

Silicon Neurons that Inhibit to Synchronize

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Abstract—We present a silicon neuron that uses shunting inhibition (conductance-based) with a synaptic rise-time to achieve synchrony. Synaptic rise-time promotes synchrony by delaying the effect of inhibition, providing an opportune period for neurons to spike together. And shunting inhibition, through its voltage dependence, inhibits neurons that are late more strongly (delaying the spike further), thereby pushing them into phase (in the next cycle). We characterize the soma (cell body) and synapse circuits, fabricated in $0.25\mu\text{m}$ CMOS. Further, we show that synchronized neurons (population of 256) spike with a period that is proportional to the synaptic rise-time.

I. NEUROMORPHIC SYNCHRONIZATION

Spike synchrony is important in neural computation, making it desirable to neuromorphic engineers, who aim to reproduce the spike-based computation of the brain in silicon. Whereas synchrony is conspicuously absent in neuromorphic systems, it is conspicuously present in numerous brain regions, in central areas involved in learning and memory (e.g., hippocampus, neocortex), as well as in peripheral areas involved in olfaction (the olfactory bulb) and vision (the retina).

Spike synchrony in the brain is more than a mere global clock: It binds neurons that represent aspects of the same object or item [1]. When two distinct groups of neurons are excited, neurons within each group synchronize, but the two groups have independent rhythms, failing to phase-lock. However, when the two groups overlap, all the neurons synchronize, signaling that these two groups represent a single object. Evidence suggests that this binding phenomenon requires distributed rhythmic synchrony generated by locally interacting inhibitory neurons [2].

Inhibition's ability to promote synchrony is counter-intuitive; it becomes clear once we take synaptic delays into account. Whereas mutual excitation advances the neuron that is late to spike [3], bringing neurons together, mutual inhibition retards spiking, pushing neurons apart. Thus excitation often results in synchrony whereas inhibition often results in asynchrony. However, these relationships can be reversed by delays. In fact, for fast rhythms (tens of hertz), the synaptic delays found in biology are significant compared to the rhythm's period. Hence, excitation can produce phase-shifts close to 180° , impeding synchrony, whereas, under similar conditions, inhibition produces 360° phase-shifts, promoting synchrony. Think of it this way: Delaying inhibition provides an opportune period for neurons to spike together.

In addition to delayed inhibition, synchrony requires inhibition to act as a shunt (i.e., nonzero conductance). Unlike a current sink, the current passed by a shunt is proportional to the voltage across it. Thus neurons that have just reset their

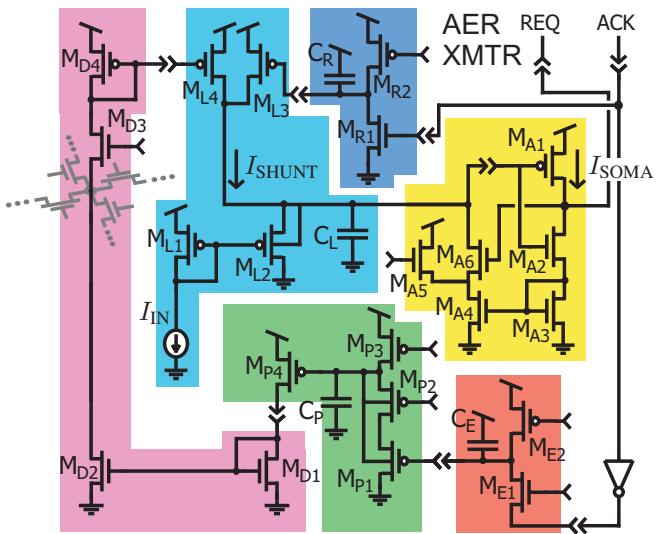


Fig. 1. The (simplified) neuron circuit comprises two modules, both based on log-domain low-pass filters: the soma and the synapse. The soma includes the membrane (cyan, M_{L1-4}), the axon hillock (yellow, M_{A1-6}), and the refractory period (blue, M_{R1-2}). The synapse includes the cleft (red, M_{E1-2}) and the receptor (green, M_{P1-4}). A diffuser with current mirrors spreads inhibition to neighboring neurons (magenta, M_{D1-4}). Single arrows represent bias voltages; double arrows represent inputs and outputs.

spikes receive negligible inhibition while those that are close to the spiking threshold receive massive inhibition. As a result, neurons that spike in synchrony (within the synaptic delay) remain unaffected while those that are late to spike (yet to invoke the axon-hillock's regenerative action) are pushed away.

