

Pinsky–Rinzel Model: Dependence of Dendritic Calcium on Sodium Conductance

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Abstract

Investigated how variations in the maximal sodium conductance (\bar{g}_{Na}) modulate the mean dendritic calcium concentration in the two-compartment Pinsky–Rinzel burster. Somatic current injection (100 pA) and soma–dendrite coupling (50 nS) evoke bursting, and I quantify calcium entry as \bar{g}_{Na} is swept from 0 to 3000 nS. Observed a rise-peak-decline relationship, suggesting an optimal range of sodium conductance for calcium homeostasis.

1. Introduction

- **Background:** Role of sodium and calcium channels in neuronal excitability and homeostatic regulation.
 - **Model choice:** The Pinsky–Rinzel two-compartment model is appropriate for studying dendritic Ca dynamics.
 - **Objective:** Quantify how mean dendritic $[\text{Ca}^{2+}]$ depends on \bar{g}_{Na} under controlled somatic drive and coupling, at different conditions.
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2. Methods

2.1. Model Equations

- Cite formula from Textbook, implemented from `pm_functions.py`
- Summarize key differential equations for soma (V_s , Na and K currents) and dendrite (V_d , Ca, K_{Ca} , AHP currents).
- Cite gating functions and calcium-handling (τ_{Ca} , conversion factor).

2.2. Parameter Values

Parameter	Symbol	Value(s)	Units	Notes
Coupling	G_{Link}	10, 50, 100	nS	soma \leftrightarrow dendrite link
Somatic drive	I_{app}	0, 50, 100	pA	constant injection
Time step	Δt	2 μs	s	Euler/RK4 integration
Simulation time	T	2 s	s	discard first 0.5 s

**Other parameters simulated are the same from textbook, tutorial 4.3*

2.3. Simulation Protocol

1. For each scale factor $\alpha \in [0.0, 3.0]$ (\times baseline \bar{g}_{Na}):
 - Set $\bar{g}_{Na} = \alpha \cdot \text{baseline_G_Na}$.
 - Integrate the model for 2 s.
2. Discard $t < 0.5$ s to remove transients.
3. Compute mean dendritic $[Ca^{2+}] = (1/T) \int_{t_0}^T Ca_d(t) dt$ over $t=0.5-2.0$ s.
4. Record voltage traces at baseline and at representative \bar{g}_{Na} values (e.g. baseline, 1.5, 3.0).
5. Alternate injected current to soma and Coupling variable and repeat.

2.4. Data Analysis & Plotting

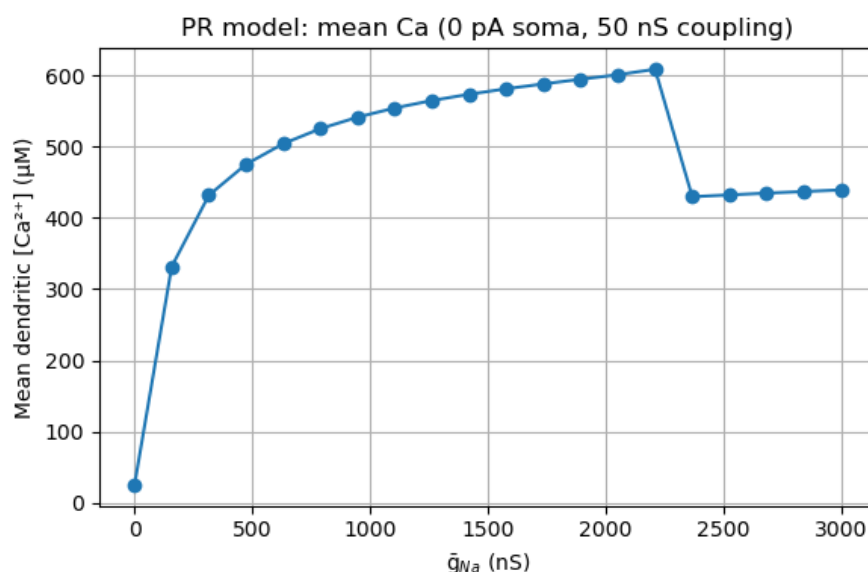
- Plot mean $[Ca^{2+}]$ vs. \bar{g}_{Na} (in nS).
- Overlay voltage traces for soma and dendrite at baseline.
- Overlay somatic voltage for selected \bar{g}_{Na} scalings.
- Compare changes in the plots with differed variables

