



Article

Assessment of somatic excitability and dendritic responses in a reduced conductance-based CA1 neuron model

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ABSTRACT

A reduced conductance-based model was assessed through somatic excitability, firing dynamics, and dendritic conductance responses. The reported baseline spike had a threshold of -36.5 mV, a baseline-to-peak amplitude of 115.1 mV, and a half-width of 0.93 ms. At $8 \mu\text{A}/\text{cm}^2$, ISIs shortened before lengthening by 17.8% relative to the minimum interval. Dendritic A-current blockade increased amplitude from 92.8 to 100.8 mV, and halving dendritic Na^+ conductance reduced it to 69.9 mV. Two new numerical experiments used a complete reference implementation with published channel kinetics. Joint waveform retuning reduced the normalized feature error by 84.7% , shifting the threshold from -39.13 to -49.36 mV and the amplitude from 114.71 to 96.76 mV. Matched zero-M and increased-M controls linked M-gate availability to late ISI lengthening, while a fixed A-inactivation gate changed the early-interval trajectory. The reported and reference baselines use their respective specified kinetics. These analyses quantify waveform trade-offs and conductance-dependent firing in the compact representation.

1. Introduction

Hippocampal CA1 pyramidal neurons participate in circuits supporting episodic memory and spatial navigation. They receive excitatory input from CA3 through the Schaffer collateral pathway and from layer III of the entorhinal cortex through the temporoammonic pathway, and project to the subiculum and entorhinal cortex. Their voltage-gated somatic and dendritic conductances shape how the neuron integrates synaptic inputs and converts them into action potentials. Conductance-based models provide a way to examine these interactions under controlled current injection. Conductance-based computational modeling, based on the foundational framework of Hodgkin and Huxley [1], provides a means of examining how ionic currents and compartmental interactions shape neuronal activity. Pinsky and Rinzel [2] developed a reduced two-compartment model for CA3 pyramidal neurons, while Traub and colleagues [3,4] established more detailed computational representations of hippocampal pyramidal neurons. These approaches provide complementary descriptions of neuronal excitability at different levels of structural and biophysical complexity. By allowing ionic conductances and intercompartmental coupling to be independently controlled, computational models complement electrophysiological experiments in

investigating the contributions of specific membrane mechanisms to action potential generation and propagation. A prominent feature of CA1 pyramidal neurons is the functional and electrical distinction between the somatic spike-initiation region and the dendritic compartments involved in synaptic integration. Direct dendritic recordings and simultaneous soma-dendrite recordings have demonstrated that action potentials initiated near the soma and axon initial segment can propagate into proximal and distal dendrites. The amplitude and extent of this backpropagation depend on the availability of dendritic Na^+ channels and the opposing action of A-type K^+ conductances, whose densities vary along the dendritic arbor [5-7]. Backpropagating action potentials provide dendritic depolarization that can interact with synaptic inputs [8], while activity- and voltage-dependent propagation influences the initiation of local dendritic spikes [9,10]. During sustained depolarizing current injection, CA1 pyramidal neurons exhibit time-dependent changes in firing frequency. Slow outward currents can reduce firing frequency, with Ca^{2+} -dependent K^+ currents contributing to accommodation [11]. Initial firing acceleration and later ISI lengthening describe different phases of the response and must be measured separately. Stronger stimulation can also produce

depolarization block in detailed CA1 models [12]. The model examined here combines a somatic regular-spiking conductance formulation from Pospischil et al. [13], the area-scaled coupling strategy of Pinsky and Rinzel [2], and a lumped dendritic Na⁺/A-type K⁺ representation informed by Migliore et al. [14]. These components were developed for different modeling purposes: the Pospischil formulation describes cortical and thalamic neuron classes, and the Pinsky–Rinzel formulation describes CA3 neurons. Their combination provides a compact test system for CA1-related response patterns; CA1-specific quantitative agreement must be assessed from its outputs.

Recent studies have further developed quantitative approaches for assessing the physiological properties of hippocampal neuron models. Sáray et al. [15] introduced HippoUnit, a standardized testing framework for comparing hippocampal neuron models with electrophysiological measurements. Tomko et al. [16] developed a reduced-morphology CA1 pyramidal-cell model and evaluated its somatic and dendritic responses using HippoUnit, while Appukuttan and Davison [17] examined the reproducibility and quantitative validation of a biologically constrained CA1 point-neuron model. In parallel, Bock et al. [18] demonstrated that somatic depolarization influences distal dendritic spike propagation through mechanisms involving A-type K⁺ channel availability. Pagkalos et al. [19] introduced a framework for incorporating dendritic processing into spiking neural networks, and Marasco et al. [20] extended simplified CA1 neuronal models to reproduce responses to both constant and variable synaptic-like inputs. Together, these studies illustrate the complementary roles of reduced neuronal representations, quantitative electrophysiological validation, and compartment-specific mechanisms in computational investigations of hippocampal neurons. Golomb et al. [21] further examined the contributions of persistent Na⁺ and M-type K⁺ currents to somatic bursting in CA1 pyramidal cells through combined experimental and modeling analyses.

This study examines how an assembled two-compartment model connects somatic excitability, firing dynamics, and local dendritic conductance. Three objectives are addressed: (i) characterize waveform and frequency-current responses; (ii) distinguish initial acceleration from post-peak adaptation and test M-current and A-current contributions; and (iii) assess parameter sensitivity and perform joint waveform retuning with an explicit reference implementation. The compact representation enables controlled comparisons of ionic mechanisms using a common set of measurement definitions.

2. Materials and methods

2.1 Model architecture

Following the reduced two-compartment strategy of Pinsky and Rinzel [2], the neuron was modeled as a somatic compartment and a single lumped dendritic compartment that were electrically coupled (Figure 1). Both compartments followed Hodgkin–Huxley-type current-balance equations [1], with a specific membrane capacitance of $C_m = 1.0 \mu\text{F}/\text{cm}^2$. The soma contained fast Na⁺, delayed-rectifier K⁺, M-type K⁺, and leak currents, whereas the dendritic compartment contained Na⁺, A-type K⁺, and leak currents. The soma occupied a fraction $p = 0.5$ of the total membrane area, with the remaining fraction assigned to the dendritic compartment. Taking outward ionic currents as positive, the somatic and dendritic membrane equations are:

$$C_m A_s \frac{dV_s}{dt} = -A_s I_{ion,s} + I_c + I_{inj} \quad (1)$$

$$C_m A_d \frac{dV_d}{dt} = -A_d I_{ion,d} - I_c \quad (2)$$

Here, V_s and V_d are membrane potentials; $A_s = pA_{tot}$ and $A_d = (1-p)A_{tot}$ are compartmental membrane areas; and $G_c = g_c A_{tot}$ is the absolute coupling conductance, with g_c normalized to total membrane area. The injected current density I_{app} is normalized to somatic area, so $I_{inj} = A_s I_{app}$.

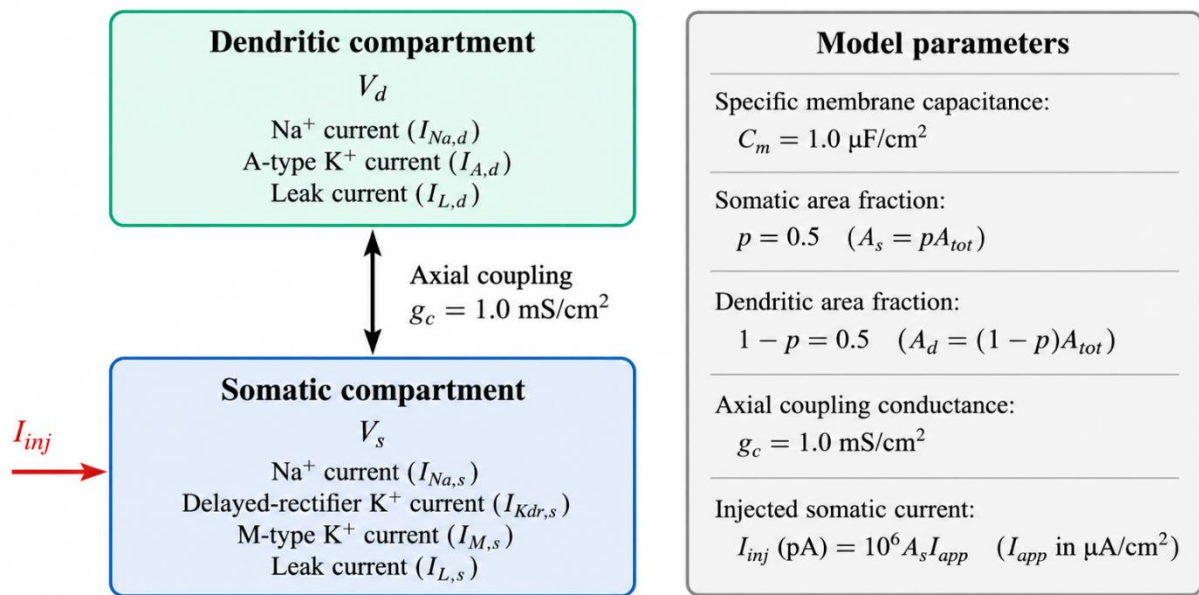


Figure 1. Two-compartment model architecture. The soma contains fast Na⁺, delayed-rectifier K⁺, M-type K⁺, and leak currents; the dendrite contains Na⁺, A-type K⁺, and leak currents. The somatic area fraction is $p = A_s/A_{tot} = 0.5$, and the total-area-normalized coupling conductance is $g_c = G_c/A_{tot} = 1.0 \text{ mS}/\text{cm}^2$. Current injection is applied at the soma, with $I_{inj} = A_s I_{app}$. Equations (1)–(2) specify the corresponding charge balance.

