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Modeling homosynaptic and heterosynaptic plasticity with a single neuromemristive synapse

Zubaer Ibna Mannan^{a,*}, Sami Azam^{b,*}, Ram Kaji Budhathoki^c, MD Nur Alam^d, Hyongsuk Kim^{e,*}

^a Department of Computer Science and Engineering, East West University, Jahurul Islam Ave., Dhaka 1212, Bangladesh

^b Faculty of Science and Technology, Charles Darwin University, NT 0810, Australia

^c Department of Electrical and Electronics Engineering, School of Engineering, Kathmandu University, Dhulikhel, Kavre, 45200, Nepal

^d Department of Artificial Intelligence, Kyungdong University Global, Geosong, Gangwon-do 46(4)-24764, the Republic of Korea

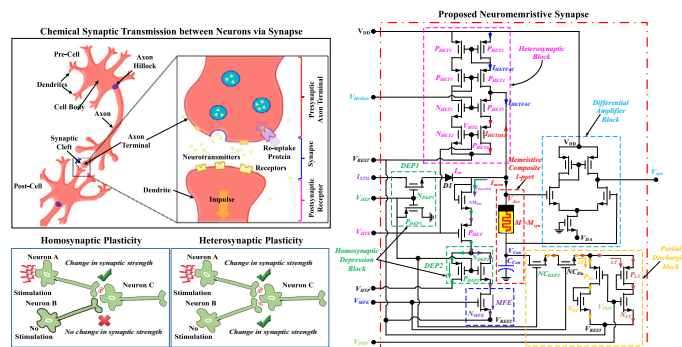
^e Division of Electronics Engineering, and Intelligent Robot Research Center, Jeonbuk National University, Jeonju, Jollabuk-do 567-54896, the Republic of Korea

HIGHLIGHTS

- An analog neuromemristive synapse is designed to mimic the bio-realistic characteristics of neurons, synapses, and synaptic plasticity. The artificial synapse uses a single memristor to support homo- and hetero- synaptic long-term and short-term plasticity.
- It imitates the homeostatic, modular input specificity, and associativity roles of the heterosynaptic plasticity. It also mimics homosynaptic short-term facilitation (STF) and depression (STD), long-term potentiation (LTP) and depression (LTD), as well as reuptake processes and strong stimulation phenomena.
- This artificial synapse can be served as a potential substitute for in-vivo and in-vitro analyses in academia. In addition, the proposed synapse functions in both volatile and nonvolatile modes, evincing it suitable for applications in Spiking Neural Networks (SNNs) to support Hebbian, anti-Hebbian, and associative learning paradigms.
- Particularly, it overcomes the limitations inherent in graphene, liquid crystal carbon nanotube composites, subthreshold analog and transistor-based CMOS neural

GRAPHICAL ABSTRACT

A neuromemristive synapse is developed that can emulate both homo- and heterosynaptic plasticity, showcasing the homeostatic and modular input-dependent roles of heterosynaptic plasticity, along with homosynaptic long-term and short-term potentiation and depression, and functioning effectively within brainwave frequency ranges.



* Corresponding authors.

E-mail addresses: zubaer.mannan@ewubd.edu (Z.I. Mannan), Sami.Azam@cdu.edu.au (S. Azam), ram.budhathoki@ku.edu.np (R.K. Budhathoki), na@kduniv.ac.kr (MD Nur Alam), hskim@jbnu.ac.kr (H. Kim).

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circuits, and traditional Von Neumann architectures.

- The power- and area-efficient artificial neuromemristive synapse can be ported in miniature CMOS ICs, which paves the way to build artificial neuronal or neural networks that exhibit human brain-like intelligence.

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ABSTRACT

Introduction: Efforts are being made to design a brain-like intelligence due to its robustness, synaptic modification (i.e., learning and memory), analog synaptic multiplication, multi-state storage, ultra-low power consumption, and parallel computation. However, current bioelectronic and biomedical technologies have yet to fully replicate brain-like intelligence. In particular, devices that can emulate both homosynaptic and heterosynaptic plasticity remain extremely limited.

Objectives: This work presents a neuromemristive synapse capable of replicating key biological features, including homosynaptic and heterosynaptic plasticity. The proposed synapse uses a memristor, a promising candidate for achieving bio-realistic features of synapse due to its low power consumption, multi-state operation, analog behavior, high data storage durability, and CMOS compatibility.

Methods: The artificial synapse is designed as a composite 1-port structure consisting of a memristor (M) and a controlled capacitor (CCon). The memristor is responsible for emulating synaptic plasticity in response to distinct brainwave patterns, while the capacitor modulates the discharge rate through the memristor. During the active input phase (synaptic potentiation), the composite 1-port charges up. During the inactive phase (synaptic depression), CCon governs the discharging of the memristor, enabling full or partial discharging through memory fading or homosynaptic and heterosynaptic depression pathways.

Results: We designed the proposed synapse in SPICE and validated its bio-functionalities through various simulations. The proposed synapse demonstrates low power consumption and replicates key neurobiological processes for learning and memory such as heterosynaptic homeostasis, modular input specificity, associativity, and homosynaptic long-term and short-term potentiation and depression (LTP, LTD, STF, STD), along with memory fading effect (MFE), and strong stimulation (SST).

Conclusion: The proposed synapse bio-realistically mimics synaptic plasticity, neurotransmitter dynamics, and neuronal responses. Implemented using off-the-shelf components, it supports both volatile and non-volatile modes, making it suitable for CMOS integration. This enables advancements in spiking neural networks, brain function analysis, and scalable neuromorphic computing systems.

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Introduction

Electronic circuits that mimic biological mechanisms are incredibly efficient, robust, and have enormous parallel processing capabilities [1]. The development of a brain-like intelligence is currently achievable because of recent developments in bioelectronics and neuromorphic engineering [2,3]. These bio-inspired neuromorphic circuits are considered to be the next generation of computing platforms. They surpass traditional Von-Neumann architectures because of their high parallel processing, low power consumption, and high adaptability [4]. Recent advancements in nano-scale CMOS technologies have made it possible for researchers to develop large-scale neuromorphic circuits using very large-scale integration (VLSI) hardware. In recent years, researchers have used different technologies to develop neuromorphic devices that include CMOS [5,6], Subthreshold CMOS [7], OxRAM [8], Switched-Capacitor (SC) [9], Spintronic [10], Graphene Transistor [11], Barristor [12], Liquid Crystal Carbon Nanotube (LC-CNT) Composites [13], and Memristor [14–25]. Among all of these elements, memristor is considered one of the most preeminent elements for designing neuromorphic integrated circuits because it works like a biosynapse and has low energy consumption, multiple-state operation, great scalability, higher durability for storing data, and CMOS compatibility [15,26,27]. Moreover, CMOS is energy inefficiency in analog synaptic operation [28], Subthreshold CMOS suffers with low operating speed [29], OxRAM requires high-voltage electro-

forming to initialize the switching medium [30], and Switched-Capacitor (SC) is prone to leakage and device variation [31]. In addition, Spintronic Devices exhibits stochastic switching [32], Graphene Transistor lacks of a band gap (i.e., zero bandgap) which makes it unsuitable for low-power neuromorphic circuits [33], Barristor suffers with manufacturing scalability, variability, and integration challenges [34], and LC-CNT Composites relies on physical alignment-assisted switching, which suffers from mechanical instability and inconsistent behavior under scaling [13]. Therefore, numerous research activities are focused on neuromorphic computation using the memristive system.

In this paper, an artificial biosynapse was developed using a composite 1-port of a memristor (M) and a controlled capacitor (CCon). The proposed neuromemristive synapse impersonates the physiological acts of homo and hetero synaptic plasticity in accordance with the distinct brain states thereby correlating to a particular frequency of the brainwaves. The memristance and voltage across the memristor represent the artificial synaptic strength and synaptic voltage, respectively. The CCon capacitor controls the rate of discharging through the memristor (M). During homo or hetero synaptic potentiation, the composite 1-port is charging in the active cycle of homo or hetero synaptic input stimuli. However, during the inactive cycle of stimulations, the CCon capacitor forces the memristor to either fully or partially discharge through M. Similar to biological synapses, the artificial synapse distinguishes between homosynaptic short-term (STF and STD) and long-term

Table 1
Abbreviations of the Synaptic Terms.

