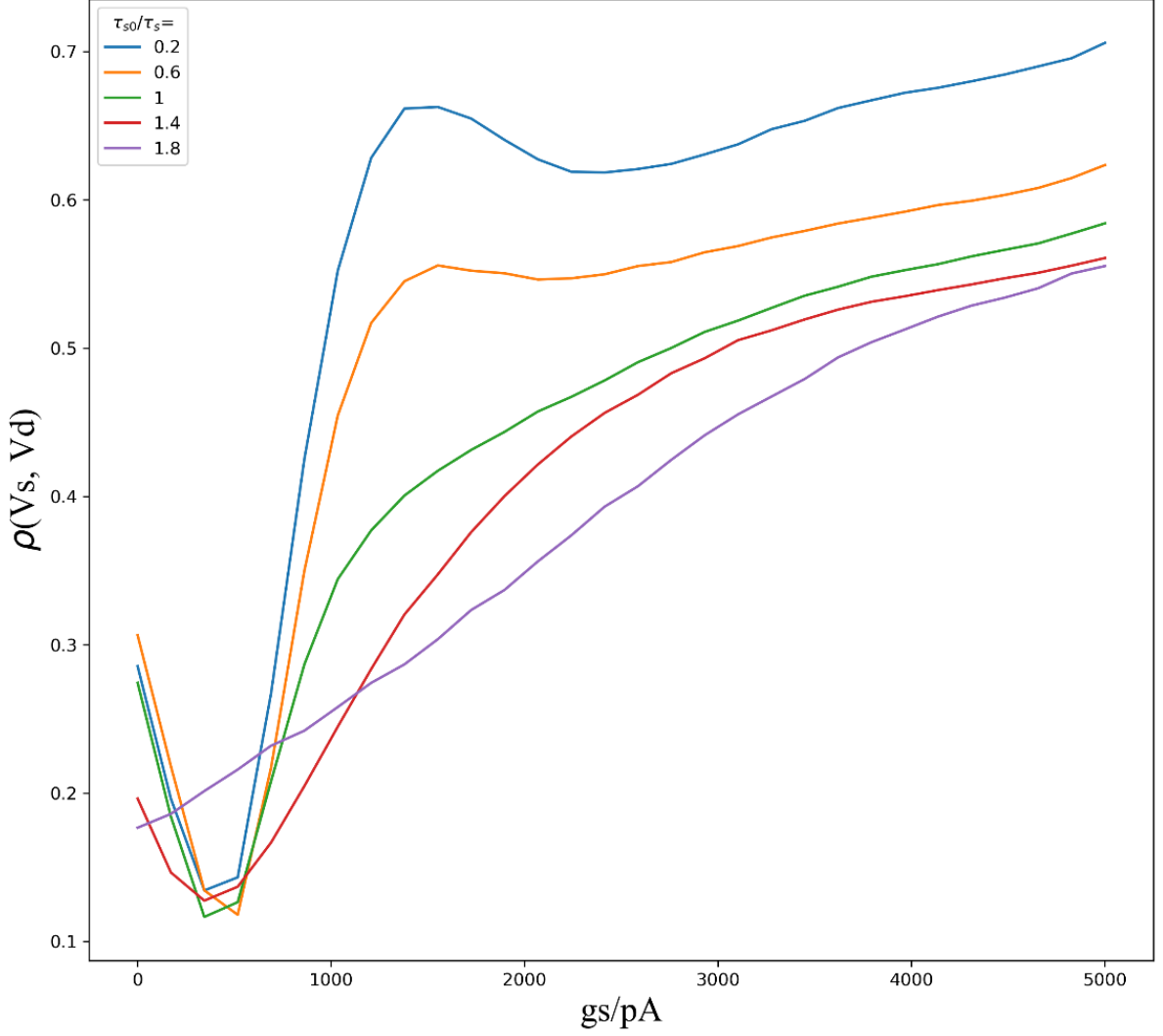


Figure 3.6: Relationship between soma-dendrite voltage correlation and active coupling to the soma. Computer simulation results show the voltage correlation coefficient between the soma and dendrite as a function of g_s (pA). Different colors represent curves obtained for different τ_s values. Here, $\tau_{d0} = 7$ ms and $\tau_{s0} = 16$ ms are kept constant as reference values. As τ_s increases, the most notable changes are as follows: first, a local minimum appears around $g_s = 300$ pA, forming a trough across the red, green, orange, and blue curves; later, this feature develops into a local maximum around $g_s = 1400$ pA, forming a crest in the orange and blue curves.

Chapter 4

Passive Model

We defined a quantitative multiplexing metric that summarizes how efficiently a single neuron reuses its spike train to represent two input streams in Chapter 3, but it is difficult to derive a closed-form expression for this index directly from the [BPM](#). To address this, we derive an approximate predictor expression for multiplexing from the passive model, rather than a direct event-based multiplexing readout. The first question is whether the derived expression can explain the relationship between multiplexing and compartmentalization within the passive model itself. The second question is whether this expression can at least qualitatively account for the multiplexing patterns observed in the [BPM](#). In the remainder of this chapter, we show that the passive model and its associated expressions could express the basic dynamics of signal integration and processing, as well as the effects of noise, phase shift, somatic input, and coupling conductances on multiplexing performance. Analytical derivations are presented in Appendix B.

4.1 Theory

Model view. The neuron is represented by two passive compartments: a dendrite and a soma. Each compartment has a leak term that relaxes its voltage toward the same leak reversal potential V_L . The compartments are coupled through an axial conductance g_c , which drives the voltages toward each other when $V_s \neq V_d$. External inputs $I_s(t)$ and $I_d(t)$ act on the soma and dendrite, respectively, and each compartment receives an independent stochastic drive (Gaussian noise). The parameter $p \in (0, 1)$ allocates the total membrane capacitance between the two compartments, so that the effective capacitances are pC_m for the soma and $(1 - p)C_m$ for the dendrite. In this density form, the inputs and coupling currents are scaled by $1/p$ and $1/(1 - p)$ in the current-balance equations.

Mechanistic intuition. In the passive model, soma–dendrite correlation arises from a balance between two simple effects. First, axial coupling promotes coordination: if one compartment is driven upward by input or noise, coupling tends to pull the other compartment in the same direction. This increases $\text{Corr}(V_s, V_d)$ as g_c grows. Second, independent inputs and independent noise promote differences between compartments. If the soma and dendrite are driven by different waveforms, or if their noise sources are uncorrelated, then V_s and V_d can fluctuate independently, lowering correlation. The capacitance split p further shapes these effects by setting how strongly each compartment responds to the same current density: smaller effective capacitance leads to faster voltage changes and stronger sensitivity to input and noise.

Readouts and metrics (concept). We use the Pearson correlation coefficient between the two voltage traces to quantify compartmentalization:

$$\text{Compartmentalization metric} = \text{Corr}(V_s, V_d).$$

Because the passive model is linear and subthreshold, it does not generate spikes or bursts. Therefore, it cannot produce event-based readouts such as ER or BF directly. Instead, the

passive model is used as a baseline to interpret how coupling, phase alignment between $I_s(t)$ and $I_d(t)$, and noise intensity shape voltage coordination. In the next section, we validate the theory prediction of $\text{Corr}(V_s, V_d)$ against discrete-time Euler simulations.

