

# A FAMILY OF FLOATING-GATE ADAPTING SYNAPSES BASED UPON TRANSISTOR CHANNEL MODELS

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We have developed a family of three analog VLSI synapses based on three types of biological channel types, ACh-excitatory, NMDA-excitatory, and GABA<sub>A</sub>-inhibitory in a 0.5 μm CMOS process. We have successfully reproduced EPSPs and IPSPs similar to what is found in biology. Presently, we manually modify synaptic strength. We will continue our work to expand self-adapting synapses. Since these synapses are relatively small in area, we anticipate having hundreds of them on a single 1.5 x 1.5 mm die.

## 1. BIOLOGICAL MODEL

The biological synapse links neurons to one another, as seen in Figure 1. Cellular signaling takes place when the first neuron fires, causing a chemical to be sent from the first cell (pre-synaptic) to the second (post-synaptic). This chemical (neurotransmitter) diffuses through a gap between the cells (synapse) and binds to receptor sites on the post-synaptic cell. Ionic currents through the post-synaptic cell activate due to binding of chemicals on these receptor sites. These currents cause an electrical response, and the post-synaptic cell has successfully received the pre-synaptic cell's signal. Although the pre-synaptic cell's electrical signal (action potential) is essentially binary in nature, it is essential that the post synaptic potential, the second cell's electrical signal, remain analog in nature [1], [2]. This post synaptic potential can be either excitatory (EPSP) or inhibitory (IPSP) and can be modified by various pharmacological and morphological factors [3]. The PSP is often modelled with Rall's alpha function

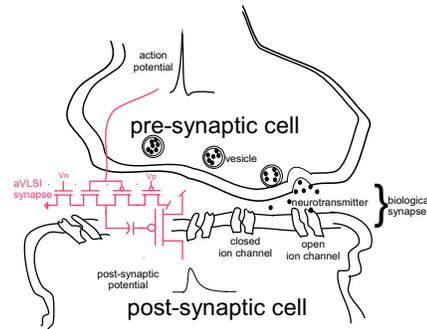
$$V_m = V_o \frac{t}{\alpha} e^{-\alpha t} = V_{max} \alpha t e^{(1-\alpha t)},$$

where  $V_{max}$  is the peak of the EPSP (or IPSP minima) [2].

The type of potential produced depends on the type of ions that flow through the cell when the neurotransmitter has binded to the cell. Ion channels, the site of ion flow through the cell and neurotransmitter reception, are akin to doors with locks. The door can open only when the proper key is used. GABA<sub>A</sub> and non-NMDA synapses employ ion channels which are keyed by the type of neurotransmitter present while NMDA synapses are keyed by the type of neurotransmitter and the voltage across the cellular membrane. Figure 2 illustrates a few of these ion channels.

## 2. SILICON MODEL

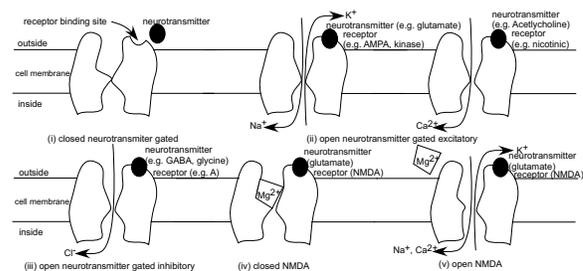
We have modified a single-transistor learning synapse (STLS) in order to create different synaptic types that are based on the types of ion channels present at the synapse. The STLS is a floating



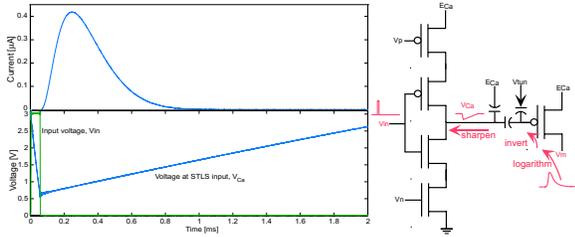
**Fig. 1.** The biological synapse is the main communication port for the nervous system. Here, an electrical response in the first (pre-synaptic) cell causes chemicals to leave and be deposited into the second (post-synaptic) cell. These chemicals, called neurotransmitters, then cause an electrical response in the second cell. Our silicon synapse has been highlighted in red to illustrate how it functionally relates to the biological synapse.