Lacking these properties, current neuromorphic synapse and neuron models synchronize poorly when using inhibition. Current-mirror synapses (CMSs) [4] lack synaptic rise-time and integrate-and-fire neurons (IFNs) [5] lack shunting inhibition. Systems composed of these elements can achieve a moderate degree of synchrony, but it is fragile, requiring excitation to rescue it [6].

To remedy these deficiencies in silicon neural models, we have developed a new silicon neuron (soma and synapse circuits), based on current-mode log-domain low-pass filters (LPFs) and pulse extenders (PEs) (Fig. 1). These neurons synchronize robustly, while being compact in size. In Section II, we describe the circuits that implement our neuron. In Section III, we characterize these circuits. In Section IV, we use a network of these neurons to generate synchrony by inhibition. In Section V, we discuss the implications of our new circuit designs and their use in other systems.

II. NEURON IMPLEMENTATION

We construct the neuron from two circuit modules based on log-domain LPFs: the soma and the synapse (Fig. 1). The soma implements membrane dynamics and spiking; the synapse supplies shunting inhibition.

A. Soma Circuit

We construct the soma from three subcircuits: the membrane, the axon hillock, and the refractory period (Fig. 1). The membrane realizes a leaky integrator (RC) response to excitatory current and shunting inhibition. An input current drives the capacitor (C_L) through a source-coupled current mirror (M_{L1-2}). As the capacitor voltage approaches M_{L2} 's gate voltage, the current decreases, compensating for the transistors' nonlinear (logarithmic) voltage-current relation. In this study, the input current is always constant, whereas the leak current, I_{SHUNT} , varies in time; it comprises the sum of a constant current (not shown), an inhibitory synaptic current (M_{L4}), and a refractory current (M_{L3}).

The membrane's output (analogous to the potential of an RC circuit) is the soma current, I_{SOMA} (M_{A1}). Increasing I_{SHUNT} reduces the steady-state output and decreases the time-constant (identical to increasing the conductance in an RC circuit). We derive the soma behavior by applying Kirchhoff's Current Law to node C_L and solving in terms of currents, which yields:

$$\tau \frac{dI_{SOMA}}{dt} = -I_{SOMA} + \frac{I_{IN} I_0}{I_{SHUNT}} \quad (1)$$

where I_{IN} is the input current, $\tau = \frac{C_L U_T}{\kappa I_{SHUNT}}$ is the soma's time-constant and I_0 is a transistor parameter as are κ and U_T (thermal voltage). In addition to the constant excitatory input and variable leak, the membrane also receives a positive feedback current for spike generation.

The axon hillock (modified from [8] by Kai Hynna) provides the positive feedback current. As I_{SOMA} increases, the feedback current (M_{A6}) turns on more strongly, overpowering the leak to cause a spike. When a spike occurs, the axon hillock initiates the process of sending an address-event off chip, which activates the refractory period.

The refractory period shunts I_{SOMA} to near zero (pulls C_L to V_{DD}) for a brief period (a few ms) after a spike, using a PE. The PE interfaces fast (about 10ns) digital signals to slow (several ms) analog ones by generating a current-pulse output (M_{L3}) from a voltage-pulse input. Its capacitor (C_R) is pulled to ground during a spike (M_{R1}), which causes M_{L3} to drive C_L to V_{DD} , until the leak through M_{R2} restores C_R .

B. Synapse Circuit

We construct the synapse from two subcircuits: the receptor and the cleft (Fig. 1). The receptor, implemented with an LPF, sets the synapse's fall-time (similar to [9]), while the cleft, implemented with a PE, sets its rise-time. The receptor's LPF differs from that of the soma. Its input (from the cleft) is a fixed-height pulse, which allows for a simpler circuit: a