Dividing Eqs. (1)–(2) by the respective areas gives coupling current densities $g_c(V_d - V_s)/p$ at the soma and $g_c(V_s - V_d)/(1 - p)$ at the dendrite; the somatic injection term is I_{app} . At $p = 0.5$ and $g_c = 1.0$ mS/cm², both coupling-density coefficients are 2.0 mS/cm². These equal and opposite absolute coupling currents conserve charge between compartments. When A_s is expressed in cm² and I_{app} in μ A/cm², the injected current in pA is I_{inj} [pA] = $10^6 A_s$ [cm²] I_{app} [μ A/cm²]. The ionic and coupling currents are defined as $I_{ion,s} = I_{Na,s} + I_{Kdr,s} + I_{M,s} + I_{L,s}$, $I_{Na,d} + I_{A,d} + I_{L,d}$, [$I_c = G_c(V_d - V_s)$]. The baseline area fraction $p = 0.5$ is a model allocation parameter. Area fraction and coupling strength are varied independently in the sensitivity analysis.

2.2 Ionic currents and channel kinetics

The somatic compartment contained a fast transient Na⁺ current I_{Na} , a delayed-rectifier K⁺ current I_{Kdr} , a slow non-inactivating M-type K⁺ current I_M , and a leak current I_L . The dendritic compartment contained a lower-density Na⁺ current, a transient A-type K⁺ current I_A , and a leak current. These currents were represented as:

$$\begin{aligned} I_{Na,j} &= g_{Na,j} m_j^3 h_j (V_j - E_{Na}), \\ I_{Kdr,s} &= g_{Kdr,s} n_s^4 (V_s - E_K), \\ I_{M,s} &= g_{M,s} z_s (V_s - E_K), \\ I_{A,d} &= g_{A,d} a_d^{q_A} b_d^{r_A} (V_d - E_K), \\ I_{L,j} &= g_{L,j} (V_j - E_L), \quad j \in \{s, d\} \end{aligned} \quad (3)$$

Here, g denotes maximal conductance density, E denotes the corresponding reversal potential, and the gating variables describe channel activation and inactivation. The exponents q_A and r_A specify the A-current gating powers. All gating variables followed first-order kinetics, expressed in a unified form as $\frac{dx}{dt} = \frac{x_\infty(V) - x}{\tau_x(V)}$.

Fast Na⁺ and delayed-rectifier K⁺ voltage-dependent kinetics follow the regular-spiking formulation of Pospischil et al. [13]. The baseline shift parameter $V_T = -58$ mV controls the voltage dependence of these gates. The M-type current uses a slowly activating, non-inactivating gate z . M-conductance perturbations are used to assess model responses in relation to the CA1 excitability mechanisms described by Golomb et al. [21].

Dendritic Na⁺ channels used the same kinetic formulation as somatic Na⁺ channels but with a lower maximal conductance. The dendritic A-type K⁺ current was described by activation and inactivation variables a and b , with rapid activation and slower inactivation. This representation follows reported dendritic A-current properties [5] and the modeling approach of Migliore et al. [14]. Table 1 lists the baseline conductances, reversal potentials, area fraction, and coupling parameter. Dendritic Na⁺ conductance is varied as a local excitability parameter; individual values are not assigned to anatomical distances. The baseline dendrite contains no separate delayed-rectifier or Ca²⁺-activated K⁺ current.

2.3 Baseline parameters and parameter assessment

The original parameter set was retained as the baseline configuration (Table 1). It included somatic Na⁺, delayed-rectifier K⁺, and M-type K⁺ conductances of 50.0, 5.0, and 0.15 mS/cm², respectively, together with dendritic Na⁺ and A-type K⁺ conductances of 8.0 and 12.0 mS/cm². Both compartments

used a leak conductance of 0.10 mS/cm², with reversal potentials of $E_{Na} = 50$ mV, $E_K = -90$ mV, and $E_L = -70$ mV. The baseline configuration gives a resting potential of -73.4 mV, a threshold of -36.5 mV under the 50 mV/ms criterion, a baseline-to-peak amplitude of 115.1 mV, and a half-width of 0.93 ms. Repetitive firing first appears at the sampled current density of 5 μ A/cm². These measurements define the reference configuration used throughout the conductance and numerical comparisons.

Table 1. Fixed model parameters

Compartment	Parameter	Symbol	Value	Units
Soma	Membrane capacitance	C_m	1.0	μ F/cm ²
Soma	Fast Na ⁺ conductance	$g_{Na,s}$	50.0	mS/cm ²
Soma	Delayed-rectifier K ⁺ conductance	$g_{Kdr,s}$	5.0	mS/cm ²
Soma	M-type K ⁺ conductance	$g_{M,s}$	0.15	mS/cm ²
Soma	Leak conductance	$g_{L,s}$	0.10	mS/cm ²
Dendrite	Na ⁺ conductance	$g_{Na,d}$	8.0	mS/cm ²
Dendrite	A-type K ⁺ conductance	$g_{A,d}$	12.0	mS/cm ²
Dendrite	Leak conductance	$g_{L,d}$	0.10	mS/cm ²
Both	Na ⁺ reversal potential	E_{Na}	50.0	mV
Both	K ⁺ reversal potential	E_K	-90.0	mV
Both	Leak reversal potential	E_L	-70.0	mV
Coupling	Somatic area fraction	p	0.5	Dimensionless
Coupling	Axial coupling conductance	g_c	1.0	mS/cm ²
Soma	Threshold-shift parameter	V_T	-58.0	mV

Joint waveform retuning was performed in the reference implementation described below. The voltage shift V_T , somatic Na⁺, delayed-rectifier K⁺, and M-type K⁺ conductances, and leak reversal potential were varied together. Rest, threshold, amplitude, and half-width were evaluated with identical definitions before and after retuning. The search also required repeated firing under current injection and a quiescent zero-current response. The reference implementation was initialized from Table 1 and completed with the published fast-current, M-current, and proximal A-current kinetics. Its baseline and returned outputs are compared directly in Section 3.8. The baseline measurements reported in Tables 2–6 retain their original protocols; the new implementation provides the explicitly specified conductance controls in Section 3.9.

2.4 Stimulation Protocols and Electrophysiological Measurements

Four baseline stimulation protocols were used. A 2 ms suprathreshold somatic pulse evoked an isolated action potential. Current steps lasting 950 ms were applied over 0 – 14 $\mu\text{A}/\text{cm}^2$ in 1 $\mu\text{A}/\text{cm}^2$ increments for F-I assessment; Table 3 reports the available selected levels. A separate 900 ms step at 8 $\mu\text{A}/\text{cm}^2$ was used to examine the ISI trajectory. Dendritic responses were compared with intact $g_{A,d} = 12 \text{ mS}/\text{cm}^2$ and blocked $g_{A,d} = 0 \text{ mS}/\text{cm}^2$ conductances. An extended current scan to 24 $\mu\text{A}/\text{cm}^2$ examined the high-current operating range. These current densities refer to somatic membrane area.

Somatic spike times were defined by upward 0 mV crossings. For each isolated spike, threshold was the first upward crossing of $dV_s/dt = 50 \text{ mV}/\text{ms}$ in the interval from 3 ms before the voltage peak to the peak; criteria of 10 and 20 mV/ms were also compared. Baseline-to-peak amplitude was $V_{\text{peak}} - V_{\text{baseline}}$, and threshold-to-peak amplitude was $V_{\text{peak}} - V_{\text{threshold}}$. Half-width was the interval between the rising and falling crossings of $V_{\text{baseline}} + (V_{\text{peak}} - V_{\text{baseline}})/2$. The afterhyperpolarization measurement was the post-spike voltage minimum.