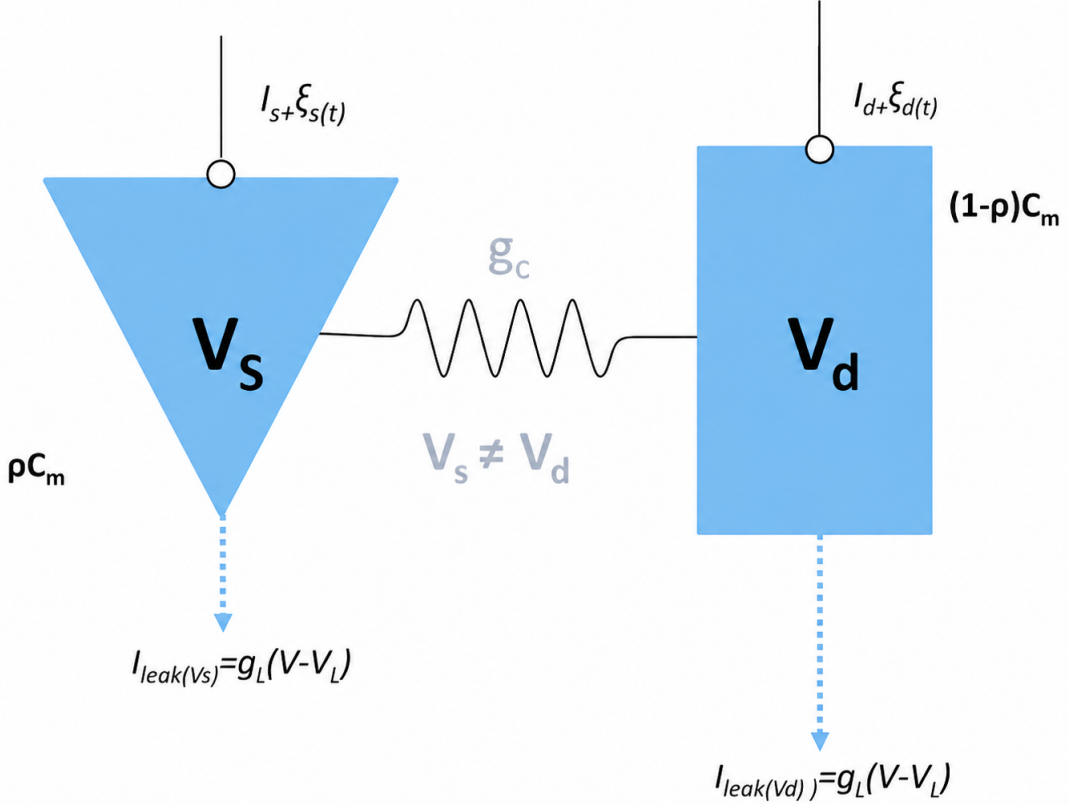


Figure 4.1: Schematic of the passive model. This diagram illustrates the structure of a canonical two-compartment model, where a somatic compartment (left) with membrane voltage V_s and area A_s receives somatic input current I_s , and a dendritic compartment (right) with voltage V_d and area A_d receives dendritic input I_d . The compartments are electrically coupled via a conductance g_c , representing the cytoplasmic and membrane properties of the connecting segment. Each compartment exhibits passive leak currents $I_{leak}(V_s)$ and $I_{leak}(V_d)$, which contribute to voltage dissipation. The inequality $V_s < V_d$ indicates a momentary voltage gradient driving current through the coupling pathway [23].

4.2 Numerical implementation and discrete-time simulation

To validate the passive-model expressions, we performed discrete-time simulations of the linear two-compartment system in Python. The membrane voltages were updated using an explicit Euler scheme with time step $\Delta t = 0.1$ ms and total duration $T = 600$ ms per run [12]. The voltages were initialized at the leak level V_L .

Inputs and phase shift. The soma and dendrite received periodic square-wave inputs with period $T_0 = 200$ ms. Each square wave switches between a low level (baseline) and a high level (amplitude). The soma duty cycle was set to $\eta_s = 0.5$, and the dendrite duty cycle was set to $\eta_d = 0.6$ (because the low segment occupies 0.4 of the period). A phase shift $\phi \in [0, 1]$ controlled the relative alignment between the two inputs. In the code, the phase shift was implemented by shifting the dendrite waveform index within each period and applying a modulo operation to keep the signal periodic.

Noise (Gaussian white noise). To test robustness under stochastic drive, independent Gaussian increments were used in the Euler discretization of white-noise drive. Specifically, at each step we drew $z_s, z_d \sim \mathcal{N}(0, \sigma^2)$ and added noise increments scaled by $\sqrt{\Delta t}$, so that noise strength scales appropriately with the simulation time step.

Discrete-time update. Using the abbreviations $r_1 = g_L/C_m$, $a = 1/(pC_m)$, and $b = 1/((1-p)C_m)$, the Euler updates take the form

$$V_s(t + \Delta t) = V_s(t) + \Delta t \left[-r_1(V_s(t) - V_L) + ag_c(V_d(t) - V_s(t)) + aI_s(t) \right] + a\sqrt{\Delta t} z_s(t), \quad (4.1)$$

$$V_d(t + \Delta t) = V_d(t) + \Delta t \left[-r_1(V_d(t) - V_L) + bg_c(V_s(t) - V_d(t)) + bI_d(t) \right] + b\sqrt{\Delta t} z_d(t). \quad (4.2)$$

Estimating correlation. We quantified compartmentalization using the Pearson correlation coefficient between $V_s(t)$ and $V_d(t)$. To avoid dependence on initial conditions, we discarded the first 200 ms (2000 steps) before computing correlation.

Parameter sweeps and visualization. We performed two-dimensional parameter sweeps by selecting two parameters as axes (e.g., phase shift versus noise level, or phase shift versus input amplitude) and holding the remaining parameters fixed. For each grid point, we computed the simulated voltage correlation and compared it to the theory prediction. Heatmaps were saved for the theory correlation, simulated correlation, and their absolute difference.

Derived correlation expressions and their numerical evaluation. Based on the passive model, we use the following derived expressions to summarize how correlations depend on input alignment, input strength, and noise level. Full derivation shows in Appendix B, here the notation “ $:=$ ” indicates dependence under the assumptions of the linear model and the fixed square-wave shapes used in simulation:

$$\rho(I_s, I_d) := \phi \tag{4.3}$$

$$\rho(\tilde{I}_s, \tilde{I}_d) := \phi \tag{4.4}$$

$$\rho_{\text{Gaussian-noise}}(V_s, V_d) := \phi, A_s, A_d, \sigma^2 \tag{4.5}$$

$$\rho_{\text{OU-noise}}(V_s, V_d) := \phi, A_s, A_d, \sigma^2 \tag{4.6}$$

$$\rho(V_s, V_d) := \phi, \frac{A_s}{\sigma}, \frac{A_d}{\sigma} \tag{4.7}$$

Meaning of the input-correlation term. In the passive-model derivation, the first quantity of interest is the correlation between the two compartment inputs, $\rho(I_s, I_d)$. Here $I_s(t)$ and $I_d(t)$ are periodic square-wave signals with the same period T_0 and fixed duty cycles. The only difference between them is a relative phase shift $\phi \in [0, 1]$, which controls how often the high (or low) segments of the two waveforms overlap in time. For these fixed

waveform shapes, the Pearson correlation between $I_s(t)$ and $I_d(t)$ is therefore determined solely by the phase shift, which is summarized by $\rho(I_s, I_d) := \phi$. Intuitively, $\rho(I_s, I_d)$ measures input alignment across compartments: it is large when the two inputs rise and fall together, and smaller when they are out of phase.

Amplitude and mean removal. We write the inputs as

$$I_s(t) = I_{s,\text{base}} + A_s q\left(\frac{t}{T_0}\right), \quad I_d(t) = I_{d,\text{base}} + A_d q\left(\frac{t}{T_0} - \phi\right),$$

where $q(\cdot)$ is a period-1 square wave that takes values in $\{0, 1\}$ (0 during the low segment and 1 during the high segment), with duty cycles matched to the simulation settings. The quantities A_s and A_d are the high–low differences of the square waves. The mean-removed signals are $\tilde{I}(t) = I(t) - \langle I \rangle$, where $\langle \cdot \rangle$ denotes the time average over the analyzed window (equivalently, over one period for stationary periodic inputs).

Voltage correlation under Gaussian white noise (this verification code). The quantity $\rho_{\text{Gaussian-noise}}(V_s, V_d)$ denotes the Pearson correlation between $V_s(t)$ and $V_d(t)$ when the passive model is driven by independent Gaussian white noise in the two compartments. In the code, the noise samples are generated as $z_s, z_d \sim \mathcal{N}(0, \sigma^2)$ at each time step and are scaled by $\sqrt{\Delta t}$ in the Euler updates. The simulated correlation is computed as $\rho_{\text{sim}} = \text{Corr}(V_s, V_d)$ after discarding an initial transient segment (first 200 ms).

Normalized dependence. In the linear model, $\text{Corr}(V_s, V_d)$ depends mainly on how large the deterministic inputs are relative to the noise level. This is why the ratios A_s/σ and A_d/σ are informative: if we scale both input amplitudes (A_s, A_d) and the noise intensity σ by the same factor, the voltage correlation remains unchanged. In contrast, changing A_s/σ or A_d/σ changes the balance between input-driven and noise-driven fluctuations and therefore changes the correlation.

Comparison between theory and simulation. Figures 4.2–4.5 compare the derived prediction ρ_{theory} with the simulated correlation ρ_{sim} over two-dimensional parameter sweeps. Each heatmap entry reports $\text{Corr}(V_s, V_d)$ for a given parameter pair (e.g., phase shift and noise intensity). We also report the error map $\Delta\rho = |\rho_{\text{sim}} - \rho_{\text{theory}}|$ to visualize discrepancies between the theory expression and the numerical simulation.

4.3 Simulation experiment and data collection

The simulations in this section were used to validate the analytical expression for [s-d voltage correlation](#) derived in Section 4.2 [derived correlation expressions](#) and Appendix B. Accordingly, Figures 4.2–4.5 follow the same three-panel structure.