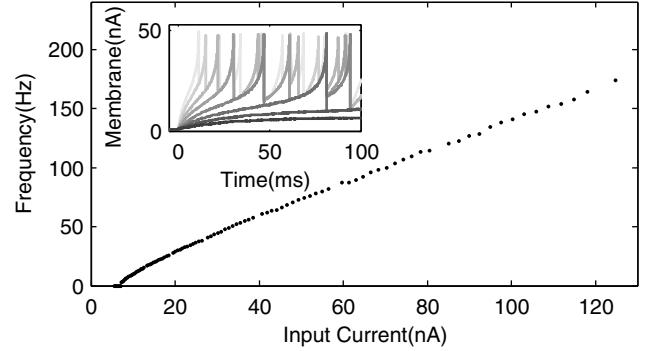


Fig. 2. The soma responds sublinearly to current (above a threshold). *Inset*: Membrane (current) traces for several step-input current levels show an RC rise and a positive feedback spike.

voltage-limited source follower (M_{P1-2}), whose voltage limit (applied to M_{P2} 's gate) sets the pulse height and hence the maximum current level that the receptor's output (M_{P4}) can achieve. It saturates at this level when driven at a high rate or with a pulse width that is long relative to its time-constant. The receptor's output drives a diffusor, which spreads the synaptic current to neighboring silicon neurons.

Our log-domain neuron and its synapse are an improvement over IFNs and CMSs. They are similar in size and complexity, while capable of modeling phenomena that depends on synaptic rise-time and shunting inhibition, such as synchrony by inhibition.

We have designed, submitted, and tested a chip with an array of our silicon neurons. The chip was fabricated through MOSIS in a 1P5M 0.25 μ m CMOS process, with just under 750,000 transistors in just over 10mm² of area. It has a 16 by 16 array of inhibitory neurons (28 by 36 μ m each) commingled with a 32 by 32 array of excitatory neurons that are not used here. The chip uses address-events [7] to transmit spikes off chip and to receive spike input. In addition, the chip includes an analog scanner that allows us to observe the state of one neuron at a time (either its soma or synapse).

III. NEURON CHARACTERIZATION

In characterizing the neuron, we focused on three aspects: the frequency-current curve (FIC), the synaptic rise-time, and the phase-response curve (PRC). The PRC summarizes the effects of rise-time and shunting inhibition on the soma.

A. Frequency-Current Curve

When various current levels are injected into the soma, its spike frequency increases sublinearly above a threshold. Below this threshold (8nA), the input current drove the soma to a steady state level too low for the positive feedback to overcome the leak (Fig. 2 *Inset*). Above it, the input current invoked sufficient positive feedback to overcome the leak resulting in a spike (which shut off the input by lowering M_{L2} 's source).

B. Synaptic Rise-time

When stimulated with a spike, the synaptic current increased initially linearly (far from the maximum level), and

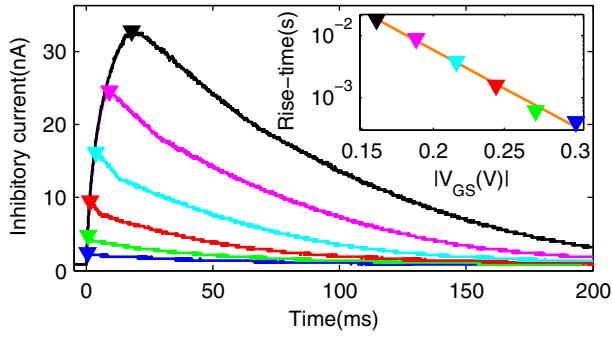


Fig. 3. The synapse responds to a spike with a low-pass filtered pulse. *Inset* The time-to-peak (triangles) depended exponentially on the gate-source voltage of the cleft's leak transistor (M_{E2} in Fig. 1).

decreased exponentially (time-constant fit was 70ms). We characterized the synaptic rise-time by varying the cleft's leak current (adjusting M_{E2} 's gate voltage) and hence the pulse-width. We measured the resulting synaptic current's rise-time, defined as the time-to-peak (Fig. 3). The rise-time depended exponentially on M_{E2} 's gate voltage, because the pulse-width is proportional to the current through this transistor. Also, the peak current increased with the pulse width, since the receptor's current had more time to rise.

C. Phase-Response Curve

The effect of synaptic inhibition depended on the phase at which it occurred. We characterized this phase-dependence by inhibiting the neuron at a random point in its cycle, once every five cycles, observing the increase in interspike interval (ISI). We repeated this process several hundred times and plotted the resulting PRC (Fig. 4 Top). The rise-time was set to 1.5ms and the time-constant was 5ms.

