A BIO-PHYSICALLY INSPIRED SILICON NEURON

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ABSTRACT

The physical principles governing ion flow in biological neurons share some interesting similarities to electron flow through the channels of a MOSFET transistor. Here we describe a circuit which seeks to exploit these similarities to produce a circuit which behaves in a manner similar to biology. Using this approach, we have developed a circuit which is extremely small in size and is capable of generating output similar to biology in shape, magnitude, and time.

The discussion in this paper describes what is believed to be a very new way of looking at the electrical properties of biological neurons. Much work has been done in the field since the early 1950's with the pioneering work of Hodgkin and Huxley. Advances have also been made in the semiconductor field, however, little of our understanding of the physics of semiconductors has carried across to our models of ion flow in biology. Here we show a model of a two channel type ($Na^+$and K^+) neuron circuit which is capable of generating action potentials, and elegantly accomplishes this with just six transistors.

Classically, modelers have used data to empirically derive equations that describe this ion flow. The approach described in this paper, however, does not rely on the implementation of empirically derived equations, but rather seeks to model biological processes by utilizing the extreme similarities between biological and silicon physics. Fig. 1 summarizes the two views.

1. BIOLOGICAL PRIMER ON CHANNELS AND MEMBRANES

In 1952, Alan Hodgkin and Andrew Huxley described the electrical activity of squid axons in a series of papers that eventually won them the Nobel Prize for medicine in 1963 [4]. They showed that two types of channels are essential to generate an action potential (the name given to the voltage spike by which neurons communicate), and they developed an electrical model to describe these channels. This model is shown in Fig. 2c. The membrane of a neuron separates charge, and these charge separation qualities are nicely modeled by the simple capacitor labeled C_{mem}. In neurons, all charge is carried by electrically charged ions. Sodium (Na^+) ions and potassium (K^+)ions are the two particular ions they found to be necessary for action potential generation, therefore we will focus on them. Both ions carry a $+1$ charge.

The power supply values, or reversal potentials of the neuron, are set by the Nernst potential equation. The Nernst potential implies that drift and diffusion currents are present in the biology, and in fact this is true. This phenomenon is also found in a p-n junction diode. When one realizes that this same macro transport phenomenon is found in both biology and semiconductors, the case for a model of this kind becomes much more apparent.

Work was partially funded through an ONR YIP grant.

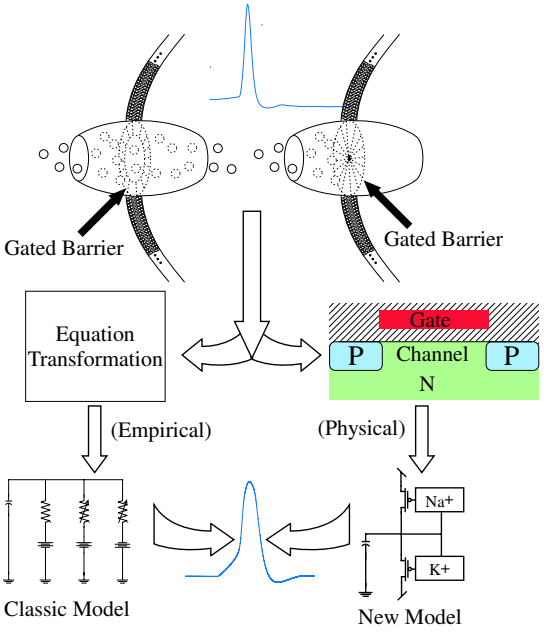

Fig. 1. Figure shows the progression from bio-physics to the corresponding silicon physics. The top shows a typical action potential from a particular snail *(helisoma trivolvis)*. Neuroscientists understand the biological channels, and have described them in some detail. From the data taken from these channels, one might extrapolate equations and empirically determine a model of the system. The other method, which is the method described in this paper, leads one to look at the direct analogy between the physics of biological channels and MOSFET transistors. Both methods can lead to action potential generation, however, the path on the right not only gives more accurate results, but also can be realized directly whereas the path on the left can not.