3. Results

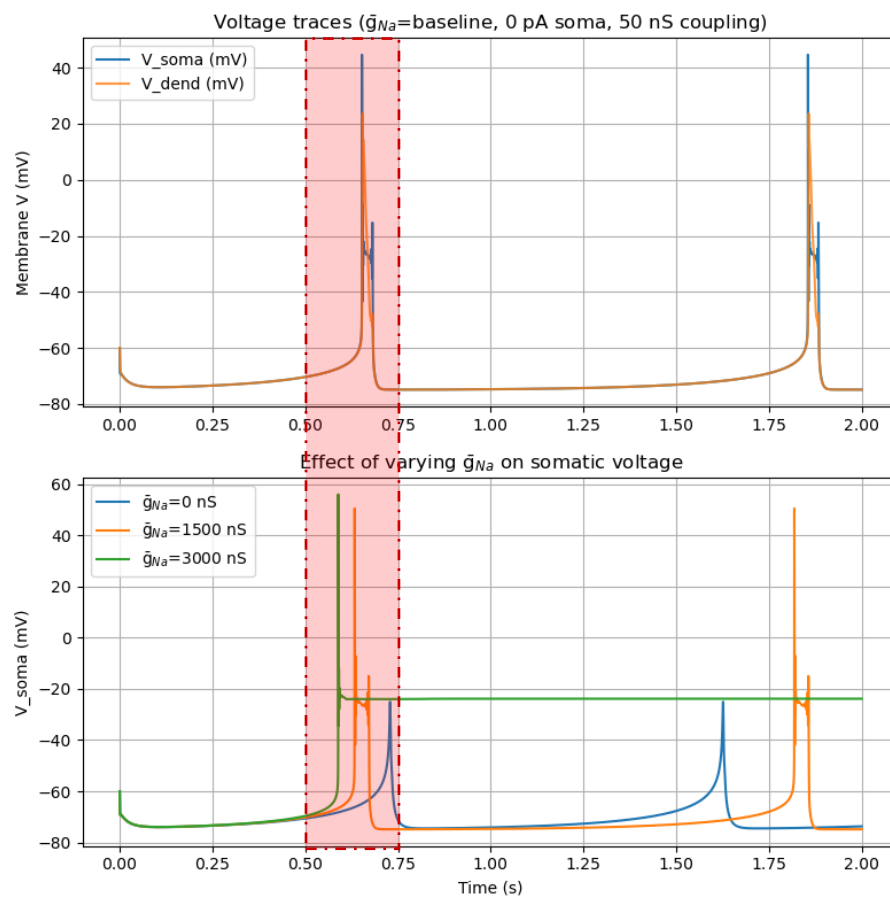
3.1 Primary results

The starting variable pair is 0 pA soma and 50 nS coupling.

In the top figure, at very low \bar{g}_{Na} (< 200 nS) the dendrite never bursts. The loop sits at a small, subthreshold equilibrium and mean $[Ca]$ rises steeply as \bar{g}_{Na} depolarizes the rest potential into the window of Ca-channel activation. Between $\sim 500-2\,000$ nS, mean $[Ca]$ asymptotes near $600\ \mu\text{M}$ as each burst grows more frequent but then saturates. Beyond $\sim 2\,500$ nS, however, the dendrite itself goes into depolarization block (its Na^+ channels inactivate), so Ca influx collapses back to a lower plateau ($\sim 430\ \mu\text{M}$) until truly extreme \bar{g}_{Na} revives only a small residual Ca

