Single organic electrochemical neuron capable of anticoincidence detection

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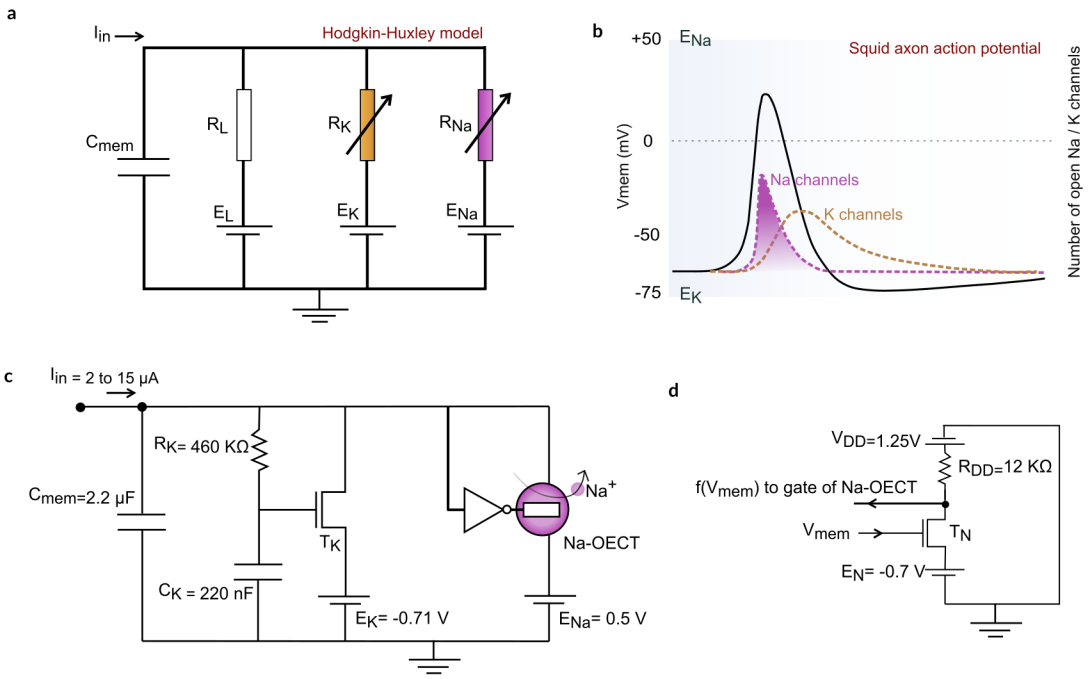

Supplementary Figure 1: **a.** Schematic of the Hodgkin-Huxley neuron model depicting sodium (Na^+) and potassium (K^+) ion channels**. b.** Graphical representation of a biological action potential with the activity profiles of Na^+ and K^+ ion channels. **c.** The c-OECN (soma) circuit with corresponding components. **d.** Detailed circuit diagram of the NMOS-based inverting amplifier utilized within the soma circuit. Both T_K and T_N are NMOS transistors (Infineon BSP295 model). Components R_K and C_K introduce the necessary delay for emulating the dynamics of the potassium channel.

Supplementary Note 1: Operation of the soma part of the circuit.

The operation of the soma part in the d-OECN circuit utilizes a conductance-based model, incorporating sodium and potassium channels that mimic the Hodgkin-Huxley model. The circuit comprises two main transistors: a sodium ion-based transistor (Na-OECT) and an NMOS transistor T_K, each connected to two distinct voltage sources, E_{Na} (500 mV) and E_K (ranging from ~0.71 to -0.75 V). These components parallel the ion channels and batteries in the original Hodgkin-Huxley model, as illustrated in Supplementary Figure 1. Voltage E_{Na} is applied to the drain of Na-OECT and E_K to the source of T_K.

In this setup, an input current I_{in}, varying between 2-15 μA, is fed into the circuit. This current is accumulated by the membrane capacitance C_{mem}, causing the membrane potential V_{mem} to rise from its baseline. Concurrently, this rise in potential drives the gate voltage of Na-OECT from approximately 1.2 V to 0.8 V via an n-type metal-oxide-semiconductor (NMOS) based inverting amplifier (Supplementary Figure 1d). This adjustment enables the device to navigate through the peak of the antiambipolar transfer curve, initiating a spike in the current which further charges the capacitor, thereby sharply increasing V_{mem} (depolarization phase).

Following this, the NMOS transistor T_K activates after a brief delay, controlled by resisitor R_K and capacitor C_K, and achieves its maximum current subsequent to the peak current of Na-OECT. The capacitor discharges through T_K, leading to a decrease in V_{mem} (repolarization phase) and returning it towards its original level. Due to the higher and longer-lasting current of the K-OECT, the voltage momentarily dips below the baseline (hyperpolarization phase). This sequence of events recurs cyclically with a constant input current, thereby sustaining the generation of action potentials.

Adjustments to the threshold T_K can be made by modifying E_K from approximately -0.71 to -0.75 V, optimizing the operational range of the soma necessary for effective integration within the d-OECN system. A larger negative E_K will operate the c-OECN in class II spiking mode necessary for the integration.