gate pFET [4]. This non-volatile method of charge storage allows for easy change of the threshold voltage of the pFET through hot-electron injection and Fowler-Nordheim tunnelling. The STLS is used as a memory device which will change its output current depending on the amount of charge on the floating gate, thus the charge on the floating gate is equivalent to the synaptic strength.

Furthermore, the forces which drive electric currents in a sub-threshold MOSFET, diffusion and drift, are the same forces which drive ionic currents through ion channels in neurons [5], [6]. Thus subthreshold MOSFETs are ideal structures to model ion channel behavior. These low, subthreshold currents allow us to have



**Fig. 2.** Examples of ion channels found in the nervous system.

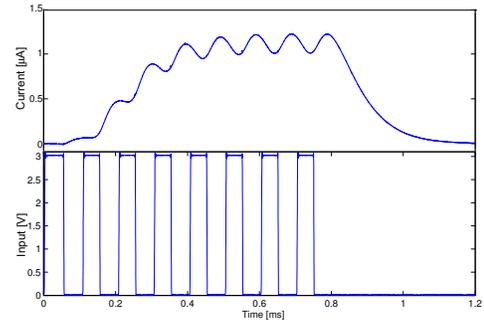


**Fig. 3.** A typical EPSC that would be found in biology modeled from Rall's alpha function on top with the log of the alpha function and a digital action potential at the bottom. The right illustrates the steps taken in developing the synapse. Important functions, nodes, and waveforms are highlighted in red.

thousands of synapses on chip which collectively use little power. Although our present implementation has not been optimized for size, its area is less than  $850 \mu\text{m}^2$  and would allow for over 600 synapses on a MOSIS TinyChip ( $1.5 \times 1.5 \text{ mm}$  die) with room for 40 pads, testing circuitry, peripheral circuitry, and wiring.

We will make further explorations in to modifying these circuits so that they will alter their synaptic strength automatically. We have already created simpler systems which can modify synaptic strength in a biological fashion [7]. Biologically, synapses strengthen through long-term potentiation (LTP) in which pharmacological and morphological changes are made improve signal transduction from the pre-synaptic to the post-synaptic cell. After LTP, the PSP is much stronger than it was before LTP. Conversely, long-term depression (LDP) decreases synaptic strength such that the PSP becomes weaker. We will be incorporating our previous work with these circuits in order to move from programmable synapses to self-adapting synapses.

To create accurate synaptic circuits with variable strength, we first investigate an EPSP that is produced by an action potential like signal. By inspection, a typical EPSP is an exponential signal with the upgoing side's time constant is much faster time constant than the downgoing side, as illustrated in Figure 3. The log of that signal is basically a triangle wave with an upgoing slope that is much faster than the downgoing slope. Since the output current of a subthreshold MOSFET is exponentially dependent on the input gate voltage, an asymmetric triangular signal placed on the gate should look like an EPSP. Since, pFETs invert the input signal instead of a fast upgoing slope and a slow downgoing slope, we should invert this signal so that it is a fast downgoing slope followed by a slow upgoing slope. A simple method of producing such an asymmetric triangular signal is by having a subcircuit which can charge a capacitor faster than it discharges it. An inverter modified with pull-up and pull-down biases can easily accomplish this task by setting the pull-up bias such that it draws current more slowly than the pull-down bias. Furthermore, this signal can be created with a digital pulse, which is an action potential-like signal. Thus we have accomplished our goal, to have an action potential like input with an EPSP like output which can be weighted according to synaptic strength. All of the other synapse types discussed here are built upon this foundation.