The sodium (Na^+) channel, one of the two channels necessary to generate an action potential, is voltage gated, meaning that it responds to changes in the voltage across the membrane (V_{mem}). This channel has an activating and an in-activating mechanism causing current to increase and then decrease as time progresses. The time constant for the opening is very fast, while the time constant for the closing is a bit slower. The step response of the Na^+channel is shown if Fig. 2 c and is derived from Hodgkin

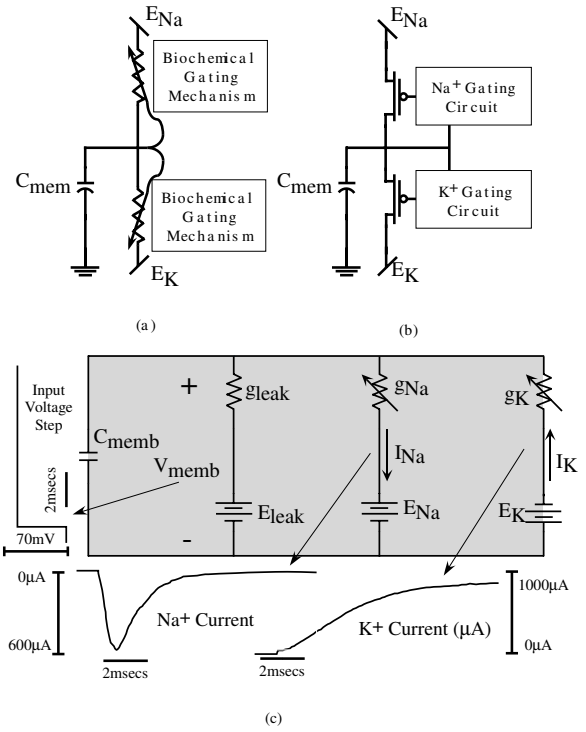

Fig. 2. (**a**) Traditionally, biological channels are modeled with dynamic resistors. Moving from biology to this resistor model linearizes the system. (**b**) Proposed is the idea of modeling channels with MOSFET transistors instead of resistors. This new model relies on the physical similarities between biology and silicon, and therefore preserves the non-linearities. Like the resistor, the transistor does not have the built-in particular dynamics that is required to model this channel. However, unlike the resistor, the MOSFET provides the means control it. (**c**) The original circuit model of neural electrical conductivity as devised by Hodgkin and Huxley. The plot on the extreme left of the figure shows the voltage step that was forced across the membrane. $Na^+$and K^+currents are also shown. Note the speed with which the Na^+channel works as compared to the K^+channel, and that the direction of current flow is opposite.

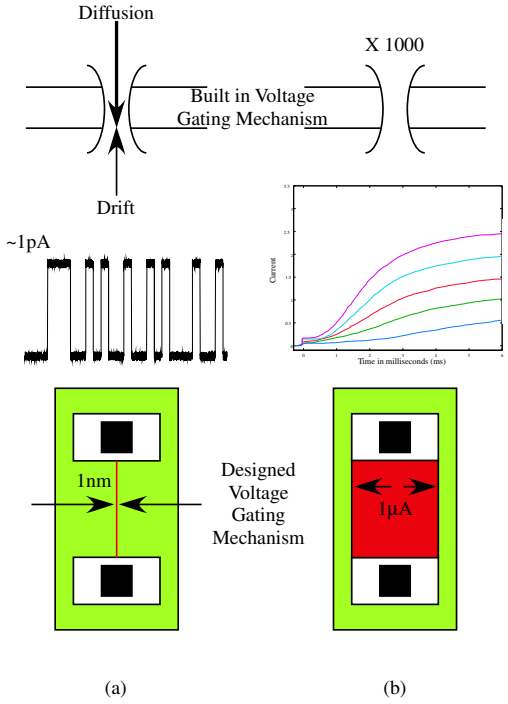

Fig. 3. (**a**) A single biological channel is stochastic in nature, as is a transistor of extremely small width (approx. 1nm in width). (**b**) However, a normal width transistor generates a smooth current. This is similar to the same way that a large population of biological channels generates a smooth current. A transistor of normal size actually models populations of biological channels instead of a single one.

and Huxley's paper [4]. Notice this output exhibits the classic step response of a bandpass filter, although to the author's knowledge, it has never been classically described as such. This gives rise to two separate time constants, which are called τ_m and τ_h for the fast and slow time constants respectively. The 'm' and 'h' were terms and variables used by Hodgkin and Huxley, and so have been maintained for comparison and clarity.