For each two-dimensional parameter sweep, the first panel shows the analytical prediction by [derived correlation expressions](#), denoted by ρ_1 ; the second panel shows the voltage correlation obtained directly from numerical simulation of the passive model, $\rho_2 = \text{Corr}(V_s, V_d)$; and the third panel shows the absolute difference between them,

$$\Delta\rho = |\rho_1 - \rho_2|. \quad (4.8)$$

Thus, the three panels are not independent measures. Rather, they show the analytical prediction, its numerical verification, and the corresponding approximation error, respectively. Figures 4.2–4.5 repeat this comparison while varying different pairs of model parameters.

In the first simulation, shown in Figure 4.2, we examined how the predicted and simulated voltage correlations vary jointly with dendritic input amplitude and noise intensity. In both the theoretical prediction and the numerical simulation, increasing the noise intensity generally reduces the correlation between the somatic and dendritic voltages, consistent with the idea that stronger independent noise decorrelates the two compartments. The dependence on dendritic input amplitude is non-monotonic: the correlation surface suggests

that there is an intermediate range of dendritic input for which the predicted voltage coordination is strongest, whereas both weaker and stronger inputs produce lower correlation.

The error map indicates that the discrepancy between theory and simulation is largest in the high-noise regime. This is expected, because stochastic fluctuations introduce greater variability into the simulated voltage trajectories than into the deterministic part of the theoretical approximation. Nevertheless, the overall error remains small ($\Delta\rho \ll 0.1$), indicating that the derived passive-model expression captures the main structure of the simulated correlation surface with good accuracy over the parameter range considered. Thus, Figure 4.2 supports the conclusion that, within the passive model, the theory provides a reliable approximation to how soma–dendrite voltage correlation depends on dendritic input strength and noise intensity.

In Figure 4.3, as conductance increases, voltage correlation becomes higher and compartmentalization decreases.

In the left panel, the analytic expression derived from the linear passive model predicts that the multiplexing metric decreases as the soma–dendrite coupling conductance g_c increases. Over the range of g_c shown, stronger coupling makes the somatic and dendritic voltages more similar, which reduces compartmentalization and therefore lowers multiplexing. For a fixed value of g_c , increasing the phase difference φ between the somatic and dendritic square-wave inputs tends to increase the multiplexing metric, but this effect is pronounced only for large phase shifts. In other words, in the linear model, weakening coupling improves multiplexing, and this improvement can be partially recovered at strong coupling only when the inputs to soma and dendrite are sufficiently out of phase.

The right panel shows numerical simulations of the same linear passive model. These simulations display the same qualitative trend: the multiplexing metric decreases with increasing coupling conductance g_c . However, the absolute values of the multiplexing metric are lower than those observed in the [BPM](#). This suggests that the linear cable approximation captures the correct direction of the effect of coupling on multiplexing, but underestimates the multiplexing capacity that can be achieved in the full nonlinear

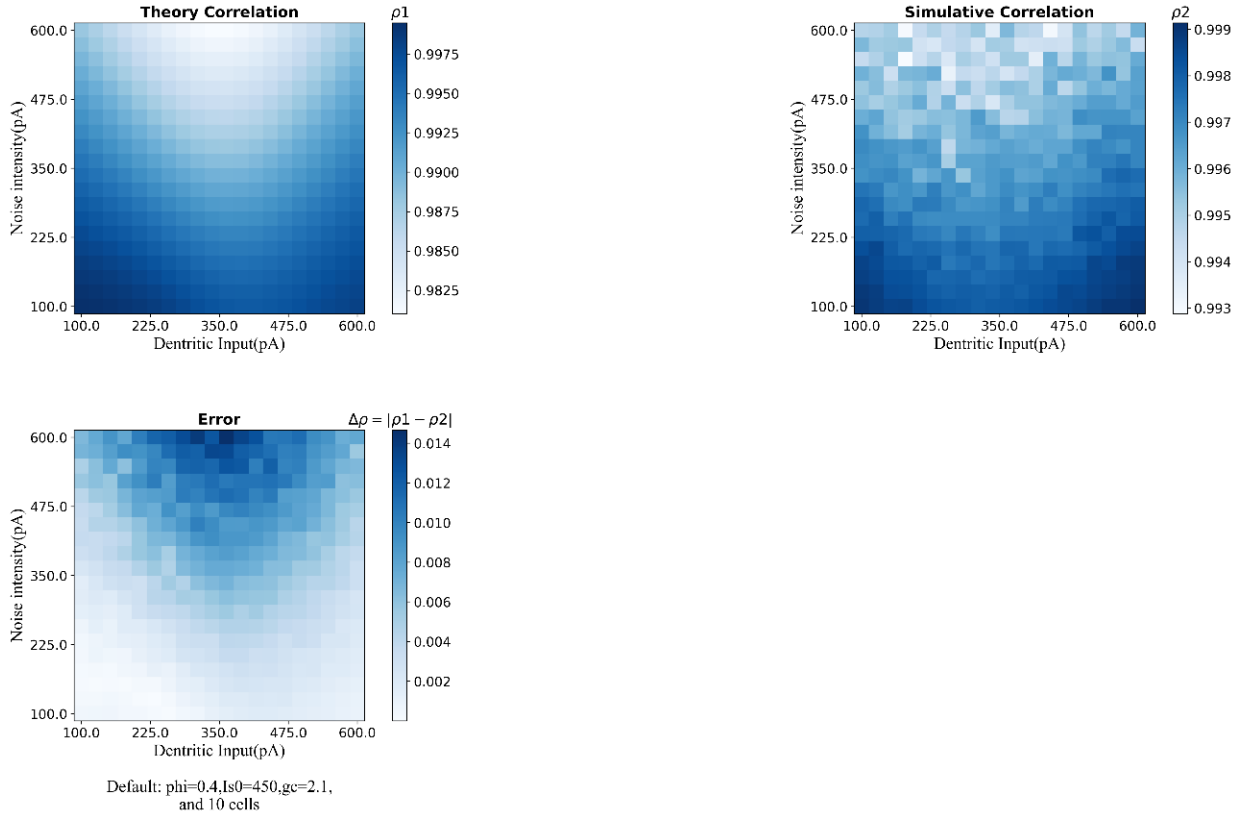


Figure 4.2: Comparison of derived approximation to the multiplexing metric as a function of dendritic input and noise intensity (left), derived from custom theoretically derived formulas derived in Section 4.2 [derived correlation expressions](#) and Appendix B. and classical passive theory simulations (right), along with an error comparison (bottom). (Top Left): Theoretical correlation values (ρ_1) derived from proposed multiplexing formula, designed to model neuronal multiplexing capacity based on input strength and noise. (Top Right): Corresponding correlations (ρ_2) from passive theory simulations show similar overall trends, though with greater variability. (Bottom): The error map ($\Delta\rho = |\rho_1 - \rho_2|$) quantifies the deviation between the theoretical and simulated models, with minor discrepancies concentrated in high-noise regimes. These results suggest that as noise intensity increases, theoretical and simulated correlations generally decrease slightly, with moderate discrepancies shown in the error map.

burst-based model.

The error plots show that the error increases with increasing levels of coupled conductance and phase shift, suggesting that the soma isolation predicted by theory is not fully achieved

in the simulations and that my equations may not fully reflect the multiplexing-related behavior in the highly coupled case and the effects of phase interactions.

In the theoretical derivation of Figure 4.4, $\rho(V_s, V_d)$ correlation decreases steadily with somatic input, while simulated correlation is less structured, possibly indicating sensitivity to somatic input at higher values of phase shift.

The soma of a neuron is not very efficient at processing a series of simultaneous current inputs across the soma, although high phase shifts enhance this ability. This reflects a trade-off between input amplitude and temporal coherence in multiplexing.

Passive theory simulations show a more diffuse pattern, with a more erratic decrease in the degree of multiplexing as soma inputs become higher and the phase difference of current inputs decreases. This suggests that the effect of I_s on multiplexing is less direct in the passive model, perhaps due to the linear assumption of input integration.

The errors are particularly pronounced at higher phase shifts, which may highlight the need for more complex temporal dynamic modeling in passive theory to accurately represent somatic multiplexing.

In Figure 4.5, as the noise intensity increases, the correlation starts to decrease. The degree of multiplexing is robust to different noise intensities, especially for low to moderate phase shifts. This means that even in the presence of noise, as long as phase coherence is maintained, under this simulation, my equation can achieve relatively stable multiplexing performance in the soma.

4.4 Insights into multiplexing from the passive model

In this chapter, we introduced a linear passive model to better quantify multiplexing and compartmentalization within neurons. By deriving analytical expressions based on passive

theory, we provided a simplified yet effective mechanism to describe how key neuronal parameters, such as coupling conductance, noise intensity, somatic input currents, and input phase shifts, affect multiplexing-related behavior. Our results show that the derived formulas match well with numerical simulations, confirming their ability to accurately capture key aspects of neuronal dynamics.

