

An Introduction to Computational Neuroscience: from Brain simulation to NeuroAI - Lab 1

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Results

1 Implement an active point neuron model in NEURON, then describe:

1.1 Currents (I_{Na} and I_{K})

- **I_{Na} (Sodium current):** I_{Na} is responsible for the rapid depolarization during an action potential. Its period length is about 10 ms. In the early phase of each period, it exhibits a sharp inward current as Na^+ flows into the cell, reaching its peak, which corresponds to the rising phase of the action potential. After this sharp peak, I_{Na} decreases rapidly due to the inactivation of Na^+ channels, contributing to the repolarization phase.
- **I_{K} (Potassium current):** I_{K} peaks slightly after the sodium current, as potassium channels open more slowly (see Figure 2). It is responsible for repolarizing the membrane following the action potential. As the K^+ ions exit the cell, the membrane potential returns to a more negative value. Similar to I_{Na} , I_{K} shows periodic peaks,

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though its onset and duration are slightly delayed and prolonged compared to the sodium current.

In particular, it's easy to notice that I_{Na} experience a drop in the first spike and a slowdown in its increasing speed in the following spikes, which is caused by the activation of potassium channel.

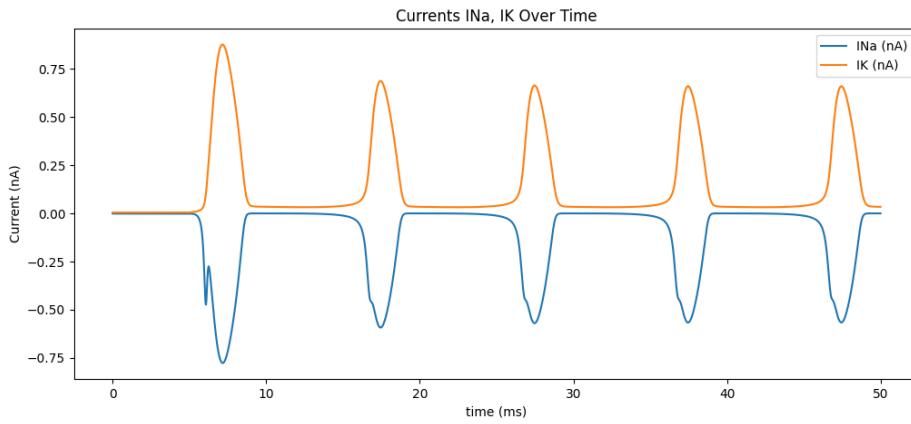


Figure 1: Currents over time

1.2 State Variables (m , h for Na^+ ; n for K^+)

- **m (Sodium activation gate):**

m increases rapidly from 0 to nearly 1 during the depolarization phase, allowing Na^+ to flow into the cell. Once the peak of the action potential is reached, m quickly returns to near 0, indicating the deactivation of sodium channels.

- **h (Sodium inactivation gate):**

h starts at a high value (near 0.6), indicating that most Na^+ channels are available for activation. As depolarization occurs, h decreases, reflecting the inactivation of sodium channels. This decrease contributes to the decline of I_{Na} after its peak. Over time, h oscillates with each action potential, decreasing during depolarization and slowly recovering during the interspike intervals.

- **n (Potassium activation gate):** n increases more slowly than m , reaching a valley after the peak of m , and reaching a peak after m has already activated and deactivated. This slower activation corresponds to the delayed opening of K^+ .

channels, allowing K^+ to exit the cell. n peaks during the falling phase of the action potential and gradually returns to lower values during the hyperpolarization phase, closing K^+ channels.

For the reason why the state variables exhibits such numerical values, see Further Analysis.

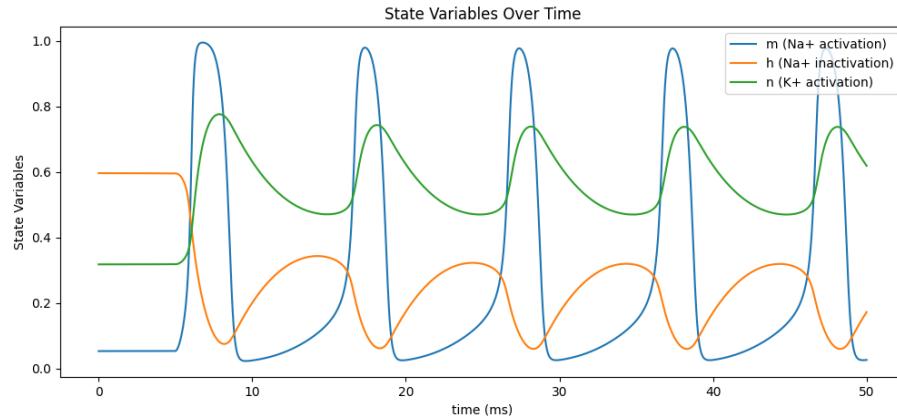


Figure 2: state variables over time

1.3 Conductance (g_{Na} and g_K)

- **g_{Na} (Sodium conductance):** g_{Na} rapidly spikes during the depolarization phase, corresponding to the activation of Na^+ channels. As Na^+ channels open, g_{Na} reaches its peak. As h (inactivation) decreases, g_{Na} also quickly decreases, explaining the sharp decline in the sodium current after its peak.
- **g_K (Potassium conductance):** g_K increases more slowly compared to g_{Na} . This conductance peaks during the repolarization phase when K^+ channels are fully activated. The slower decay of g_K reflects the sustained opening of potassium channels, which helps restore the membrane potential back to its resting state.

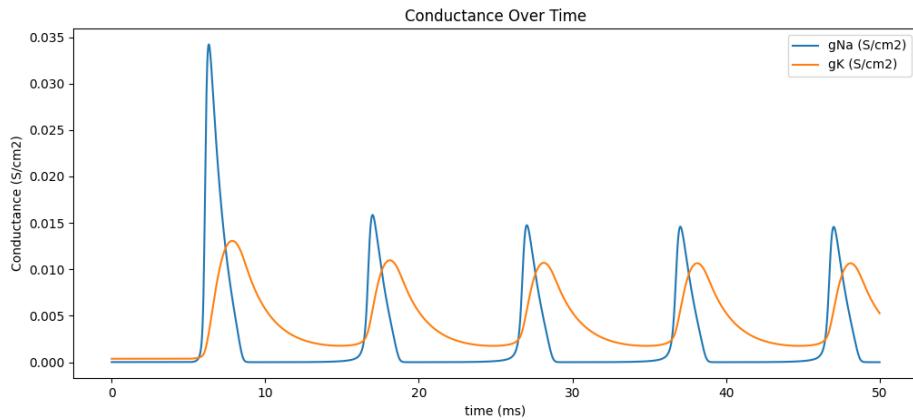


Figure 3: Conductance over time

1.4 Voltage (Membrane potential)

The membrane potential (figure 4) shows periodic spikes, namely the action potentials. The sharp depolarization phase can be attributed to the peak of the sodium current (I_{Na}), driven by the rapid opening of Na^+ channels. Similarly, the repolarization phase corresponds to the activation of potassium channels, allowing K^+ to exit the cell and bring the membrane potential back down.

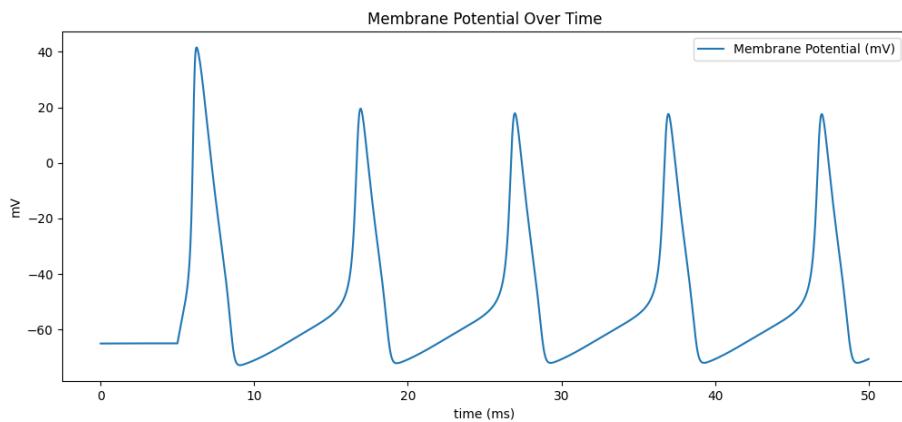


Figure 4: Voltage over time

In particular, the first peak of current and membrane voltage is higher than the following. This can be explained by the state variables: Before the first peak (when the current hadn't been injected), all the channels start in their resting state. As a result, the initial depolarization triggers a strong inward I_{Na} and outward I_K . After the first action poten-

tial, the state variable h (below 0.3) is lower than its resting value (about 0.6), meaning the sodium channel has a lower probability to stay in open state (inactivation). Similarly, the state variable n stays higher than its resting value, and the first period lasts longer than the following, resulting in the slightly higher potassium conductance in the first action potential.

2 Use the dynamics of state variables to explain the mechanisms underlying the five phases of the action potential.

2.1 The Rising Phase (Depolarization)

During the rising phase, the membrane potential depolarizes (becomes more positive). This is driven by the activation of sodium channels, represented by the activation variable m , which increases rapidly. As m increases, sodium channels open, allowing Na^+ ions to flow into the cell, causing a rapid depolarization. During this phase:

- m increases sharply as the membrane potential reaches the threshold.
- h is relatively high, keeping sodium channels available for activation.
- n increases more slowly than m , corresponding to the fact that the potassium channels take longer to open.

2.2 The Peak Phase

The peak phase occurs when the membrane potential reaches its maximum value. At this point:

- m is near its maximum value, meaning that the majority of sodium channels are open.
- h begins to decrease as sodium channels start to inactivate, reducing the Na^+ influx.
- n continues to rise as potassium channels gradually open, preparing for repolarization.

2.3 The Falling Phase (Repolarization)

During the falling phase, the membrane potential becomes more negative, returning to the resting potential. This phase is driven by:

- m drops sharply and h continues to decrease, leading to the inactivation of sodium channels and the termination of Na^+ influx.
- n reaches its peak, fully activating potassium channels, forming K^+ ions outflux, which drives the membrane potential back towards negative values (repolarization).

2.4 Afterhyperpolarization (the membrane potential temporarily becomes more negatively charged than when at rest.)

- h is still low, meaning sodium channels remain largely inactivated.
- n remains high, keeping potassium channels open, contributing to K^+ oulflux which drives the membrane potential below the resting potential before the potassium channels gradually close.

2.5 The Refractory Period

The Refractory Period consists of two parts:

- **Absolute Refractory Period:** During the early part of repolarization and afterhyperpolarization, h is very low (almost zero), meaning most sodium channels are inactivated and cannot reopen. During this period, no new action potential can be triggered.
- **Relative Refractory Period:** As h starts to recover and potassium channels (n) begin to close, the membrane potential approaches the resting level. However, due to relatively high n and incomplete recovery of h , a stronger-than-normal stimulus is required to initiate a new action potential.

As shown in the figures 5, 6 and 7, the increase of constant injected current amplitude makes the neuron return to a relatively higher hyperpolarized potential. As a result, the

stable value of state variables h (or limit average) becomes higher, making it harder to make the neuron get out of its refractory period, which leads to the damping of voltage oscillation amplitude or just failure to spike again. To recover the periodical spike under fixed large injected current amplitude, see Exercise 5.