For spike times t_k , $\text{ISI}_k = t_{k+1} - t_k$ and instantaneous frequency is $1000/\text{ISI}_k$ in spikes/s when ISIs are in ms. Initial frequency uses the first ISI; peak frequency uses the minimum ISI; late frequency is 1000 divided by the mean of the final five ISIs. Stimulus-averaged frequency is $N_{\text{spikes}}/T_{\text{stim}}$ in seconds. Post-peak adaptation is defined as $(\text{ISI}_{\text{late}} - \text{ISI}_{\text{min}})/\text{ISI}_{\text{min}}$. Within-current variation is $\text{CV} = \text{SD}(\text{ISI})/\text{mean}(\text{ISI})$, calculated separately for each current and analysis window. These deterministic CVs describe within-train timing variation, including transients. The F-I regression uses only the six reported suprathreshold levels from 5 to 14 $\mu\text{A}/\text{cm}^2$.

2.5 Dendritic and conductance-control analyses

The principal dendritic analysis uses the single lumped compartment. Dendritic spike amplitude is measured relative to its own pre-spike baseline. Na^+ conductance is varied from 4 to 12 mS/cm^2 , and A-type K^+ conductance from 0 to 18 mS/cm^2 . Comparisons at intact and zero A-conductance isolate the effect of that conductance within the specified model architecture. A separate cable-discretization comparison is included among the numerical analyses. It reports distal amplitude and propagation delay as the segment number is increased while geometry and channel distributions are held fixed. This comparison assesses discretization dependence; the conductance sweep of the lumped compartment remains a separate analysis.

2.6 Numerical implementation and reproducibility

NEURON provides an established environment for compartmental neuronal modeling [22]. Its numerical tools support efficient implementation and spatial discretization [23]. Principles of model construction and compartmental cable representation are described in The NEURON Book [24]. The reported baseline calculations used Python 3 and adaptive LSODA integration with relative tolerance 10^{-6} and absolute tolerance 10^{-8} . The new reference implementation integrated the same ten-state compartment architecture with fourth-order Runge-Kutta at 0.020 ms during parameter search and 0.010 ms for final measurements. Both implementations initialized gates at steady state and equilibrated for 150 ms from -70 mV. The new implementation and its channel-rate conventions are

specified in Section 2.7. Parameter retuning and gate-control conditions shared the same stimulus and measurement definitions. Step sizes of 0.020 and 0.010 ms were compared for peak, threshold, and half-width. New results were exported as complete voltage-gate trajectories, ISI series, and parameter records.

2.7 Reference implementation and joint retuning

The new numerical experiments use the Table 1 conductance architecture with explicitly completed kinetics at 36 °C. Fast-channel rates and M-gate kinetics were taken from ModelDB 123623; proximal A-current kinetics were taken from ModelDB 2796 [13,14]. This reference implementation fixes the previously unspecified A-current powers to $q_A = r_A = 1$ and M-gate τ_{max} to 1000 ms. Its computed outputs define the new reference baseline.

For $u = V - V_T$, define $F(x, y) = \frac{x}{\exp(x/y) - 1}$, $F(0, y) = y$. The fast-channel rates in ms^{-1} are $\alpha_m \& = 0.32F(13 - u, 4)$, $\beta_m \& = 0.28F(u - 40, 5)$, $\alpha_h \& = 0.128 \exp\left(\frac{17-u}{18}\right)$, $\beta_h \& = \frac{4}{1 + \exp\left(\frac{40-u}{5}\right)}$, $\alpha_n \& = 0.032F(15 - u, 5)$, $\beta_n \& = 0.5 \exp\left(\frac{10-u}{40}\right)$. Each gate obeys $\frac{dx}{dt} = \alpha_x(1 - x) - \beta_x x$. The M-gate has $z_{\infty} = \frac{1}{1 + \exp\left(\frac{V+35}{10}\right)}$, and $\tau_z = \frac{1000}{3.3 \exp\left(\frac{V+35}{20}\right) + \exp\left(\frac{-V+35}{20}\right)}$ ms. For the proximal A-current at 36 °C, let $\zeta(V) = -1.5 - \frac{1}{1 + \exp\left(\frac{V+40}{5}\right)}$, $c = \frac{96.48}{8.315(273.16+36)}$, $A(V) = \exp[\zeta(V)(V - 11)c]$, and $B(V) = \exp[0.55\zeta(V)(V - 11)c]$. Then $a_{\infty} = \frac{1}{1 + A(V)}$, $\tau_a = \max\left[0.1, \frac{B(V)}{5^{1.2} \times 0.05 \times (1 + A(V))}\right]$ ms, $b_{\infty} = \frac{1}{1 + \exp[3(V+56)c]}$, and $\tau_b = \max[2, 0.26(V + 50)]$ ms. Both gates obey $\frac{dx}{dt} = \frac{x_{\infty} - x}{\tau_x}$, with the A-type potassium current expressed as $I_A = g_A a b (V - E_K)$. Coupling densities are $\frac{g_c(V_d - V_s)}{p}$ and $\frac{g_c(V_s - V_d)}{1-p}$.

An isolated spike was evoked by a 50 $\mu\text{A}/\text{cm}^2$ somatic pulse from 10 to 12 ms after equilibration. Joint targets were rest -65 mV, threshold -52.5 mV, baseline-to-peak amplitude 95 mV, and half-width 1.5 ms, selected from the article's comparison ranges. The objective was the sum of squared target errors divided by scales of 5 mV, 5 mV, 10 mV, and 0.5 ms, respectively. Bounds were:

$$V_T \& \in [-80, -55] \text{ mV},$$

$$g_{\text{Na},s} \& \in [20, 70] \text{ mS}/\text{cm}^2,$$

$$g_{\text{Kdr},s} \& \in [1, 8] \text{ mS}/\text{cm}^2,$$

$$g_{\text{M},s} \& \in [0.03, 0.25] \text{ mS}/\text{cm}^2,$$

$$E_L \& \in [-73, -60] \text{ mV}$$

A seeded 360-candidate search followed by 30 local coordinate proposals evaluated the joint objective. Candidates were penalized for fewer than five spikes during a 300 ms step at 8 $\mu\text{A}/\text{cm}^2$ or any spikes during a 950 ms zero-current check.

The gate-control experiment applied identical 900 ms steps at 8 $\mu\text{A}/\text{cm}^2$ to the reference baseline, zero M-conductance, increased M-conductance ($\setminus(0.225 \text{ mS}/\text{cm}^2 \setminus)$), zero A-conductance, and a condition holding the A-inactivation gate b at its post-equilibration value. All other parameters were fixed. Initial, minimum and final-five-ISI measurements were taken from each condition's own spike train. Complete voltage and gate trajectories were recorded at 0.010 ms; displayed gate trajectories use 25 ms means.

3. Results

3.1 Action potential waveform and gating dynamics

A 2 ms suprathreshold somatic pulse evoked a single action potential (Figure 2A). The voltage rose from -73.4 mV to +41.7 mV, giving a baseline-to-peak amplitude of 115.1 mV and a half-width of 0.93 ms (Table 2). The detected threshold was -36.5 mV at 50 mV/ms, so the corresponding threshold-to-peak amplitude was 78.2 mV. These amplitude conventions describe the same spike and must be distinguished in literature comparisons.

Table 2. Baseline measurements, feature definitions, and physiological interpretation

Property	Reported baseline	Measurement definition	Interpretation
Resting potential	-73.4 mV	Equilibrated soma	Reference operating point
AP threshold	-36.5 mV	50 mV/ms upstroke crossing	Depolarized relative to -55 to -50 mV [6,7]
AP amplitude	115.1 / 78.2 mV	Peak - baseline / peak - threshold	Different reference voltages
AP half-width	0.93 ms	Half baseline-to-peak amplitude	Comparison range approximately 1-2 ms [6]
Firing onset	5 $\mu\text{A}/\text{cm}^2$	950 ms step; sampled resolution	Onset in [4,5] $\mu\text{A}/\text{cm}^2$
F-I gain	29.33 spikes/s per $\mu\text{A}/\text{cm}^2$	Six levels from 5 to 14 $\mu\text{A}/\text{cm}^2$	$R^2 = 0.9936$
Post-peak lengthening	17.8%	(7.8 6.62)/6.62	900 ms step at 8 $\mu\text{A}/\text{cm}^2$
Dendritic Na sensitivity	24.7% decrease	$g_{\text{Na,d}}: 8 \rightarrow 4 \text{ mS}/\text{cm}^2$	Local conductance response

Amplitudes are defined relative to their reference voltage. The model’s baseline-to-peak and threshold-to-peak values are not interchangeable. Literature waveform comparisons require matching measurement criteria, recording methods, and temperature; current-density measurements also require assuming a membrane area before converting to pA. Na⁺ activation m increased rapidly during the upstroke, while the Na⁺ inactivation variable h decreased during the upstroke and recovered following the spike (Figure 2B). Delayed-rectifier activation n increased more slowly and remained elevated during repolarization. The post-spike voltage minimum was -73.5 mV, only 0.1 mV below the resting baseline. Thus, the baseline model produces a shallow afterhyperpolarization.