Through simulation experiments, we found that multiplexing performance generally declines with increased coupling conductance, supporting the prediction that moderate s-d coupling enhances the neuron’s ability to independently process multiple signals. Additionally, our analyses highlight an optimal range of dendritic input current, where multiplexing peaks before diminishing, particularly in high-noise environments. Phase coherence acts as a critical factor: moderate-to-high phase shifts between dendritic and somatic inputs influence multiplexing efficiency and somatic signal stability.

Finally, our theoretical (eq (4.3-4.7)) and simulated correlation comparisons consistently demonstrated high accuracy and reliability, with minor deviations under extreme noise and coupling conditions. These findings underscore the passive model’s effectiveness in describing compartmentalized signal integration and multiplexing, providing a robust theoretical foundation for understanding how neurons manage complex signaling scenarios.

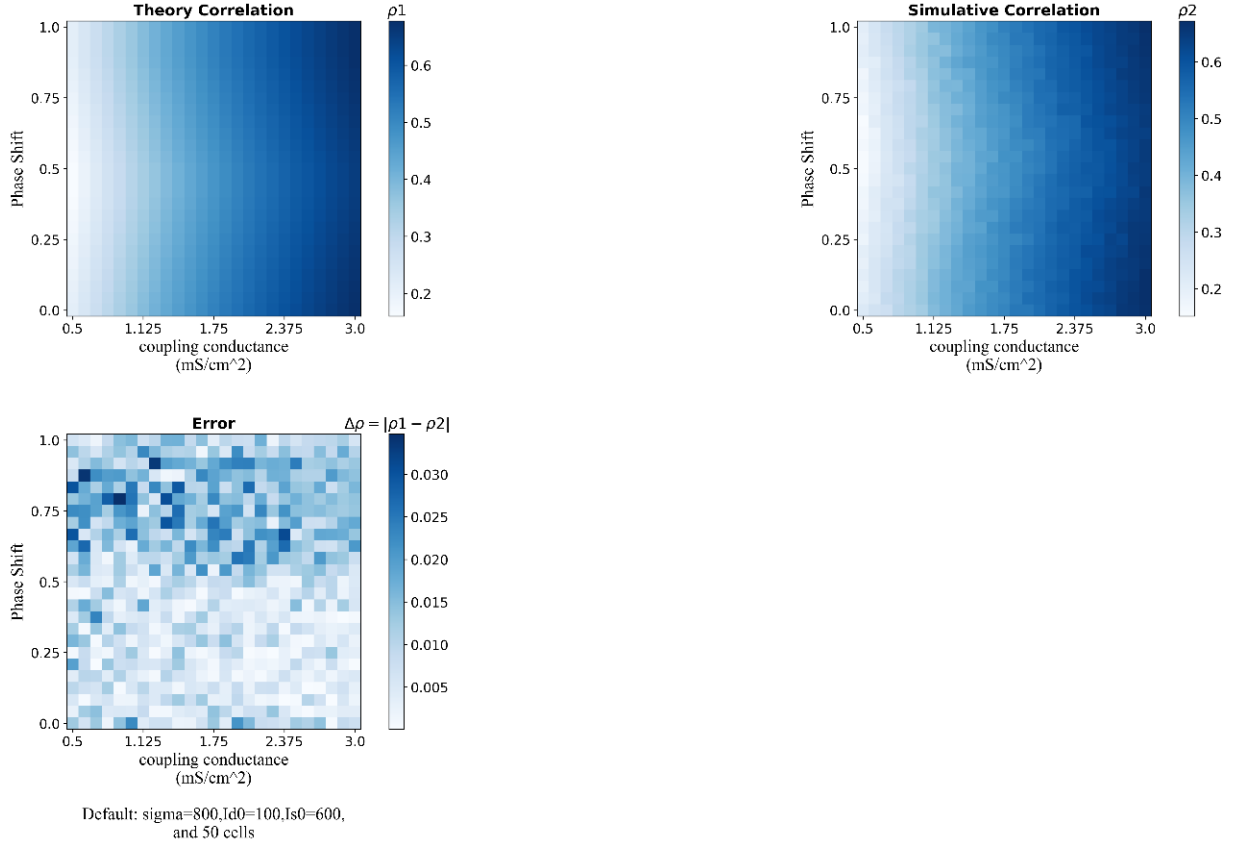


Figure 4.3: Comparison of derived approximation to the multiplexing metric patterns generated using a custom theoretical derived formulas (left panels) and classical passive theory simulations (right panels) across varying coupling conductance and phase shift parameters. Top Left: Correlation coefficients (ρ_1) predicted by the proposed multiplexing formula, derived in Section 4.2 [derived correlation expressions](#) and Appendix B. Top Right: Correlation coefficients (ρ_2) computed using theory simulations. Bottom Left: Absolute error map ($\Delta\rho = |\rho_1 - \rho_2|$) quantifying the difference between theoretical and simulative predictions, showing strong alignment with minor deviations at higher phase shifts.

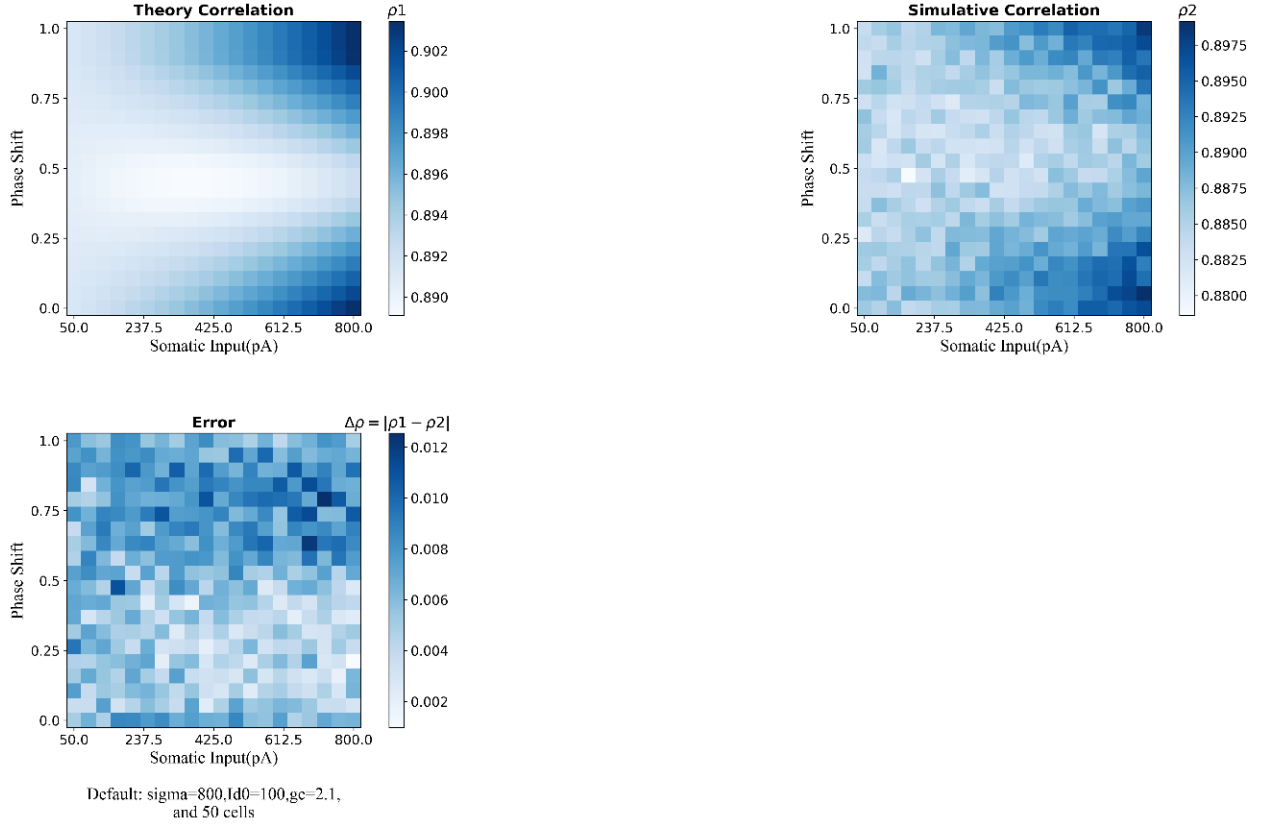


Figure 4.4: Somatic input-dependent modulation of derived approximation to the multiplexing metric under phase shift variations: comparison between a theoretical derived formula and classical passive theory simulations. (Top Left): Theoretical correlation (ρ_1) derived in Section 4.2 [derived correlation expressions](#) and Appendix B, calculated using a proposed multiplexing formula reveals a smooth, phase-sensitive modulation pattern. Multiplexing capacity increases with somatic input but declines under high phase shift conditions, reflecting diminished temporal coherence in signal integration. (Top Right): Correlation values (ρ_2) from passive theory simulations exhibit similar trends, though with increased irregularity, particularly at moderate to high phase shifts. This may reflect the limitations of passive theory in capturing phase-sensitive somatic dynamics. (Bottom): The absolute error map ($\Delta\rho = |\rho_1 - \rho_2|$) shows that the greatest deviations between theory and simulation occur under conditions of high phase shift, supporting the conclusion that phase coherence is a critical factor in somatic multiplexing performance.