**Fig. 4.** Similar to biological findings, an increase in the frequency of the signal results in the aggregation of basic EPSPs.

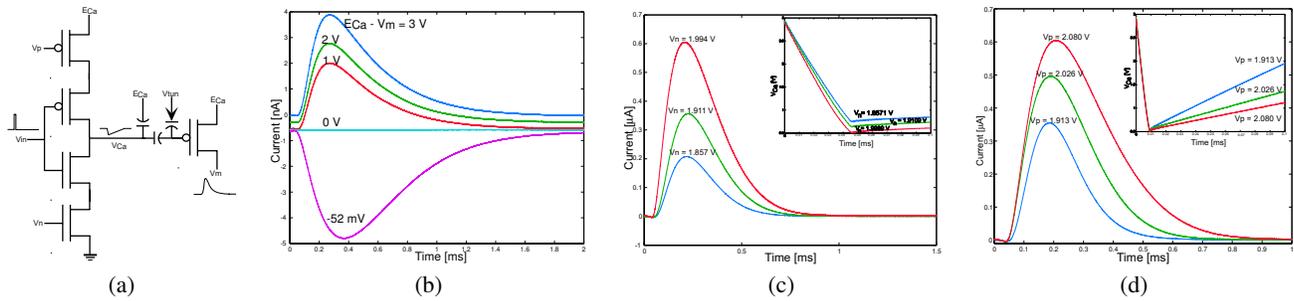
### 3. EXCITATORY SYNAPSE

There are many varieties of excitatory channels in the nervous system as seen in Figure 2. Acetylcholine channels allow  $\text{Ca}^{2+}$  to rush into the cell in the presence of acetylcholine, while glutamate channels bring  $\text{Na}^+$  in and drive  $\text{K}^+$  out. This circuit topology can emulate many types of excitatory synapses by simply changing the relative values of the constant voltages (Figure 5). For instance, while  $E_{\text{Ca}}$  and ground are used to represent a collection of ACh channels, they could be replaced by  $E_{\text{Na}}$  and  $E_{\text{K}}$  respectively to emulate a family of glutamate channels. Of course, the digital input to the synapse should be scaled accordingly in order to maintain proper operation.

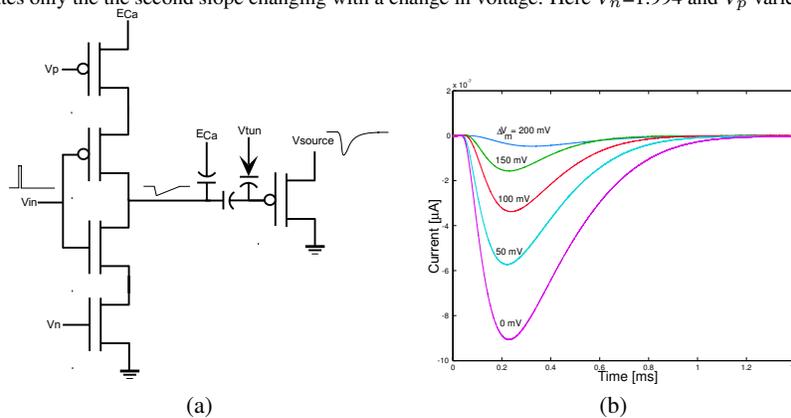
Figures 5c and 5d illustrate how we can easily create a variety of EPSPs by simply modifying our pull-down ( $V_n$ ) and pull-up ( $V_p$ ) biases. Although the voltage levels given are accurate to three decimal places, the operating point within the  $V_n, V_p$  parameter space need not be so accurate. Our experiments were repeatable and stable using 12-bit DACs and hand-turned potentiometers. As expected,  $V_n$  changes the EPSP's rising time constant while  $V_p$  changes the falling time constant. Therefore by changing our location in the  $V_n, V_p$  parameter space, we can emulate the many of the natural stimuli which modify EPSP shape and size [1], [2], [3].