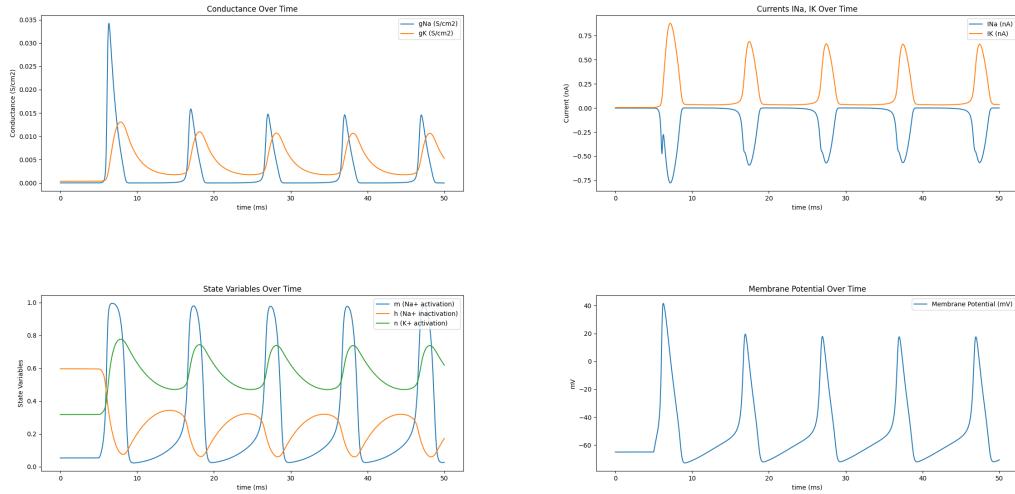


Figure 5: Conductance, current, state variable and membrane voltage when injected current is 0.1 nA

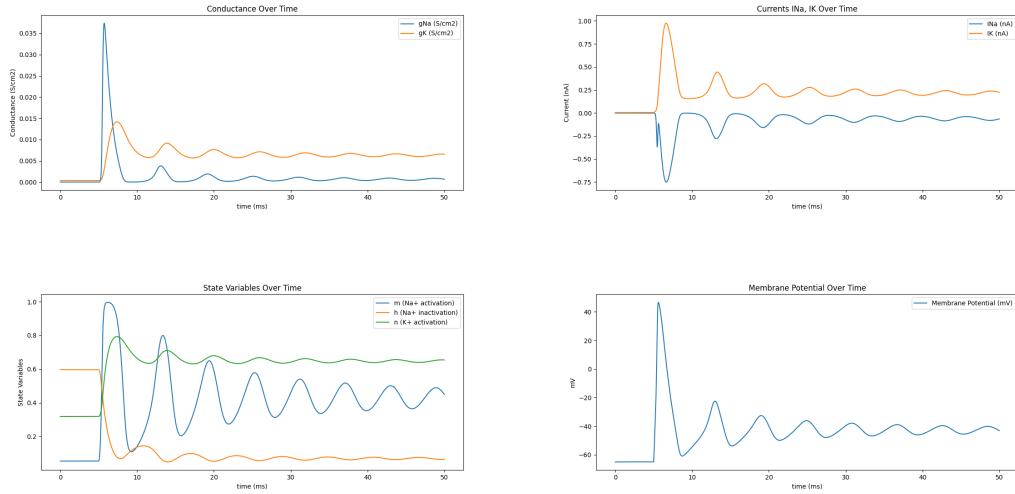


Figure 6: Conductance, current, state variable and membrane voltage when injected current is 0.5 nA

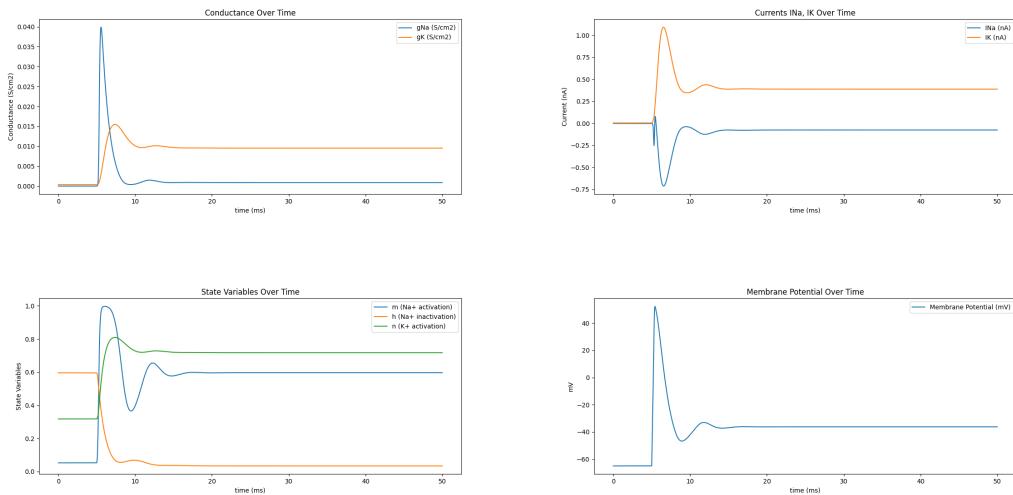


Figure 7: Conductance, current, state variable and membrane voltage when injected current is 1.0 nA

3 Current injection protocols and neuronal response

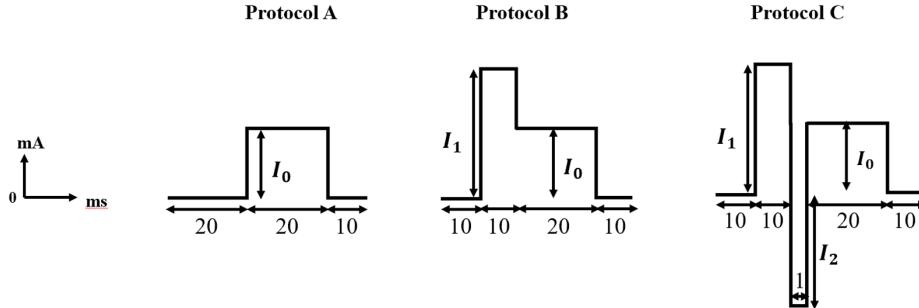


Figure 8: Protocols

3.1 Protocol A

3.1.1 Expectation

Similar to Exercise 1, the neuron responds with immediate action potentials.

3.1.2 Result

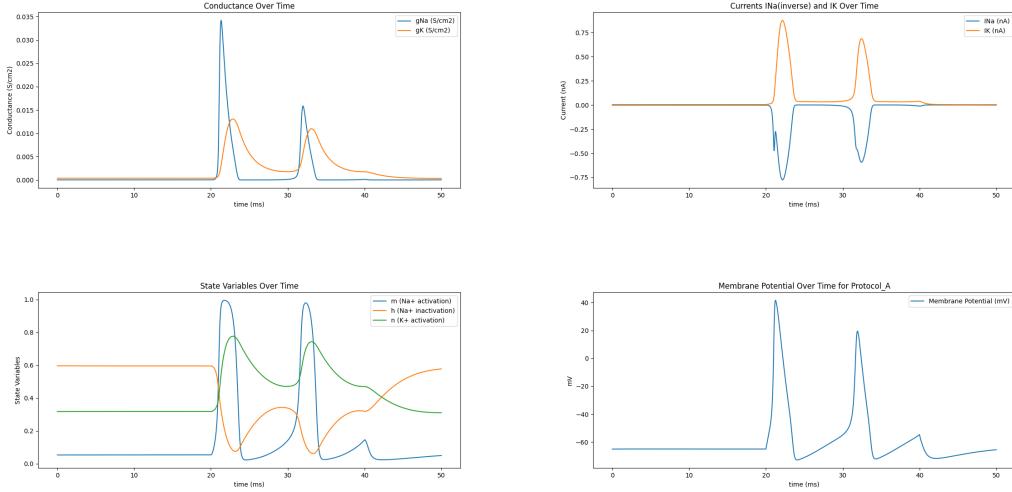


Figure 9: ProtocolA

3.2 Protocol B

3.2.1 Expectation

Given the simulated results in Exercise 1, the neuron will first generate an immediate strong action potential and be repressed by it, namely staying in the absolute refractory period. When the current amplitude drops to 0.1 nA, the neuron is release from the absolute refractory period and form weaker spikes.

3.2.2 Result

The membrane potential plot shows that the neuron fires an action potential almost immediately after the stronger current I_1 is applied. The membrane depolarizes rapidly and reaches a peak around 40 mV at approximately 10 ms. After this initial spike, the membrane repolarizes due to sodium channel inactivation (low h value) and potassium channel activation (high n value). When the current switches to I_0 at 20 ms, the neuron gradually recovers from the refractory period and produces a second and third action potential with decreased amplitude.

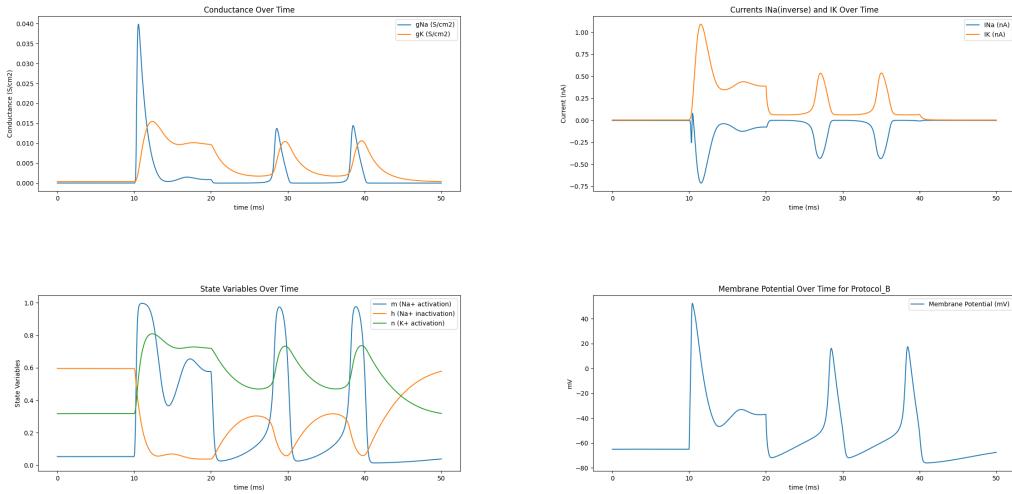


Figure 10: Protocol B

3.3 Protocol C

3.3.1 Expectation

The only difference of Protocol C from B is that there is a negative current pulse after 10 ms of I_1 . So the state of ion channel may change but not that fast. Since it brings an outflow of current, the membrane voltage instantly drops, and then recovers as the current returns to positive.

3.3.2 Result

When the hyperpolarizing current I_2 is applied, the membrane potential dips significantly, reaching below -125 mV. This means the membrane voltage goes from above the E_k to below E_k , contributing to the significant change in the direction of the potassium ion flow. Compared with protocol B, the latter two spikes come earlier thanks to the negative current which accelerates the rebounce of h and m , as well as the decrease of n . Consequently, the second spike in Protocol C occurs before that in Protocol B, meanwhile has a larger amplitude.