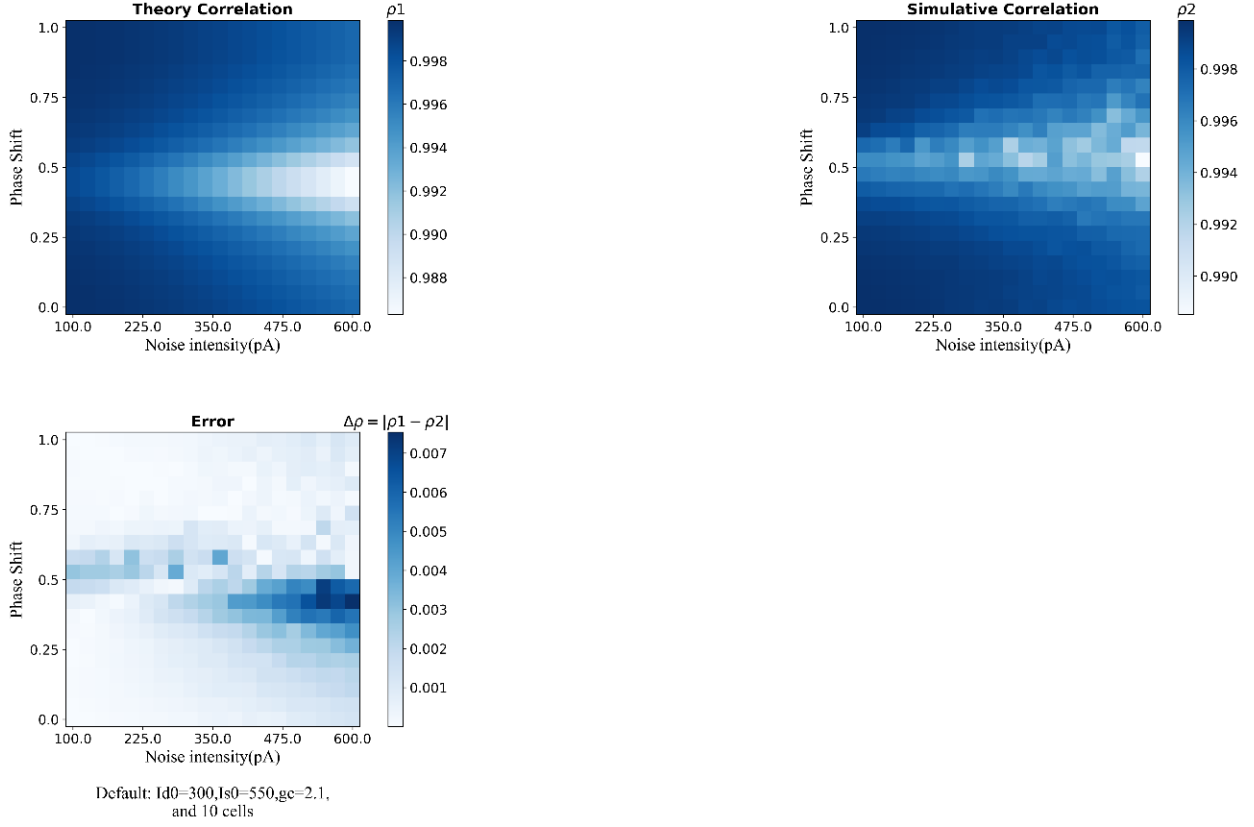


Figure 4.5: Impact of noise intensity and phase shift on derived approximation to the multiplexing metric: comparison between a theoretical derived formula (left panels) and classical passive theory simulations (right panels), with error quantification (bottom). (Top Left): Theoretical correlation (ρ_1) computed in Section 4.2 [derived correlation expressions](#) and Appendix B shows a narrow band of reduced correlation centered around phase shift ≈ 0.5 , with multiplexing remaining high across varying noise intensities. (Top Right): passive theory simulations (ρ_2) reveal a qualitatively similar structure, though slightly more sensitive to fluctuations in both phase shift and noise. This reflects the classical model’s treatment of signal interference under phase asynchrony, especially under stochastic input. (Bottom): The absolute error ($\Delta\rho = |\rho_1 - \rho_2|$) highlights the deviation between models, with the largest discrepancies occurring at moderate to high phase shifts and noise intensities, yet remaining below 0.007 overall. These results suggest that the theoretical derived formula reliably approximates passive theory under complex noise-phase interaction regimes. Shows strong correlations at higher noise intensities in the theoretical model, but simulated correlations are less stable, with error focused at higher phase shifts. This suggests that both the formulas and the passive model capture an optimal temporal coherence window disrupted by intermediate phase shifts [16].

Chapter 5

Comparison of Burst Propagation Model and Passive Model

The integration of these models provides a powerful toolkit for studying neuronal dynamics. Fine-tuning the model parameters can significantly enhance the performance of neuromorphic systems.

In the burst propagation model ([BPM](#)), key parameters are analyzed for their impact on multiplexing ability. These include input-related factors (input currents to the dendrite and soma, and noise affecting both), transmission properties (active coupling to the soma and passive coupling to the dendrite), and an intrinsic cellular characteristic (τ_s). By evaluating how changes in these parameters influence multiplexing, the model effectively explores temporal dynamics such as spike timing, and nonlinear responses to synaptic inputs.

Correlation formulas from the passive model, such as current and voltage correlation coefficients, provide insights into the effects of noise, phase shifts, and input currents on signal propagation. These formulas serve as benchmarks for assessing deviations caused by nonlinearities or external disturbances, making them valuable for validating more complex

models.

The passive model shows that as noise increases, correlation stability decreases, and the multiplexing rate drops sharply with rising phase shifts and noise intensity. This suggests that traditional models struggle to maintain multiplexing under such conditions, likely due to the linear and attenuation-driven nature of cable theory: two voltage traces recorded at different locations become less similar when the input varies faster or when the distance between the sites is larger.

This behavior may be consistent with the linear passive model: both signal and noise are weakened with distance along the dendrite, so voltage correlations drop more under higher noise than in the nonlinear [BPM](#). However, since $\Delta\rho \ll 0.1$ between the theoretical prediction and the passive-model simulation, the theoretical formula still effectively describes the passive model's behavior under varying noise levels and input phase differences.

Chapter 6

Conclusion and Future Directions

The findings of this thesis combine the two-compartment model with advanced computational approaches to unravel the complexities of soma-dendrite interactions. This work not only highlights the fundamental principles of neuronal compartmentalization and multiplexing but also integrates sophisticated modeling techniques to optimize predictions and elucidate intricate dynamics.

6.1 Conclusion

The burst propagation model extends the two-compartment framework by integrating dynamic, nonlinear components to capture the temporal and spatial aspects of spike and burst generation.

It uses ordinary differential equations (ODEs) to describe the interaction between somatic and dendritic compartments. Key factors such as conductances, impedance mismatch and time constants are included to represent realistic neuronal behavior [17]. In our simulations, bursts were identified using an inter-spike-interval (ISI) criterion: consecutive spikes

separated by ISIs ≤ 15 ms were assigned to the same burst. Such bursts typically occur when dendritic input is strong or temporally clustered, so that the resulting dendritic depolarization repeatedly drives the somatic membrane potential above spike threshold. This model also highlights the importance of dendrite-to-soma coupling, demonstrating how specific adjustments can enhance or inhibit the transmission of bursts. By linking bursts to synaptic inputs, it provides insight into how neurons handle rapid, high-frequency signals critical for neural coding [15].

In this thesis, the passive model was adapted and compared to nonlinear models, revealing its strengths in predicting steady-state signal attenuation and its limitations in capturing complex, dynamic phenomena such as burst propagation and multiplexing (the passive model does not generate spikes). Notice that the linearity of the passive model offered a controlled baseline for simulations, but it was less adept at representing the intricate temporal dynamics captured by the burst propagation model. For instance, the passive model’s predictions were less accurate under high noise intensities or strong dendro-somatic coupling, where nonlinear dynamics dominate [30].