Terms	Abbreviations	Terms	Abbreviations
Long-Term Potentiation	LTP	Non-plastic Synaptic Response	NSR
Long-Term Depression	LTD	Strong Stimulation	SST
Short-Term Facilitation	STF	Heterosynaptic Facilitation	HTE_FAC
Short-Term Depression	STD	Heterosynaptic Depression	HTE_DEP
Memory Fading Effect	MFE	Memristive Synaptic Efficacy	M _{Syn}

(LTP and LTD) plasticity, shown in Table. 1, based on repetition time of the input cycle. The proposed synapse also features separate pathways through M and C_{con} to facilitate the different synaptic weakening rates for homo and hetero synaptic depressions and reuptake processes.

The proposed neuromemristive synapse is beneficial than existing state-of-the-art artificial neuromorphic synapses in a number of ways. Neuromorphic excitatory synapses shown in [5] need twice as much hardware as compared to our proposed synapse in order to support only STDP (spike-timing-dependent plasticity). The self-sufficient proposed model is more compatible and uses less space than the digitally controlled neuromorphic circuit in [6], which needs more circuits to implement synaptic plasticity. The sub-threshold CMOS iono-neuromorphic model in [7] requires nonvolatile digital storage to store synaptic modification and is unable to exhibit short-term synaptic plasticity compared to our proposed synapse. To imitate the STP and LTP phenomena, the proposed neuro-memristive synapse requires only a single memristor, unlike the OxRAM synapse in [8], which requires 10 and 20 OxRAM devices, respectively. Moreover, the proposed synapse is bio-realistically more effective, energy-efficient, and area-efficient than the switched-capacitor-based conductive synapse in [9], which requires multiple switches, a couple of capacitors, and an amplifier to implement a single synaptic conductance. Both the hybrid spintronic-CMOS synapse in [10], the twisted bilayer graphene transistor synapse in [11], and the CMOS-memristive synapse in [19] exhibit only STDP mechanisms. The synaptic baristor in [12] exhibits LTP, LTD, STP, and STD plasticity but doesn't exhibit heterosynaptic plasticity. The liquid crystal-carbon nanotube (LC-CNT) synapse in [13] and the neuro-transistor in [20] are unable to exhibit homo and hetero synaptic depressions, whereas the solid-state PEN-based memristive synapse in [18] only demonstrates heterosynaptic plasticity. The memristive synapses in [21–23] are unable to exhibit heterosynaptic plasticity. In contrast to these synapses, the proposed neuro-memristive synapse demonstrates both homo and hetero synaptic facilitation and depression, as well as memory-fading effects. Unlike the mathematical [16] and macro [17] models of the memristive synapse, our proposed synapse is implemented in a circuit with off-the-shelf components. In addition, the proposed neuro-memristive synapse can operate at different frequency range and pulse width (1 ~ 3 ms) of the brainwaves. In contrast, the WSe₂-based memtransistor in [24] and LiNbO₃ memristor in [25], which required 3 V ~ 20 V and ±4 V pulse amplitude, respectively, with a 50 ms pulse width.

The main contribution of this paper is the design of an analog neuromemristive synapse that mimics bio-realistic characteristics of neurons, synapses, and synaptic plasticity. The artificial synapse uses a single memristor to support homo- and hetero- synaptic long-term and short-term plasticity. It imitates the homeostatic, modular input specificity, and associativity roles of the heterosynaptic plasticity. It also mimics homosynaptic short-term facilitation (STF) and depression (STD), long-term potentiation (LTP) and depression (LTD), as well as reuptake processes and strong stimulation phenomena. This artificial synapse can be served as a poten-

tial substitute for in-vivo and in-vitro analyses in academia. In addition, the proposed synapse operates in both volatile and non-volatile modes. This makes it suitable for Spiking Neural Networks (SNNs), where it can support Hebbian, anti-Hebbian, and associative learning paradigms. Particularly, it overcomes the limitations of graphene, liquid crystal carbon nanotube composites, sub-threshold analog and transistor-based CMOS neural circuits, and traditional Von Neumann architectures. This advantage allows it to be integrated into miniature CMOS ICs, paving the way for artificial neural networks that exhibit human brain-like intelligence.

The rest of the paper is organized as follows: Section 2 describes the bio-synapse and its plasticity, and Section 3 discusses the operation of the proposed neuro-memristive synapse. Section 4 presents the simulations and results, followed by concluding remarks in Section 5.

Synapse, plasticity and brainwaves

Neurons or nerve cells are the building blocks of the nervous system that transmit information electrical-chemically via synapse. Synapse, a junction between two neurons, plays a crucial role in transmitting signals between neurons, allowing for the integration of information and the coordination of various brain functions. Synapse allows the release of neurotransmitters (such as acetylcholine, dopamine, serotonin, glutamate, and so on) from the presynaptic neuron into the synaptic cleft,¹ shown in Fig. 1(a), where they bind to receptors on the postsynaptic neuron, shown in Fig. 1(b), initiating a series of opening and closing of ion channels [35,36]. The release of different types of neurotransmitters have an impact on the ion channel behavior and the outcomes of postsynaptic cells. For instance, excitatory neurotransmitters such as acetylcholine or glutamate depolarize postsynaptic cells, increasing the probability of postsynaptic firings, a phenomenon known as synaptic potentiation, while inhibitory neurotransmitters such as GABA or dopamine suppress post-firings by hyperpolarizing the postsynaptic cell, leading to synaptic depression [37]. When the postsynaptic neuron depolarizes, its membrane potential increases from resting membrane potential (typically -70 mV) and when it reaches the threshold (typically -55 mV), it generates an all-or-none electrical impulse, namely action potential (AP). Generation of such action potentials in synaptic transmission is proportional to the probability and pattern of neurotransmitter diffusion from presynaptic neuron and the receptor sensitization of postsynaptic neuron. The continual diffusion of neurotransmitters increases the synaptic efficacy or strength of the synapse between two neurons [38,39]. However, synapse utilizes the reuptake process to remove the neurotransmitters from the synaptic cleft to regulate the strength of synapse or to terminate the synaptic transmission of signals between neurons. Reuptake process, shown in Fig. 1(b), absorbs the diffused neurotransmitters from synaptic cleft through neurotransmitter transporters and brings back to presynaptic neurons. The reuptake

¹ Synaptic cleft, shown in Fig. 1(b), is a small gap between pre- and post-synaptic neurons (≈ 20 nm wide) and forms a junction between neurons.

