1. Introduction

Neurodegenerative diseases and aging may often lead to synaptic degeneration. People affected by such pathologies indeed exhibit cognitive deficit and loss of synaptic plasticity, i.e., the ability of neurons to adapt and respond to external stimuli. This mechanism is at the base of learning and memory capabilities of the brain: the periodic stimulation of the presynaptic neuron causes the strengthening of the synaptic connection and a long-term potentiation of the postsynaptic end. On the other hand, low-frequency stimuli might cause the weakening of the synapse and a long-term depression (LTD) state. Recent studies reported a strong correlation between the decrease of synaptic density and memory loss, even though the exact mechanism is still unknown. Among possible hypothesis, it seems that neurodegeneration is related to a decrease of the expression of proteins involved in synaptic functioning (i.e., presynaptic vesicles transporters, postsynaptic regulators). However, the complexity of the brain and neuronal connections prevents the studies of biological mechanisms underlying cognitive impairment. In this scenario, in vitro platforms have attracted significant interest as they could provide simplified biomimetic models of neuronal systems and allow the investigation of synaptic proteins' role in neurodegeneration. In this context, organic electrochemical transistors (OECTs) have recently emerged as neuromorphic devices that exhibit synaptic plasticity and adaptive behavior as biological synapses. OECTs are three terminal devices with source and drain electrodes connected by an organic semiconductor channel, that offers an efficient ionic-to-electronic current transduction and in situ signal amplification, while being able to interface with biological cells. Most of these devices are based on poly(3,4-ethylenedioxythiophene) doped with poly(styrene sulfonate) (PEDOT:PSS) and can be modulated from an initial conductive state to a dedoped (and less conductive) one when the gate terminal is biased with a positive pulsed voltage. Here, cations from the electrolyte are injected into the PEDOT:PSS channel hindering the conjugation between PEDOT and PSS molecules. In case of high-frequency pulses, the number of ions trapped into the polymer progressively increases, reducing the conductance and determining a short-term depressive (STD)
behavior which resembles the short-term plasticity of the brain.

Lately, PEDOT:PSS-based OECTs have also shown neurotransmitter-mediated long-term plasticity: here the oxidation of dopamine secreted from cells directly interfaced with the neuromorphic platform produces cations which can be retained in the PEDOT:PSS channel over long time, emulating the LTD of biological synapses. These studies demonstrated the ability of OECTs of emulating the learning process of neurons showing long-lasting memory upon periodic stimulation. However, these artificial synapses still lack biomimetic features such as neurotransmitters receptors and postsynaptic regulators which could recapitulate the same architecture of biological synapses promoting the seamless integration of organic neuromorphic devices within the neuronal network. In this context, the use of supported lipid bilayers (SLBs) represents a straightforward approach to implement in vitro platforms with the same lipid composition of cell membranes. Furthermore, these artificial double layers can be functionalized with proteins of interest (i.e., transmembrane receptors and ion channels), preserving their conformation and native environment. Therefore, combining organic neuromorphic devices with SLBs represent a promising approach toward the development of biomimetic in vitro synapses, which present on the surface the same composition of neuronal membrane and display synaptic plasticity.

Here, we investigated the role of synthetic membranes on the short-term plasticity of OECTs where the presence of SLBs might hinder the passage of ions and modulate the conductance of the neuromorphic device. Furthermore, different positionings of the gate electrode were explored to additionally regulate the ion flow through the membrane and enhance the short-term memory. We expect that such biomembrane-based organic neuromorphic transistor could represent a first step toward the implementation of fully biomimetic in vitro systems, which resemble composition and functionalities of neuronal networks and as such, could contribute to unwind the role of synaptic proteins in neurodegeneration.

2. Results and Discussion

Organic neuromorphic devices were fabricated using glass substrates with indium tin oxide (ITO) contacts: here, PEDOT:PSS was deposited through a selective dry etching procedure as described in the Experimental Section. Then, employing a solvent-assisted lipid bilayer (SALB) technique, a phosphatidylcholine-based supported lipid bilayer was assembled from 1-palmitoyl-2-oleoyl-glycero-3-phosphocholine (POPC) that is one of the major component of biological membranes and facilitates the formation of a fluid and homogeneous bilayer.