The passive model captures passive signal transmission, showing how voltages decay and spread between dendrite and soma under coupling and noise. The **BPM** extends this baseline by adding active dendrite-to-soma drive and spike-triggered feedback, allowing us to study how nonlinear dendritic events shape firing patterns and multiplexed read-outs. By combining a linear passive model with the **BPM**, this thesis connects an interpretable passive baseline to a nonlinear model that generates spikes and bursts. Together, the two models provide complementary insight: the passive model clarifies parameter-to-correlation relationships, while the **BPM** tests whether these relationships persist when burst-generating mechanisms are present.

Compartmentalization is quantified by $\text{Corr}(V_s, V_d)$. Therefore, the linear model provides an analytic baseline for linking coupling and input statistics to compartmentalization, and for interpreting how compartmentalization relates to multiplexing.

In the Burst Propagation Model, we defined the metrics of compartmentalization and

multiplexing and came to the most important conclusion: the two metrics are negatively correlated. These metrics further suggest that optimal neuronal efficiency depends on balanced interactions between soma and dendrites.

6.2 Future Directions

Because the passive model does not generate spikes or bursts, it cannot directly produce the event-based multiplexing metric used in the Burst Propagation Model, although it can still quantify compartmentalization through $\text{Corr}(V_s, V_d)$.

Incorporating additional compartments, such as axonal or inhibitory regions, into the two-compartment and burst propagation models could enhance biological realism. In the Burst Propagation Model, another parameter that has not yet been systematically tested is the phase shift φ between the dendritic input I_d and the somatic input I_s . This parameter was not included in the present analysis because the influence of φ on the overall model output is too large, which makes the influence of φ on the overall model output less accurate. From a biological point of view, if the dendritic and somatic compartments receive two signals that are widely separated in time, the two-compartment system can distinguish them relatively easily. In that case, the distinction between the two input streams becomes trivial, which is less informative about the intrinsic multiplexing capacity of the system itself.

In addition to the metric of multiplexing and the metric of compartmentalization degree, there are actually more indicators that can be tested: $\text{Corr}(\text{BF}, I_d)$, $\text{Corr}(\text{ER}, I_s)$ (these two indicators are actually the components of the metric of multiplexing, which were introduced in the methodology section). What is the relationship between these two indicators and I_d , I_s , g_s , c_d , σ , τ_s respectively, and what impact does the change of the six parameters have on these two indicators? And when φ is included in future analyses, how do the seven parameters affect these two indicators?

In the passive model, we did not draw a simulation graph of $\text{Corr}(V_s, V_d)$ for each parameter

separately as in the previous model, but instead chose to vary two parameters at the same time. This work of determining the precise impact of each parameter on cable theory may also be added to future tasks.

In artificial intelligence, these models offer methods to simulate hierarchical learning and signal processing.

The application of these models may extend to artificial neural networks, where burst-like mechanisms can enhance computational efficiency [3]. Exploring the role of burst propagation and s-d coupling in diseases such as schizophrenia, autism, or epilepsy could lead to new therapeutic strategies [31].

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APPENDICES

Appendix A

Supplementary information

Dynamics of dendritic and somatic compartments			
E_L	-70	mV	
τ_s	16	ms	
C_s	370	pF	
$\tau_{(\omega s)}$	100	ms	
b	200	pA	<i>strength of spike</i> – <i>triggered adaptation</i>
V_T	-50	mV	
τ_d	7	ms	
g_d	1200	pA	
C_d	170	pF	
c_d	2600	pA	
$\tau_{\omega d}$	30	ms	
a	13	$nS = nA/V$ $= pA/mV$	
E_d	-38	mV	
D_d	6	mV	
V_r	-70	mV	<i>reset voltage after a spike</i>
α	1		<i>Noise parameter settings</i>

Table A.1: Model Parameters.

Time Step		
$time$	600	ms
dt	0.1	ms
N	<i>round</i>	$time/dt$
t	$np.arange(0, N + 1) * dt$	$[0, 0.1, 0.2, \dots, N * dt]$

Table A.2: Time accuracy and length setting.

Background Input		
t_{d0}	0.1	ms
<i>low ratio d</i>	0.4	
t_{s0}	200	ms
<i>low ratio s</i>	0.5	

Table A.3: Noise-free signal input setup.

Appendix B

Derivation

Our linear passive model is:

$$\dot{V}_s = -\frac{g_L}{C_m}(V_s - V_L) + \frac{g_c}{pC_m}(V_d - V_s) + \frac{(I_s + \xi_1)}{pC_m} \quad (1)$$

$$\dot{V}_d = -\frac{g_L}{C_m}(V_d - V_L) + \frac{g_c}{(1-p)C_m}(V_s - V_d) + \frac{(I_d + \xi_2)}{(1-p)C_m} \quad (2)$$

The square-wave somatic input is

$$I_s(t) = \begin{cases} I_{s-base}, & 0 \leq t < \eta_1 T \\ I_{s0}, & \eta_1 T < t \leq T \end{cases}$$

which is periodic, i.e., $I_s(t + T) = I_s(t)$, where T is the period. In the simulation, we set $\eta_1 = 0.5$ and $T = 200$ ms.

The dendritic square-wave input is

$$I_d(t - T) = \begin{cases} I_{d-base}, & 0 \leq t < \eta_2 T \\ I_{d0}, & \eta_2 T < t \leq T \end{cases}$$

The phase shift between the two signals is denoted by $\phi \in [0, 1]$. In the simulation, we set $\eta_2 = 0.4$.

Introduce $A_s = I_{s0} - I_{s-base}$ and $A_d = I_{d0} - I_{d-base}$ to represent the high-low differences of the square waves, so that:

B.1 Derivation of the Correlation Coefficient for Noise-Free Input Signal

$$\text{Var}(I_s) = \langle I_s^2 \rangle - \langle I_s \rangle^2$$

“ $\langle * \rangle$ ” represents the time average over one cycle, so we obtain:

$$\text{Var}(I_s) = \text{Var}(I_s - I_{s-base}) = (\eta_1 - \eta_1^2) A_s^2 = 0.25 A_s^2.$$

Similarly, $\text{Var}(I_d) = 0.24 A_d^2$. Continuing, we compute

$$\text{Cov}(I_s, I_d) = \langle I_s I_d \rangle - \langle I_s \rangle \langle I_d \rangle$$

and the correlation coefficient

$$\rho(I_s, I_d) = \frac{\text{Cov}(I_s, I_d)}{\sqrt{\text{Var}(I_s) \text{Var}(I_d)}}.$$

We find

$$\rho(I_s, I_d) = f(x) = \begin{cases} \frac{10}{\sqrt{6(0.2-\phi)}}, & 0 \leq \phi \leq 0.4 \\ -\frac{\sqrt{6}}{3}, & 0.4 \leq \phi \leq 0.5 \\ \frac{10}{\sqrt{6(\phi-0.7)}}, & 0.5 \leq \phi \leq 0.9 \\ \frac{\sqrt{6}}{3}, & 0.9 \leq \phi \leq 1 \end{cases} \quad (3)$$

This expression depends only on ϕ ; we simplify and rewrite it as $\rho(\phi)$. Then

$$\text{Cov}(I_s, I_d) = xy \rho(\phi) \quad (4)$$

where $x = \sqrt{\text{Var}(I_s)} = 0.5A_s$ and $y = \sqrt{\text{Var}(I_d)} = \sqrt{0.24}A_d$.

B.2 Derivation of Correlation Coefficient for Noisy Input Signal

Signals with noise:

$$\tilde{I}_s(t) = I_s(t) + \xi_1, \quad \tilde{I}_d(t) = I_d(t) + \xi_2.$$

The noise terms ξ_1 and ξ_2 are independent and are also independent of I_s and I_d . Thus,

$$\begin{aligned} \text{Var}(\tilde{I}_s) &= \text{Var}(I_s) + \text{Var}(\xi_1) = x^2 + \sigma^2, \\ \text{Var}(\tilde{I}_d) &= y^2 + \sigma^2, \end{aligned}$$

$$\text{Cov}(\tilde{I}_s, \tilde{I}_d) = \text{Cov}(I_s, I_d) = xy \rho(\phi).$$

Therefore,

$$\rho(\tilde{I}_s, \tilde{I}_d) = \frac{xy \rho(\phi)}{\sqrt{(x^2 + \sigma^2)(y^2 + \sigma^2)}} = \frac{\rho(\phi)}{\sqrt{\left[1 + \left(\frac{\sigma}{x}\right)^2\right] \left[1 + \left(\frac{\sigma}{y}\right)^2\right]}} \quad (5)$$

Note that $\frac{\sigma}{x} = \frac{2\sigma}{A_s}$, so

$$\rho(\tilde{I}_s, \tilde{I}_d) \text{ depends on } \phi \text{ and the signal-to-noise ratios } \frac{A_s}{\sigma}, \frac{A_d}{\sigma}.$$

B.3 Derivation of voltage correlation coefficient of simplified linear model

B.3.1 The Ornstein-Uhlenbeck Noise

Properties of the noise are controlled by these two parameters: α and σ , all positive numbers.