One natural stimulus which greatly affects the PSP is the driving force that a particular ion has to propel it through the channel. The larger this driving force, the easier it is for the ion to move through the channel. This driving force is dependent on the membrane voltage ( $V_m$ ) and the ion's concentration gradient between the inside and outside of the cell. This gradient can be represented as a voltage such as  $E_{\text{Ca}}$  for calcium. Biological experiments to explore this biological phenomena are known as voltage clamp measurements. Figure 5b shows how we can modify our voltage levels to recreate biological voltage clamp data [1], [2], [8].

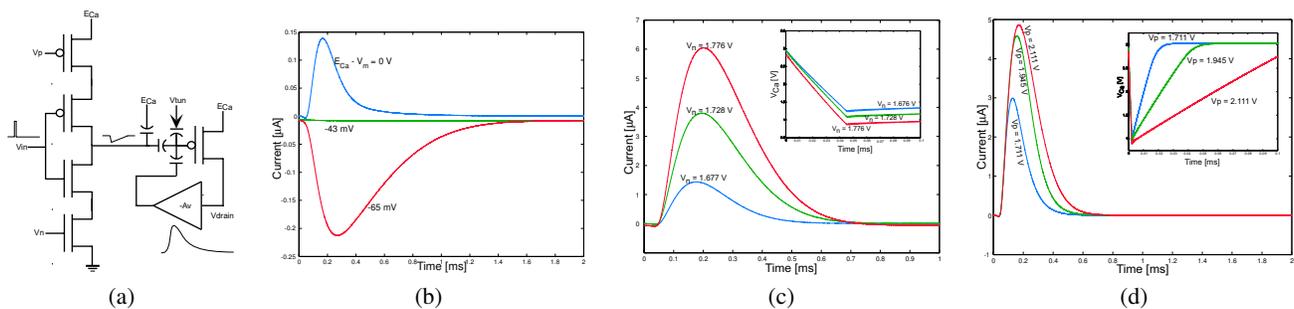
Another biological phenomena is that of temporal summation. EPSPs combine to form one large aggregate signal. This effect is much like charging a capacitor with a series of small pulses. Each individual pulse is not enough to charge the capacitor, but if the period is small enough, the capacitor does not have enough time to completely discharge. Over time, the capacitor will be completely charged with some ripple. Figure 4 shows that we can achieve results similar to what is found in biology.



**Fig. 5.** (a) The input of the basic excitatory circuit is basically an inverter with an output that has been slowed down by the capacitor to  $E_{Ca}$ . The net synaptic strength is proportional to the charge on the floating gate and like its biological analogue, this synaptic strength can be modified. (b) Changing  $V_d$  in excitatory synapse. In biology, decreasing the membrane voltage decreases the size of the basic EPSP. Here decreasing the voltage across the synapse channel transistor, decreases the size of the EPSP. Here  $V_n=1.259$  and  $V_p=2.238$ .(c) The rate at which initial, upgoing side of the basic EPSP increases by increasing  $V_n$ . Note the inset, a plot of the voltage on the input of the STLS, illustrates only the the beginning slope changing with a change in voltage. Here  $V_n$  varies and  $V_p=2.080$ .(d) The rate at which second, downgoing side of the basic EPSP increases by increasing  $V_p$ . Note the inset, a plot of the voltage on the input of the STLS, illustrates only the the second slope changing with a change in voltage. Here  $V_n=1.994$  and  $V_p$  varies.



**Fig. 6.** (a) The inhibitory synapse is similar to the basic excitatory, with the exception of the location of the output terminal. The floating gate source draws current, rather than having the drain provide current. Thus, we have an inhibiting effect on any subcircuit that this synapse is connected to. (b) As expected, these  $GABA_A$  inhibitory results are basically the mirror of the excitatory synapses where this IPSP decreases for decreasing voltage across the STLS in this voltage clamp experiment. Here  $V_n=1.867$  and  $V_p=2.109$ .