3.2 Repetitive firing and F-I characteristics

Under sustained suprathreshold stimulation, the model generated repetitive somatic action potentials, as illustrated by the spike train and corresponding phase-plane trajectory in Figure 3. During 950 ms current steps, no spikes were detected at 0-4 $\mu\text{A}/\text{cm}^2$, whereas repetitive firing first occurred at the sampled current density of 5 $\mu\text{A}/\text{cm}^2$ (Table 3). The firing rate subsequently increased with injected current, reaching 281.1 spikes/s at 14 $\mu\text{A}/\text{cm}^2$ (Figure 4). At 8 $\mu\text{A}/\text{cm}^2$, the stimulus-averaged rate was 123.2 spikes/s and the first-ISI rate was 67.0 spikes/s (Table 3). The former includes the entire 950 ms response; it is not the late instantaneous rate. Ordinary least-squares regression of

mean rate against current at the six reported suprathreshold levels (5, 6, 8, 10, 12, and 14 $\mu\text{A}/\text{cm}^2$) gives a slope of 29.33 spikes s⁻¹ per $\mu\text{A}/\text{cm}^2$ and $R^2 = 0.9936$. This summarizes the sampled range rather than the local gain near onset. The transition from no spikes at 4 $\mu\text{A}/\text{cm}^2$ to firing at 5 $\mu\text{A}/\text{cm}^2$ places the sampled firing-onset current in [4, 5] $\mu\text{A}/\text{cm}^2$; the 1 $\mu\text{A}/\text{cm}^2$ resolution does not resolve the onset bifurcation.

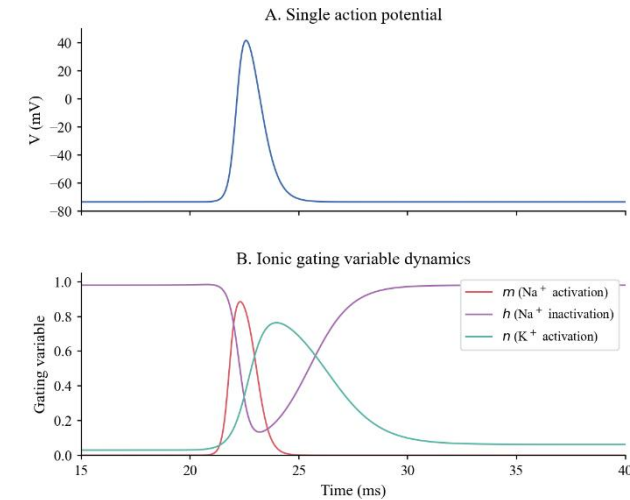


Figure 2. Isolated somatic response to a 2 ms suprathreshold pulse. (A) Somatic voltage, with peak +41.7 mV and pre-spike baseline -73.4 mV. (B) Somatic Na⁺ activation m, Na⁺ inactivation h, and delayed-rectifier K⁺ activation n. Both panels share the labeled time axis; voltage is in mV, and gates are dimensionless

3.3 Initial firing acceleration and post-peak adaptation

During the separate 900 ms step at 8 $\mu\text{A}/\text{cm}^2$, the model generated 110 spikes (Figure 5A), corresponding to a stimulus-averaged rate of 122.2 spikes/s. The first ISI was 14.9 ms (67.1 spikes/s), and the minimum was 6.62 ms around the fourteenth interval (151.1 spikes/s). The late ISI approached 7.8 ms (128.2 spikes/s; Figure 5B). Relative to the minimum ISI, late ISI lengthened by approximately 17.8%, equivalent to a 15.1% fall in instantaneous rate. The late rate nevertheless remained above the first-ISI rate. Initial acceleration and subsequent post-peak adaptation therefore coexist in the same response. The baseline response shows modest adaptation after a larger initial acceleration. M-conductance dependence is assessed separately in Section 3.6 using the zero-conductance condition and increased-conductance conditions. This distinguishes the observed ISI trajectory from the effect of a specific model current.

3.4 Dendritic backpropagation and a-current regulation

Somatic excitation produced a dendritic spike with baseline-to-peak amplitude 92.8 mV, compared with 115.1 mV at the soma (Figure 6), a soma-to-dendrite reduction of approximately 19.4%. Removing dendritic A-type K⁺ conductance increased dendritic amplitude to 100.8 mV, an 8.6% increase, and prolonged repolarization. Thus, the A-current opposes spike amplitude and contributes to dendritic recovery in this model. The magnitude of the recovery effect also depends on the baseline architecture, which lacks a separate dendritic delayed-rectifier K⁺ current. Reducing dendritic Na⁺ conductance from 8 to 4 mS/cm² reduced dendritic amplitude from 92.8 to 69.9 mV, a 24.7% reduction (Figure 7).

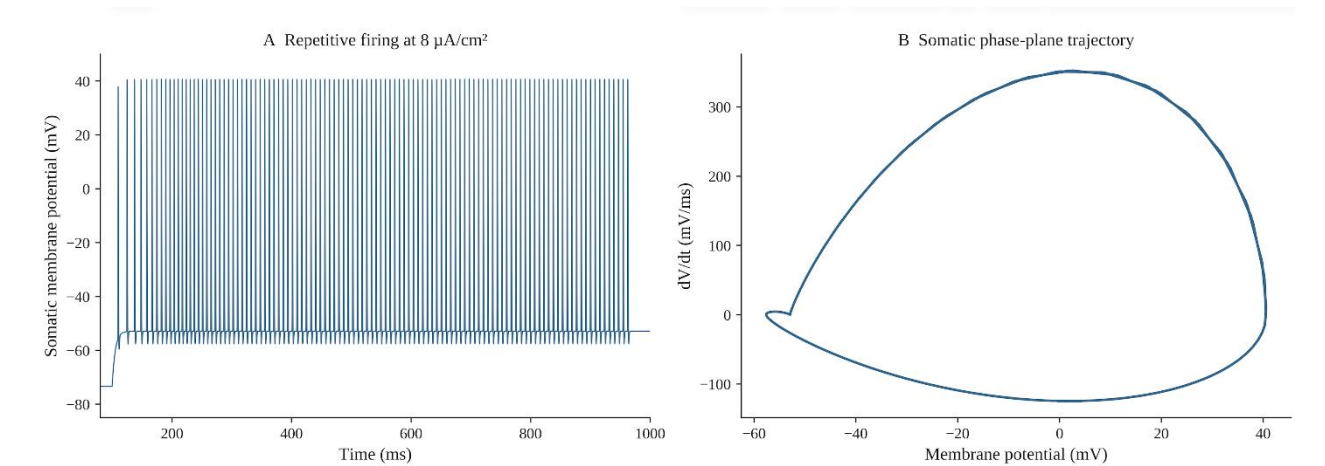


Figure 3. Repetitive somatic firing at 8 $\mu\text{A}/\text{cm}^2$. (A) Somatic voltage during the 950 ms current step. (B) Somatic phase-plane trajectory, with membrane potential in mV and dV_s/dt in mV/ms. The labeled axes give the temporal and voltage scales

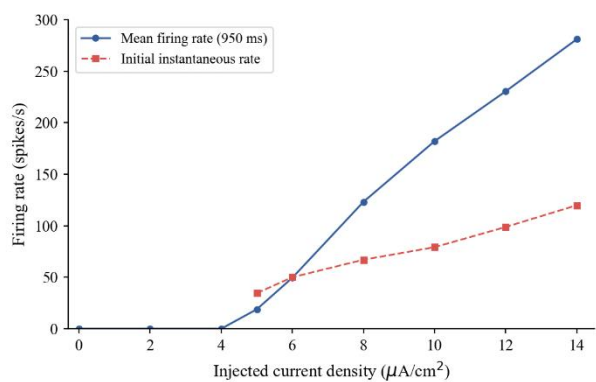


Figure 4. Baseline F-I relationship. Filled circles show spike count divided by the 950 ms stimulus duration; open squares show the inverse first ISI. Current density is normalized to somatic area. Lines connect the reported levels. The mean-rate regression uses 5–14 $\mu\text{A}/\text{cm}^2$ (six levels), with slope 29.33 spikes s^{-1} per $\mu\text{A}/\text{cm}^2$ and $R^2 = 0.9936$.

Across the complete 4–12 mS/cm² sweep, amplitude increased from 69.9 to 103.5 mV. This is a local conductance sensitivity, not an anatomical-distance measurement. Experimental work identifies dendritic Na⁺ availability and A-type K⁺ conductance as interacting determinants of backpropagation [5,6].

3.5 ISI statistics and high-current firing behavior

ISI statistics were evaluated separately at 6, 8, 10, and 12 $\mu\text{A}/\text{cm}^2$ during 950 ms stimulation. Full-train CV includes the initial transient, while late CV describes the final firing phase. Statistics are kept separate across current levels because pooling different mean firing periods would conflate between-condition differences with within-train variation.