The second channel type, the potassium (K^+) channel, is also voltage gated. However, unlike the Na^+channel, this channel, once opened, will not close until the voltage on the membrane decreases. Again, the step response is shown in Fig. 2c [4]. This has the very characteristic low pass response to a step input. While the time constant is still quite fast, it is clear from comparing the data in Fig. 2 that the time constant, τ_n, for I_K is much slower than either of the time constants found in I_{Na}.

The interaction of these two currents causes the characteristic action potential. The currents are in opposition to each other. The Na^+current responds quickly and charges the membrane fast. The slower K^+current reduces the membrane to rest, but since it is so slow to turn on/off, the K^+channel actually causes a hyperpolarization to occur. Although many other types of currents contribute to the details of the action potential waveform, the two currents being discussed are the minimum needed to actually produce an action potential, and therefore are the only ones discussed.

These channels have classically been described using variable conductances (Fig. 2c), which looks nice and simple. While, admittedly, this model is nice for gaining intuition about the operation of cells, the equations [4] and, therefore the model describing the conductances is very complex. Aside from the complexity issue, use of this empirical method actually linearizes the solution, does not take advantage of the similarities of the physics, and ultimately cannot be directly realized (meaning that no circuit element exists which has the properties described by Hodgkin and Huxley). [1]

2. CIRCUIT OVERVIEW

Biological channels have a non-linear current relationship to the voltage on the membrane, in fact they have an exponential relationship. This relationship simply cannot be accomplished using a resistor.

The circuit model described here replaces the linearized conductances that Hodgkin and Huxley used with nonlinear conductances. To replace these elements with elements which also have an exponential relationship between voltage and current would be ideal. This brings to mind two types of devices: a BJT transistor,

[1]For a discussion of $Na^+$and K^+equations which are based on the physics, see [1].

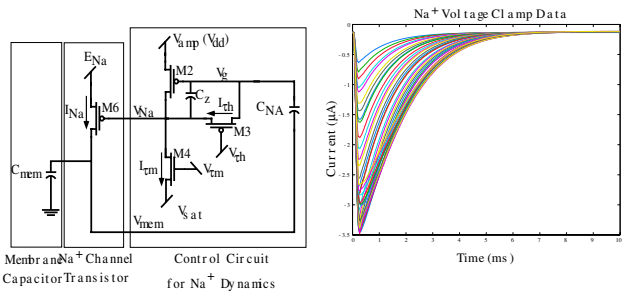

Fig. 4. The Na^+circuit and experimental data from it. One can easily see the channel transistor and membrane capacitor. Connected to the channel transistor is the circuit which controls its dynamics. It is a bandpass filter with a gain term (set by the relationship between C_{Na} and C_z). The experimental data from the Na^+voltage clamp (step response) experiments performed in lab are also shown here. These responses are indicative of the bandpass filter that was being implemented. Although, not shown here, when the input step voltage exceeds E_{Na}, the current starts flowing in the opposite direction, as one would expect.