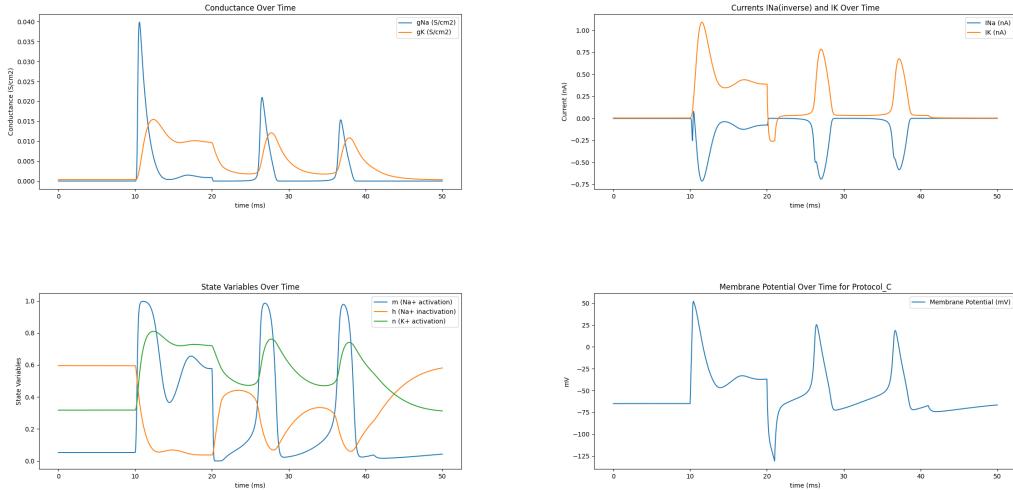


Figure 11: Protocol C

4 Add CaT channels to the HH model

4.1 Difference in cat1i,cat1g,cat1h

	vhalfn	vhalfl	kn	kl
cat1g	-51.73	-85.4	6.53	-5.4
cat1h	-43.15	-73.9	5.34	-2.76
cat1i	-60.7	-93.2	8.39	-5.4

Table 1: parameters of different CaT Channels

	taun	taul
cat1g	$(0.5 + 0.124 * \exp(-v/15.8)) / qt$	$(10.4 + 0.0118 * \exp(-v/7.85)) / qt$
cat1h	$(0.774 + 0.14 * \exp(-v/13.27)) / qt$	$(22.25 + 0.0455 * \exp(-v/7.46)) / qt$
cat1i	$(2.71 + 0.028 * \exp(-v/9.34)) / qt$	$(110 + 0.0009 * \exp(-v/5.16)) / qt$

Table 2: τ of different CaT channels

4.2 Parameter Analysis

The state variable dynamics equations for the CaT channel are:

$$\begin{cases} \frac{dn}{dt} = \frac{n_\infty - n}{\tau_n} \\ \frac{dl}{dt} = \frac{l_\infty - l}{\tau_l} \end{cases} \quad (1)$$

where n_∞ and l_∞ are the steady-state activation and inactivation variables, and τ_n and τ_l are the time constants. The specific formulas are :

$$n_\infty = \frac{1}{1 + \exp\left(\frac{-(v - v_{half,n})}{k_n}\right)} \quad (2)$$

$$l_\infty = \frac{1}{1 + \exp\left(\frac{-(v - v_{half,l})}{k_l}\right)} \quad (3)$$

4.2.1 Impact of Parameters on Dynamics

The parameters in the table affect the dynamics of the CaT channel as follows:

- **$v_{half,n}$ and $v_{half,l}$:** determine the half-maximal voltage points of the activation and inactivation curves. A more negative $v_{half,n}$ facilitates activation and shifts the activation curve to the left, while a more negative $v_{half,l}$ inhibits activation and shifts the inactivation curve to the left.
- **k_n and k_l :** determine the slope of the activation and inactivation curves. Larger k_n and k_l values result in a more gradual curve, while smaller values result in a steeper curve.
- **τ_n and τ_l :** tau is the time constant for state variables. A larger tau increases a state variable's sensitivity to voltage. Obviously, we have the order of $taun > taul > cat1i > cat1h > cat1g$.

4.3 Simulated Voltage Response

4.3.1 With constant current injection

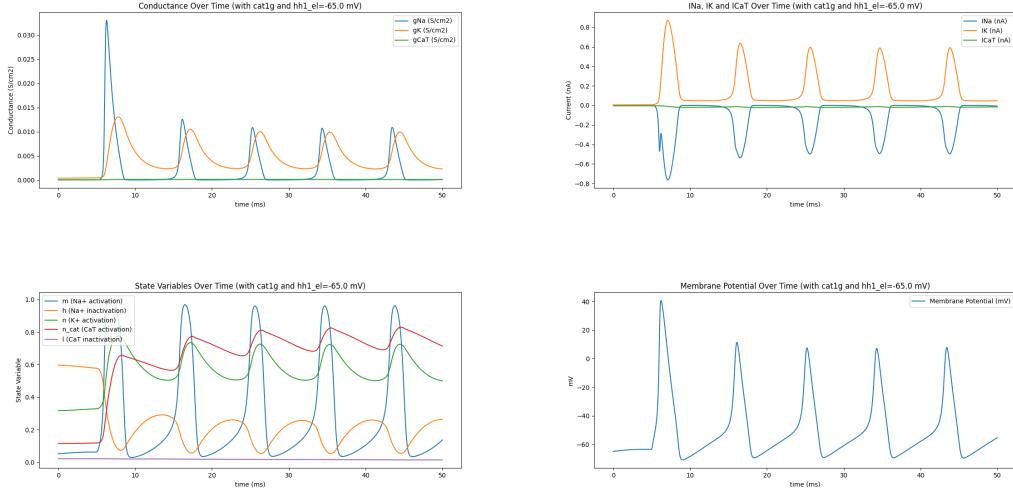


Figure 12: Conductance, current, state variable and membrane voltage with cat1g, -65mV, current injection

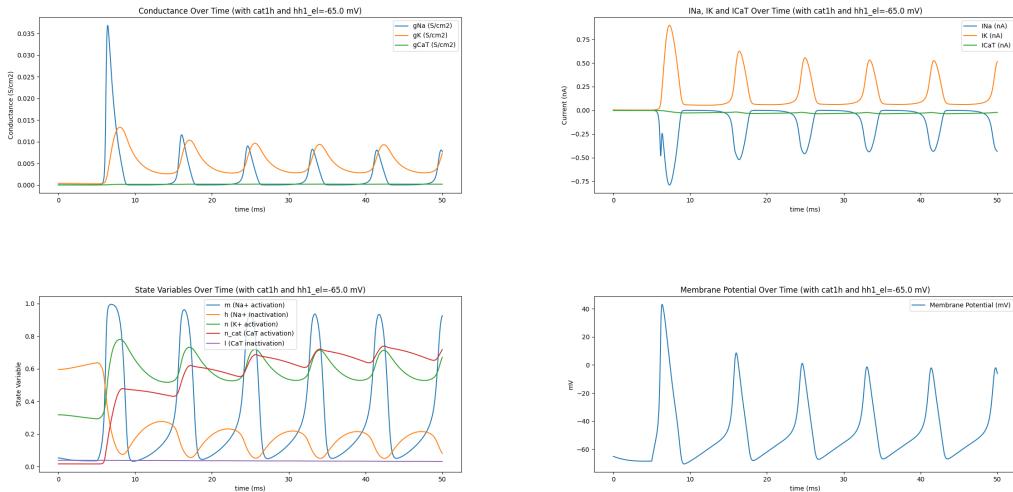


Figure 13: Conductance, current, state variable and membrane voltage with cat1h, -65mV, current injection

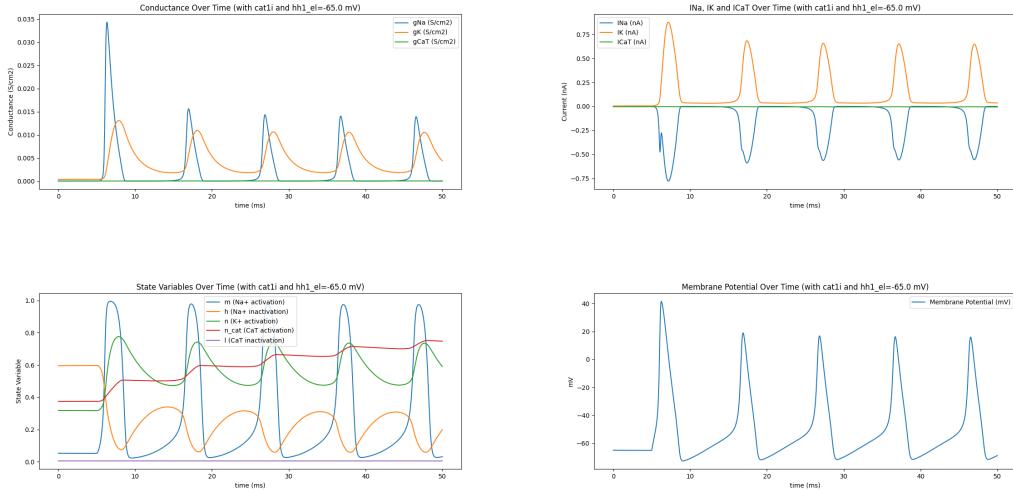


Figure 14: Conductance, current, state variable and membrane voltage with cat1i, -65mV, current injection

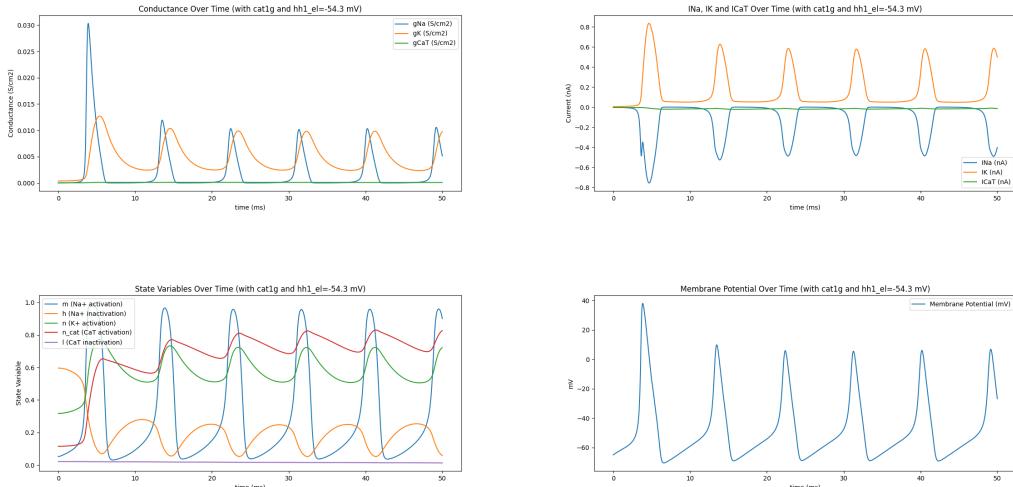


Figure 15: Conductance, current, state variable and membrane voltage with cat1g, -54.3mV, current injection