Figure 8 and Table 4 show that mean ISI fell from 20.10 ms at 6 $\mu\text{A}/\text{cm}^2$ to 4.30 ms at 12 $\mu\text{A}/\text{cm}^2$. Full-train CV ranged from 0.042 to 0.111, and late CV from 0.007 to 0.009. These values describe deterministic timing variation rather than trial-to-trial irregularity. The reported ISI counts correspond to 47, 117, 173, and 219 spikes, respectively, consistent with the rounded mean rates in Table 3.

Table 3. Baseline F-I measurements at the reported selected levels from the 0–14 $\mu\text{A}/\text{cm}^2$ scan. Mean rate uses the full 950 ms step; initial rate uses the first ISI. A dash indicates that no ISI exists. Regression is restricted to the six displayed suprathreshold levels (5–14 $\mu\text{A}/\text{cm}^2$), giving $R^2 = 0.9936$

Injected current ($\mu\text{A}/\text{cm}^2$)	Mean firing rate (spikes/s, 950 ms)	Initial instantaneous rate (spikes/s)
0	0.0	—
2	0.0	—
4	0.0	—
5	18.9	34.6
6	49.5	50.0
8	123.2	67.0
10	182.1	79.2
12	230.5	98.8
14	281.1	119.9

Table 4. Within-current ISI statistics for 950 ms steps. N is the number of ISIs, one fewer than the spike count. Values after $\pm$ are ISI standard deviations, not uncertainty across replicate trials. Full CV includes all intervals; late statistics describe the final firing phase.

Current ($\mu\text{A}/\text{cm}^2$)	N (ISI)	Full-train ISI (ms)	Full CV	Late ISI (ms)	Late CV
6	46	20.10 $\pm$ 1.92	0.096	19.30 $\pm$ 0.17	0.009
8	116	8.08 $\pm$ 0.90	0.111	7.80 $\pm$ 0.07	0.009
10	172	5.43 $\pm$ 0.31	0.057	5.38 $\pm$ 0.04	0.007
12	218	4.30 $\pm$ 0.18	0.042	4.27 $\pm$ 0.03	0.007

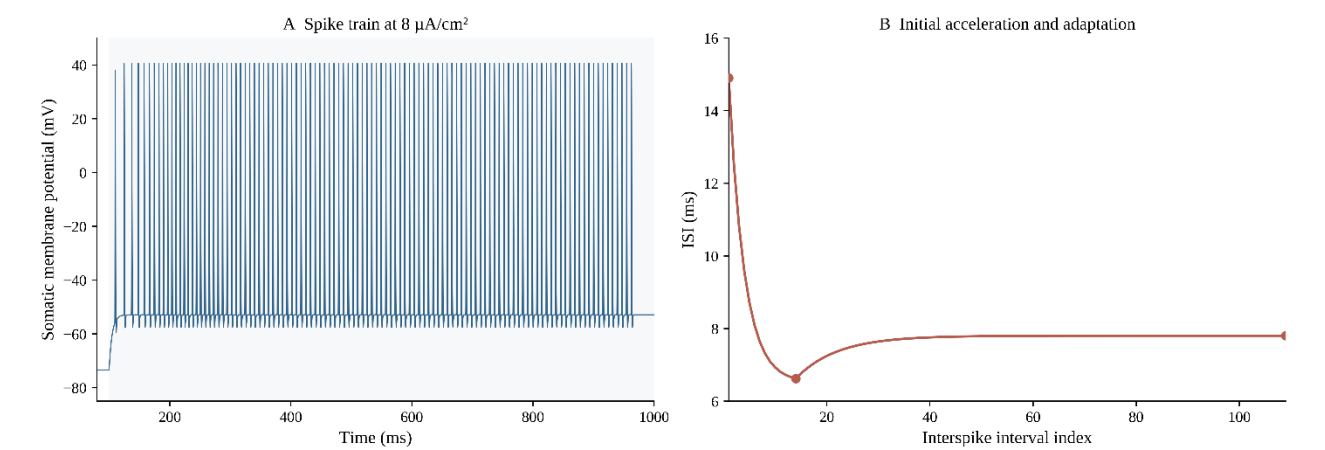


Figure 5. Somatic response to a 900 ms step at 8 μA/cm². (A) Voltage trace, containing 110 spikes. (B) Successive ISIs: first, 14.9 ms; minimum, 6.62 ms; late plateau, approximately 7.8 ms. ISI index distinguishes the early acceleration from later lengthening. This 900 ms protocol is separate from the 950 ms F-I protocol.

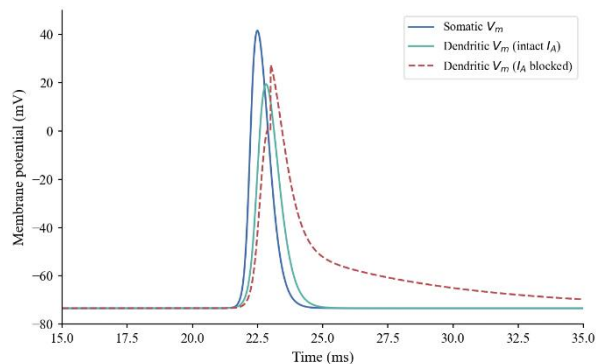


Figure 6. Somatic and lumped dendritic voltage responses to somatic excitation. The solid somatic trace is compared with dendritic traces at intact $g_{A,d}$ = 12 mS/cm² and blocked $g_{A,d}$ = 0. Dendritic baseline-to-peak amplitudes are 92.8 and 100.8 mV, respectively. The labeled time and voltage axes show the scales; the blocked condition exhibits slower recovery.

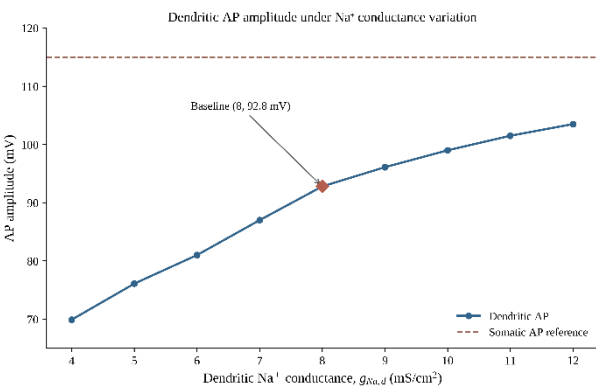


Figure 7. Dendritic Na⁺ conductance sensitivity in the lumped compartment, with $g_{A,d}$ fixed at 12 mS/cm². Dendritic amplitude is peak minus its own pre-spike baseline. The baseline point is $g_{Na,d}$ = 8 mS/cm², amplitude 92.8 mV; the dashed line is the somatic amplitude reference (approximately 115 mV). The horizontal axis denotes conductance, with no anatomical-distance mapping.

The extended scan produced mean rates of 303.2 and 310.5 spikes/s at 16 and 18 μA/cm², followed by 257.9, 85.3 and 3.2 spikes/s at 20, 22 and 24 μA/cm² (Figure 9). The response therefore turns downward above 18 μA/cm². Depolarization block is defined by cessation of repetitive discharge during sustained depolarization; Figure 9 summarizes the rate response.

3.6 Parameter sensitivity and interaction analysis

The sensitivity analysis varied p , g_c , $g_{M,s}$, $g_{A,d}$, $g_{Na,d}$, and V_T (Figure 10; Table 5). Relative to baseline, p spans ±20%, g_c spans ±50%, $g_{A,d}$ includes blockade and a 50% increase, and $g_{Na,d}$ spans ±50%. The M-conductance scan includes zero conductance and values up to 50% above baseline. Increasing p from 0.40 to 0.60 changed mean firing rate from 114 to 132 spikes/s. Increasing g_c from 0.5 to 1.5 mS/cm² increased dendritic amplitude from 85.0 to 96.7 mV and reduced response latency from 0.52 to 0.26 ms. Increasing $g_{M,s}$ from 0 to 0.225 mS/cm² reduced the late rate from 156 to 111 spikes/s; the post-peak adaptation index increased from 0.012 to 0.225. Thus, the zero-M condition provides within-model control of conductance. The scan supports an M-current contribution to late firing and post-peak adaptation, but this summary does not isolate the mechanism of the preceding acceleration.