In fact, we first investigated how the alternating glass/PEDOT:PSS areas could affect the SLB assembly at the device’s surface, its outward architecture, and uniformity. In particular, exploiting the optical transparency of the device, the bilayer fluidity was characterized by means of fluorescence recovery after photobleaching (FRAP), which confirmed the high lateral mobility of lipid molecules with a complete recovery of fluorescence within 5 min. Additionally, fluorescence profiles of SLB formed on glass and PEDOT:PSS show a maximum fluorescence intensity ($F_{\text{max}}$) that is lower when the bilayer is formed on the polymeric film ($F_{\text{max-PEDOT:PSS}} \approx 30$, $F_{\text{max-glass}} \approx 80$). This suggests that the presence of the conductive polymer

![Figure 1](https://example.com/figure1.png)

**Figure 1.** A) FRAP results of POPC membrane on OECT, interfacing PEDOT:PSS substrates. Fluorescence intensity recovery after photobleaching is shown at $t = 0 \text{ s}$ (i), $60 \text{ s}$ (ii), and 5 min (iii). Scale bar: 50 µm. The corresponding normalized fluorescence intensity profiles on PEDOT:PSS substrate are shown in (iv). B) AFM 3D images of the surface morphology of bare PEDOT:PSS (i) and a POPC bilayer formed on PEDOT:PSS (ii). Glass surface instead displays a coarse morphology (iii), whose domains become more defined and sphere-like, after the bilayer formation (iv).
might attenuate the fluorophore activity inserted within the SLB ($\lambda_{\text{em}} = 615$ nm, red fluorescence). In fact, the transmittance of PEDOT:PSS films is maximum for incident light with wavelengths in the blue region of the visible spectrum, while it decreases when moving into the red region.[16] Furthermore, the estimation of the SLB diffusion coefficients on PEDOT:PSS and glass (Table S1, Supporting Information) suggests that the different surface morphologies and rigidities might affect the lipid mobility within the membrane. Here, the area of the SLB which interfaces the PEDOT:PSS film presented higher fluidity, compared to bilayer regions in contact with the rigid glass, indicating a “cushion effect” of the polymer which reduces the frictional coupling between the lipid membrane and the underlying substrate.[12] Furthermore, we estimated the local roughness of the SLB through atomic force microscopy (AFM). As shown in Figure 1B-i, the bare PEDOT:PSS films displayed a characteristic fuzzy surface morphology with sharp-cornered structures[13,14] after the bilayer formation instead, the indented surface texture became smoother, implying a homogeneous lipid covering (Figure 1B-ii).

Notably, the analysis of the root-mean-square (RMS) roughness ($R_q$) highlights a less predominant unevenness of the bilayer structure when formed on PEDOT:PSS ($R_q = 27\%$) than in the case of glass surfaces ($R_q = 31\%$), (Figure 1B-iii,iv), demonstrating how the substrate morphology affects the SLB structure.

A pattern of positive voltage pulses was applied at the gate electrode, emulating an action-potential-like presynaptic stimulus in order to investigate how the capacitive/resistive properties of the SLB might regulate the passage of ions through the membrane and consequently modulate the short memory of the organic neuromorphic device.

Such presynaptic input is characterized by the voltage amplitude ($A$) which drives the ions from the electrolyte into the PEDOT:PSS channel, the pulse width (PW) and the time interval between consecutive pulses ($\Delta t$) (Figure 2A). The latter, in particular, has emerged as fundamental parameter for the modulation of the OECT conductance (Figure S2, Supporting Information): the application of the pulsed input at the gate electrode induces a reversible dedoping of the PEDOT:PSS channel due to cations injected into the bulk of the polymer film.[15] When $\Delta t$ is long enough to allow the charge equilibrium before the application of a second pulse, the polymer is doped to its initial state; high-frequency pulses, instead, force cations to remain trapped in the PEDOT:PSS channel causing a “memory effect,” intended as a cumulative dedoping, resulting in the short-term depressive behavior.[16]

In addition to the variable input signal at the PEDOT:PSS gate electrode, its position in respect to the channel was varied to obtain a planar (Figure 2B) and a top-gate OECT (Figure 2C). Notably, in the case of the planar OECT, the SLB covers both gate and channel, while in the top configuration, the gate surface is not hindered by the membrane.