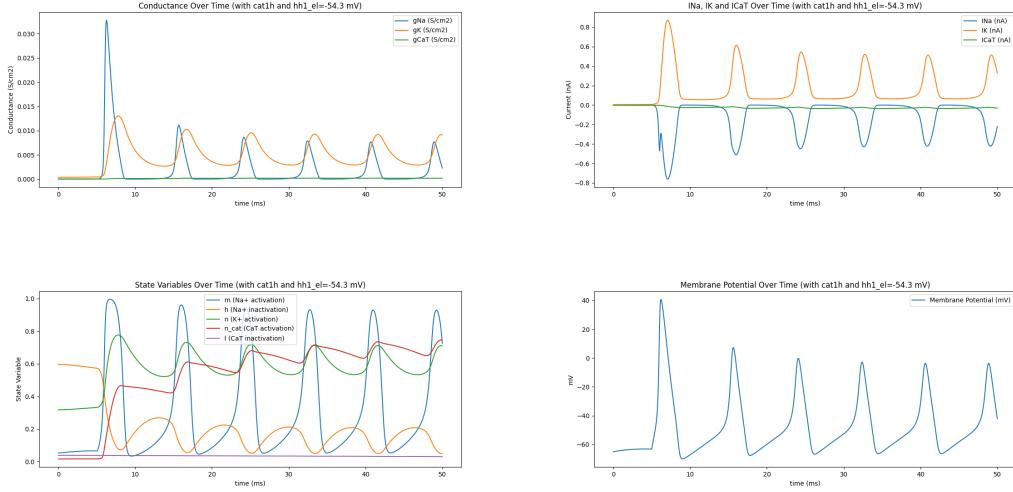


Figure 16: Conductance, current, state variable and membrane voltage with cat1h, -54.3mV, current injection

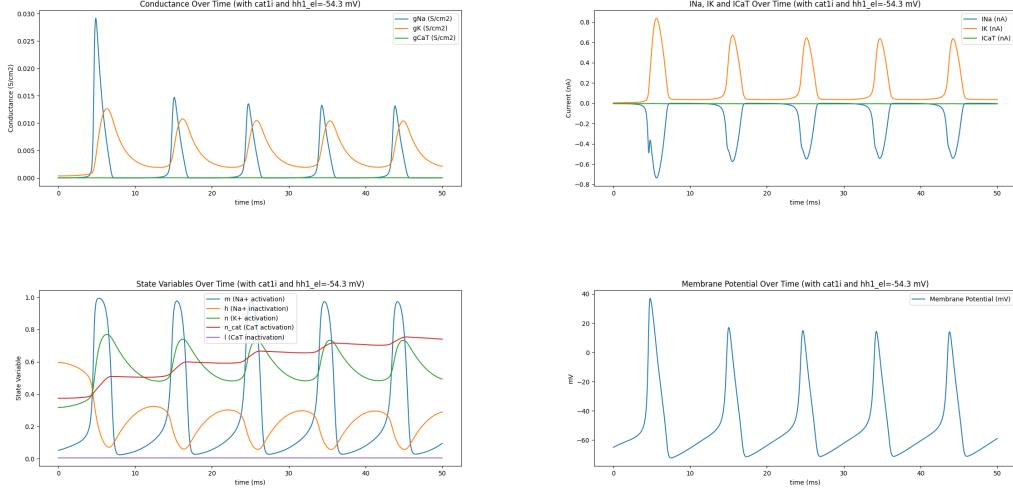


Figure 17: Conductance, current, state variable and membrane voltage with cat1i, -54.3mV, current injection

From the numerical simulation above, all three CaT channels have relatively small contribution to membrane passing current compared with sodium channel and potassium channel. This corresponds to the value of g_{bar} ($g_{\text{cabar}}=0.008(\text{mho}/\text{cm}^2)$, $g_{\text{nabar}}=0.12(\text{mho}/\text{cm}^2)$, $g_{\text{kbar}} = 0.036(\text{mho}/\text{cm}^2)$), as well as the continuously low l of CaT. Under constant current input which can trigger periodical spike, the state variable n goes in a rising trend.

Furthermore, corresponding to the parameter table, we get the observation that n_{cat1i} has a more gradual curve and less sensitivity to voltage resulting in a higher action potential and longer spike period, which testifies the prediction above. Similarly, the n curve is steeper in $cat1h$ and the steepest in $cat1g$.

Here, the $hh1_{el}$ doesn't make much difference in the numerical simulation.

4.3.2 Without current injection

For further exploration, we test the neuron under the circumstance without current injection.

The equation for the membrane potential V considering the $hh1$ channel (with Na and K components) and the CaT channel, along with the leak current, is:

$$I = C_m \frac{dV}{dt} - g_{Nabar} m^3 h (V - V_{Na}) - g_{Kbar} n^4 (V - V_K) - g_{CaTbar} n l (V - V_{CaT}) - g_l (V - V_l) \quad (4)$$

The leak current contributes to the resting membrane potential. If V_l is increased, the driving force ($V - V_l$) becomes smaller (assuming V is initially less than V_l), which reduces the leak current. This can lead to a higher resting membrane potential, making the neuron more depolarized at rest. A higher V_l also means the membrane potential is closer to the threshold for action potential generation. This can make the neuron more excitable, as less depolarization is needed to reach the threshold.

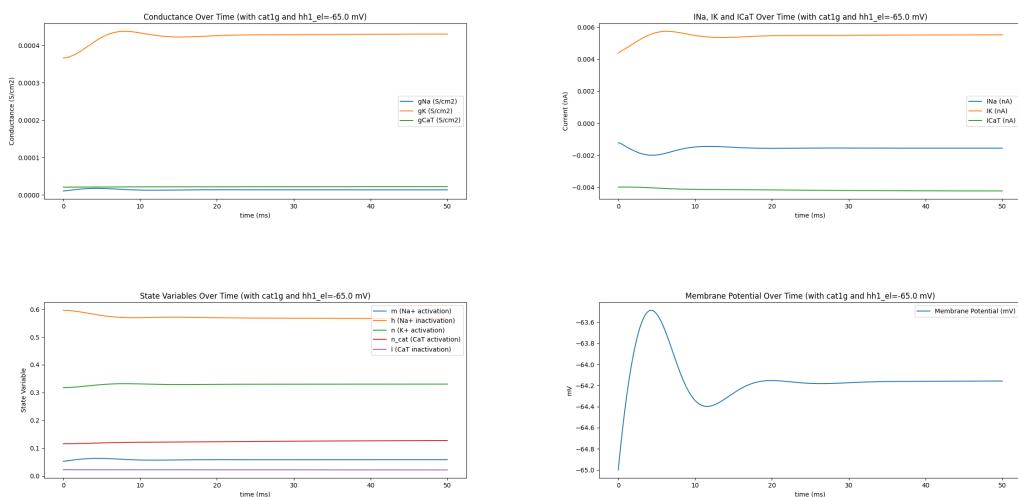


Figure 18: Conductance, current, state variable and membrane voltage with cat1g, -65mV, no current injection

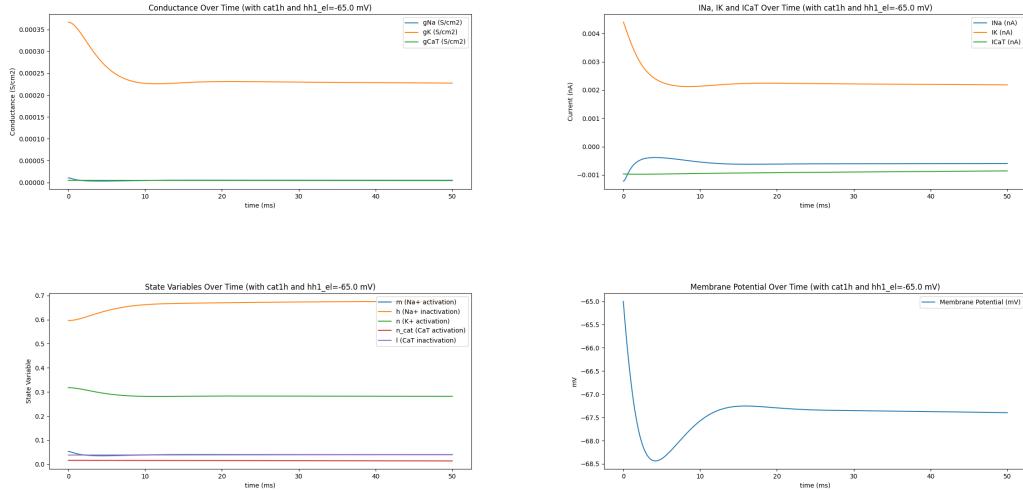


Figure 19: Conductance, current, state variable and membrane voltage with cat1h, -65mV, no current injection

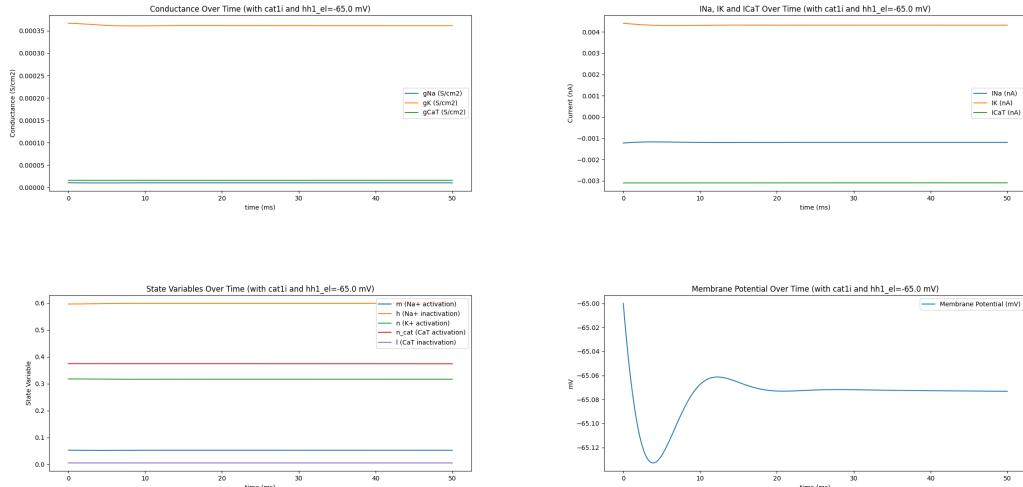


Figure 20: Conductance, current, state variable and membrane voltage with cat1i, -65mV, no current injection

When $hh1_{el} = -65mV$, all three neurons fail to generate spikes. Ideally, the stable membrane potential can be calculated from the v_{half} of every state variable.