Table 5. Parameter sensitivity and electrophysiological responses

Parameter	Scanned range	Principal response
p	0.40–0.60	Normalized input resistance: 0.86–1.17; firing rate: 114–132 spikes/s
g_c	0.5–1.5 mS/cm²	Dendritic AP amplitude: 85.0–96.7 mV; response latency: 0.52–0.26 ms
$g_{M,s}$	0–0.225 mS/cm²	Late firing frequency: 156–111 spikes/s; adaptation index: 0.012–0.225
$g_{A,d}$	0–18 mS/cm²	Dendritic AP amplitude: 100.8–88.9 mV
$g_{Na,d}$	4–12 mS/cm²	Dendritic AP amplitude: 69.9–103.5 mV
V_T	–61 to –55 mV	AP threshold: –39.8 to –33.1 mV; rheobase: 4–7 μA/cm²

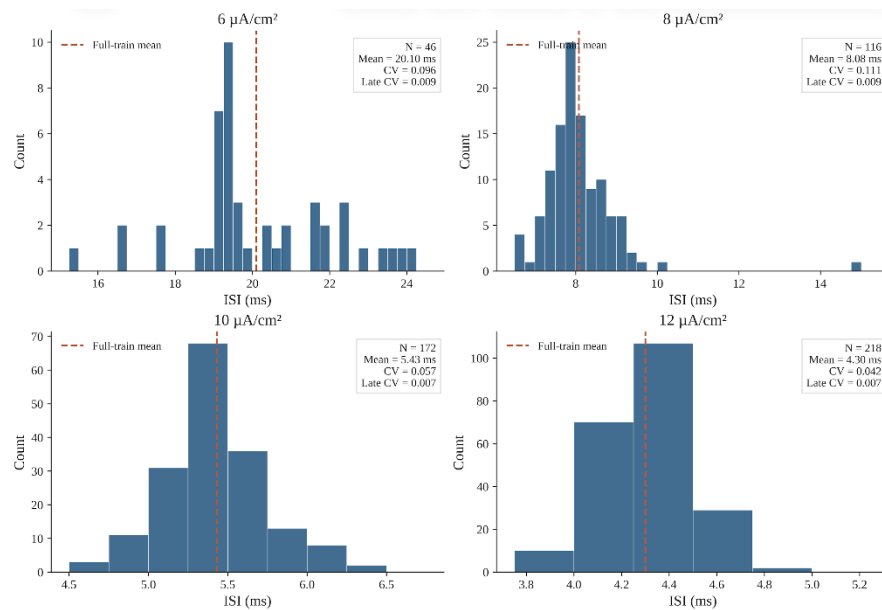


Figure 8. Current-specific ISI histograms for 950 ms somatic steps. Panels identify current density, ISI count N, full-train mean and CV, and late CV. The dashed line marks each full-train mean. Counts are 46, 116, 172, and 218 at 6, 8, 10, and 12 $\mu\text{A}/\text{cm}^2$. Horizontal axes are in ms; vertical axes show interval counts.

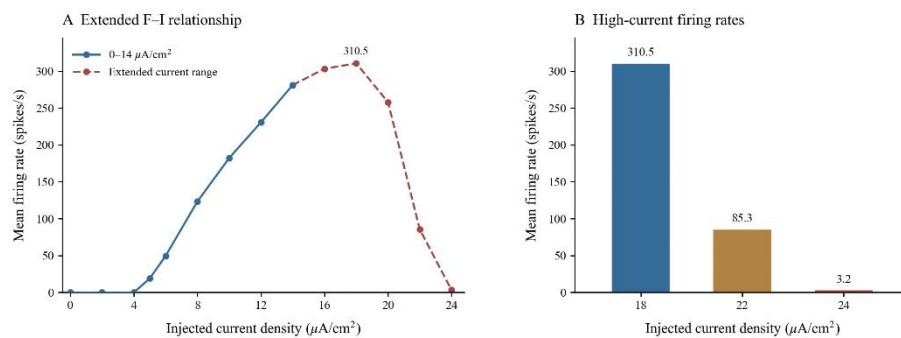


Figure 9. High-current mean-rate response. (A) Extended F-I relationship over 0–24 $\mu\text{A}/\text{cm}^2$. (B) Mean rates at 18, 22, and 24 $\mu\text{A}/\text{cm}^2$, illustrating the high-current downturn.

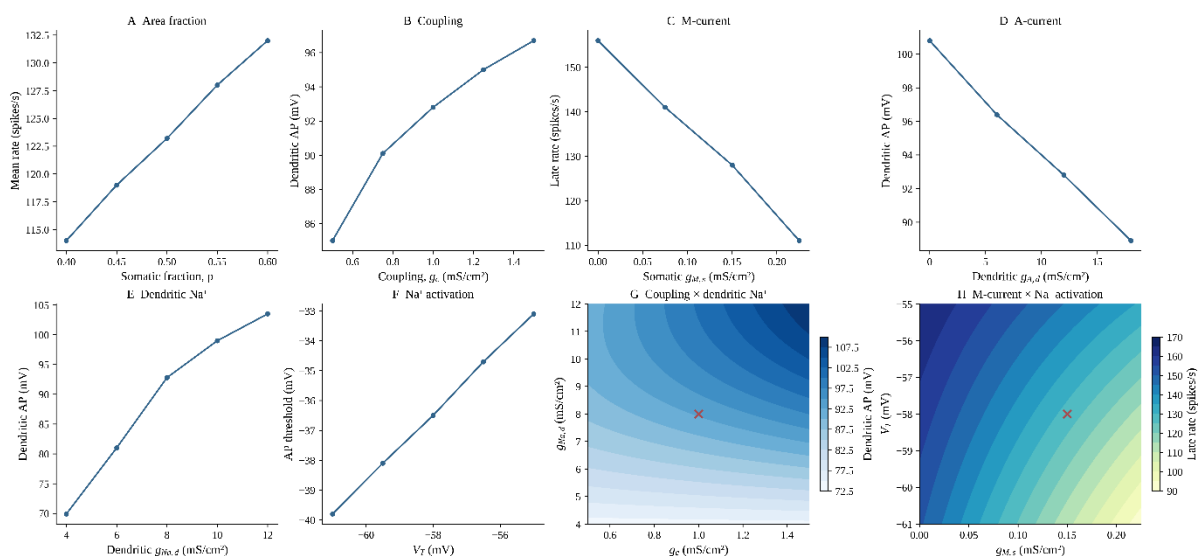


Figure 10. Parameter sensitivity and interaction analysis. (A) Somatic area fraction and firing frequency. (B) Coupling conductance and dendritic action potential amplitude. (C) Somatic M-type K^+ conductance and late firing frequency. (D) Dendritic A-type K^+ conductance and spike amplitude. (E) Dendritic Na^+ conductance and spike amplitude. (F) Na^+ activation voltage shift and action potential threshold. (G) Interaction between coupling and dendritic Na^+ conductance. (H) Interaction between M-type K^+ conductance and Na^+ activation voltage shift.

Pairwise scans illustrate how dendritic amplitude depends jointly on g_c and $g_{Na,d}$, and late rate on $g_{M,s}$ and V_T (Figure 10G,H). These response surfaces show that a parameter's effect depends on the background conductance. They are interpreted as joint parameter responses rather than a separately estimated interaction coefficient. The V_T scan shifted the detected threshold from -39.8 to -33.1 mV and sampled firing-onset current from 4 to $7 \mu\text{A}/\text{cm}^2$. Joint waveform retuning is evaluated separately in Section 3.8 using the complete reference kinetics.

3.7 Numerical convergence and measurement robustness

Measurement and numerical comparisons varied threshold criteria, output sampling interval, relative solver tolerance, and cable segment number (Figure 11, Table 6). Temporal and tolerance comparisons concern the two-compartment waveform, whereas the segment-number comparison concerns the auxiliary cable discretization. Action potential threshold estimates depended on the voltage-derivative criterion. Thresholds of -46.2 , -43.1 , and -36.5 mV were obtained using dV/dt criteria of 10 , 20 , and 50 mV/ms, respectively. Increasing the derivative criterion shifted the detected threshold toward more depolarized values along the same action potential upstroke. As output interval decreased from 0.100 to 0.005 ms, estimated peak approached $+41.7$ mV, and half-width approached 0.93 ms. The 0.010 and 0.005 ms conditions agreed at the displayed precision. Tightening relative tolerance from 10^{-4} to 10^{-7} likewise reduced changes in waveform measurements; the 10^{-6} and 10^{-7} conditions agreed at the reported precision.

For the auxiliary cable comparison, increasing segmentation from 5 to 40 raised distal amplitude from 63.9 to 66.6 mV and reduced delay from 2.81 to 2.55 ms. Refinement to 80 segments produced no further change at the displayed precision. These results establish convergence within that numerical comparison and are separate from the lumped-compartment Na^+ conductance sweep. Together, the comparisons distinguish measurement-convention effects from numerical-resolution effects. A lower derivative criterion gives a more hyperpolarized threshold on the same spike upstroke, while the peak and half-width stabilize as output sampling and solver tolerance are refined.