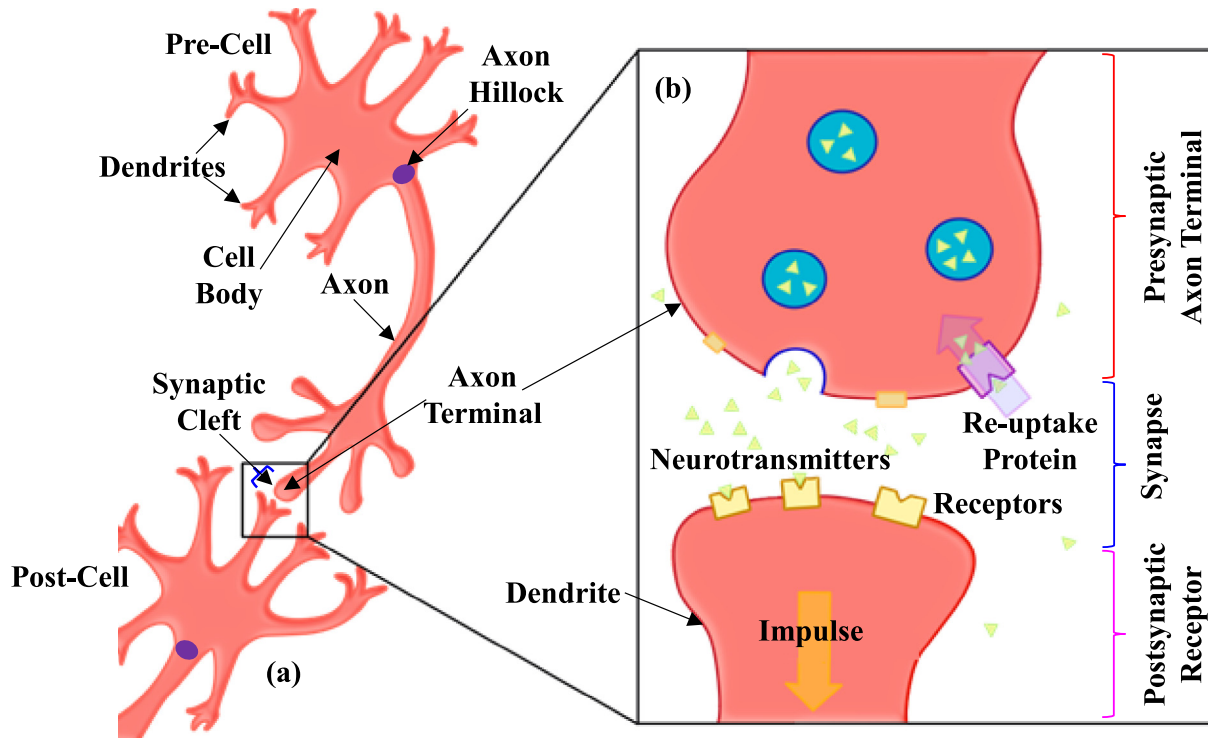


Fig. 1. Schematic illustration of neuron–neuron communication and synaptic structure. (a) It depicts the primary components of a neuron, including the cell body, dendrites, axon, and axon terminal. (b) It provides a detailed cross-section of a synapse, illustrating the process of synaptic transmission, neurotransmitter release, receptor binding, and reuptake.

process plays a crucial role in regulating the concentration of neurotransmitters in the synaptic cleft, thereby influencing the strength (*i.e.*, decreasing the synaptic strength) and duration of synaptic transmission [37]. The physiological attributes of synaptic depression caused by reuptake and inhibitory neurotransmitters should be addressed separately. This is because inhibitory neurotransmitters hyperpolarize the post-cell, which results in post-membrane potential being reduced to $-110\text{ mV} \sim -150\text{ mV}$, whereas in the reuptake process, post-membrane potential remains same as resting potential. The reuptake process can be related to the memory fading effect (MFE) in an electronic circuit [21,22].

Synaptic plasticity is the biological mechanism by which specific patterns of synaptic activities lead to changes in synaptic

strength or efficacy. It is the ability of synapses to increase or decrease its efficacy and is thought to be one of the most important neurochemical foundations of learning and memory [37]. Depending on the input specificity, synaptic plasticity of a synapse is characterized in two types: homosynaptic plasticity and heterosynaptic plasticity, as shown in Fig. 2. Homosynaptic plasticity is input-specific or associative, *i.e.*, synaptic efficacy changes only at post-synaptic targets specifically stimulated by a presynaptic neuron. It is a form of synaptic plasticity where changes occur specifically at the synapses that are activated, as opposed to neighboring or unrelated synapses, as shown in Fig. 2(a). In contrast, heterosynaptic plasticity, shown in Fig. 2(b), represents the ability of the synapse to modify its strength as a result of activity in another neighboring

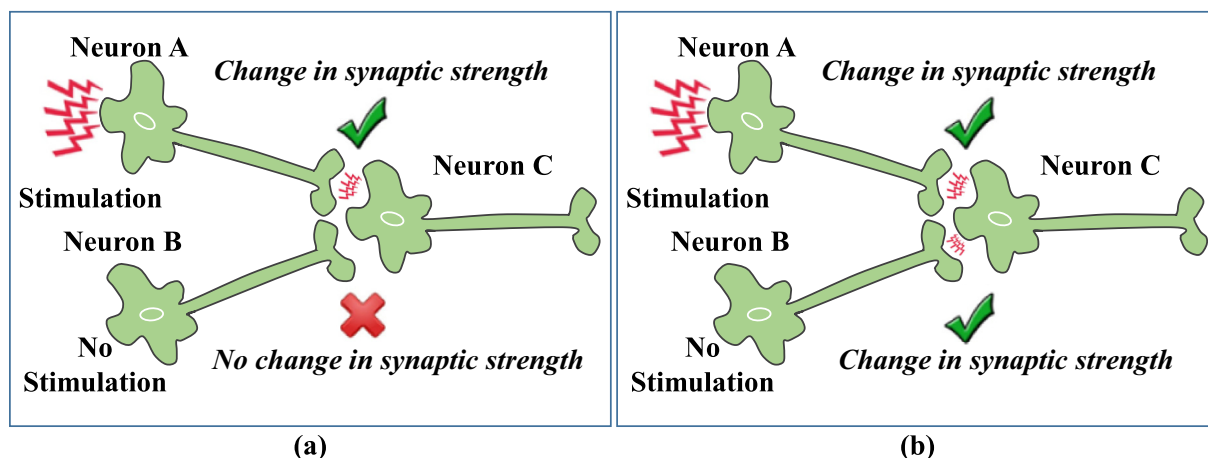


Fig. 2. (a) Illustration of homosynaptic plasticity where concurrent firing in Neuron A only induces changes in the synaptic connection from Neuron A to Neuron C. (b) A model of heterosynaptic plasticity, where concurrent firing in Neuron A induces a change in the synaptic connection from Neuron B to Neuron C.

neuron or pathway [40–42]. The homosynaptic and heterosynaptic plasticity can be categorized in two types: potentiation or facilitation and depression, based on synaptic modification of the synapse. Moreover, depending on the durability of synaptic modification, plasticity is categorized into two types: short-term plasticity (short-term facilitation (STF) and short-term depression (STD)) and long-term plasticity (long-term potentiation (LTP) and long-term depression (LTD)). STF occurs when the repeatedly stimulated presynaptic neuron releases neurotransmitters at a faster rate, which causes a temporary enhancement in the strength of a synapse between neurons [37]. However, STD induces with the depletion of neurotransmitters and their vesicles despite the presence of presynaptic stimulations, thus weakens the strength of a synapse. Both STF and STD occur over a shorter period of time, lasting only tens of milliseconds to a couple of minutes [37,43]. LTP is a type of activity-dependent plasticity that persistently strengthens a synapse based on recent patterns of synaptic activity, thus producing a long-lasting increase in signal transmission between two neurons. LTD persistently weakens specific synapses in order to build a productive use of synaptic strengthening caused by LTP, and stabilizes neuronal circuit to facilitate encoding of new information [37,44]. Both LTP and LTD last hours or longer based on the patterns of stimulation. Homosynaptic STF, STD, LTP, and LTD are input specified which means that if a synapse has these plasticity traits, it doesn't spread to other synapses and only propagates synaptic transmission between two succeeding neurons [45]. In contrast, heterosynaptic LTP and LTD are associative. Because weak stimulation of a single pathway alone is not enough to induce LTP, continuous stimulation of another pathway can induce LTP in both pathways [46]. Heterosynaptic LTD weakens a synapse independently, despite the activity of the pre- or post-synaptic neurons, as a result of a modulatory interneuron firing to stabilize the synaptic transmission [37,44]. Heterosynaptic LTD (which might occur along with homosynaptic LTP) has the ability to sustain synaptic competition as well as balancing plasticity modifications [42]. Therefore, long-term and short-term plasticity are considered as one of the major neuromorphic foundation of learning and memory in neuronal communication as memories are thought to be encoded by synaptic modification [41,44].

Homosynaptic depression weakens the strength of synapse more rapidly than heterosynaptic depression, and the homosynaptic reuptake process occurs after a synapse exhibits LTP or STF. In homosynaptic depression, the rapid depletion of neurotransmitter vesicles in the presynaptic terminal causes synapses to weaken swiftly. This is due to the direct response to a synapse caused by repeated presynaptic activation, which causes a quicker onset of depression [47,48]. In contrast, heterosynaptic depression tends to be mediated by diffusible factors or modulators that act indirectly on the synaptic strengths of the multiple synapses and, therefore, operates on a slower timescale [49,50]. The reuptake process after LTP or STF is slower because it involves more complicated cellular processes, such as recycling vesicles, the re-synthesis of neurotransmitters, and membrane trafficking. This makes the weakening of the synapses slower than homosynaptic depression [47,51]. Additionally, homosynaptic depression causes the synapse to deplete essential resources (like neurotransmitters or calcium ions) more quickly, which in turn accelerates fatigue [52]. In contrast, heterosynaptic depression indirectly deplete resources at the affected synapse and homosynaptic reuptake and recovery are mediated by transporter proteins, vesicle reformation, and reloading of neurotransmitters, which take time and further slow-down the synaptic weakening process [53].