Introduce $g = \sqrt{2\alpha\sigma}$, the differential form of the noise is given as follows:

$$d\xi = -\alpha\xi dt + dW$$

Here dW is a stochastic Gaussian increment, satisfying:

$$\text{Var}(\Delta W) = \langle (\Delta W)^2 \rangle = dt$$

The noise integral is in the form of:

$$\xi(t) = \xi(0)e^{-\alpha t} + ge^{-\alpha t} \int_0^t e^{\alpha t} dW$$

When t is large enough, the first term on the right side of the above formula can be ignored, so:

$$\xi(t) = ge^{-\alpha t} \int_0^t e^{\alpha t} dW \quad (6)$$

The noise of the two compartments is recorded as:

- **Soma:** $\xi_1(t) = ge^{-\alpha t} \int_0^t e^{\alpha t} dW_1$
- **Dendrite:** $\xi_2(t) = ge^{-\alpha t} \int_0^t e^{\alpha t} dW_2$

Next, we solve the differential equations (1) and (2).

First, we convert them into matrix form:

$$\frac{d}{dx} \begin{pmatrix} \tilde{V}_s \\ \tilde{V}_d \end{pmatrix} = \begin{pmatrix} -(\lambda_1 + ag_c) & ag_c \\ bg_c & -(\lambda_1 + bg_c) \end{pmatrix} \begin{pmatrix} \tilde{V}_s \\ \tilde{V}_d \end{pmatrix} + \begin{pmatrix} a\tilde{I}_s \\ b\tilde{I}_d \end{pmatrix}.$$

In this expression, $\tilde{V}_s = V_s - E_L$, $\tilde{V}_d = V_d - E_L$, $\lambda_1 = \frac{gL}{C_m}$, $a = \frac{1}{pC_m}$, and $b = \frac{1}{(1-p)C_m}$.

Let $\lambda_2 = \lambda_1 + (a + b)g_c$. Then λ_1 and λ_2 are the eigenvalues of the coefficient matrix, corresponding to the eigenvectors $\begin{pmatrix} 1 \\ 1 \end{pmatrix}$ and $\begin{pmatrix} a \\ -b \end{pmatrix}$, respectively. Therefore, we can solve:

$$\begin{pmatrix} \tilde{V}_s \\ \tilde{V}_d \end{pmatrix} = \frac{1}{a+b} \begin{pmatrix} e^{-\lambda_1 t} & ae^{-\lambda_2 t} \\ e^{-\lambda_1 t} & -be^{-\lambda_2 t} \end{pmatrix} \begin{pmatrix} c_1 + \int_0^t ab(\tilde{I}_s + \tilde{I}_d)e^{\lambda_1 t} dt \\ c_2 + \int_0^t (a\tilde{I}_s - b\tilde{I}_d)e^{\lambda_2 t} dt \end{pmatrix} \quad (7)$$

c_1 and c_2 are constants. In the long-time limit $t \rightarrow \infty$, the terms involving c_1 and c_2 can be ignored. Expanding the integrals in the expression above, we encounter the following form:

$$e^{-\lambda t} \int_0^t I(t)e^{\lambda t} dt = e^{-\lambda t} \left[\int_0^t I(t)e^{\lambda t} dt + e^{-\lambda t} \int_0^t \xi(t)e^{\lambda t} dt \right] \quad (8)$$

Using integration by parts, the first term on the right-hand side can be expanded as

$$e^{-\lambda t} \int_0^t I(t)e^{\lambda t} dt = \frac{1}{\lambda} I(t) + Ae^{-\lambda t} \sum_{i=0}^{\infty} (-1)^i \int_0^t e^{\lambda t_i} \delta(t - t_i) dt.$$

Here, A is the difference between the high and low levels of the square-wave signal $I(t)$, t_i is a discontinuity point of $I(t)$, and $\delta(t)$ is the Dirac delta function.

In the long-time limit $t \rightarrow \infty$, the second term on the right-hand side collapses into infinitely narrow bounded peaks, which do not contribute to the correlation coefficient and can be ignored.

For the second term on the right-hand side of the expression above, using integration by parts and the O-U noise in differential form, we obtain

$$e^{-\lambda t} \int_0^t \xi(t)e^{\lambda t} dt = \frac{1}{\lambda - \alpha} \xi(t) - \frac{1}{\lambda - \alpha} \dot{\xi}(t) - \frac{1}{\lambda - \alpha} \xi(0)e^{-\lambda t}.$$

The last term can be dropped for sufficiently large t . Here, $\dot{\xi}(t) = ge^{-\lambda t} \int_0^t e^{\lambda t} dW$ has a similar form to Eq. (6). We study the covariance of $\xi(t)$ and $\xi'(t)$:

$$\text{Cov}(\xi(t), \xi'(t)) = g^2 e^{-(\lambda+\alpha)t} \text{Cov} \left(\sum e^{\lambda t} dW, \sum e^{\alpha t} dW \right).$$

Using $\text{Cov}(\Delta W, \Delta W) = dt$, the expression above can be converted into an integral. Taking t sufficiently large to discard small terms, we obtain

$$\text{Cov}(\xi(t), \xi'(t)) = \frac{g^2}{\lambda + \alpha} = \frac{2\alpha\sigma^2}{\lambda + \alpha}. \quad (9)$$

Define

$$\xi_{ij} = ge^{-\lambda_j t} \int_0^t e^{\lambda_j t} dW_i, \quad i, j \in \{1, 2\}.$$

Thus,

$$\text{Var}(\xi_{ij}) = \frac{g^2}{2\lambda_j} = \frac{\alpha\sigma^2}{\lambda_j}. \quad (10)$$

Now we have three sets of variables of interest:

$$\{I_s, I_d\}, \{\xi_1, \xi_{11}, \xi_{12}\}, \{\xi_2, \xi_{22}, \xi_{21}\}.$$

Two variables from different sets are independent, while two variables within the same set are correlated. As in the derivation of Eq. (9), we obtain:

$$\text{Cov}(\xi_i, \xi_{ij}) = \frac{2\alpha\sigma^2}{\lambda_j + \alpha} \quad (11)$$

$$\text{Cov}(\xi_{i1}, \xi_{i2}) = \frac{2\alpha\sigma^2}{\lambda_1 + \lambda_2} \quad (12)$$

Based on the above conclusions, we simplify equation (8):

$$e^{-\lambda t} \int_0^t \tilde{I}(t) e^{\lambda t} dt = \frac{1}{\lambda} I(t) + \frac{1}{\lambda - \alpha} (\xi(t) - \xi'(t)) \quad (13)$$

We can expand the right-hand side of Eq. (7) by defining $X = (a+b)\tilde{V}_s$ and $Y = (a+b)\tilde{V}_d$. Then,

$$\begin{aligned} X = & \left(\frac{ab}{\lambda_1} + \frac{a^2}{\lambda_2} \right) I_s + ab \left(\frac{1}{\lambda_1} - \frac{1}{\lambda_2} \right) I_d + \left(\frac{ab}{\lambda_1 - \alpha} + \frac{a^2}{\lambda_2 - \alpha} \right) \xi_1 \\ & + ab \left(\frac{1}{\lambda_1 - \alpha} - \frac{1}{\lambda_2 - \alpha} \right) \xi_2 \\ & - \left(\frac{ab}{\lambda_1 - \alpha} \xi_{11} + \frac{a^2}{\lambda_2 - \alpha} \xi_{12} + \frac{ab}{\lambda_1 - \alpha} \xi_{21} - \frac{ab}{\lambda_2 - \alpha} \xi_{22} \right) \end{aligned} \quad (14)$$

$$\begin{aligned} Y = & ab \left(\frac{1}{\lambda_1} - \frac{1}{\lambda_2} \right) I_s + \left(\frac{ab}{\lambda_1} + \frac{b^2}{\lambda_2} \right) I_d + ab \left(\frac{1}{\lambda_1 - \alpha} - \frac{1}{\lambda_2 - \alpha} \right) \xi_1 \\ & + \left(\frac{ab}{\lambda_1 - \alpha} + \frac{b^2}{\lambda_2 - \alpha} \right) \xi_2 \\ & - \left(\frac{ab}{\lambda_1 - \alpha} \xi_{11} - \frac{ab}{\lambda_2 - \alpha} \xi_{12} + \frac{ab}{\lambda_1 - \alpha} \xi_{21} + \frac{b^2}{\lambda_2 - \alpha} \xi_{22} \right) \end{aligned} \quad (15)$$

From Eqs. (4) and (9)–(12), we can calculate

$$\text{Var}(X) = \text{Cov}(X, X), \quad \text{Var}(Y), \quad \text{and} \quad \text{Cov}(X, Y).$$