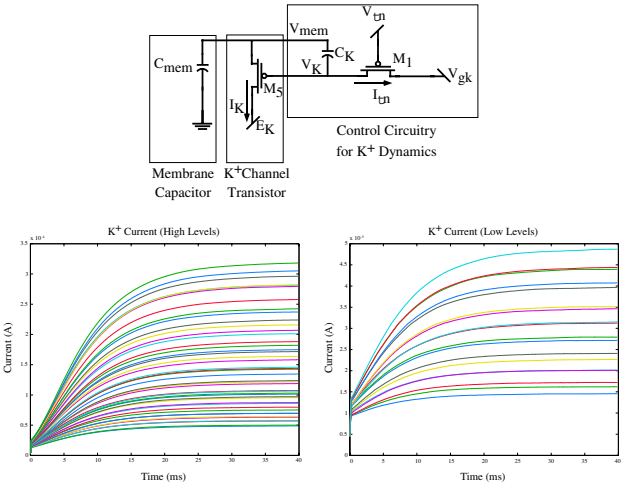

Fig. 5. The K^+circuit. Again, it is easy to see the channel transistor and the membrane capacitor. The circuit connected to the channel transistor is a lowpass filter, as is needed from observing the step response for a biological channel shown in Fig. 2. There are two plots of the K^+data to show that the dynamics are preserved even at low current levels (the right plot is for small input voltage steps). Note that both show a low pass response with the proper time response.

or a sub-threshold MOSFET transistor. The MOSFET transistor has been used for several reasons, one of which is the extremely low amounts of power dissipated by it in sub-threshold. A second significant reason is the fact that the current levels from a sub-threshold MOSFET transistor are naturally comparable in magnitude to those seen in biology.

There is a great deal of correlation between the physics of neurons and the physics of a sub-threshold MOSFET. Biological channels are really composed of two high level parts: the actual channel (the physical structure that ions flow through) and the gating mechanism which controls the opening and closing of the channel. Sub-threshold MOSFETs have this same idea, with one major difference. The channel of the MOSFET is gated by a voltage, however, where the biological channel has an inherent activating/deactivating mechanism (i.e. built into the protein structure itself) the activating/deactivating mechanism of a MOSFET must be designed.

The authors acknowledge that current through an individual biological channel is a stochastic phenomenon. However, this is also true for a MOSFET of sufficiently small width. One could build MOSFETs of extremely small width (all of which would exhibit this stochastic nature),place them in parallel, and observe the smooth currents usually seen from a transistor. Since this is analogous to a MOSFET of reasonable width, the transistors being used in this circuit can be said to model populations of biological channels. Fig.3 summarizes this idea.

It is easy to see why Hodgkin and Huxley would have conceptualized this channel as a variable conductance resistor in 1952. The transistor would have been unknown to anyone other than a few people at that time, and certainly not to those whose primary research was in the biological world. Now, however, the transistor is quite a common component, and our knowledge of them has increased greatly. Several successful attempts have been made to build neuron models on chip before, however in each case, the modelers have sought to implement the Hodgkin and Huxley equations [6][7][3]. While this is a valid method and can lead to action potential generation, it also leads to large circuit models which are based on equations, not biology. Since one of the ultimate goals of this work is to implement a chip with 1000's of channel models on it, small circuit size is of paramount important to success.

2.1. Na^+Circuit

Since it is known that the Na^+channel response is that of a bandpass filter, a bandpass circuit which would control a MOSFET channel in a similar way needs to be developed.

Fig. 4 shows the Na^+circuit including a bandpass control circuit which controls the Na^+channel transistor. This tunable bandpass filter has poles which can be moved based on voltages placed on the nodes $V_{\tau m}$ and $V_{\tau h}$. The tunability of this circuit enables the poles to be placed wherever they are needed to match the speed of the biological Na^+channel.

2.2. K^+Circuit

Similar to the terms τ_m and τ_h, Hodgkin and Huxley used the term τ_n to described the time constant of the activation of the K^+channel. A similarly named term V_{τ_n} is present in the K^+channel model. This term is the bias which controls the time constant of the activation of the K^+channel. In other words, it controls where τ_n is. Again, our results closely match the biological data.

2.3. Neuron Circuit

By tying these two circuits together, a complete, although simple, circuit model can be realized. However, tying these circuits together yields another point to consider; biological resting voltage. At that voltage nothing happens because the net current is $0A$. The resting voltage of this circuit is determined by the interplay of the two smaller circuits. This point can also be tuned (by setting V_{amp}, V_{sat}, and V_{gk}), and cannot be neglected as there is only a small regime in which the two circuits will cause an action potential. In other regions the circuit will be silent as one or the other overpowers the competition.