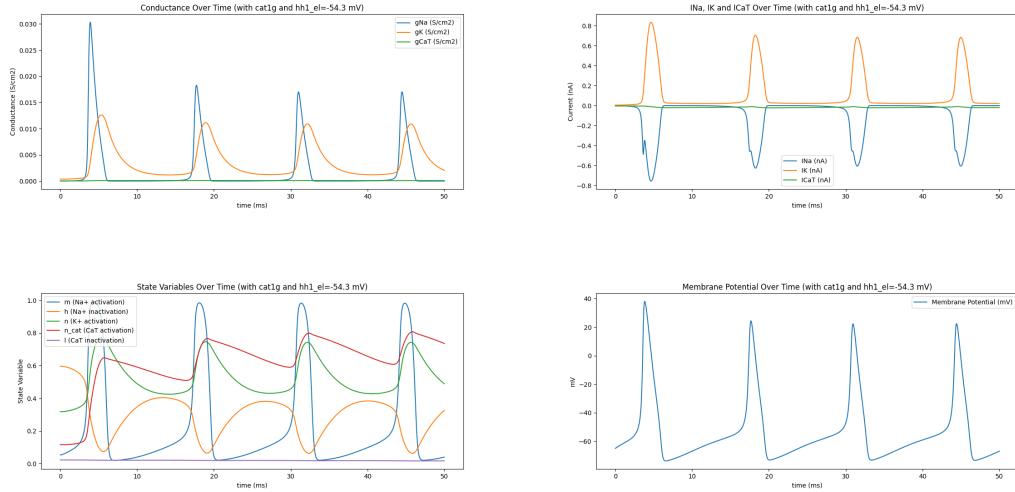


Figure 21: Conductance, current, state variable and membrane voltage with cat1g, -54.3mV, no current injection

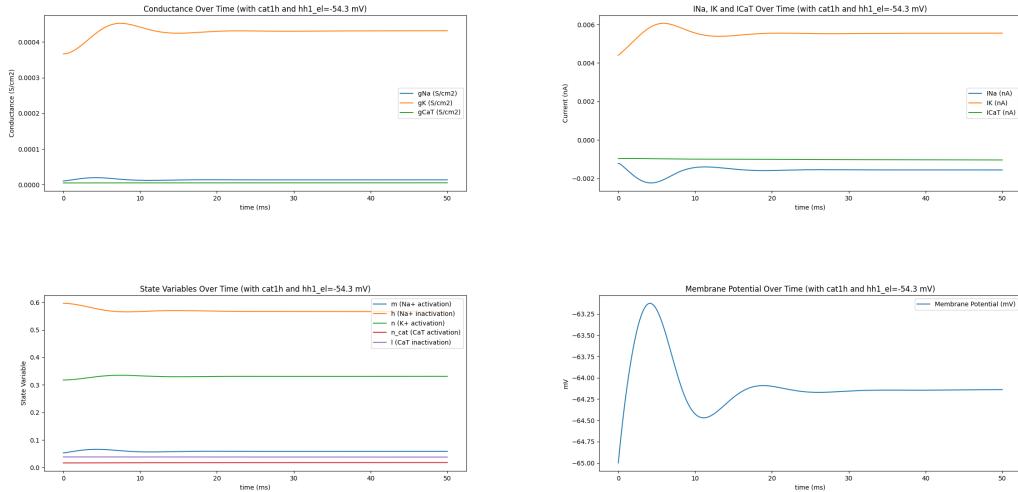


Figure 22: Conductance, current, state variable and membrane voltage with cat1h, -54.3mV, no current injection

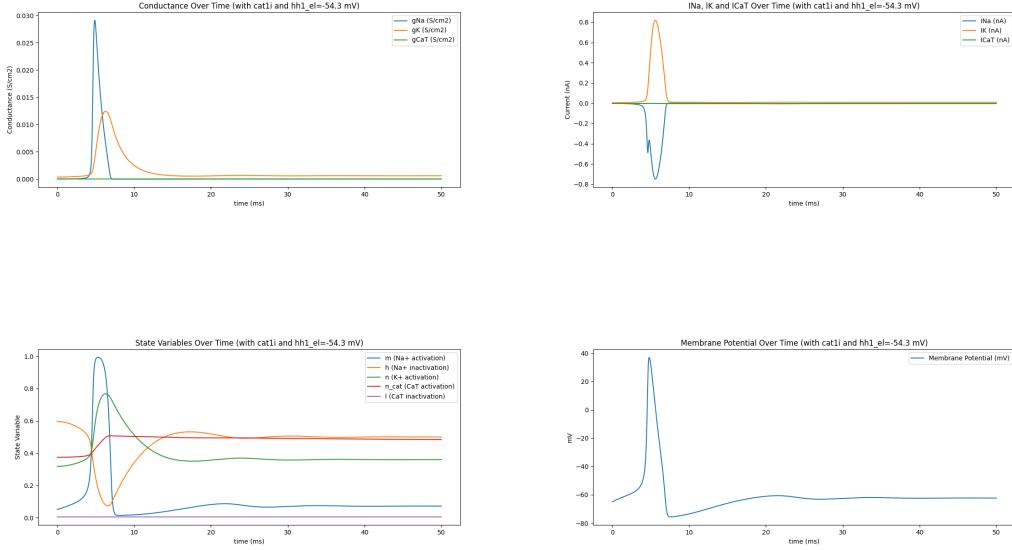


Figure 24: Conductance, current, state variable and membrane voltage with cat1i, -54.3mV , no current injection

When $hh1_{el}$ is set to be -54.3eV , the neuron with cat1g succeeded in generating periodical spikes, the one with cat1i generates a spontaneous spike, while that with cat1h fails to get excited.

To be specific, cat1g and cat1i have v_{half} below the leaking threshold voltage. This can explain the result that they successfully generated spike(s). However, with a relatively lower v_{half} , its membrane potential reaches a lower voltage after the neuron is recovered from the action potential, making it harder to spike again.

4.4 Finding Interpretation:(In)Activation of Ion Channels

We take the model with three CaT channels inserted as example.(since the difference is the most significant) Other models are similar to it.

While there is no notable difference in m , h and n act unusually with CaT channels. After the first time of h decrease, h does not increase to the level as high as the original. Similarly, after the first time of n increase, n only drops to a relatively high level. In other words, sodium channel has a lower probability to stay in open state, the state variable n stays higher than its value in hh1. Membrane potential changes more quickly at a cost of lower voltage peak.

This can explain why we see shorter period length and less maximum in CaT model.

4.5 Spontaneously Spike at -54.3mV

As calcium spikes provide the driving force for a long burst of rapidly emitted sodium spikes and T-type calcium channels can be activated at a low voltage, the -54.3 mV might serve as an initial driving force for calcium channels to activate, and then Na channel as well as K channel are activated, ultimately generate spontaneously spikes.

4.6 Exploration: Minimum Voltage of Spontaneous Spike

According to the observations above, we find that model with three CaT channels will spontaneously spike at -54.3 mV. Thus, we try to figure out the min-max voltage needed for spontaneous spike.

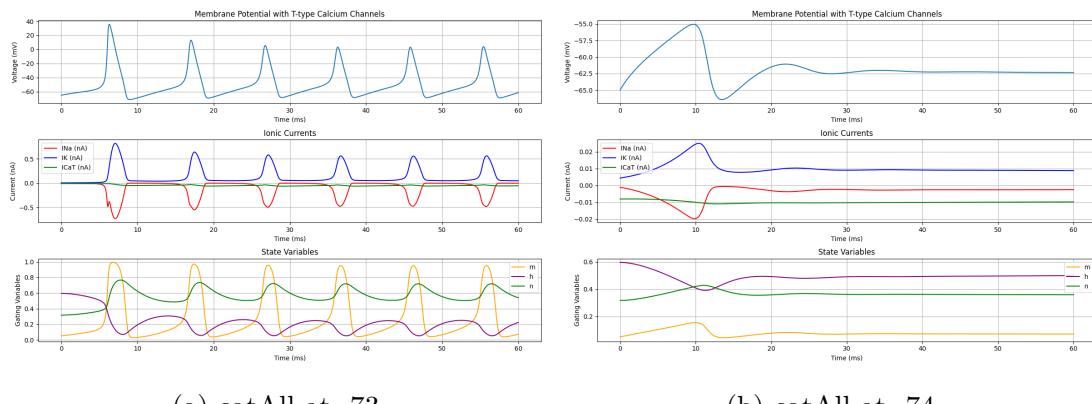


Figure 25: Minimum Voltage of Spontaneous Spike

We figure out the min-max voltage is between -74 mV and -73 mV. At -73 mV, the soma can continuously spike without current injection. Also, voltage shape and ion channel behavior are quite the same with voltage ranging from -73 to -54.3 mV (We haven't try voltage above, but we assume that will be similar) Yet we did try voltage below -73, we find that as voltage get lower, the curve becomes flatter.

Our hypothesis is CaT channels help soma to spike at a low voltage, and the specific impact can be influenced by voltage. After a certain spot(-73mV in our experiment), the impact will not significantly change, while before the spot, CaT will assist soma to gradually form a spike.

4.7 Experiment on neurons with two CaT channels and more

When a constant current is injected, the phenomenon doesn't change much compared with those with only one CaT channel, since the state of the whole neuron is mostly determined by the current injection. When no current is injected, only the neuron with cat1i and cat1g succeeds in generating spike under -65 mV, while all generates spike under -54.3 mV. For relevant figures, see Extended Data.

Finally we insert all three channels into the same neuron. the maximum of voltage spikes after the first two significantly decreases to below zero (about -16mV), and state variable m no longer increase to almost 1, resulting in decreased sodium conductance. Surprisingly, when there are no current injection, the peak of membrane potential spike can reach above zero.

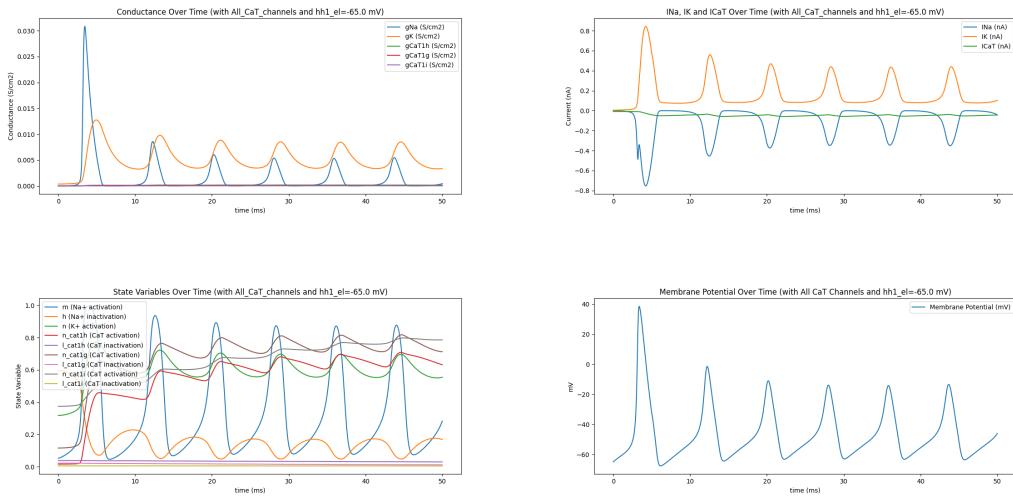


Figure 26: Conductance, current, state variable and membrane voltage with cat1h, -65mV, With current injection

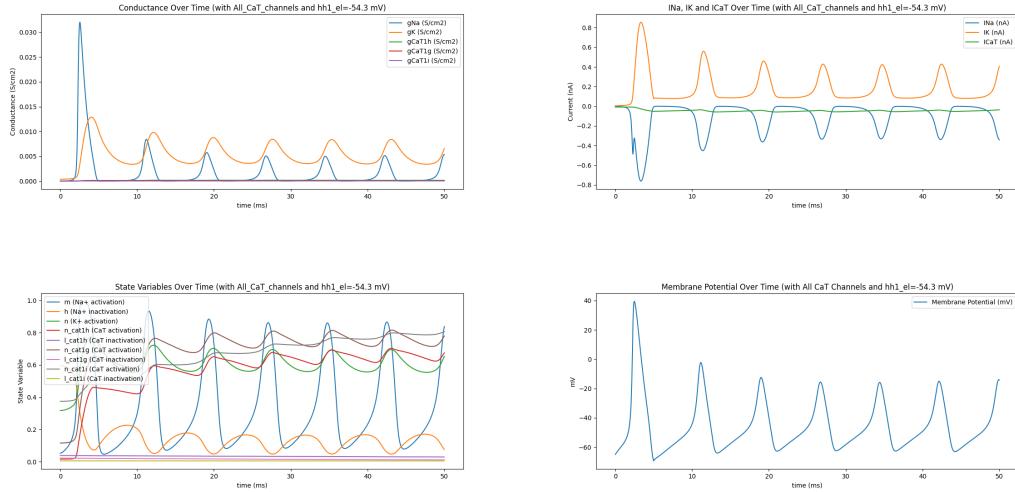


Figure 27: Conductance, current, state variable and membrane voltage with all CaT channels, -54.3mV, with current injection