Figure 2D shows the temporal response of the POPC-functionalized OECT upon the application of a 6-pulses gate bias (fixed amplitude and width) with decreasing $\Delta t$. Here, we expect that the application of a positive voltage at the gate electrode drives $Na^+$ present in the electrolyte into the bulk of the (polymeric) neuromorphic channel which is therefore dedoped to a less conductive state: indeed, cations can compensate the negative charges on the sulfonate groups of PSS which, in the doped state, neutralize the holes of the PEDOT backbone.[17] In particular, when $\Delta t = 7$ s, a reversible dedoping of the PEDOT:PSS channel is observed, inducing a complete

![Figure 2](image_url)
recovery of the initial channel conductance (Figure 2D-i), therefore no memory effect is observed. For $\Delta t = 1 \text{ s}$, the device starts displaying a short-term depressive behavior (i.e., conductance decrease) (Figure 2D-ii) as $\Delta t$ is insufficient to allow cations injected in the polymeric channel to return into the electrolyte before the application of the subsequent pulse. Here, the conductance modulation $\Delta G$ is further enhanced when decreasing $\Delta t$ from 1 to 0.5 s (Figure 2D-iii). Interestingly, in presence of the SLB, the top-gate OECT features higher conductance modulation compared to the planar gate configuration (Figure 2E), while in case of the unfunctionalized OECT, the gate position does not have any relevant effect on the short-term conductance depression (Figure S3, Supporting Information).

Further characterizations on the neuromorphic operation of the POPC-coupled device demonstrate how the presence of the bilayer at the gate electrode (i.e., planar configuration) causes a decrease in the number of cations injected from the gate; on the other hand, in the top-gate OECT, the SLB enhances the charge retention into the PEDOT:PSS channel. These results further corroborate the role of the top-gate electrode in complementing more effectively the STD compared to the planar gate configuration (Figure S4, Supporting Information).

The short-term depressive behavior of the OECT is regulated by the migration of ions from the electrolyte into the PEDOT:PSS channel: such mechanism strongly depends on the response time ($\tau$) of the OECT, a crucial parameter which describes the ability of the device in switching from the doped (on-state) to the dedoped state (off-state) and vice versa. The movement of ionic species is usually modeled through an equivalent ionic resistor–capacitor (RC) circuit where the resistor and the capacitor are connected in series (Figure 3A). Here, the resistive part accounts for the ion flow through the electrolyte, while the capacitance models the accumulation of charges inside the bulk of the conducting polymer. Therefore, the electrochemical doping/dedoping of the PEDOT:PSS channel can be described as the charge/discharge of the equivalent RC circuit and is characterized by a time constant ($\tau$). In particular, such parameter also refers to the time needed to charge the

Figure 3. A) Schematics of the electrical circuit modeling ionic conduction in OECTs where $R_S$ takes into account the ion flow through the electrolyte and $C_{CH}$ describes the accumulation of cations within the polymeric channel. B,C) Channel current response after the application of a voltage pulsed input with $\Delta t = 7 \text{ s}$ for planar (B) and top-gate (C) OECTs with and without a biomembrane. Both graphs show how, in the presence of the bilayer (pink curve in the graph in (B) and yellow curve in the graph in (C)), the pulse shape is smoother on the edges, suggesting slower doping/dedoping of the PEDOT:PSS channel. The value of $\tau_{OECT-SLB}$ was calculated setting a threshold at the 63% of the maximum channel current (corresponding to 63% of charge of the RC circuit model) represented by the dashed horizontal line in the graphs in (B) and (C). D) Mean values of $\tau_{OECT-SLB}$ ($N = 3$) calculated for planar and top-gate configurations with and without the lipid bilayer: here, the top-gate OECT functionalized with the POPC bilayer displays the highest value of $\tau_{OECT-SLB}$, which confirms the hindered passage of ions through the membrane.
equivalent circuit from the discharged state to the 63.2% of its maximum charge, or equivalently, as the time needed to discharge about 37% of its fully charged state.[5] As previously reported, the presence of a SLB increases the response time \( \tau \) of the OECT (i.e., slower switching from on- to off-state), hindering the ion movement toward the PEDOT:PSS channel.[9]

Here, to perform the computation of \( \tau \) in the biomimetic synapse (named as \( \tau_{\text{OECT-SLB}} \)), a voltage step was applied at the gate electrode while monitoring the channel current: as shown in Figure 3B,C, in presence of the POPC membrane, the pulse shape of the output signal is smoother compared to the ones obtained for the unfunctionalized OECT, for both planar (Figure 3B) and top configurations (Figure 3C), suggesting a delay in the output current caused by the artificial membrane.