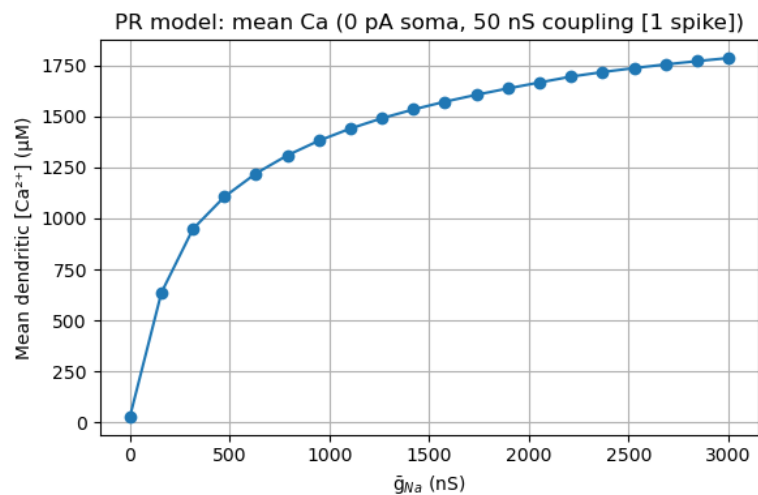


current.



Looking at the bottom panel, with baseline \bar{g}_{Na} (blue), there are two clear dendritic Ca^{2+} -spike bursts at ~ 0.65 s and ~ 1.8 s in the top subpanel, and these propagate through the 50 nS coupling to produce corresponding somatic depolarizations. At intermediate $\bar{g}_{Na} = 1\,500$ nS (orange), the bursts are larger and arrive just a hair earlier, driving bigger somatic spikes. At high $\bar{g}_{Na} = 3\,000$ nS (green), the soma is held depolarized around -25 mV by the persistent dendritic drive and inactivated Na^+ channels, so no discrete spikes appear.