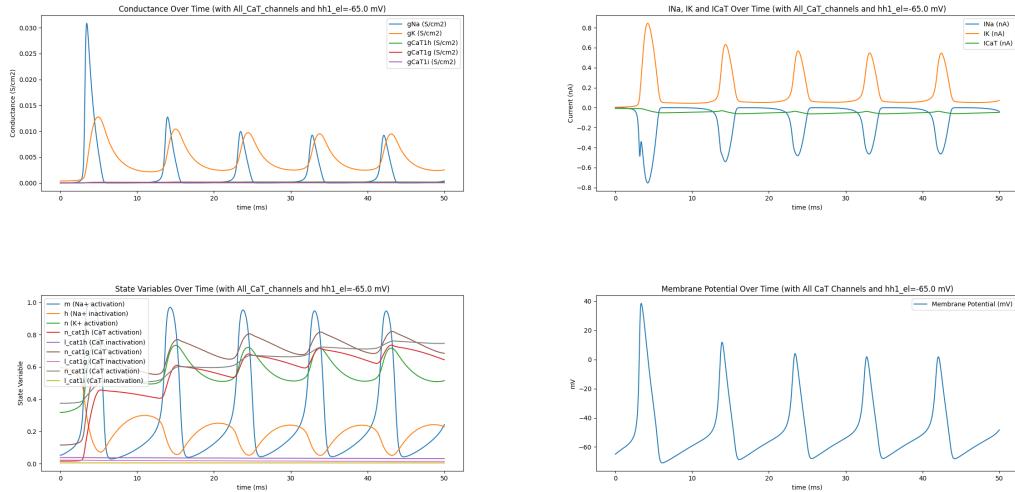


Figure 28: Conductance, current, state variable and membrane voltage with cat1h, -65mV, no current injection

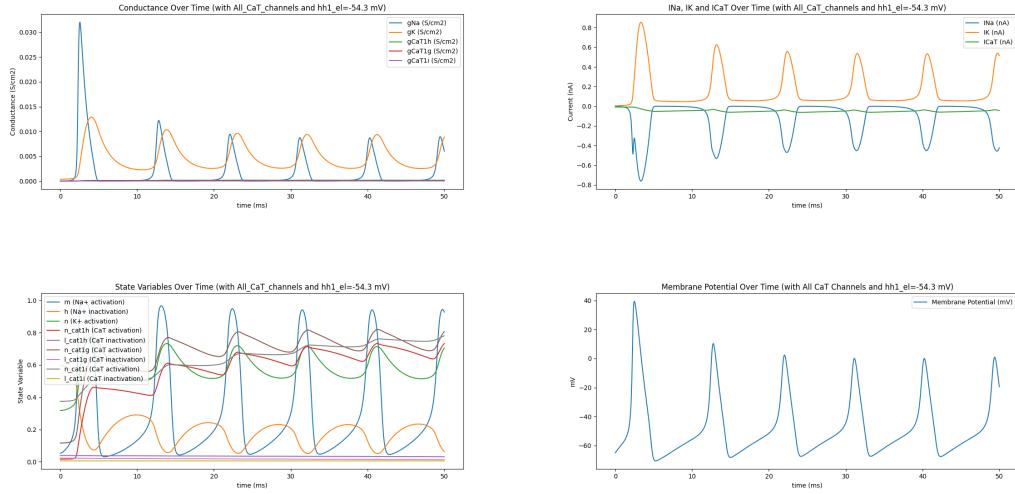


Figure 29: Conductance, current, state variable and membrane voltage with all CaT channels, -54.3mV, no current injection

5 Fast Spiking

5.1 Give a stronger current injection

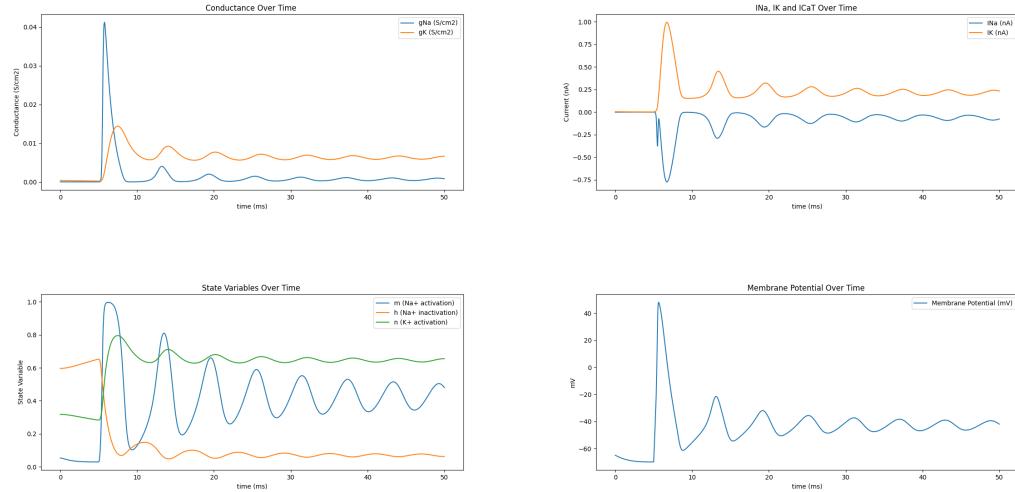


Figure 30: Conductance, current, state variable and membrane voltage with $I=0.5$, $g_{Na}=0.12$

When $I = 0.5$, the oscillation amplitude of membrane voltage gradually diminishes. To enable the model to generate fast spikes, we increase $gbar$ from 0.12 to 0.50, which promotes the sodium current influx, enabling the membrane potential to pass its threshold, thus generating fast spikes.

5.2 enable the model to generate fast spikes

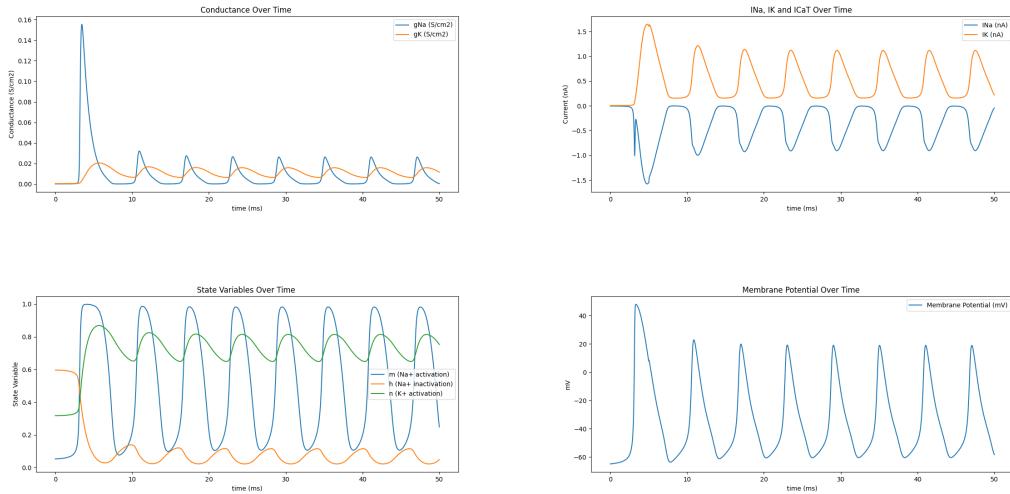


Figure 31: Conductance, current, state variable and membrane voltage with $I=0.5$, $g_{Na}=0.5$

Further Analysis

Why sodium channel opens faster than potassium channel

The Hodgkin-Huxley model simulates the ion channel dynamics through differential equations that define the rate constants (α and β) and the time constants (τ) of the gating variables. The activation and inactivation gates of Na^+ and K^+ channels differ significantly in their temporal dynamics, which explains the delayed opening of K^+ channels compared to Na^+ channels.

Rate Constants and Time Constants

The time constant τ and the steady-state value (∞) for each gating variable are defined by the equations:

$$\begin{aligned}\tau_x &= \frac{1}{\alpha_x + \beta_x}, \\ x_\infty &= \frac{\alpha_x}{\alpha_x + \beta_x},\end{aligned}$$

where x represents the gating variables m , h , or n , each associated with different ion channels. α_x and β_x are the rate constants corresponding to the opening and closing of the channels, respectively.

Na⁺ Channels:

- Activation gate (m):

$$\alpha_m = \frac{0.1(V + 25)}{e^{\frac{V+25}{10}} - 1}, \quad \beta_m = 4e^{-\frac{V}{18}}$$

- Inactivation gate (h):

$$\alpha_h = 0.07e^{-\frac{V+30}{20}}, \quad \beta_h = \frac{1}{e^{\frac{V+30}{10}} + 1}$$

K⁺ Channels:

- Activation gate (n):

$$\alpha_n = \frac{0.01(V + 10)}{e^{\frac{V+10}{10}} - 1}, \quad \beta_n = 0.125e^{-\frac{V}{80}}$$

Comparison of Dynamics

1. **Rate Constant Dynamics:** The rate constants α_m and β_m for the Na⁺ channel change rapidly as the membrane potential approaches the action potential threshold, causing the m-variable to quickly approach its maximum value, thus opening the channels swiftly. In contrast, the α_n and β_n for the K⁺ channel change more slowly, causing a slower increase in the n-variable.

2. **Time Constants:** The time constants τ_m and τ_h for Na⁺ channels are typically smaller than τ_n for K⁺ channels. This implies that the state variables for Na⁺ channels reach

their steady-state values faster than those for K^+ channels, contributing to the delay in K^+ channel opening.

3. State Variabel Dynamics: During the typical initiation of an action potential, the m-variable of Na^+ channels rapidly approaches 1, allowing for a quick influx of Na^+ ions, while the n-variable for K^+ channels increases more gradually, delaying their opening until after the Na^+ channels have activated.

Conclusion

When the state variables change, they affect the ion channel conductance by controlling the open/close probability of the channel, which in turn increase/decrease the ion flux, altering the membrane potential, and eventually modifying the state variable (and conductance). In short, the relationship between state variables and ion channel conductance forms a feedback loop that is fundamental to the neural excitability and the generation of action potentials. Generally, a higher membrane peak requires a longer period to get prepared.

Contribution

Aiyu Wang: the simulation of neurons with 2 kinds of CaT channels (see Extended data).

Yueying Luo: coding for Ex1-3, Ex5; simulation for neurons with 1 kind of CaT channels and Ex 5, report for sections 4.4 and 4.5.

Jiarui Sun: coding for Ex1-5, the rest of the report and simulation.