**Fig. 7.** (a) Like the biological NMDA synapse, this circuit's response depends on the membrane voltage as well as the carrier flow. Here, we use a  $C^4$  amplifier to mimic the effect of  $Mg^{2+}$ . (b) As expected, the NMDA voltage clamp experiment results are similar to the basic excitatory synapse where the EPSP decreases, then goes negative for decreasing voltage across the STLS. Here  $V_n=1.259$  and  $V_p=2.238$ . (c) The rate at which initial, upgoing side of the NMDA EPSP increases by increasing  $V_n$ . Note the inset, a plot of the voltage on the input of the STLS, illustrates only the the beginning slope changing with a change in voltage. Here  $V_n$  varies and  $V_p=2.080$ . (d) The rate at which second, downgoing side of the NMDA EPSP increases by increasing  $V_p$ . Note the inset, a plot of the voltage on the input of the STLS, illustrates only the the second slope changing with a change in voltage. Here  $V_n=1.777$  and  $V_p$  varies.

#### 4. INHIBITORY SYNAPSE

The inhibitory synapse produces a decrease in membrane voltage in response to an action potential in the pre-synaptic cell. Thus, the IPSP is effectively the mirror image of the EPSP. This IPSP is caused by a flow of  $Cl^-$  ions through  $GABA_A$  gated channels. Glycine gated channels operate in a similar fashion and these silicon synapses can be considered to be similar to glycine biological synapses as well.

Figure 6a shows that we simply take a basic excitatory synapse and use a different node for the output. Since we want a net flow of negative current, we use the STLS source node for output rather than the STLS drain node. Current is now drawn rather than provided. The resulting effect is shown in Figure 6b. Figure 6b is a repetition of the voltage clamp experiment done above in the excitatory circuit section where we have effectively changed the driving force of the ions through the channel in an inhibitory synapse.

#### 5. NMDA EXCITATORY SYNAPSE

Biological NMDA synapses are doubly gated by the type of neurotransmitter present and the voltage across the cell membrane. We have reproduced this gating mechanism by adding a feedback mechanism to the basic excitatory synapse. As illustrated in Figure 7a, the feedback is a  $C^4$  amplifier with a gain of -1 to a second input to the STLS. The  $C^4$  is a tunable bandpass filter in which each corner can be adjusted with a bias [9]. We pushed the corners as far out as possible, thus allowing the  $C^4$  to act like an all-pass amplifier. As the output of the synapse increases, the output of the feedback decreases, thus decreasing the voltage at the input of the pFET floating gate. A decrease in the pFET input effectively increases the output, thus repeating this cycle. In the biological NMDA synapse, the presence of neurotransmitter initially opens other non-NMDA channels, which begins to depolarize the cell. Once the cell has depolarized enough, a  $Mg^{2+}$  ion is forced out of the NMDA channel and the channel fully opens. The initial depolarization of the cell is like the initial voltage change on the output of the circuit while the feedback which augments this change on the circuit effectively like the  $Mg^{2+}$  ion being forced from the channel.

Figures 7c and 7d illustrate that we can easily create a variety of EPSPs by simply changing  $V_n$  and  $V_p$ . Similar to the basic excitatory synapse data shown above in Figures 5c and 5d we can easily choose  $V_n$  and  $V_p$  to create an EPSP to suit a variety of applications. Figure 7b illustrates that we can drive the circuit from a positive to negative EPSP with a voltage clamp experiment. This is effectively the same as initially driving  $K^+$  out and  $Na^+$  in, not moving the ions much at all, and finally driving  $K^+$  in and  $Na^+$  out.

#### 6. CONCLUSION

We have added new circuits to the available models of synapses. These circuits transform a binary signal in to an analog EPSP. These artificial excitatory synapses respond to equivalent stimuli in the same fashion as biological excitatory synapses. Such stimuli include, and are not limited to, changing the membrane voltage and frequency of the input.

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