3.8 Joint waveform retuning

The reference baseline generated a resting potential of -70.85 mV, a threshold of -39.13 mV, a peak of $+43.86$ mV, a baseline-to-peak amplitude of 114.71 mV, and a half-width of 0.935 ms (Figure 12). These outputs were obtained from the fully specified reference kinetics; the original reported baseline remains -73.4 mV, -36.5 mV, $+41.7$ mV, 115.1 mV and 0.93 ms, respectively (Table 2). The two baselines correspond to their stated implementations and are compared within their own protocols. The retuned set was $V_T = -70.4050$ mV, $g_{Na,s} = 21.2094$ mS/cm², $g_{Kdr,s} = 5.1450$ mS/cm², $g_{M,s} = 0.214171$ mS/cm², and $E_L = -64.6691$ mV. Other parameters remained at Table 1 values. Rest became -65.58 mV, threshold -49.36 mV, peak $+31.18$ mV, amplitude 96.76 mV and half-width 0.856 ms. The normalized objective fell from 13.68 to 2.10 (84.7% reduction). The half-width target error is shown with the other feature errors in Figure 12B.

Table 6. Numerical convergence and measurement robustness

Analysis	Parameter	Setting	AP threshold (mV)	AP peak (mV)	AP half-width (ms)	Distal amplitude (mV)	Propagation delay (ms)
Threshold criterion	dV/dt threshold	10 mV/ms	-46.2	—	—	—	—
Threshold criterion	dV/dt threshold	20 mV/ms	-43.1	—	—	—	—
Threshold criterion	dV/dt threshold	50 mV/ms	-36.5	—	—	—	—
Temporal resolution	Output interval	0.100 ms	—	40.8	1.00	—	—
Temporal resolution	Output interval	0.050 ms	—	41.4	0.96	—	—
Temporal resolution	Output interval	0.020 ms	—	41.6	0.94	—	—
Temporal resolution	Output interval	0.010 ms	—	41.7	0.93	—	—
Temporal resolution	Output interval	0.005 ms	—	41.7	0.93	—	—
Solver convergence	Relative tolerance	10^{-4}	—	41.2	0.96	—	—
Solver convergence	Relative tolerance	10^{-5}	—	41.6	0.94	—	—
Solver convergence	Relative tolerance	10^{-6}	—	41.7	0.93	—	—
Solver convergence	Relative tolerance	10^{-7}	—	41.7	0.93	—	—
Spatial convergence	Dendritic segments	5	—	—	—	63.9	2.81
Spatial convergence	Dendritic segments	10	—	—	—	65.8	2.63
Spatial convergence	Dendritic segments	20	—	—	—	66.4	2.57
Spatial convergence	Dendritic segments	40	—	—	—	66.6	2.55
Spatial convergence	Dendritic segments	80	—	—	—	66.6	2.55

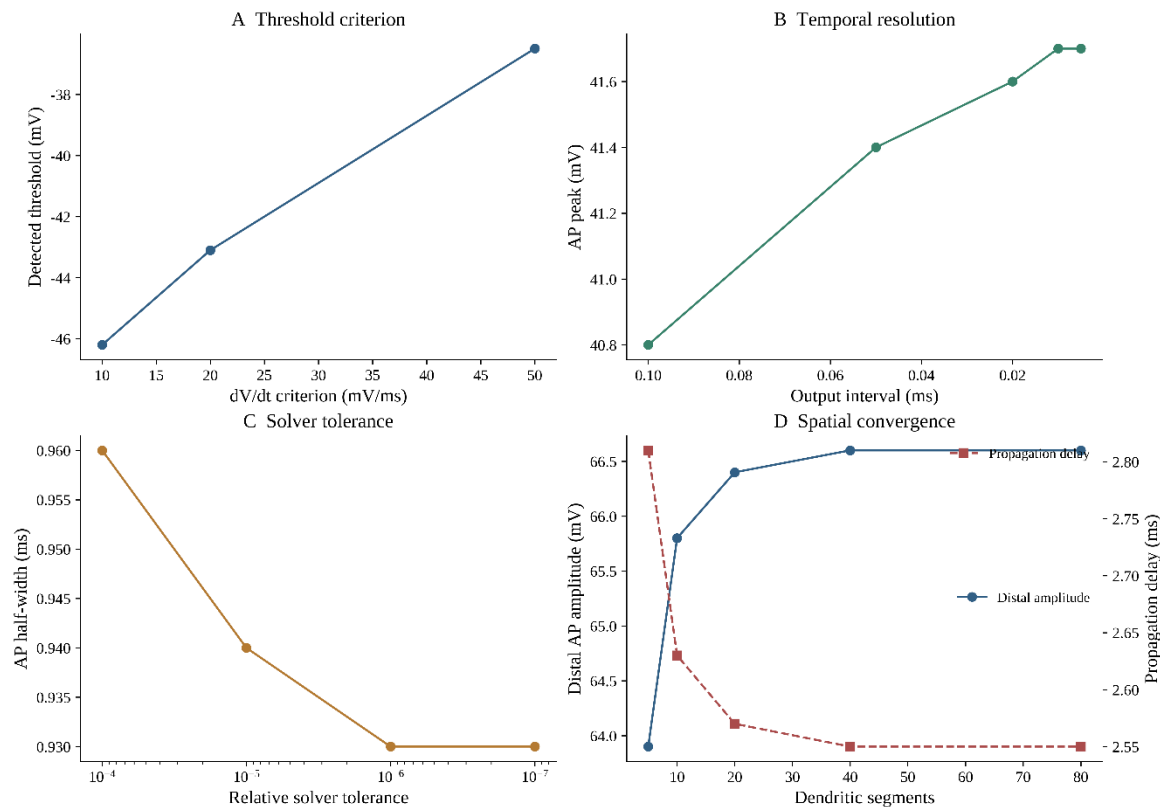


Figure 11. Numerical convergence and measurement robustness. (A) Sensitivity of detected action potential threshold to voltage-derivative criteria. (B) Effects of output sampling interval on action potential peak voltage. (C) Effects of solver tolerance on action potential half-width. (D) Spatial convergence of dendritic spike amplitude and propagation delay.

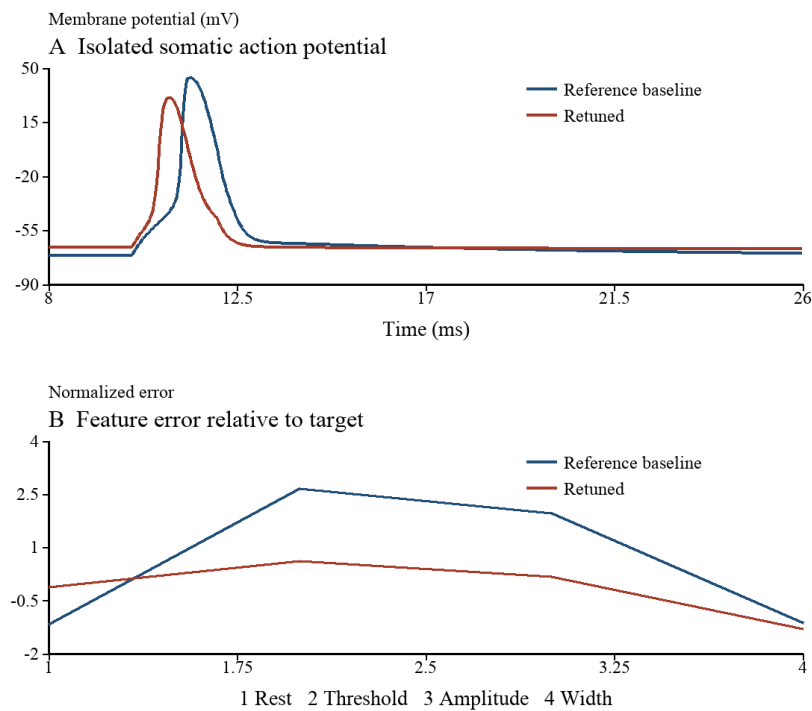


Figure 12. Joint waveform retuning in the explicit reference implementation. (A) Isolated somatic responses to the same 2 ms, 50 $\mu\text{A}/\text{cm}^2$ pulse. (B) Target errors normalized by the scales in Section 2.7; zero denotes the target value. All voltages and amplitudes use the stated reference conventions.

A $0.25 \mu\text{A}/\text{cm}^2$ scan gave firing-onset intervals of $(4.00, 4.25] \mu\text{A}/\text{cm}^2$ for the reference baseline and $(0.00, 0.25] \mu\text{A}/\text{cm}^2$ for the returned set, using at least two spikes during a 950 ms step. The zero-current check produced no spikes in either accepted condition. Halving the integration step changed peak by less than 0.01 mV and half-width by less than 0.001 ms; threshold differences were below 0.3 mV under the sampled 50 mV/ms crossing criterion.