3.2 Single Burst

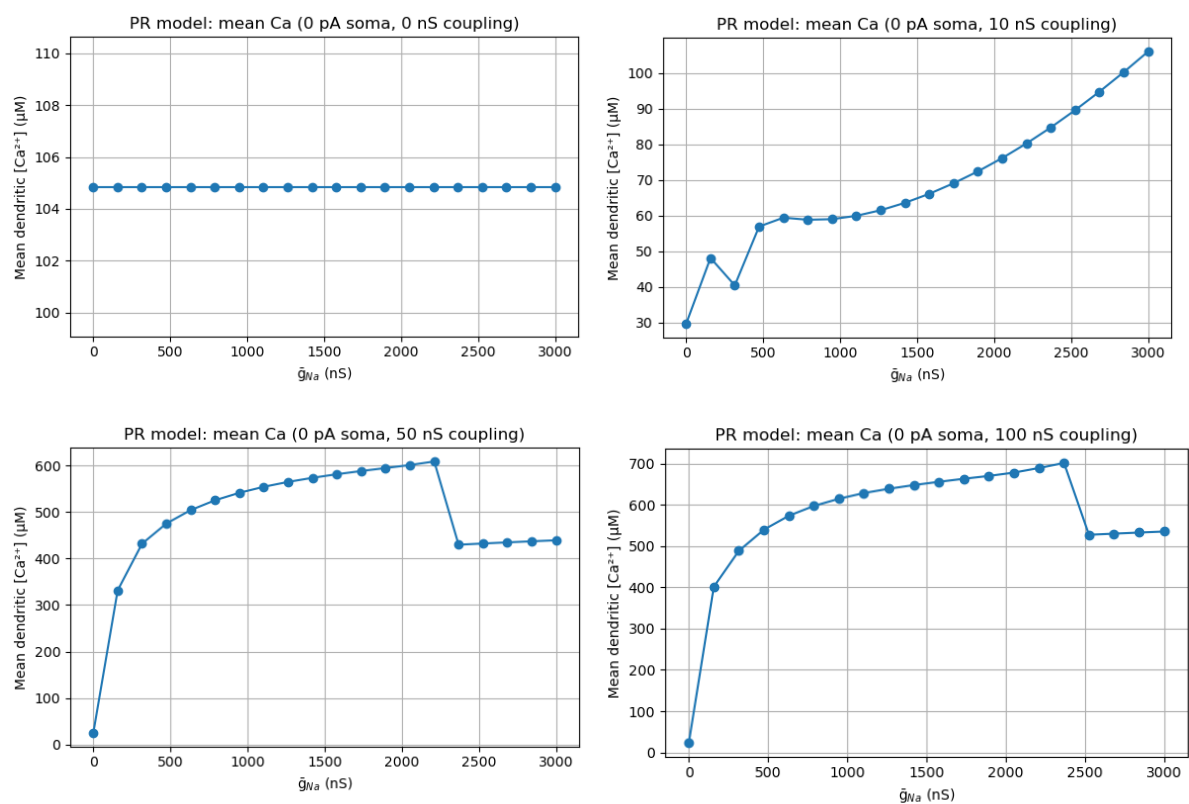


From the bottom panel I found that at this condition, a single burst occurs at all \bar{g}_{Na} levels. Thus, the following figure is generated by restricting the averaging window to the single burst between 0.5 s and 0.75 s. By constraining the analysis to a single event, the amplitude of Ca^{2+} influx per spike is decoupled from the frequency of bursts.

As \bar{g}_{Na} increases beyond threshold, each action potential becomes larger (higher peak) and/or broader (longer duration), driving more Ca^{2+} entry through the dendritic channels. Hence the mean Ca^{2+} climbs smoothly toward $\sim 1800 \mu M$ for a single-spike window.

Since only one spike is measured, and it is before depolarization-block can set in, the downturn does not appear when averaging over longer bursting or multiple spikes.