The numerical value of \( \tau_{\text{OECT-SLB}} \) was obtained setting a threshold in the channel current during postprocessing to identify the time needed to charge the neuromorphic channel up to the 63% (Figure 3B,C): here, both planar and top-gate biomembrane-based OECTs revealed higher time response compared to their unfunctionalized counterpart. Interestingly, in presence of the SLB, the top-gate device displays higher values of \( \tau_{\text{OECT-SLB}} \) compared to the planar gate one (Figure 3D).

This result suggests that the position of the gate electrode might elicit diverse ionic paths through the bilayer where cations are forced to cross the SLB barrier to reach the PEDOT:PSS channel, causing a significant delay in the top-gate OECT response (\( \tau \) increases of 45%). On the other hand, in the planar gate device, ions might move in the aqueous cushion placed at the interface between the lipid bilayer and the PEDOT:PSS channel where the hindrance caused by the SLB slightly affects the response time of the device (\( \tau \) increases of 25%).

The time constant \( \tau \) describing the ionic conduction in OECTs can be exploited to modulate the neuromorphic properties of artificial synapses as the electrical RC circuit can be fully charged/discharged after \( 5 \tau \) (Text S1, Supporting Information). Furthermore, as the STD behavior of OECTs can be enhanced decreasing the time interval between pulses (\( \Delta t \)), varying \( \Delta t \) as multiple values of \( \tau_{\text{OECT}} \) (from \( 1 \tau_{\text{OECT}} \) to \( 5 \tau_{\text{OECT}} \)) reduces the conductivity of the PEDOT:PSS channel, switching the polymer from fully to partial dedoped state.

In particular, prior to the formation of the SLB, high-frequency pulses (i.e., \( \Delta t = 1 \tau_{\text{OECT}} \)) enabling only 37% of ionic discharge[20] elicits a conductance modulation of 1%, while a pulsing regime set with \( \Delta t = 5 \tau_{\text{OECT}} \), allowing charge equilibrium, induces a conductance decrease of only 0.5% (Figure S5, Supporting Information). Here, to investigate if the increased response time (i.e., slower doping/dedoping) of the biomembrane-based OECT affects the neuromorphic functions of the device, a pattern of voltage pulses with \( \Delta t \) equal to multiple values of \( \tau_{\text{OECT-SLB}} \) was applied at the gate electrode. Figure 4 shows the STD behavior of the POPC-coated device when \( \Delta t \) is equal to \( 1 \tau_{\text{OECT-SLB}} \) and to \( 5 \tau_{\text{OECT-SLB}} \), both for the planar and top-gate architectures (see Figure S6 in the Supporting Information for data relative to \( 2 \tau_{\text{OECT-SLB}}, 3 \tau_{\text{OECT-SLB}}, \) and \( 4 \tau_{\text{OECT-SLB}} \)). As expected, enhanced STD behavior is observed when \( \Delta t = 1 \tau_{\text{OECT-SLB}} \) (Figure 4A-i,B-i), while the gate bias with \( \Delta t = 5 \tau_{\text{OECT-SLB}} \) induces lower conductance modulation (Figure 4A-ii,B-ii). Also in this case, the highest memory effect is observed in case of the top-gate OECT (Figure 4C). Graph in Figure 4C reports also \( \Delta G \) obtained for the unfunctionalized OECT, with \( \Delta t \) equal to multiple values of \( \tau_{\text{OECT}} \) (output channel conductance reported in Figure S5 in the Supporting Information). Interestingly, the POPC-coated device exhibited amplified short-term memory for both gate architectures and for all voltage patterns (\( \tau, 2 \tau, 3 \tau, 4 \tau, \) and \( 5 \tau \)). This result further confirms that the presence of the SLB hinders the ionic discharge of the PEDOT:PSS channel, therefore enhancing neuromorphic behavior. As mentioned above, \( \Delta t = 1 \tau \) should elicit a discharge of the polymeric channel of 37% (according to the RC model circuit); however, the higher \( \Delta G \) values observed for the biomembrane-functionalized OECT suggest that here \( \Delta t = 1 \tau \) induces lower ionic discharge (Figure 4D,E).

