

## ABSTRACT

## 1. INTRODUCTION

Action potential occurs when the membrane potential of a specific axon location rapidly rises and falls. The potential is the voltage across the membrane cells caused by cell depolarization. Depolarisation propagate to adjacent locations to give rise to similarly depolarise.

During the generalization of the action potential,  $Na^+$  and  $K^+$  gates as the response to the signal from other neuron.  $Na^+$  channels opens and closes at the start of the action potential with the membrane reaching its threshold potential while  $K^+$  channels opens, moving into the axon and causes the deepolarization. There also exists the repolarization of  $K^+$ , opening the  $K^+$  channels and enabling the ion to move out of the axon[1]. Such repolarization creates a change in polarity between the outside and inside side of the cell. Meanwhile, the potassium currents exceeds the sodium current resetting the voltage back to its normal resting value, typically -70 mV. However, if the voltage increases and past the 15mV higher than the resting value, the sodium current dominates.[2]

Each excitable cell is treated as The electrical element by the typical Hodgkin-Huxley model. Written as  $C_m$ , The lipid bilayer is represented as a capacitance,  $g_l$  (electrical conductances) and  $g_n$  (where  $n$  is the specific ion channel), represented by electrical conductances, voltage-gated ion channels depends on both voltage and time.

The equation involved:  $I_c = C_m * d(V_m)/dt$  stands for the current flowing through the lipid bilayer and  $I_i = g_i * ((V_m) - V_i)$ , where  $V_i$  is the reversal potential of the  $i$ th ion channel. Thus, for a cell with sodium and potassium channels, the total current through the membrane is given  $I = C_m d(V_m)/dt + g_K * (V_m) - V_K) + g_{Na} * (V_m) - V_{Na}) + g_l * (V_m - V_l)$  where  $I$  is the total membrane current per unit area,  $C_m$  is the membrane capacitance per unit area,  $g_K$  and  $g_{Na}$  are the potassium and sodium conductances per unit area, respectively.  $(V_K)$  and  $(V_{Na})$  are the potassium and sodium reversal potentials, respectively.  $(V_m)$ ,  $(g_{Na})$  and  $(g_K)$  are

time dependent elements where the last two depends on voltage as well.

Furthermore, one process cannot be ignored is the myelination. The myelination occurs mainly in vertebrates, The correlated efficient transduction of electrical signals is fast and efficient. During the myelination, the membrane capacitance is reduced by the myelin starch and increased by the membrane resistance in the inter-node intervals.[3] Specifically, in the myelinated segments of the axon, action potentials cannot propagate through the membrane. The fast conduction speed and energy efficiency are the two important advantages. For instances, due to the confined ionic currents (to the nodes of Ranvier), far fewer ions 'leak' across the membrane, saving metabolic energy. Such kind of the significant selective advantage, since the human nervous system uses approximately 20 percent of the body metabolic energy. Parental education positively provides a partial protection against the impact of SED and the early socio-economic disadvantage, a vulnerability factor for the risk factor for aberrant myelin growth during a critical developmental period. In order to characterize voltage-gated channels, voltage clamp data are fitted. For such voltage-clamp. For a derivation of the Hodgkin-Huxley equations under, see[4]:  $m(t) = m_0 - [(m_0 - m_{inf}) * (1 - exp(-t/m))]$   $h(t) = h_0 - [(h_0 - h_{inf}) * (1 - exp(-t/h))]$   $n(t) = n_0 - [(n_0 - n_{inf}) * (1 - exp(-t/n))]$  Thus, for every value of membrane potential  $V_m$  the sodium and potassium current can be described as:  $I_{Na}(t) = g_{Na} m(V_m)^3 * h(V_m) * (V_{Na} - E_{Na})$   $I_{K(t)} = -g_K * (n_{V_m}^4 * (V_m - E_K)$  The classical Hodgkin-Huxley(HH) neglects the time-dependence of ion concentrations in spiking dynamics. The dynamics is therefore limited to the membrane capacitance and resistance determinant model. In the reality, the concentration, pumps and buffers are also time dependent. Fluxes across the neuronal membrane change either intra-extracellularly. In the real biology study, the bifurcation structure and thus the phase space structure is required to be combined with the activation and inhibition of the new excitability emerging in ion homeostasis. To be more specific, the dynamics is determined by the cell volume-to-surface-area ratio the slower backward buffering buffering, especially related to the pathological conditions

in epileptiform burst activity, and spreading depression in stroke and etc.

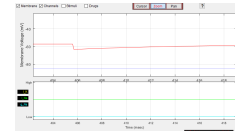
Due to the threshold, the action potential can be induced. If depolarization reaches -55 mV (but sensitive to the voltage at -50 mV), then the action potential continues and runs all the way to +30 mV, at which  $K^{[+]}$  ion causes repolarization. For  $Na^{+}$ , the activation gate opens when the membrane potential reaches -55 mV while the inactivation gate closes after a specific period of time, on the scale of a fraction of a millisecond.

