**Motivation & Introduction**

- The biological neuron has complex and highly nonlinear dendritic computations – dendritic spiking.
- These dendritic spikes are key to initiate precisely timed axonal AP (action potential) and hence empower the input-output transformation among neurons.
- Three model neuron configurations:
  1. Neuron 1 - clustered synapses onto same branch.
  2. Neuron 2 - distributed synapses on different branches.

**Model Neuron Setup**

- Each model neuron has circuit components: 18 synapses, 3 dendritic branches, soma, and axon hillock (~300 transistors).
- Non-linear two-stage dendritic arbor implemented.
- Circuit designed using CNFET (carbon nanotube field-effect transistor) SPICE model.

**CNFET Simulation Results**

**Effect of dendritic spike on output AP timing**

- Dendritic spike (Neuron 1) leads to larger dv/dt in the somatic potential compared to PSP alone (Neuron 2).
- Large dv/dt in the somatic potential results in relatively invariant IO delay and less AP jitter.

**Effect of synaptic synchronization on output AP timing**

- ISI (inter-stimuli interval) between inputs AP and AP+, varies.
- Dendritic spike requires spatiotemporal clustered inputs.

**Effect of synaptic activation level on output AP timing**

- Number of synapses activated in Neuron 1 and Neuron 3 varies.

**Results and Discussion**

- When ISI increases, output AP jitter and IO delay in both model neurons increase.
- The rate of increase is higher in Neuron 1 because dendritic spike is more sensitive to the degree of input synchrony.
- When the active dendrites are absent (Neuron 3), the AP jitter and IO delay are highly dependent on the number of synapses activated.
- With passive dendrites only, it would require more neurons to achieve accurate signal transformation.