To further investigate the different ionic paths influenced by the presence of the biomembrane, electrochemical impedance spectroscopy (EIS) was performed, where a Ag/AgCl reference electrode was placed above the SLB, while the neuromorphic channel was used as working electrode (see the Experimental Section). Here, the bilayer induces a significant shift of the curve and the appearance of a semicircle at high frequencies (Figure S7A, Supporting Information). In addition, the frequency response of the neuromorphic device was evaluated by using the PEDOT:PSS gate and channel as reference and working electrodes, respectively. In this case, the presence of the bilayer causes a variation in the slope of the Nyquist plot curve (Figure S7B, Supporting Information). The Nyquist curves of EIS and planar impedance were then fitted with the equivalent electrical circuit shown in Figure S7C (Supporting Information), describing the electrolyte resistance (\( R_\text{e} \)), the electrical double layer (\( R_{\text{DL}} \) and \( C_{\text{DL}} \)), and the conducting polymer nonidealities (\( Z_{\text{m}} \))[21] the analysis revealed that the presence of the bilayer elicits an increase of \( R_{\text{DL}} \) (Table S2, Supporting Information), confirming the SLB ionic hindrance effect.

Interestingly, the SLB resistance \( R_{\text{DL}} \) found from the EIS doubles the one calculated from the planar frequency measurement: this result together with the charge analysis data shown in Figure 4D,E suggests that in presence of a top electrode (i.e., Ag/AgCl in EIS or PEDOT:PSS top gate in the OECT configuration), the biomembrane behaves like a “resistive barrier” to the ion flow, and in the operation of the neuromorphic device, such barrier hinders the ionic discharge of the PEDOT:PSS channel enhancing the percentage of charges retained in the organic polymer and therefore the conductance modulation. Additionally, the EIS analysis is also in good agreement with the time constant \( \tau_{\text{OECT-SLB}} \) values: the top-gate configuration is indeed characterized by a larger response time due to a more resistive ionic path. It is important to note that even though the EIS data disclose the same increased resistance, there are no models able to directly correlate the frequency response of the device to the OECT modulation. In conclusion, although a more sophisticated electrical modeling might be required to describe the doping/dedoping of OECTs coupled with lipid bilayers, the enhanced conductance modulation observed in the POPC-coupled neuromorphic device validates the role of biomembranes as modulators for the short-term depressive behavior in artificial synapses.
3. Conclusion

We have investigated how the presence of artificial biomembranes influences the neuromorphic behavior of OECTs, with particular focus at the short-term plasticity. In particular, the channel conductance depression was evaluated exploiting a planar gate electrode and a top-gate device. First of all, the formation and morphology of the bilayer were investigated by means of FRAP and AFM which confirmed a fluid homogeneous structure. The investigation of the electrical behavior of the SLB was conducted by applying a pattern of high-frequency pulses at the gate electrode: here, the top-gate biomimetic synapse displayed higher STD compared to the corresponding planar architecture. This result is further supported by EIS and frequency response measurements where the biomembrane exhibited the same capacitive behavior but with higher resistivity in case of the electrochemical characterization. Such resistive behavior together with the enhanced time response and STD found for the top-gate architecture suggests that the lipid bilayer might induce different conducting paths for the cations present in the electrolyte according to the gate electrode configuration. Although the ionic pathways underlying the doping/dedoping mechanism require further investigation, it is clear that biomembrane-based OECTs exhibit enhanced short-term depressive behavior, paving the way toward the implementation of synapses resembling OECTs, a new class of devices able to mimic both functionalities and composition of neurons, therefore providing reliable in vitro models for the investigation of neurodegenerative disorders involving synaptic plasticity loss.
4. Experimental Section