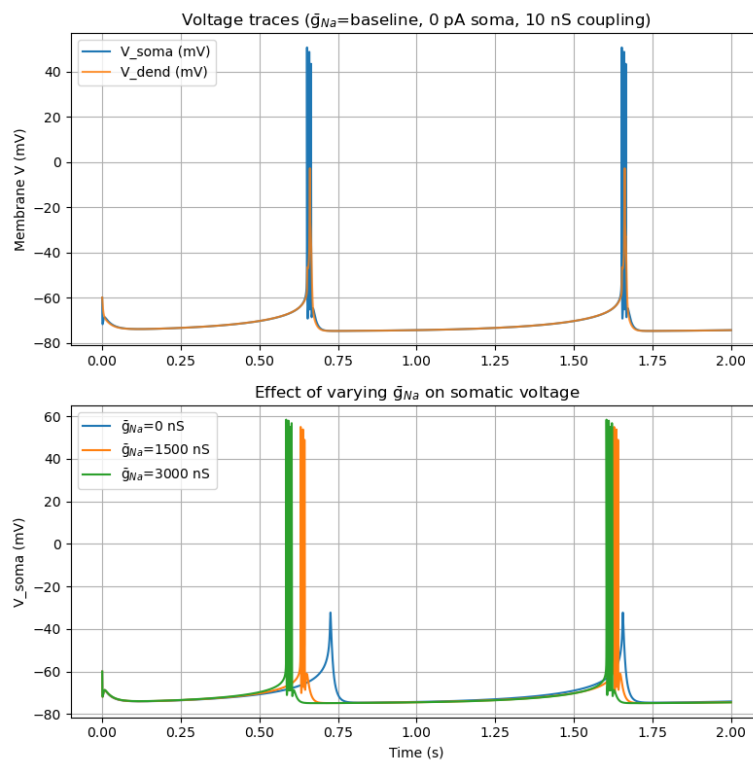
3.3 Coupling Effects



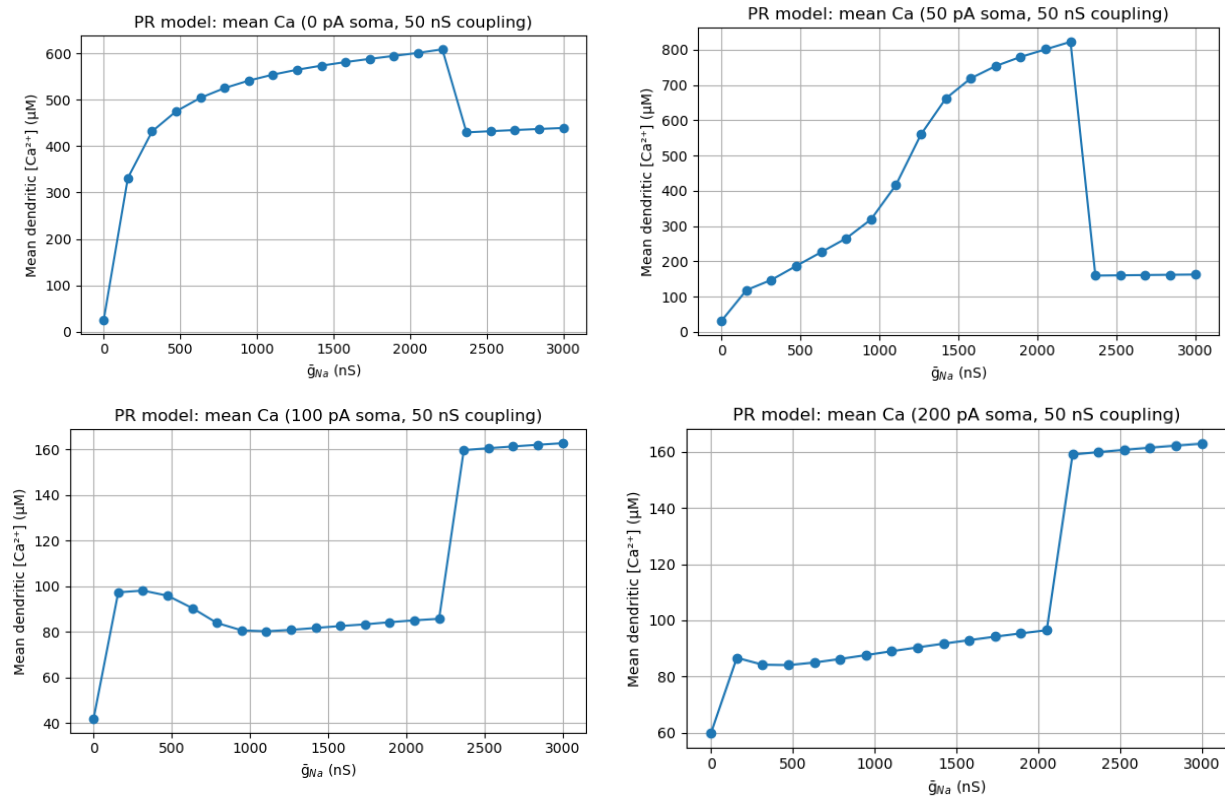
While without any injected current, set G_{link} to 0nS, 10nS, 50nS and 100nS.

At 0nS, the dendritic Ca^{2+} current simply sits at its tiny leak-equilibrium, so mean Ca^{2+} is perfectly flat ($\sim 105 \mu M$) for all \bar{g}_{Na} . The plot from 10nS is interesting to look at: it looks that it behaves differently at this condition, with steadily increasing Ca^{2+} along with \bar{g}_{Na} .

But it could be explained if we look at the voltage panel of it: since the coupling is not strong enough, the sodium ions have not accumulated too much which will cause the soma to keep being depolarized. And at 100nS, the relation acts pretty like the primary results.