Heterosynaptic plasticity modifies the synaptic strengths between adjacent synapses depending on the overall network activity and contributes to the homeostatic regulation. When a

synapse exhibits long-term potentiation (LTP), neighboring synapses experience long-term depression (LTD) to prevent over-excitation and subsequent neuron excitotoxicity [53,54]. However, in the case of modular input-specific roles, heterosynaptic plasticity facilitates synaptic competition by allowing increased activity or allocating specific pathways to reinforce (i.e., strengthening the synapse) the active synapse while inhibiting less active ones. This synaptic pruning of synapses refines connectivity and facilitates selective and effective information processing, which is especially important during development and adaptive learning [55,56].

The interaction between homo- and hetero- synaptic plasticity emphasizes the complexity of synaptic modification. Heterosynaptic plasticity enables the modulation of multiple synaptic pathways, in conjunction with highly active synapses (homosynaptic plasticity), thereby simplifying the information storage process of the brain. Consequently, this leads to an increase in brain connections, which in turn increases the complexity of network interactions necessary for memory encoding, association, and storage. In addition, during associative learning, activating two different pathways simultaneously (due to homosynaptic and heterosynaptic connections) can lead to the potentiation of one synapse while also altering the surrounding synaptic environment. Heterosynaptic changes are important for associative learning because they assist in embedding information in distributed networks and form an association between a conditioned stimulus and an unconditioned stimulus [57,58].

Neurons produce electrical pulses, namely action potentials, when activated individually (initiating homosynaptic plasticity) or collectively (initiating heterosynaptic plasticity) in synaptic transmission. These synchronized, rhythmic, or repetitive electrical pulses that result from mass or individual neural communication are called brainwaves. The CNS recognizes five kinds of brainwaves with different frequency bands: delta (δ), theta (θ), alpha (α), beta (β), and gamma (γ) [59]. Each brainwave, shown in Table 2, contributes to different neural activities (known as brain states) through synaptic modification. The mammalian hippocampal has a neutral frequency of 10 Hz, at which repetitive stimulation does not modify synaptic efficacy. However, higher-frequency bands of brainwaves induce synaptic modification for memory and learning [60]. Therefore, the brainwaves and their corresponding brain-states for regular and plasticity-dependent synaptic transmission are different. For instance, gamma brainwaves, or the coupling of theta and gamma brainwaves, facilitate the timing and synchronization of neuronal firing, thereby enhancing the efficacy of synaptic transmission and promoting long-term plasticity [61]. However, identifying the exact brainwaves responsible for specific types of synaptic plasticity is difficult and challenging due to the complexity of interacting brain rhythms [62], task and region specificity of brainwaves and plasticity [63], state-dependent brain activity [64], diverse plasticity mechanisms [65], technical limitations in measurement tools [66], and plasticity beyond synapses [67]. Therefore, we used delta, theta, and alpha ($f \leq 10$ Hz) brainwaves to show the normal synaptic response and beta and gamma brainwaves to show the plasticity (both homo- and hetero- synaptic) responses of the proposed synapse.

Table 2
Characteristics and Frequency Bands of Brainwaves.

Band	Range	Brain-states
Delta (δ)	< 4 Hz	Sleep / Loss of bodily awareness
Theta (θ)	4 – 8 Hz	Inner peace / Meditation / Healing
Alpha (α)	8 – 12 Hz	Passive attention / Learning
Beta (β)	12 – 35 Hz	Anxiety dominant / Active / Learning
Gamma (γ)	> 35 Hz	Concentration / Awareness / Intelligence

Architecture of neuromemristive synapse

The proposed neuromemristive synapse, shown in Fig. 3, contains several blocks. A composite 1-port that consists of a memristor (M) and a capacitor (C_{CON}) in series is the main core of the neuromemristive synapse. The composite 1-port has a current-controlled push-pull architecture where the memristor is used to mimic the bio-realistic attributes of a synapse and its plasticity, whereas the capacitor is used to control the ratio of discharging through memristor M. In the active cycle of homosynaptic ($I_{mem} = I_{in} = I_{STM}$) or heterosynaptic ($I_{mem} = I_{HTEFAC}$) stimulation, both memristor M and capacitor C_{CON} will be charged by I_{mem} current (i.e., the devices are pulling I_{mem} to charge themselves) and in the inactive cycle of $I_{mem} = I_{in} = I_{STM}$ or $I_{mem} = I_{HTEFAC}$ (shown in blue arrowheads in heterosynaptic block), the stored charge in C_{CON} will be forced to discharge either partially or fully through memristor M (i.e., C_{CON} is pushing M to discharge through homosynaptic ($I_{HomoDIS}$, shown in purple arrowheads) or heterosynaptic (I_{HTEDEP} , shown in red arrow-

heads in heterosynaptic block) discharge pathways based on synaptic activity and their corresponding plasticity phenomena. The I_{mem} can be defined as,

$$I_{mem} = \begin{cases} I_{in} = I_{STM}, V_{DEP} > 0 \&\& V_{HTE} > 0 \&\& V_{HteSyn} < 0 \\ I_{HTEFAC}, V_{DEP} > 0 \&\& V_{HTE} > 0 \&\& V_{HteSyn} > 0 \\ I_{HomoDIS} = \frac{V_{CON} - V_{Acc}}{M}, (V_{DEP} < 0 \text{ or } V_{MFE} > 0) \&\& V_{HTE} < 0 \&\& V_{HteSyn} > 0 \\ I_{HTEDEP} = \frac{V_{CON} - V_{Acc}}{M}, V_{DEP} = 0 \&\& V_{MFE} = 0 \&\& V_{HTE} < 0 \&\& V_{HteSyn} < 0 \end{cases} \quad (1)$$

where V_{Acc} is the accumulated voltage of the memristive composite 1-port, V_{CON} is the voltage of the control capacitor, and $M = M_{Syn}$ is the memristance of the artificial neuromemristive synapse. The memristance (M_{Syn}) can be define as

$$M = M_{Syn} = M(0) + V_{Ct} R_T \quad (2)$$

where, $M(0)$ is the initial state and, V_{Ct} (V_{Ct} depends on inputs) and R_T are intrinsic parameters of the memristor as shown in Fig. A1 in the Appendix. The diode D_1 in Fig. 3 is used to block the reverse