Appendix

Modeling K⁺ and Na⁺ Channels

K⁺ Channel

$$\begin{aligned}
 g_K &= g_{K_{\max}} n^4 \\
 \frac{dn}{dt} &= \alpha_n(1 - n) - \beta_n n \\
 n &= n_\infty - (n_\infty - n_0)e^{-\frac{t}{\tau_n}} \\
 n_\infty &= \frac{\alpha_n}{\alpha_n + \beta_n} \\
 \tau_n &= \frac{1}{\alpha_n + \beta_n} \\
 \alpha_n &= \frac{0.01(V + 10)}{e^{\frac{V+10}{10}} - 1} \\
 \beta_n &= 0.125e^{\frac{V}{80}}
 \end{aligned}$$

Na⁺ Channel

$$\begin{aligned}
g_{Na} &= g_{Na_{max}} m^3 h \\
\frac{dm}{dt} &= \alpha_m(1 - m) - \beta_m m \\
\frac{dh}{dt} &= \alpha_h(1 - h) - \beta_h h \\
m &= m_\infty - (m_\infty - m_0)e^{-\frac{t}{\tau_m}} \\
h &= h_\infty - (h_\infty - h_0)e^{-\frac{t}{\tau_h}} \\
m_\infty &= \frac{\alpha_m}{\alpha_m + \beta_m} \\
\tau_m &= \frac{1}{\alpha_m + \beta_m} \\
\alpha_m &= \frac{0.1(V + 25)}{e^{\frac{V+25}{10}} - 1} \\
\beta_m &= 4e^{-\frac{V}{18}} \\
h_\infty &= \frac{\alpha_h}{\alpha_h + \beta_h} \\
\tau_h &= \frac{1}{\alpha_h + \beta_h} \\
\alpha_h &= 0.07e^{-\frac{V+30}{20}} \\
\beta_h &= \frac{1}{e^{\frac{V+30}{10}} + 1}
\end{aligned}$$

Extended data

Two CaT channels inserted for Ex4

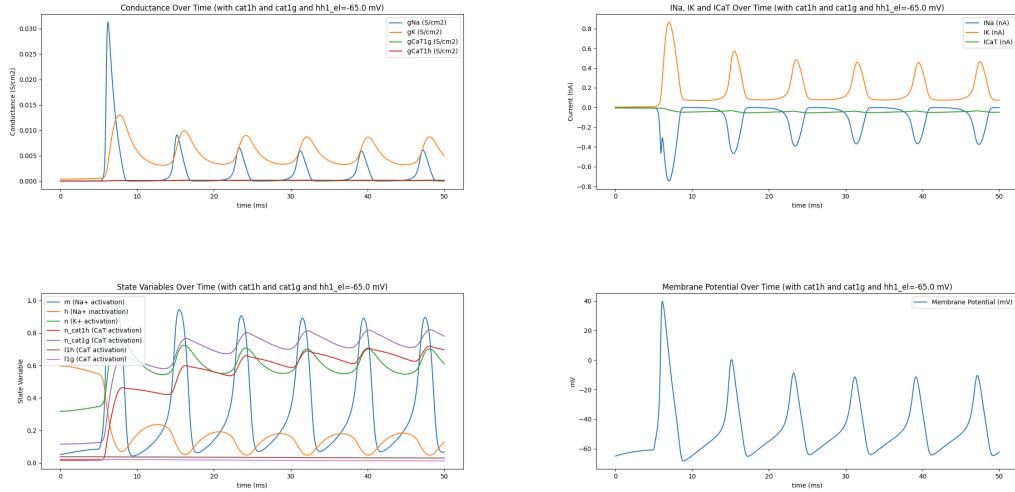


Figure 32: Conductance, current, state variable and membrane voltage with cat1g and cat1h, -65mV, current injection

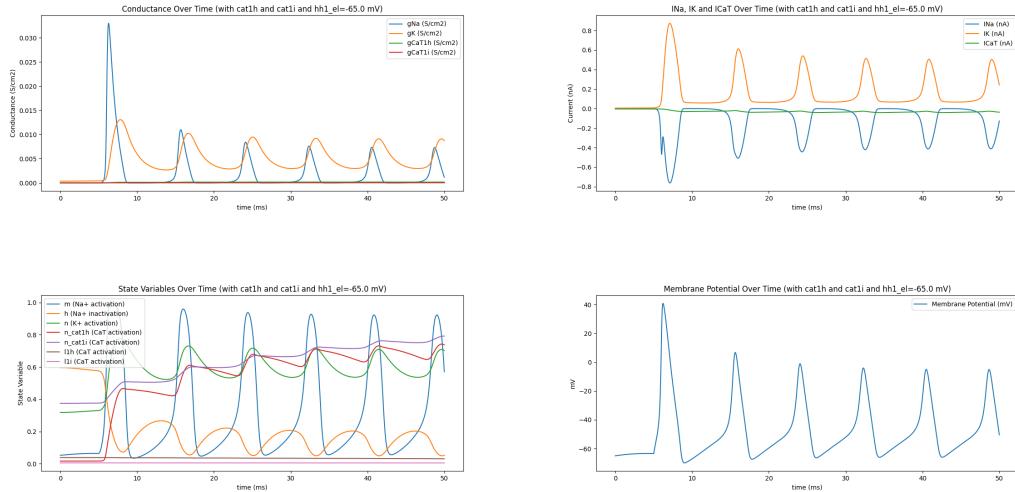


Figure 33: Conductance, current, state variable and membrane voltage with cat1i and cat1h, -65mV, current injection

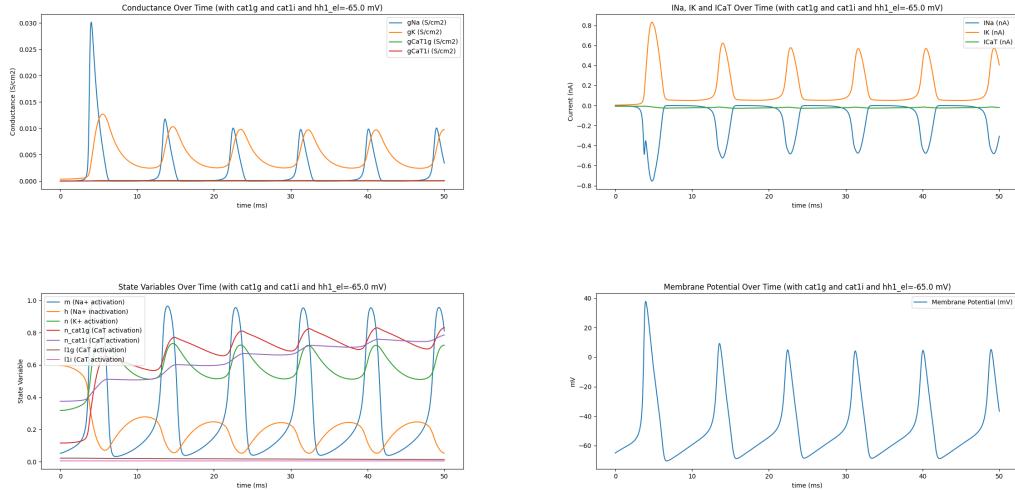


Figure 34: Conductance, current, state variable and membrane voltage with cat1i and cat1g, -65mV, current injection

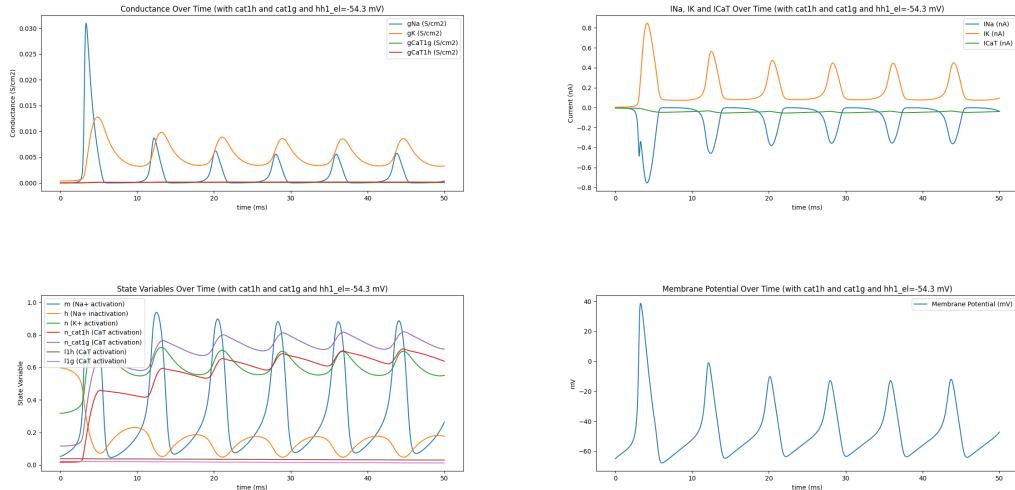


Figure 35: Conductance, current, state variable and membrane voltage with cat1g and cat1h, -54.3mV, current injection

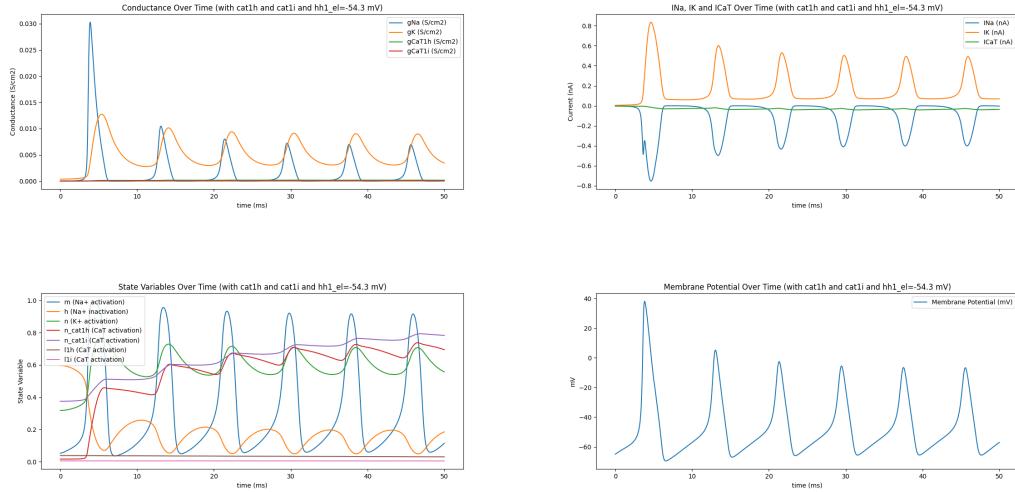


Figure 36: Conductance, current, state variable and membrane voltage with cat1i and cat1h, -54.3mV, current injection

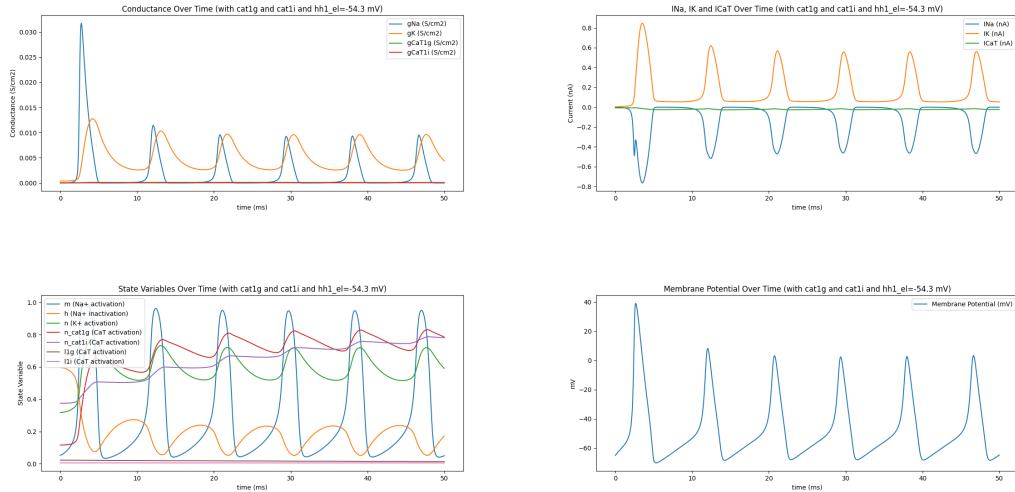


Figure 37: Conductance, current, state variable and membrane voltage with cat1i and cat1g, -54.3mV, current injection