A current clamp experiment (current step) on the circuit affords the observation of an action potential; current is injected into the node V_{mem}, and the voltage response is measured. For low

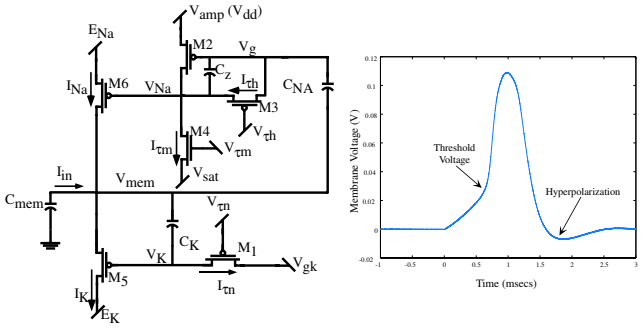

Fig. 6. When the two previously mentioned circuits are tied together, the basic "neuron" circuit is formed. Notice that in terms of size, it is roughly that of an "AND" gate. The graph shows an experimentally measured Action Potential generated by the circuit. Due to the tunability of the circuit, there can be significant variation in shape from one action potential (under a certain set of bias conditions) to the next. However, within a fixed set of biases, the action potentials all look alike regardless of the input current magnitude (obviously within the boundary conditions of the circuit).

amplitudes of input current, an action potential is not produced. A slight depolarization can be observed, but the voltage never reaches the threshold voltage where the Na^+channel really turns on. However, once enough current is injected, an action potential can be seen, Fig. 6. This figure show experimental results. Due to the fact that this circuit can be tuned to many different regions, the action potentials can be made to look quite different from each other. However, for a fixed set of biases, the action potentials will look very similar to each other regardless of the magnitude of the input current. Only the spike frequency will be affected.

Figure 7 shows simulated data, correlating to experimental IC measurements showing frequency versus current results. This circuit was simulated in SPICE, using the EKV model [2] to accurately model the sub-threshold behavior. We obtained results that closely matched experimental biological and circuit data. Simulation data for several different input currents is shown on the left of Fig. 7. Notice that the spikes look very similar from one to the next, save that the approach to the threshold voltage is much faster. The spikes in the third graph have decreased in size, but the input current is $21\mu A$ which is huge for this circuit, and has a good chance of killing a real cell.

It is worth noting that all of the currents seen here are quite high compared to biological channels and neurons. This is due to the fact that the devices used in this system had $\frac{W}{L}$ ratios of near 1500, and the capacitors were discrete instead of on chip. Simulated results for smaller size transistors show that the concept works even when the transistors are small (i.e. action potentials can be generated), and that the magnitudes of the currents is appropriate for the biology that is being modeled. A new IC with smaller sized transistors and integrated capacitors is in fab.

One may ask what changes happen to the action potential as input current is increased. The simple answer is that spike frequency changes. Since the voltage on a capacitor is related to the current onto it, the more current the faster it charges and the faster the neuron model spikes.

3. CONCLUSION

Using the numerous similarities between biology and silicon physics, a circuit which closely models biological neurons has been developed using just six transistors. We believe that this is a better way to model neurons for several reasons. Using this model one can simulate neurons in real time, do it in a way that closely resembles the physics of the system, potentially fit thousands of these channels onto a single chip (leaving us room for much more complex/complete neuron models), and do it all with low power.

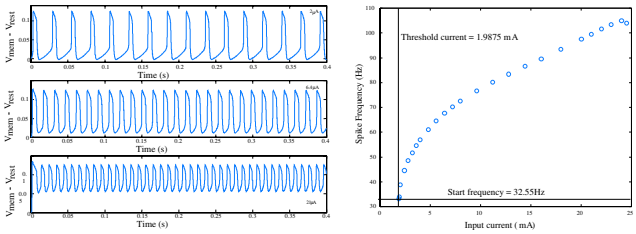

Fig. 7. Simulated data using an EKV model for the transistors. The graph on the left clearly shows the spike rate changing for different input currents (2μA, 6.4μA, and 21μA respectively). Notice that the general shape does not change, only the frequency. The graph on the right shows the relationship between spike rate and input current over a wider range of currents.

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