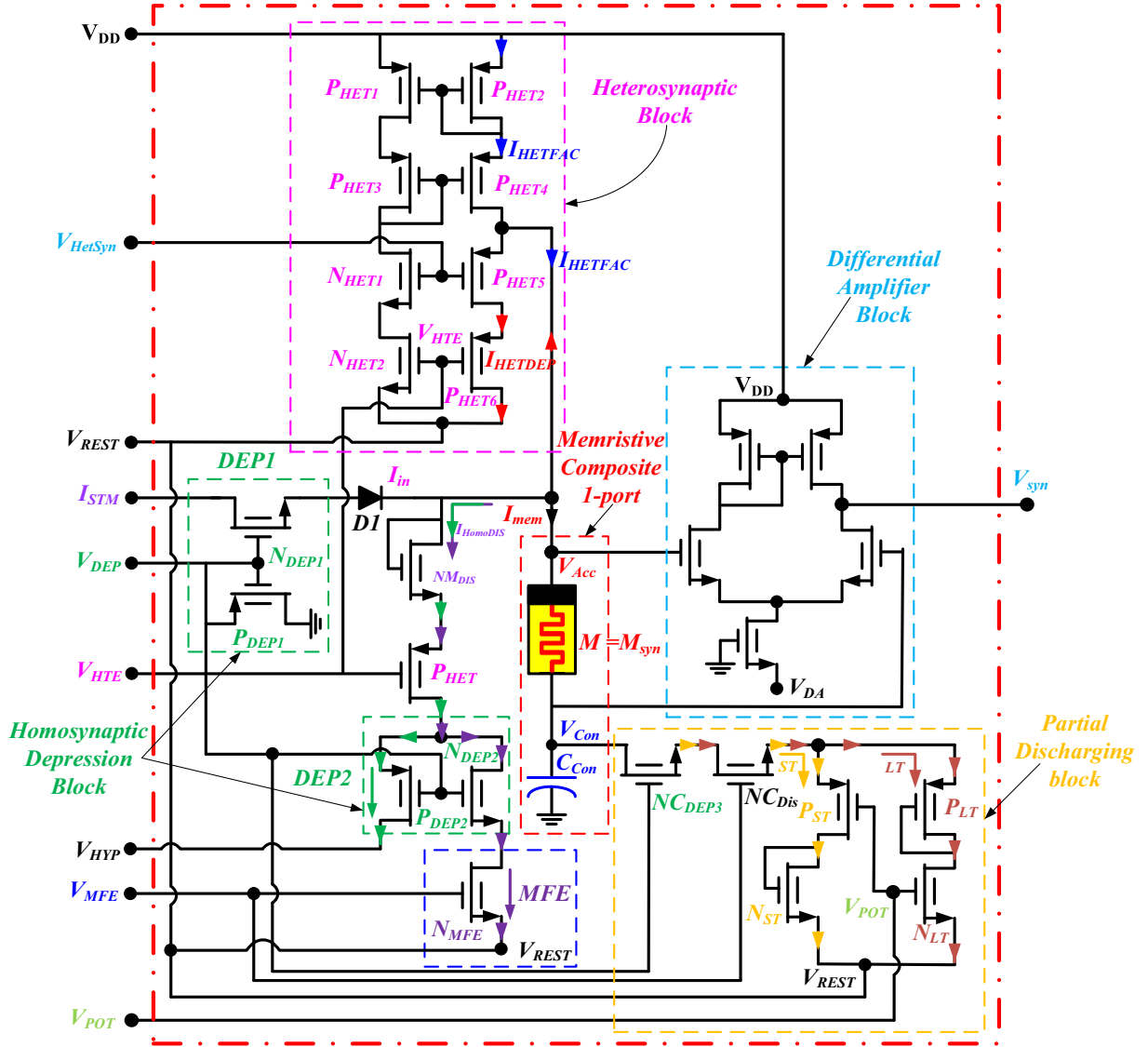


Fig. 3. Circuit diagram of the proposed neuromemristive synapse. The design comprises several functional blocks: a memristive composite 1-port that mimics the bio-realistic attributes of a synapse and its plasticity; a homosynaptic depression block for short-term and long-term depression (STD/LTD); a partial discharging block for short-term and long-term potentiation (STF/LTP); a heterosynaptic block for heterosynaptic facilitation and depression; and an amplifier block that produces the final synaptic output, V_{syn} .

directional current. The artificial synaptic voltage (V_{Syn}) is equal to the voltage across the memristor (V_{mem}) and is extracted using a differential amplifier as shown in Fig. 3 where,

$$V_{\text{Syn}} = V_{\text{mem}} = I_{\text{mem}}M = V_{\text{Acc}} - V_{\text{Con}} \quad (3)$$

To implement the homosynaptic potentiation (STF and LTP) and depression (STD and LTD) and reuptake (i.e., equivalent to MFE in electronic circuit), we implement separate discharging paths through memristor M and capacitor C_{CON} . According to section 3, the pattern, activity, durability, and the ratio of the decrement in synaptic efficacy of a synapse for the MFE and homosynaptic STD or LTD are quite different. Therefore, we designed two separate discharging paths to implement the MFE and homosynaptic STD or LTD phenomena. The composite 1-port will be discharged through HOMO_MFE pathway ($C_{\text{CON}} \rightarrow M \rightarrow \text{NM}_{\text{DIS}} \rightarrow \text{P}_{\text{HET}} \rightarrow \text{N}_{\text{DEP2}} \rightarrow \text{N}_{\text{MFE}} \rightarrow V_{\text{REST}}$) for MFE, as shown with purple arrowheads in Fig. 3. In contrast, the composite 1-port will be discharged through HOMO_DEP pathway ($C_{\text{CON}} \rightarrow M \rightarrow \text{NM}_{\text{DIS}} \rightarrow \text{P}_{\text{HET}} \rightarrow \text{P}_{\text{DEP2}} \rightarrow V_{\text{HYP}}$) for homosynaptic STD or LTD, as shown with green arrowheads in Fig. 3. V_{REST} and V_{HYP} represent the resting membrane potential (-70 mV) and hyperpolarize membrane potential (-110 mV), respectively. Moreover, the accumulation of synaptic efficacy and the rate of abolishment of neurotransmitters from synaptic cleft for homosynaptic STF and LTP are quite different according to section 2. Therefore, we implement two separate discharge paths through C_{CON} capacitor. When the composite 1-port exhibits homosynaptic LTP, the stored charge in C_{CON} will mostly discharged through HOMO_LT_PART_DIS pathway ($C_{\text{CON}} \rightarrow \text{NC}_{\text{DEP3}} \rightarrow \text{NC}_{\text{DIS}} \rightarrow \text{P}_{\text{LT}} \rightarrow \text{N}_{\text{LT}} \rightarrow V_{\text{REST}}$), as shown with burgundy arrowheads, whereas it will marginally discharge through HOMO_MFE pathway ($C_{\text{CON}} \rightarrow M \rightarrow \text{NM}_{\text{DIS}} \rightarrow \text{P}_{\text{HET}} \rightarrow \text{N}_{\text{DEP2}} \rightarrow \text{N}_{\text{MFE}} \rightarrow V_{\text{REST}}$), as shown with purple arrowheads, and partially discharge through HOMO-ST_PART_DIS pathway ($C_{\text{CON}} \rightarrow \text{NC}_{\text{DEP3}} \rightarrow \text{NC}_{\text{DIS}} \rightarrow \text{P}_{\text{ST}} \rightarrow \text{N}_{\text{ST}} \rightarrow V_{\text{REST}}$), as shown with yellow arrowheads in Fig. 3, for homosynaptic STF. We implemented additional depression switches DEP1 (N_{DEP1} and N_{DEP2}) and NC_{DEP3} , MFE switch (N_{MFE}), C_{CON} partial discharging switch (NC_{DIS}), and heterosynaptic switch (P_{HET}) to ensure that the neuromemristive synapse shouldn't exhibit two different types of plasticity phenomena at the same time similar to biological synapses.

The heterosynaptic facilitation and depression are implemented using the heterosynaptic block, shown in Fig. 3, which are connected to the memristor M of the composite 1-port. When the neuromemristive synapse exhibits heterosynaptic facilitation (i.e., active cycle of V_{HTE} where $V_{\text{HTE}} = V_{\text{H}}$), the hetero-synapse input V_{HteSyn} ($V_{\text{HteSyn}} = V_{\text{H}}$) generates I_{HTEFAC} current (where, $I_{\text{mem}} = I_{\text{HTEFAC}}$) and charges the memristor M and capacitor C_{CON} through HET_FAC pathway ($V_{\text{DD}} \rightarrow \text{P}_{\text{HET2}} \rightarrow \text{P}_{\text{HET4}} \rightarrow M \rightarrow C_{\text{CON}} \rightarrow 0$). In con-

trast, for heterosynaptic depression (i.e., inactive cycle of V_{HTE} where $V_{\text{HTE}} = V_{\text{L}}$ and $V_{\text{HteSyn}} = V_{\text{L}}$), memristor M and capacitor C_{CON} are discharged through HET_DEP pathway ($0 \rightarrow C_{\text{CON}} \rightarrow M \rightarrow \text{P}_{\text{HET5}} \rightarrow \text{P}_{\text{HET6}} \rightarrow V_{\text{REST}}$).