Then the voltage correlation is

$$\rho(V_s, V_d) = \rho(X, Y) = \frac{\text{Cov}(X, Y)}{\sqrt{\text{Var}(X) \text{Var}(Y)}}.$$

For convenience, we simplify Eqs. (14) and (15) to the following form:

$$\begin{aligned}
X &= AI_s + BI_d + (C_1 + C_2)\xi_1 + (D_1 - D_2)\xi_2 \\
&\quad - (C_1\xi_{11} + C_2\xi_{12} + D_1\xi_{21} - D_2\xi_{22}) \\
Y &= BI_s + EI_d + (D_1 - D_2)\xi_1 + (C_1 + C_3)\xi_2 \\
&\quad - (D_1\xi_{11} - D_2\xi_{12} + C_1\xi_{21} + C_3\xi_{22})
\end{aligned}$$

Then,

$$\begin{aligned}
Var(X) &= A^2x^2 + B^2y^2 + 2ABxy\rho(\phi) + C_X\sigma^2 \\
Var(Y) &= B^2x^2 + E^2y^2 + 2EBxy\rho(\phi) + C_Y\sigma^2 \\
Cov(X, Y) &= ABx^2 + BEy^2 + (AE + B^2)xy\rho(\phi) + C_{XY}\sigma^2
\end{aligned}$$

In the formula,

$$\begin{aligned}
C_X &= (C_1 + C_2)^2 + (D_1 - D_2)^2 + \frac{\alpha}{\lambda_1}(C_1^2 + D_1^2) + \frac{4\alpha}{\lambda_1 + \lambda_2}(C_1 C_2 - D_1 D_2) + \frac{\alpha}{\lambda_2}(C_2^2 + D_2^2) \\
&\quad - \frac{4\alpha}{\alpha + \lambda_1} [C_1(C_1 + C_2) + D_1(D_1 - D_2)] \\
&\quad - \frac{4\alpha}{\alpha + \lambda_2} [C_2(C_1 + C_2) - D_2(D_1 - D_2)], \\
C_Y &= (C_1 + C_3)^2 + (D_1 - D_2)^2 + \frac{\alpha}{\lambda_1}(C_1^2 + D_1^2) + \frac{\alpha}{\lambda_2}(C_3^2 + D_2^2) \\
&\quad + \frac{4\alpha}{\lambda_1 + \lambda_2}(C_1 C_3 - D_1 D_2) \\
&\quad - \frac{4\alpha}{\alpha + \lambda_1} [C_1(C_1 + C_3) + D_1(D_1 - D_2)] \\
&\quad - \frac{4\alpha}{\alpha + \lambda_2} [C_3(C_1 + C_3) - D_2(D_1 - D_2)], \\
C_{XY} &= (2C_1 + C_2 + C_3)(D_1 - D_2) + \frac{2\alpha C_1 D_1}{\lambda_1} - \frac{\alpha(C_2 + C_3)D_2}{\lambda_2} \\
&\quad + \frac{2\alpha}{\lambda_1 + \lambda_2} [D_1(C_2 + C_3) - 2C_1 D_2] \\
&\quad - \frac{2\alpha}{\alpha + \lambda_1} [D_1(2C_1 + C_2 + C_3) + 2C_1(D_1 - D_2)] \\
&\quad - \frac{2\alpha}{\alpha + \lambda_2} [-D_2(2C_1 + C_2 + C_3) + (C_2 + C_3)(D_1 - D_2)].
\end{aligned}$$

Finally, the correlation coefficient between voltages is:

$$\frac{AB \left(\frac{x}{\sigma}\right)^2 + BE \left(\frac{y}{\sigma}\right)^2 + (AE + B^2) \left(\frac{x}{\sigma}\right) \left(\frac{y}{\sigma}\right) \rho(\phi) + C_{XY}}{\sqrt{\left[A^2 \left(\frac{x}{\sigma}\right)^2 + B^2 \left(\frac{y}{\sigma}\right)^2 + 2AB \left(\frac{x}{\sigma}\right) \left(\frac{y}{\sigma}\right) \rho(\phi) + C_X\right] \left[B^2 \left(\frac{x}{\sigma}\right)^2 + E^2 \left(\frac{y}{\sigma}\right)^2 + 2EB \left(\frac{x}{\sigma}\right) \left(\frac{y}{\sigma}\right) \rho(\phi) + C_Y\right]}}$$

Thus, it depends only on the signal-to-noise ratios $\frac{x}{\sigma}$ and $\frac{y}{\sigma}$, and the phase difference ϕ .

B.3.2 The Gaussian Noise

We start from the differential form of equations (1) and (2):

$$\begin{aligned}dV_s &= -\lambda_1(V_s - E_L)dt + ag_c(V_d - V_s)dt + a(I_sdt + \sigma dW_1) \\dV_d &= -\lambda_1(V_d - E_L)dt + bg_c(V_s - V_d)dt + b(I_ddt + \sigma dW_2)\end{aligned}$$

where Gaussian increment: $dW_1 \sim \sqrt{dt}\mathcal{N}(0, 1)$, $dW_2 \sim \sqrt{dt}\mathcal{N}(0, 1)$

Look at the following expression:

$$\begin{aligned}&e^{-\lambda t} \int_0^t e^{\lambda t} (I dt + \sigma dW) \\&= \frac{1}{\lambda} I(t) + \xi(t) - \frac{1}{\lambda} I(0) e^{-\lambda t} - \frac{1}{\lambda} e^{-\lambda t} \int_0^t e^{\lambda t} dI(t)\end{aligned}$$

where $\xi(t) = \sigma e^{-\lambda t} \int_0^t e^{\lambda t} dW$ has same form as noise

As analyzed in 3.1, the last two terms on the right side can be ignored in the long term.

Define:

$$\xi_{ij} = \sigma e^{-\lambda_j t} \int_0^t e^{\lambda_j t} dW_i$$

Then,

$$\begin{aligned}
Var(\xi_{ij}) &= \frac{\sigma^2}{2\lambda_j} \\
Cov(\xi_{i1}, \xi_{i2}) &= \frac{\sigma^2}{\lambda_1 + \lambda_2} \\
Cov(\xi_{.1i}, \xi_{.2j}) &= 0
\end{aligned}$$

Using the matrix solution idea of 3.1, we can get:

$$\begin{aligned}
X &= AI_s + BI_d + D\xi_{11} + E\xi_{12} + D\xi_{21} - D\xi_{22} \\
Y &= BI_s + CI_d + D\xi_{11} - D\xi_{12} + D\xi_{21} + F\xi_{22}
\end{aligned}$$

In the formula, $X = (a + b)(V_s - E_L)$, $Y = (a + b)(V_d - E_L)$, $A = \left(\frac{ab}{\lambda_1} + \frac{a^2}{\lambda_2}\right)$,

$$B = ab\left(\frac{1}{\lambda_1} - \frac{1}{\lambda_2}\right), \quad C = \left(\frac{ab}{\lambda_1} + \frac{b^2}{\lambda_2}\right), \quad D = ab, \quad E = a^2, \quad F = b^2$$

Then we have:

$$\begin{aligned}
Var(X) &= A^2x^2 + B^2y^2 + 2ABxy\rho(\phi) + C_X\sigma^2 \\
Var(Y) &= B^2x^2 + C^2y^2 + 2BCxy\rho(\phi) + C_Y\sigma^2
\end{aligned}$$

$$Cov(X, Y) = ABx^2 + BCy^2 + (AC + B^2)xy\rho(\phi) + C_{XY}\sigma^2$$

where

$$C_X = \frac{D^2}{\lambda_1} + \frac{E^2 + D^2}{2\lambda_2} + \frac{2D(E - D)}{\lambda_1 + \lambda_2}$$

$$C_Y = \frac{D^2}{\lambda_1} + \frac{D^2 + F^2}{2\lambda_2} + \frac{2D(F - D)}{\lambda_1 + \lambda_2}$$

$$C_{XY} = \frac{D^2}{\lambda_1} - \frac{D(E + F)}{2\lambda_2} + \frac{D(E + F - 2D)}{\lambda_1 + \lambda_2}$$

Then, we got the correlation coefficient:

$$\begin{aligned} \rho_{\text{Gaussian}}(V_s, V_d) &= \rho_{\text{Gaussian}}(X, Y) = \frac{\text{Cov}(X, Y)}{\sqrt{\text{Var}(X)\text{Var}(Y)}} \\ &= \frac{ABx^2 + BCy^2 + (AC + B^2)xy\rho(\phi) + C_{XY}\sigma^2}{\sqrt{[A^2x^2 + B^2y^2 + 2ABxy\rho(\phi) + C_X\sigma^2] \cdot [B^2x^2 + C^2y^2 + 2BCxy\rho(\phi) + C_Y\sigma^2]}} \end{aligned}$$