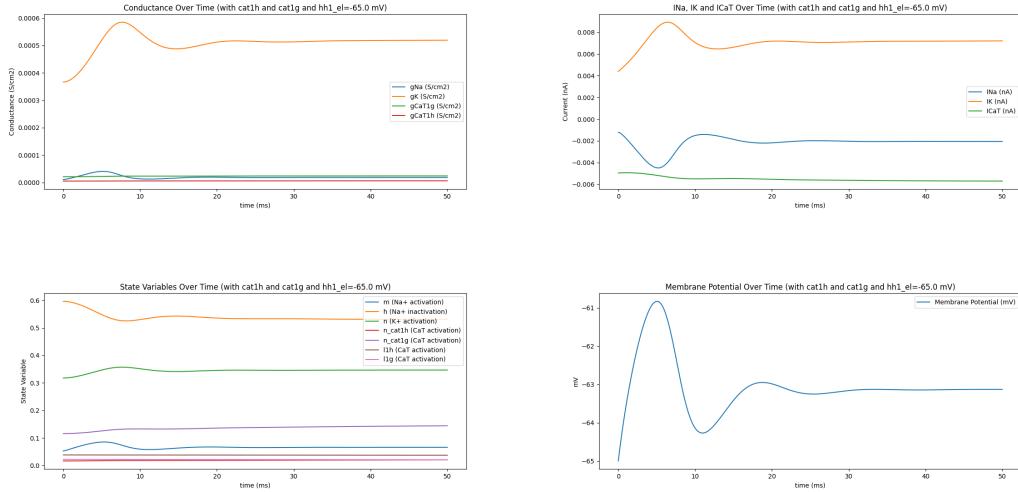


Figure 38: Conductance, current, state variable and membrane voltage with cat1h and cat1g, -65mV, no current injection

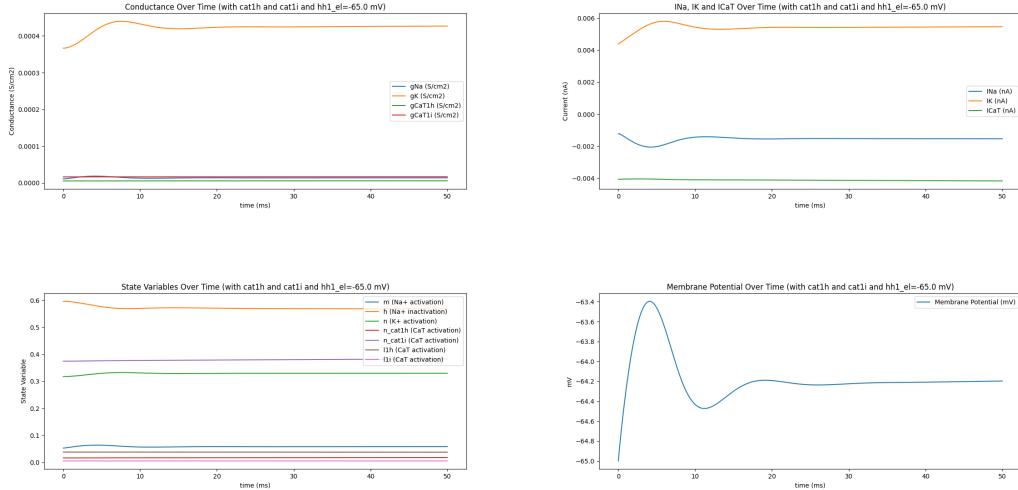


Figure 39: Conductance, current, state variable and membrane voltage with cat1h and cat1i, -65mV, no current injection

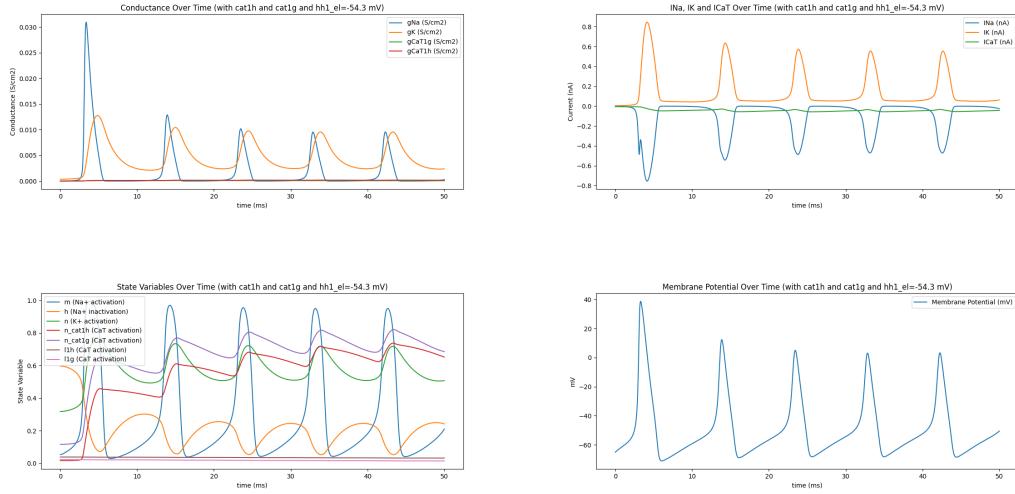


Figure 41: Conductance, current, state variable and membrane voltage with cat1h and cat1g, -54.3mV, no current injection

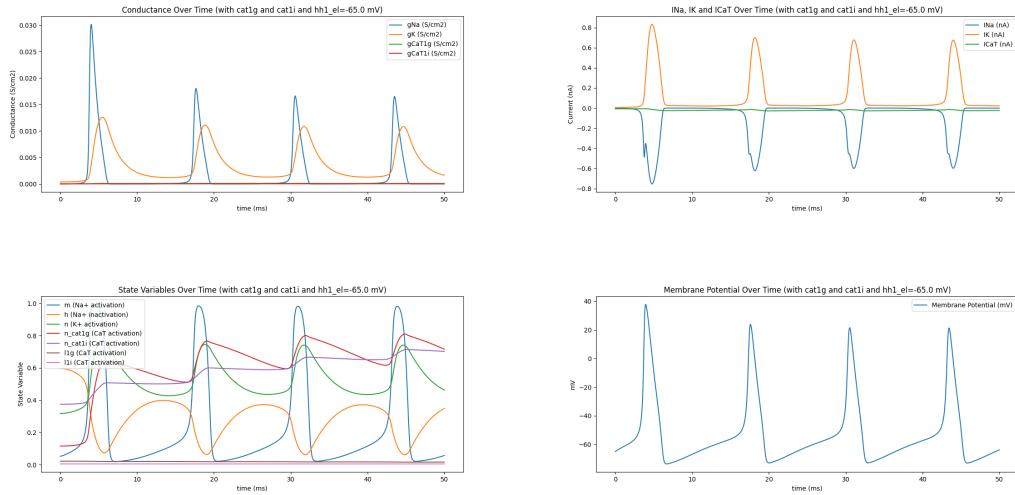


Figure 40: Conductance, current, state variable and membrane voltage with cat1i and cat1g, -65mV, no current injection

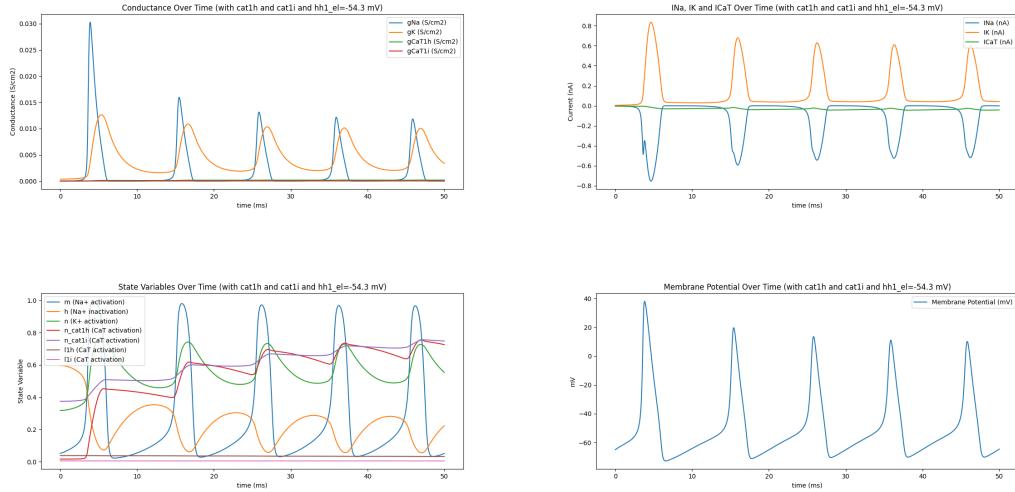


Figure 42: Conductance, current, state variable and membrane voltage with cat1i and cat1h, -54.3mV, no current injection

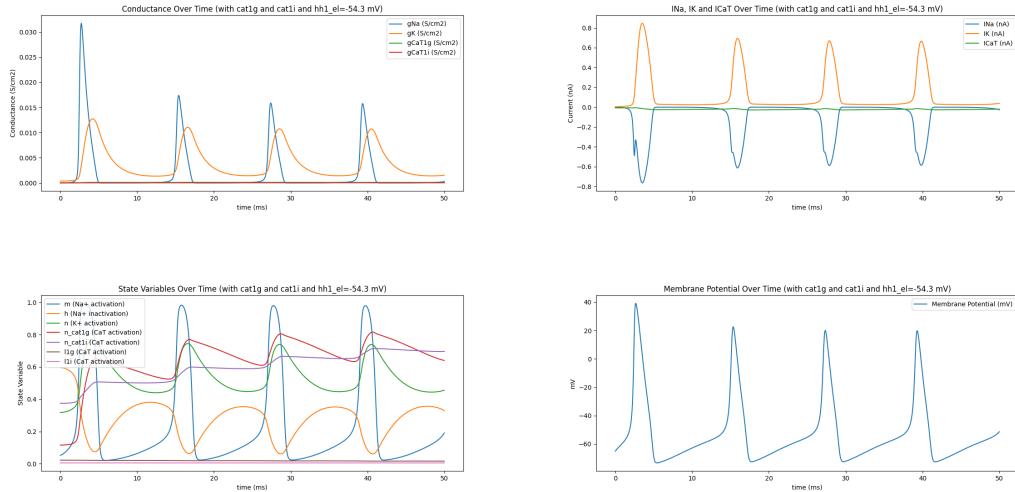


Figure 43: Conductance, current, state variable and membrane voltage with cat1i and cat1g, -54.3mV, no current injection