Results and discussions

A modified memristor emulator [68] was used in this paper to design the proposed neuromemristive synapse as the memristor is commercially unavailable. We operated the memristor emulator in the linear region ($2 \text{ K} \sim 14 \text{ K}$) to avoid the unintended consequences of nonlinear ionic dopant drift at its boundaries [69,70] as shown in Fig. A1(b) in the appendix. The memristive fingerprints of the modified memristor emulator ($M(0) = 2 \text{ K}$, $C_{\text{T}} = 2 \mu\text{F}$, and $R_{\text{T}} = 4 \text{ K}$), shown in Fig. A1 (a), for bipolar inputs are shown in Figs. A1 (c) and (d). We conducted various SPICE simulations to verify the bio-realistic features of the proposed neuromemristive synapse. Table 4 lists the circuit parameters used to obtain the subsequent experimental results of the proposed synapse, using the operating signals mentioned in Table 3.

Non-plastic synaptic responses

We used a stimulating pulse current $I_{\text{in}} = I_{\text{stm}} = 100 \mu\text{A}$ with $\text{PW} = 1 \text{ ms}$ along with brainwave frequencies $\delta = 3.5 \text{ Hz}$ ($\text{PP} = 286 \text{ ms}$), $\theta = 7 \text{ Hz}$ ($\text{PP} = 142.86 \text{ ms}$), and $\alpha = 10 \text{ Hz}$ ($\text{PP} = 100 \text{ ms}$), which can be seen in magenta in Fig. 4(a), (e), and (i). This resulted in the proposed memristive synapse responding in a manner that does not exhibit synaptic plasticity. In contrast, we used the same $I_{\text{in}} = 100 \mu\text{A}$ ($\text{PW} = 1 \text{ ms}$ and $\text{PP} = 40 \text{ ms}$) and β -brainwaves ($\beta = 25 \text{ Hz}$) to show the response of our proposed architecture to plasticity, as shown in Fig. 4(m). Figs. 4(a), (e), (i), and (m) show the 1's complement MFE signals ($V_{\text{MFE}} = V_{\text{H}} = 1 \text{ V}$) in blue. The inset in the middle shows that V_{MFE} is inactive when I_{in} is active and vice versa. The proposed synapse does not exhibit either homosynaptic depression or heterosynaptic plasticity, as evidenced by the values of $V_{\text{DEP}} = V_{\text{H}} = 1 \text{ V}$, $V_{\text{HTE}} = V_{\text{L}} = -1 \text{ V}$, and $V_{\text{HteSyn}} = V_{\text{O}} = 0 \text{ V}$, as shown in Fig. 4 (b), (f), (j), and (n). The artificial synapse doesn't build up synaptic strength (M_{syn}) for $\delta = 3.5 \text{ Hz}$, $\theta = 7 \text{ Hz}$, and $\alpha = 10 \text{ Hz}$ brainwaves (Figs. 4(c), (g), and (k)). However, M_{syn} builds up significantly for $\beta = 25 \text{ Hz}$ (Fig. 4(o)). Like the biosynapse, the neuromemristive synapse doesn't change the strength of the synapse below the neutral frequency ($\leq 10 \text{ Hz}$); instead, it changes the efficacy of the synapse above the neutral frequency ($> 10 \text{ Hz}$). The artificial synaptic voltage (V_{Syn}) consequently increases in each active cycle of I_{in} , as shown in Fig. 4(p). This is because the synaptic strength (M_{syn}) builds up at a non-neutral frequency of $\beta = 25 \text{ Hz}$. This is in contrast to Figs. 4(d), (h), and (l), where V_{Syn} remains the same ($V_{\text{Syn}} = 220 \text{ mV}$) due to constant M_{syn} at $\delta = 3.5 \text{ Hz}$, $\theta = 7 \text{ Hz}$, and $\alpha = 10 \text{ Hz}$. We can conclude from Fig. 4 that the proposed arti-

Table 3
Operating modes of the proposed neuromemristor synapse.

Synapse Type	Memory Type	Synaptic Mode	Synaptic Response	Operating Signals				
				V_{HTE}	V_{HteSyn}	V_{DEP}	V_{MFE}	V_{POT}
Homosynaptic	Volatile	Facilitation/ Potentiation	NSR + MFE	V_{L}	V_{O}	V_{H}	V_{H}	V_{O}
			STF + MFE	V_{L}	V_{O}	V_{H}	V_{H}	V_{L}
			LTP + MFE	V_{L}	V_{O}	V_{H}	V_{H}	V_{H}
		Depression	STD	V_{L}	V_{O}	V_{L}	V_{O}	V_{L}
			LTD	V_{L}	V_{O}	V_{L}	V_{O}	V_{H}
			NSR	V_{L}	V_{O}	V_{H}	V_{O}	V_{O}
	Nonvolatile	Facilitation/ Potentiation	STF	V_{L}	V_{O}	V_{H}	V_{O}	V_{O}
			LTP	V_{L}	V_{O}	V_{H}	V_{O}	V_{O}
			LTP	V_{L}	V_{O}	V_{H}	V_{O}	V_{O}
Heterosynaptic	Volatile	Facilitation	HTE_FAC	V_{H}	V_{H}	V_{H}	V_{O}	V_{O}
		Depression	HTE_DEP	V_{L}	V_{L}	V_{H}	V_{O}	V_{O}

Table 4

Circuit parameters of the proposed neuromemristive synapse.

Parameters	Value	Parameters	Value
V_{DD}	2.5 V	V_{SS}	-2.5 V
V_H	0.4 ~ 1.0 V	V_L	-1.0 ~ -0.4 V
V_{REST}	-70 mV	V_{HYP}	-110 mV
V_0	0 V	C_{Con}	12 μ F

ficial synapse qualitatively mimics the biorealistic attributes of a biosynapse.

Homosynaptic plasticity

In this paper, we implemented both homosynaptic short-term and long-term plasticity, as shown in Figs. 5 and 6, respectively.