The neuron was most sensitive to inhibition between 15 and 30ms after it spiked (its uninhibited ISI was 38ms). In this sensitive region, each inhibitory spike added more than 8ms to the neuron's ISI. During this phase of its cycle the neuron's membrane (current) was high, resulting in more effective shunting inhibition (Fig. 4 Bottom). On the other hand, inhibition applied less than 5 or more than 32ms after it spiked added less than 4ms to the neuron's ISI. During these phases, either its membrane potential was low, so shunting inhibition was less effective, or the inhibition did not have time to rise to its peak effectiveness. And near the cycle's end, the positive feedback from the axon hillock turned on, overpowering the inhibition.

IV. APPLICATION TO SYNCHRONY

Having characterized an individual neuron's properties, we tested the (16 by 16) network's ability to synchronize for two different rise-times. We drove each neuron with a constant current (31nA) and configured it to inhibit itself and all of its neighbors, using a diffusor biased to spread synaptic current globally. Because the amplitude of the synaptic current depends on the pulse width, we increased the amplitude of

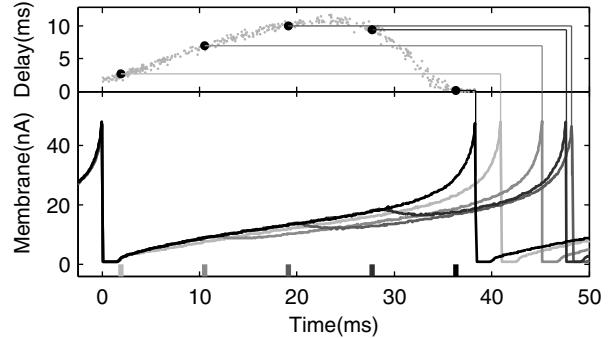


Fig. 4. *Bottom* Membrane (current) traces of a neuron that we drove with a constant current and inhibited at various phases; the increase in interspike interval depends on when inhibition occurs (vertical bars). *Top* The phase-response curve shows that inhibition is most effective between 15 and 30ms after the neuron spikes, adding about 8ms to its interspike interval (38ms).

inhibition (by increasing the maximum level) when the rise-time was fast, so that the neurons received about the same inhibition, spiking at about the same rate, with about the same number of neurons active in both cases. The average rate was 36Hz versus 38Hz and the active fraction (spiked at least once in 250ms) was 45% versus 47%.

Synchrony by inhibition required a synaptic rise-time. Using a fast rise-time (0.1ms), the network did not synchronize (Fig. 5a), whereas using a slow rise-time (11.6ms), the network synchronized at 38Hz (Fig. 5b). We quantified synchrony by calculating the network's vector strength (VS). VS is a normalized sum of unit-length vectors, one for each spike: Their angles correspond to the spike's phase relative to the strongest frequency (from an FFT of the population histogram). If all of the neurons' spikes lined up at the same phase (perfect synchrony), VS would equal one. Conversely, if the neurons' spikes distributed themselves at random phases (asynchronous), VS would approach zero. Unlike other synchrony measures, VS does not penalize suppression of neurons, which is useful in our system. VS equaled 0.18 and 0.83 for the fast and slow rise-times, respectively.

To confirm the synaptic rise-time's pivotal role in synchrony, we varied it and measured the network period (the inverse of the strongest frequency). The network period was one to two times the rise-time, depending on the fall-time (receptor's time-constant), plus an offset, caused by the axon hillock's positive feedback overpowering inhibition shortly before a spike. With a rise-time of 11.6ms, and a receptor time-constant of 5ms (same as Fig. 5), the network period (26ms), minus an offset (10ms), was 1.5 times the rise-time (Fig. 6). This same proportionality constant yielded a good fit for rise-times ranging from 3 to 100ms. The network is synchronous (VS > 0.5) for rise-times between 10 and 60ms.

V. DISCUSSION

Our silicon neurons synchronize using shunting inhibition with a rise-time, verifying that synchrony by inhibition is robust to neuronal variability (from transistor mismatch). When inhibiting each other, 45 to 47% of neurons were active.