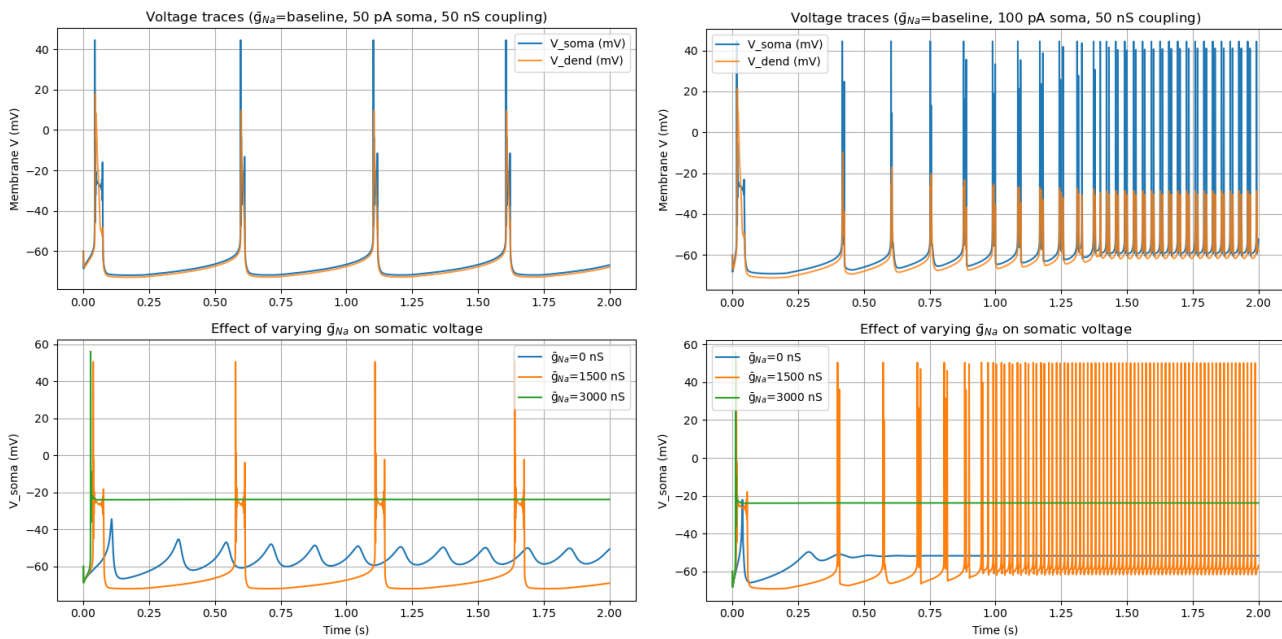
3.4 Injected Current



While keeping the G_{link} constantly at 50 nS, alternated the injected current to be 0pA, 50pA, 100pA and 200pA. By averaging from 0.5 to 2.0 s, both how many bursts occur and how much Ca each burst delivers are captured.

At 50 pA, a small somatic drive dramatically increases burst frequency once \bar{g}_{Na} crosses ~ 200 nS. With very strong drive at 100pA or 200pA applied, bursts speed up again (you squeeze in more bursts over 1.5 s), so the mean climbs gradually from $\sim 90 \mu\text{M}$ up to $\sim 160 \mu\text{M}$ right before block. After $\sim 2.2 \mu\text{S}$, block shuts off bursts and the final jump takes place.

We might need to look at the voltage traces to explain the behaviors.



At 50 pA of somatic drive, it seems to be near the optimal frequency for the intrinsic Ca burst oscillator. Although the intrinsic burst frequency increases as larger drives, single spikes are way narrower and hence lower Calcium transients' amplitude.

4. Conclusion

In this final project, I varied the maximal sodium conductance in the two-compartment Pinsky–Rinzel model to quantify its effect on dendritic calcium dynamics under different somatic drive and coupling conditions. When no somatic current was injected, increasing \bar{g}_{Na} first depolarized the resting potential enough to engage the intrinsic Ca^{2+} oscillator before driving the dendrite into depolarization block at very high conductance.

Introducing a modest somatic injection (50 pA) dramatically increased burst frequency, yielding the largest overall Ca^{2+} accumulation, whereas stronger injections (100–200 pA) shifted the oscillator's period and reduced per-window Ca^{2+} despite higher burst rates. Varying coupling strength further tuned the sensitivity of dendritic Ca^{2+} to changes in \bar{g}_{Na} , from flat equilibrium at

zero coupling to steep recruitment and block at moderate-to-strong coupling.

The relationship between sodium conductance and calcium influx demonstrated can be concluded: there exists an optimal \bar{g}_{Na} range that maximizes Ca entry without triggering inactivation, while generally increasing sodium conductance will lead to increasing in dendritic Ca^{2+} until the ion accumulation keeps the neuron from hyperpolarization to causes a constant depolarizing block.

References

1. Textbook, Chapter 4 "Conductance-based models".
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