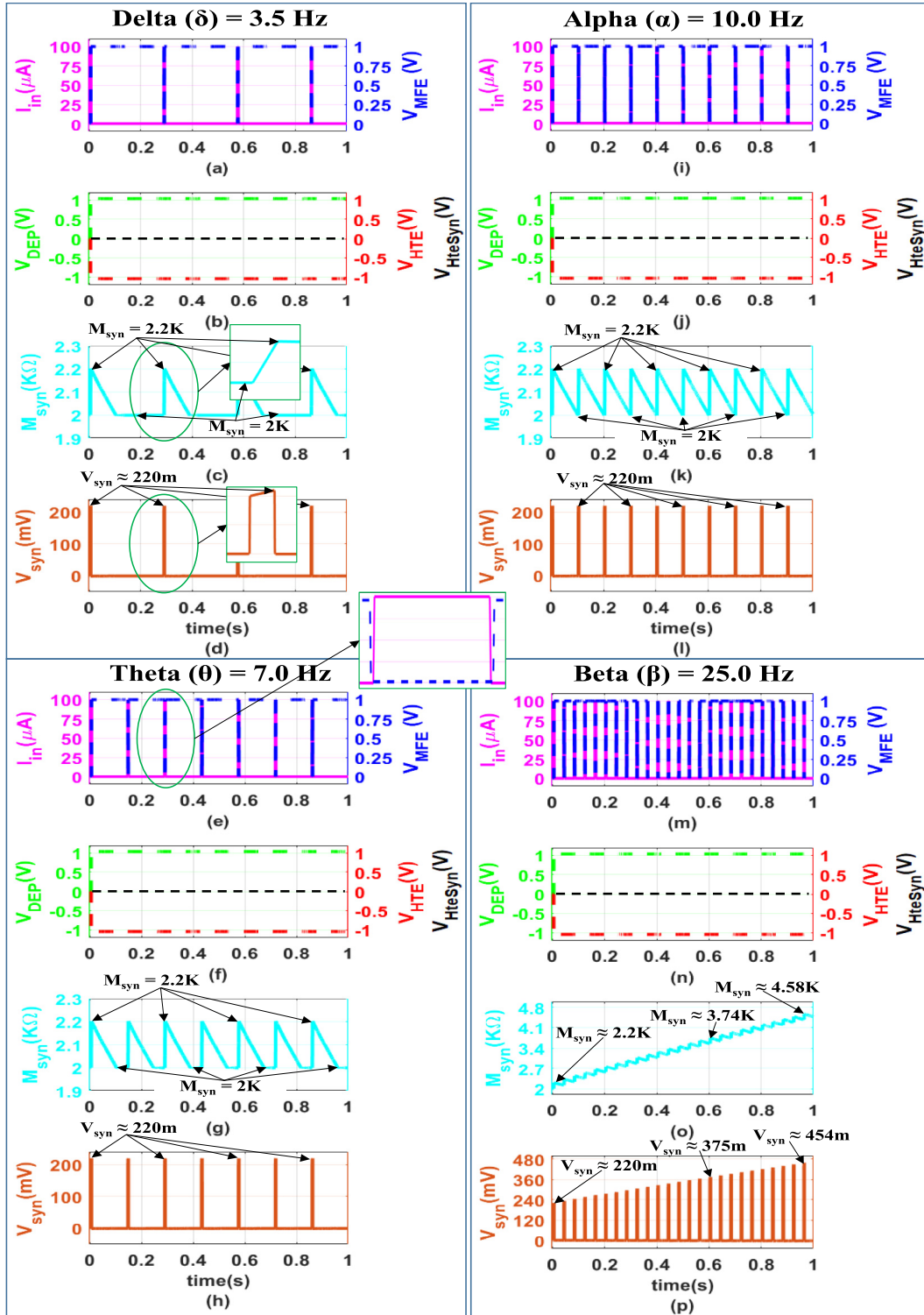


Fig. 4. Synaptic response of the proposed neuromemristive synapse for Delta (δ), Theta (θ), Alpha (α) and Beta (β) brainwaves. Input stimulation (I_{in}) and memory fading effect (V_{MFE}) for (a) $\delta = 3.5$ Hz, (e) $\theta = 7$ Hz, (i) $\alpha = 10$ Hz, and (m) $\beta = 25$ Hz. Control signals for homosynaptic depression (V_{DEP}) and heterosynaptic plasticity (V_{HTE}) for (b) $\delta = 3.5$ Hz, (f) $\theta = 7$ Hz, (j) $\alpha = 10$ Hz, and (n) $\beta = 25$ Hz. Rate of change of the synaptic strength (M_{syn}) for (c) $\delta = 3.5$ Hz, (g) $\theta = 7$ Hz, (k) $\alpha = 10$ Hz, and (o) $\beta = 25$ Hz. Artificial synapse voltage (V_{syn}) for (d) $\delta = 3.5$ Hz, (h) $\theta = 7$ Hz, (l) $\alpha = 10$ Hz, and (p) $\beta = 25$ Hz.

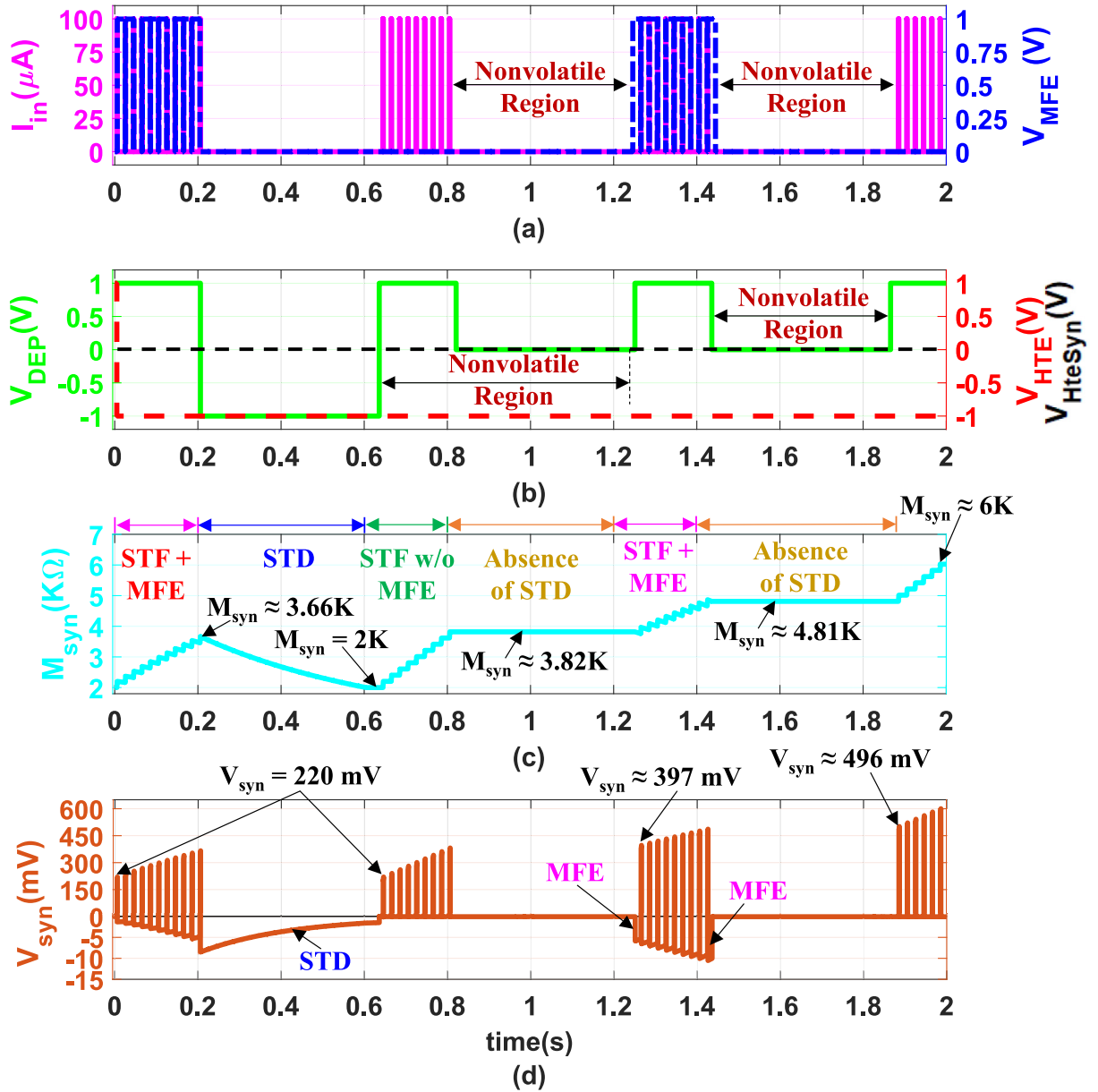


Fig. 5. Short-term plasticity response of the proposed neuromemristive synapse for Gamma-brainwave (50 Hz). (a) Input stimulus (I_{in}) and memory fading effect (V_{MFE}), (b) control signals for homosynaptic depression (V_{DEP}) and heterosynaptic plasticity (V_{HTE}), (c) rate of change of the synaptic strength (M_{syn}), and (d) synaptic voltage (V_{syn}) of the proposed artificial synapse.

We showed short-term plasticity by stimulating the proposed neuromemristive synapse with a range of different conditions, including short-term facilitation (STF), short-term depression (STD), and non-volatility stimuli. On the contrary, for long-term plasticity, we provided long-term potentiation (LTP) and long-term depression (LTD) stimuli.