**OECT Fabrication:** OECT fabrication was carried out on 25 × 25 mm wide glass substrates, with four 10 × 10 mm ITO squares at each corner (Xinyan Technology Ltd.). PEDOT:PSS (Hereaus, Clevios PH1000) aqueous solution was prepared by adding 5 vol% ethylene glycol (Sigma-Aldrich), 1 vol% 3-glycidoxypropyl)trimethoxysilane (Sigma-Aldrich), and 0.02 vol% dodecylbenzenesulfonic acid (Sigma-Aldrich). Glass substrates were treated with oxygen plasma (Tecno-Service) for 2 min at 80 W. Subsequently, the PEDOT:PSS solution was spin-coated on the substrate at 2000 rpm for 2 min. Thermal annealing at 140 °C on hot plate was performed. OECT PEDOT:PSS gate and channel were patterned through oxygen plasma dry etching technique for 15 min, at 100 W. The masks used to define the transistor geometry during the etching procedure were made of poly(dimethylsiloxane) (PDMS), mixed in ratio 10:1 w/w with a cross-linker, and cured at 80 °C for 1 h (PDMS, Sylgard 184). The outcomes of the etching procedure were two symmetrical PEDOT:PSS stripes 7 × 17 mm wide, 2 mm apart. Then, the devices were immersed in milli-Q water, for 1 h, to allow for the complete swelling of the PEDOT:PSS. A PDMS microfluidic channel 17 mm long, 4 mm wide, and 0.4 mm high was created and attached on the top of the device through a two component glue (Picodent Twinsil).

**Lipid Preparation:** The lipid mixture consisted of 100 mol% of POPC (Avanti Polar Lipids), and 0.5% (mol/mol) of Texas Red 1,2-dihexadecanoyl-sn-glycero-3-phosphoethanolamine triethylammonium (Thermo Fisher, USA) salt as a fluorescent probe. The lipids were mixed in chloroform at a desired concentration. The chloroform was first evaporated under a nitrogen stream, and then in a desiccator under vacuum for 2 h. The resultant lipid thin film was rehydrated with a mixture of 70% v/v of water to a final concentration of 0.5 mg mL⁻¹ as final concentration.

The solution was gently vortexed and then sonicated on ice for 25 min. Finally, the suspension was extruded 15 times through a 100 nm pore polycarbonate membrane (Sigma-Aldrich, USA) by using a mini extruder (Sigma-Aldrich, USA). The lipid vesicle solution was stored at 4 °C.

**Bilayer Formation:** Lipid bilayer was obtained using the SALB method, where the SLB was formed into a microfluidic channel by means of a solvent exchange procedure.[19] The microfluidic chamber was made of polydimethylsiloxane, mixed in ratio 10:1 w/w with its cross-linker, cured at 80 °C for 1 h (PDMS, Sylgard 184).

Prior the membrane formation, the OECT was treated with oxygen plasma (Diener electronic, Germany) at a pressure of 1 mbar with power of 20 W for 2 min. The channel was placed covering both gate and channel of the OECT. The liposome solution was diluted with isopropanol/water mixture (3:7 v/v) to a final concentration of 0.5 mg mL⁻¹ and incubated for 30 min inside the chamber. A buffer solution (10 × 10⁻³ M Tris, 100 × 10⁻³ M NaCl, pH 7.5) was delivered into the microfluidic channel with a flow rate of 50 µL min⁻¹ for 2 h to remove excess vesicles while forming the bilayer.

**FRAP:** The fluidity of the bilayer was evaluated using the FRAP technique with a Leica TCS SPS gated with stimulated emission depletion (STED) microscope (Leica Microsystems, Germany) equipped with a 25× water immersion objective. A 20 µm wide circular spot was bleached by 114 mW 592 nm laser beam for 1.3 s. The recovery of the bleached spot was then monitored for 5 min. The fluorescence intensity of the spot was measured and normalized to a reference spot bleached by 114 mW 592 nm laser beam for 1.3 s. The recovery of the fluorescence intensity was monitored with a 25× objective at a rate of 1/2 for 2 h to remove excess vesicles while forming the bilayer.

The authors declare no conflict of interest.

Supporting Information

Supporting Information is available from the Wiley Online Library or from the author.

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Conflict of Interest

The authors declare no conflict of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Keywords

biohybrid synapses, organic electrochemical transistors, organic neuromorphics, supported lipid bilayers
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