3.9 Gate trajectories and conductance controls

The reference baseline produced 60 spikes, first ISI 7.95 ms, minimum ISI 7.95 ms and late ISI 16.61 ms during the 900 ms step (Figure 13A). Removing M-conductance produced 129 spikes, first ISI 7.39 ms, minimum ISI 6.93 ms and late ISI 6.93 ms; increasing M-conductance to $0.225 \text{ mS}/\text{cm}^2$ produced 37 spikes, first ISI 8.29 ms, minimum ISI 8.29 ms and late ISI 29.08 ms. Minimum-to-late ISI lengthening was 109.0%, 0.000% and 251.0%, respectively. The matched zero-M intervention directly links the reference implementation's late slowing to M-current availability. Holding A-current inactivation at its post-equilibration value produced 51 spikes, first ISI 9.19 ms, minimum ISI 9.19 ms and late ISI 19.77 ms. Complete A-current removal produced 117 spikes, first ISI 4.80 ms, minimum ISI 4.26 ms, and late ISI 8.74 ms. The baseline minimum occurred at ISI 2, whereas the frozen-inactivation minimum occurred at ISI 1. The early interval sequence therefore depends on dendritic A-current availability as well as the coupled voltage dynamics. Figure 13B presents the M-gate and A-inactivation trajectories recorded in the same baseline train. These gate-controlled runs use the explicit reference kinetics in Section 2.7. The original 900 ms train showed 17.8% minimum-to-late ISI lengthening (Figure 5); the new reference train shows a different adaptation magnitude. Channel-specific conclusions are drawn from interventions within the same implementation and stimulus.

4. Discussion

4.1 Somatic waveforms and physiological correspondence

At the 50 mV/ms criterion, the reported baseline threshold was -36.5 mV , compared with the cited -55 to -50 mV range; half-width was 0.93 ms compared with approximately 1–2 ms [6,7]. The original baseline therefore exhibits a waveform calibration mismatch. Lower derivative criteria yielded thresholds of -46.2 and -43.1 mV . Baseline-to-peak amplitude was 115.1 mV and threshold-to-peak amplitude was 78.2 mV, emphasizing the role of the reference voltage in amplitude comparisons.

Joint retuning in the explicit reference implementation reduced the normalized waveform error from 13.68 to 2.10. Threshold shifted from -39.13 to -49.36 mV , rest from -70.85 to -65.58 mV , and baseline-to-peak amplitude from 114.71 to 96.76 mV. Half-width changed from 0.935 to 0.856 ms. The joint objective quantifies the combined improvement and the residual width error; the two parameter sets are fully specified in Section 3.8.

4.2 Ionic mechanisms of spike-frequency adaptation

The $8 \mu\text{A}/\text{cm}^2$ response accelerates from a first-ISI rate near 67 spikes/s to a peak near 151 spikes/s and then slows to approximately 128 spikes/s. The latter slowdown corresponds to 17.8% ISI lengthening relative to the minimum interval. This sequence explains why the late rate can remain above the initial rate despite post-peak adaptation. The 123.2 spikes/s value in Table 3 is the mean over 950 ms; approximately 128 spikes/s describes late instantaneous frequency in the separate 900 ms protocol. Their different windows and definitions account for the difference. The reference gate-control experiment connects M-gate accumulation with progressive ISI lengthening. Zero M-conductance removed most post-peak lengthening, while increased M-conductance enlarged it.

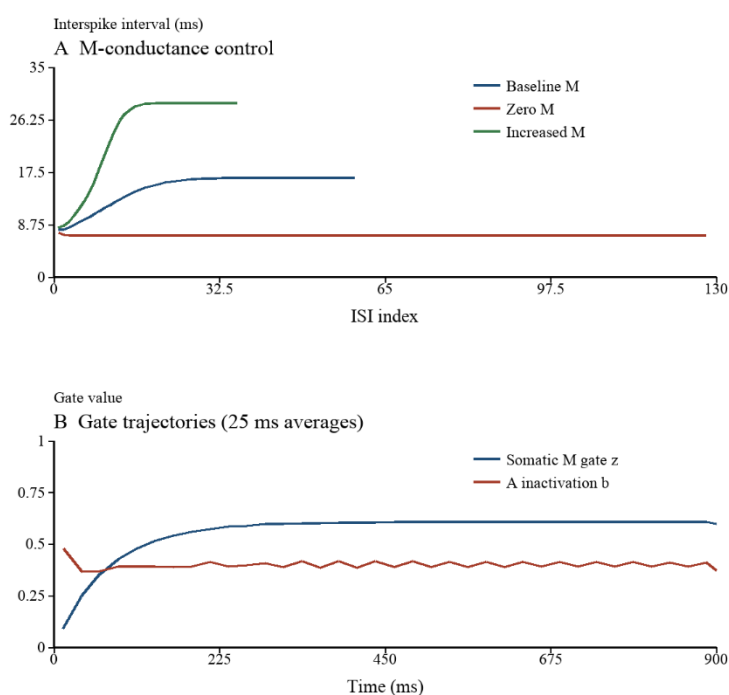


Figure 13. Matched gate-control experiment at $8 \mu\text{A}/\text{cm}^2$ for 900 ms. (A) Successive ISIs for baseline, zero-M and increased-M conditions. (B) Somatic M-gate z and dendritic A-inactivation gate b in the baseline response, displayed as 25 ms averages. Voltage-gate trajectories were sampled at 0.010 ms.

Holding dendritic A-current inactivation at its resting value changed the early firing trajectory. These interventions identify distinct roles for slow somatic outward current and dendritic gate availability under the specified reference kinetics (Section 3.9). Ca^{2+} -dependent K^+ currents provide a further adaptation pathway in CA1 [11].

4.3 Dendritic Conductance Effects

Dendritic Na^+ and A-type K^+ conductances have opposing effects on local spike amplitude. A-current blockade raises amplitude by 8.6%, whereas halving dendritic Na^+ conductance lowers it by 24.7%. The prolonged recovery after A-current blockade should be interpreted within a dendrite that lacks a separate delayed-rectifier K^+ current. It therefore demonstrates dependence on the implemented outward conductance, not the isolated recovery mechanism of biological CA1 dendrites. Experimental and detailed modeling studies place these local effects within spatially distributed dendritic channel populations [5,14,18].

The baseline dendritic amplitude is already approximately 19.4% below somatic amplitude. This compartmental difference is distinct from the 24.7% change caused by reducing dendritic Na^+ conductance. Neither percentage defines attenuation over 440 μm . Cable filtering, taper, branching, and A-current gradients are spatial mechanisms that a single lumped compartment cannot encode. The auxiliary segmentation comparison addresses numerical convergence only; anatomical attenuation requires a geometrically specified and physiologically constrained cable model.

These conductance effects can be placed within the broader context of pyramidal-neuron dendritic structure and synaptic integration [25]. Direct recordings have demonstrated regenerative responses in CA1 dendrites [26], and experimental and modeling analyses have examined dendritic spike initiation and propagation [27]. Detailed CA1 models have also related dendritic processing to a two-layer computational organization [28] and characterized nonlinear subthreshold synaptic summation [29]. Slow recovery of dendritic Na^+ channels from inactivation provide a further mechanism for activity-dependent attenuation of backpropagating action potentials [30].

4.4 Scope of the Reduced Model

The reduced architecture separates somatic spike generation from local dendritic excitability. The new experiments provide a complete channel-rate specification, a jointly returned waveform configuration, and matched gate interventions. Waveform fitting, repetitive firing and dendritic propagation address different features of the model; the fitted parameter set is evaluated on all reported waveform measurements, and its remaining errors are retained in the results. The lumped dendritic analysis concerns local conductance responses, while spatial propagation depends on cable geometry and channel distributions.

5. Conclusion

This study assessed an assembled two-compartment conductance-based model using somatic waveform measurements, F-I responses, ISI dynamics, dendritic conductance perturbations, and parameter and numerical comparisons. The assessment identifies which response patterns the compact representation generates and which quantitative features differ from CA1 electrophysiology. The baseline spike had threshold -36.5 mV under the 50 mV/ms criterion, baseline-to-peak amplitude 115.1 mV, and half-

width 0.93 ms. Firing first appeared at the sampled density of $5 \mu\text{A}/\text{cm}^2$. At $8 \mu\text{A}/\text{cm}^2$, firing accelerated initially and then showed 17.8% post-peak ISI lengthening. Increased M-conductance reduced late firing and increased the adaptation index, while dendritic A-current blockade increased local spike amplitude by 8.6%. Dendritic Na^+ reduction changed local excitability without defining anatomical distance. Joint waveform retuning lowered the normalized feature error by 84.7%. Matched gate-control calculations supported an M-current contribution to late ISI lengthening and showed that A-current availability changes the initial firing trajectory. Together, the baseline measurements and explicit reference experiments quantify the conductance-dependent responses and the waveform trade-offs of the reduced representation.

Ethical issue

The author is aware of and complies with best practices in publication ethics, specifically regarding authorship (avoidance of guest authorship), dual submission, manipulation of figures, competing interests, and compliance with research ethics policies. The author adheres to publication requirements that the submitted work is original and has not been published elsewhere. All survey participants provided informed consent before participating, and the anonymity and confidentiality of all respondents were strictly maintained throughout the research process.

Data availability statement

The manuscript contains all the data. However, additional data will be provided by the corresponding author upon reasonable request.

Conflict of interest

The author declares no potential conflict of interest.

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