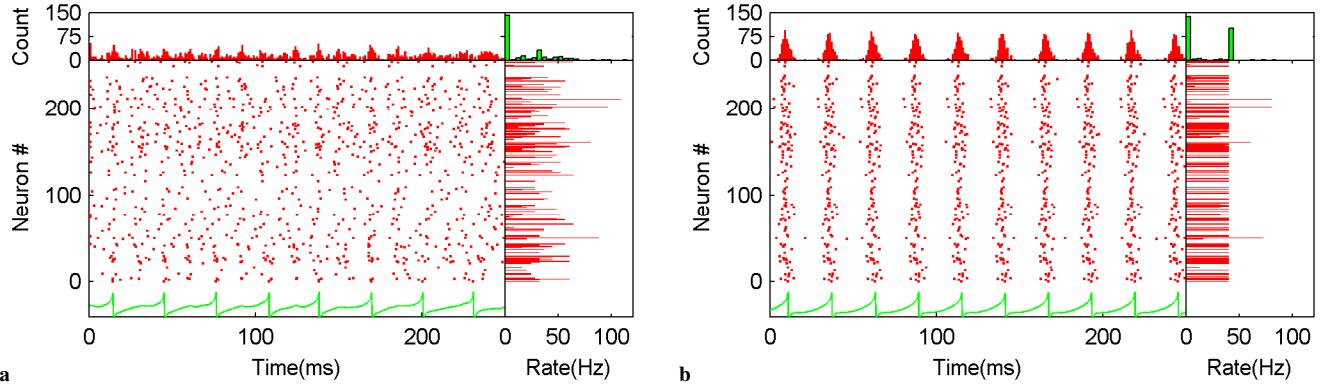


Fig. 5. Synchrony requires finite synaptic rise-time. **a** With a fast rise-time (0.1ms), the neurons spiked asynchronously ($VS = 0.18$, see text). **b** With a slow rise-time (11.6ms), the neurons spiked synchronously ($VS = 0.83$). For both panels: *Bottom left* Rasters of all 256 neurons and membrane (current) of one representative neuron. *Top left* Histogram of spike activity (1ms bins). *Bottom right* Average spike rate for each neuron. *Top right* Distribution of spike rates for all neurons.

In the asynchronous case (fast rise-time), these neurons had a frequency coefficient of variation (CV) of 0.52, whereas in the synchronous case (slow rise-time), the CV decreased to 0.28 (in both cases the CVs were 0.24 when neurons only inhibited themselves). Augmenting inhibition with fast excitation (gap junctions) among neurons may increase the number of active neurons by providing additional current to less excitable neurons, giving them the chance to fire before being inhibited.

The rise-time is not only necessary for synchrony; it determines the network period. We found the network period to be proportional to the rise-time, which delayed inhibition by between a quarter- and a half-cycle (depending on the receptor's time-constant). This explains rise-time's role in setting the period, as ideally network activity should be 180° out of phase with inhibition [10]. In addition to rise-time, other sources of delay can influence the network period: Axonal propagation is the primary source of delay in biology.

Inhibitory interactions, which generate synchrony in a spatially distributed manner, can mediate binding, which is not possible with a global clock. In the data we presented, these

interactions were far-reaching (mediated by a diffusive grid). Synchrony is still achieved when inhibition spreads only to nearby neurons (data not shown). In this situation, we posit that groups of neurons that are separated by a distance greater than the spreading range will not synchronize; only neurons within the same group will synchronize. However, if the groups come within range, they will start to synchronize, and synchrony will be robust when there is overlap, realizing binding. We are currently exploring this behavior with our chip by activating neurons in patches.

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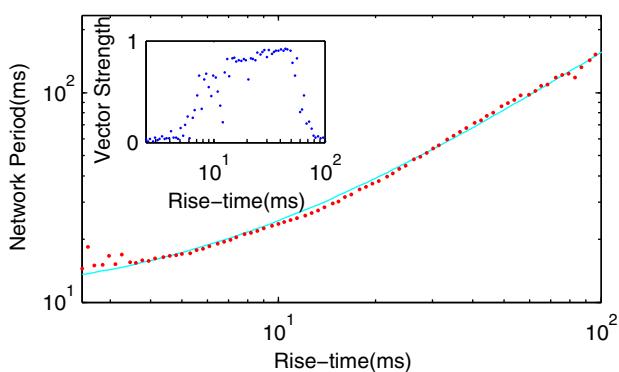


Fig. 6. The rise-time determines the network frequency. The network period is 1.5 times the rise-time (plus an offset of 10ms), determined using a linear fit (cyan). *Inset* The network is synchronous for rise-time values ranging between 10 and 60ms, with 21 to 66% of neurons active.