Furthermore, one process cannot be ignored is the myelination. The action potential travels down the axon as voltage-gated ion channels are opened by the spreading depolarization. In unmyelinated axons, this happens continuously with voltage-gated channels throughout the membrane. The myelination occurs mainly in vertebrates. The fast and efficient transduction of electrical signals are related. During the myelination, the membrane capacitance is reduced by the myelin sheath and increased by the membrane resistance in the inter-node intervals.[3] Specifically, in the myelinated segments of the axon, action potentials cannot propagate through the membrane. The fast conduction speed and energy efficiency are the two important advantages. For instance, due to the confined ionic currents (to the nodes of Ranvier), far fewer ions 'leak' across the membrane, saving metabolic energy. Such kind of significant selective advantage, since the human nervous system uses approximately 20 percent of the body metabolic energy. Parental education positively provides a partial protection against the impact of SED and the early socio-economic disadvantage, a vulnerability factor and the risk factor for aberrant myelin growth during a critical developmental period.

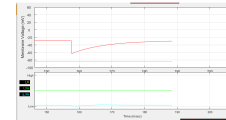
Physiologically, action potential frequencies of up to 200-300 per second (Hz) are routinely observed. However, the maximum frequency is ultimately limited by the absolute refractory period. A limit to the highest frequency at which neurons can respond to strong stimuli exists. The absolute refractory period is 1 ms. That is, the absolute refractory period limits the maximum number of action potentials generated per unit time by the axon. The strength of the stimulus must be very high in order to ensure that the duration of the action potential is as short as the duration of the absolute refractory period. To overcome the relative refractory period, stimulus is required to be stronger than normal.

## 2. ACTION POTENTIALS

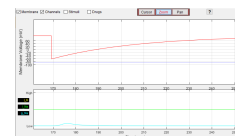
Because of the fact that the threshold to induce the action potential is around -50 mV, here all three groups of the signal activated at around that value and thus the default value setting gives rise to the action more easily for K and cl ions which are -57.2 mV, -72.1 mV and for the Na ion, the half



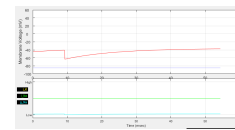
**Fig. 1.** cl action potential



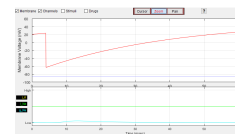
**Fig. 2.** cl-h action potential



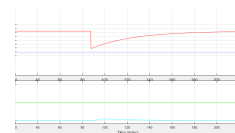
**Fig. 3.** k action potential



**Fig. 4.** k-h action potential

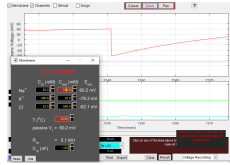


**Fig. 5.** Na action potential

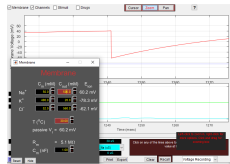


**Fig. 6.** Na-h action potential

of the default induces the activation easier, which is around 26 mV. In addition, to gain the highest frequency, the Na ion's temperature is set to be 30 degree and the concentration inside the cell is set to be kept as 50 mM with the one outside the cell set as 500 mM. The potential is thus 60.2 mV. This

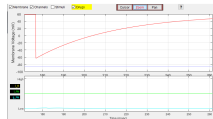


**Fig. 7.** Na-highest action potential

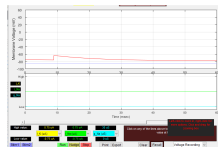
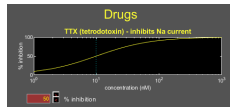


**Fig. 8.** Na-highest action potential

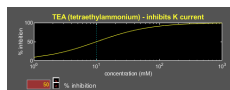
set gives out the result around :  $f = 1000 \text{ circles} / 4 \text{ ms} = 250 \text{ Hz}$ .



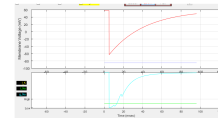
**Fig. 9.** drug1



**Fig. 11.** drug2



The drug 1 inhibits the Na current significantly. After setting the inhibition as 50 percent, the Na activation cannot



**Fig. 13.** drug3



be observed as activated. For the drug2, the TEA inhibits the K current. The drug set as 50 percent inhibition totally inhibits the K current as well. For the last drug, the ponase inhibitor destroys the inactivation gate of the Na current and makes the Na current no longer inactivated. For all those three drugs, the only affected quantity is the current either in activation or inactivation phase effectively. However, the voltage is all kept unchanged.

### 3. SUMMARY

In summary, the project explores the action potential of K, Na, and Cl ion channels. The gating of these channels is opened with stimuli of -50mV and for Na, there is the inactivation gate while no inactivation gate for K and Cl ions. The temperature affects the frequency of the action potential effectively while the concentration does not work significantly. With the drugs applied to the current, the phase of activation and inactivation can be inhibited effectively.

### REFERENCES

- [1] Bullock, Orkand Grinnell 1977. Voltage-Gated Ion Channel
- [2] Stevens, CF (1966). Neurophysiology: A Primer. New York: John Wiley and Sons. LCCN 66015872. OCLC 1175605.
- [3] Zalc B (2006). "The acquisition of myelin: a success story". Novartis Found. Symp. Novartis Foundation Symposia. 276: 1521, discussion 215, 547, 27581. doi:10.1002/9780470032244.ch3. ISBN 978-0-470-03224-4. PMID 16805421.