To demonstrate the short-term plasticity of the proposed synapse, stimuli were applied using homosynaptic spike trains with specific parameters. An input STF stimuli $I_{in} = 100 \mu A$ (PW = 1 ms) with γ -brainwaves ($\gamma = 50$ Hz (PP = 20 ms)) was used along with active memory fading effect signal V_{MFE} on the first and third cycles, as shown in Fig. 5(a). Fig. 5(b) shows the homosynaptic STD (V_{DEP}) and the inactive heterosynaptic plasticity ($V_{HTE} = V_L = -1$ V and $V_{HteSyn} = V_0 = 0$ V) signals. It can be seen in Fig. 5(c) that the close successive stimulus gradually raises the synaptic efficacy (M_{syn}), even when V_{MFE} is active (1st cycle, $t \leq 0.2$ s). In contrast, the artificial synapse gradually decreases its synaptic efficacy in the active period of STD (0.2 s $< t < 0.64$ s) with a similar passion

to that of the volatile biosynapse. However, a non-volatility increase in M_{syn} is seen in the active cycle of I_{in} and the inactive cycle of V_{MFE} (0.64 s $\leq t < 0.84$ s), and M_{syn} remains unchanged ($M_{syn} = 3.82$ K) until $V_{DEP} \geq 0$ and $V_{MFE} = 0$ (0.84 s $\leq t \leq 1.24$ s). Similar like the biological synapse, the synaptic efficacy (M_{syn}) of the proposed neuromemristive synapse decreases in the inactive cycle of I_{in} and active cycle of memory fading effect (V_{MFE}) along with the active cycle of homosynaptic STD (V_{DEP}). In contrast, the proposed synapse increases its synaptic efficacy (M_{syn}) in the active cycle of I_{in} despite the presence of V_{MFE} and in absence of V_{DEP} . Fig. 5 (c) also shows that when the input stimulus I_{in} returns, the synaptic efficacy (M_{syn}) rises in three stages: namely, a volatile rise (1.24 s $< t \leq 1.46$ s), a hold-off rise (1.46 s $< t \leq 1.8$ s), and a nonvolatile rise ($t > 1.8$ s). Synaptic voltage (V_{syn}), shown in Fig. 5(d), produces a higher voltage ($t \geq 1.2$ s) compared to $t \leq 0.84$ s due to higher buildup in M_{syn} .

To determine the outcome of the long-term plasticity of the artificial synapse, we stimulated the proposed synapse with

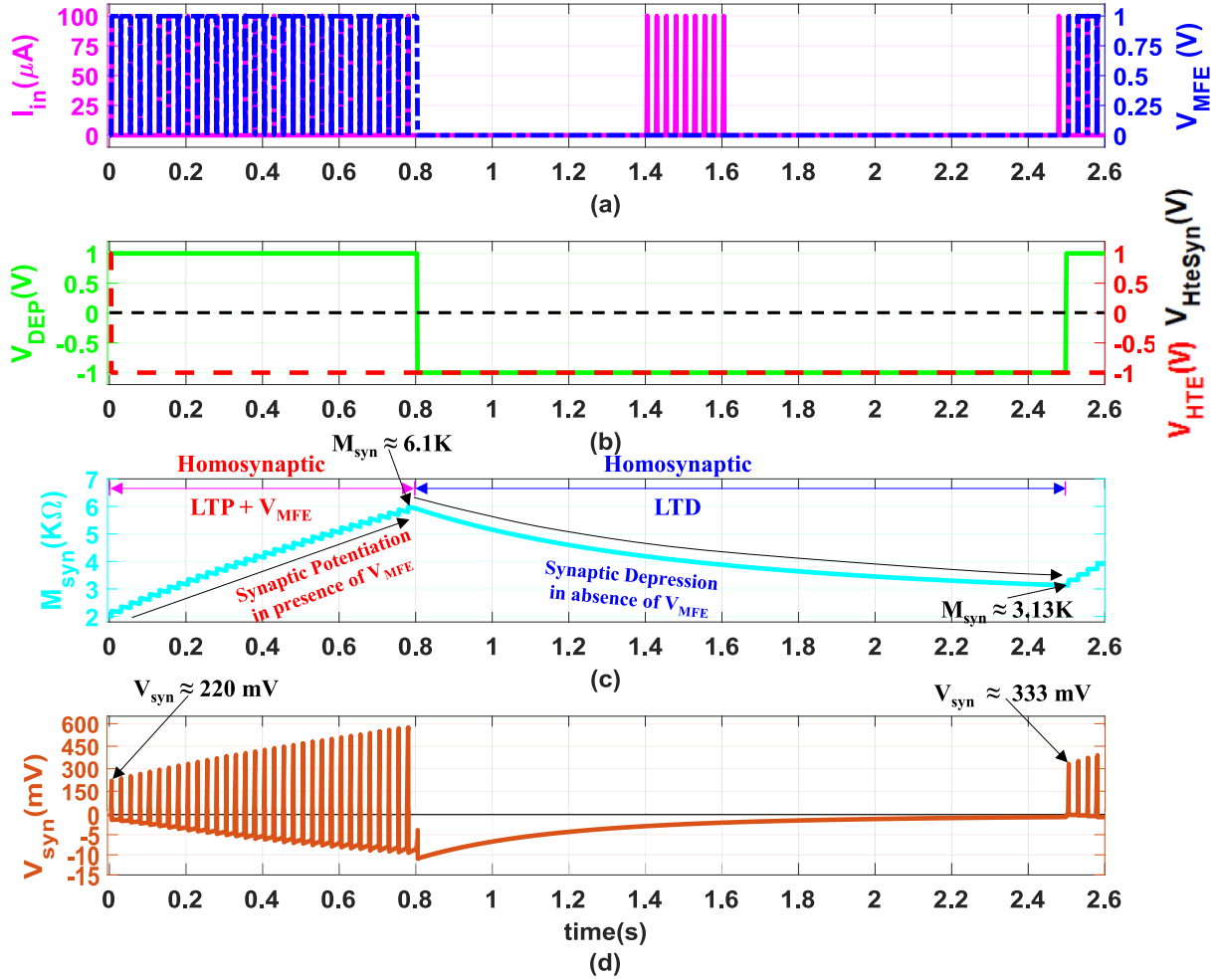


Fig. 6. Long-term plasticity response of the proposed neuromemristive synapse for Gamma-brainwave (40 Hz). (a) Input stimulus (I_{in}) and memory fading effect (V_{MFE}), (b) control signals for homosynaptic depression (V_{DEP}) and heterosynaptic plasticity (V_{HTE}), (c) rate of change of the synaptic strength (M_{syn}), and (d) synaptic voltage (V_{syn}) of the proposed artificial synapse.

homosynaptic LTP stimuli $I_{in} = 100 \mu A$ (PW = 1 ms) and γ -brainwaves ($\gamma = 40$ Hz (PP = 25 ms)) shown in magenta solid, and the V_{MFE} is shown in dotted blue in Fig. 6(a). Fig. 6(b) shows the homosynaptic LTD (V_{DEP}) and the inactive heterosynaptic plasticity ($V_{HTE} = V_L = -1V$ and $V_{HteSyn} = V_0 = 0V$) in solid green and dotted red and black curves, respectively. Fig. 6(c) shows that the synaptic efficacy (M_{syn}) swiftly increases in the potentiation cycle despite the presence of active V_{MFE} and gradually decreases in the depression cycle (active presence of V_{DEP}) despite the active presence of I_{in} ($1.4s \leq t \leq 1.62s$). The memristive synapse can't go back to its initial state $M(0) = 2K$ during the LTD period because synaptic efficacy has already grown during the LTP period. Hence, the proposed synapse produces higher V_{syn} (shown in Fig. 6(d)) when the presynaptic stimulations return to the active state after $t \geq 2.4s$ than that in the initial period ($t < 0.1s$).

We can conclude from Fig. 6 that the neuromemristive synapse effectively imitates the long-term plasticity phenomenon of the biosynapse, as long-lasting enhancements in synaptic efficacy were developed in both the LTP and LTD processes, similar to that of biological synapses.

Heterosynaptic plasticity

To implement the heterosynaptic plasticity, we provide a homosynaptic stimuli $I_{in} = 100 \mu A$ (PW = 1 ms) with γ -

brainwaves ($\gamma = 50$ Hz (PP = 20 ms)) and active duty cycle of 40 %, shown in Fig. 7(a), to the proposed synapse. The homosynaptic stimuli was provide for the readers' better understanding. Fig. 7(b) shows the 1-s complement V_{MFE} and V_{DEP} . The control signals for heterosynaptic facilitation ($V_{HTE-FAC}$) and depression ($V_{HTE-DEP}$) are shown in Fig. 7(c). Fig. 7(d) shows the synaptic input voltages for heterosynaptic facilitation ($V_{syn-FAC}$) and depression ($V_{syn-DEP}$). Combination of these synaptic inputs and control signals are responsible for the generation of heterosynaptic facilitation and depression in the proposed neuromemristive synapse. We provide only 40 % of $V_H = |V_L| = 1V$ as synaptic inputs for facilitation ($V_{syn-FAC} = 400$ mV) and depression ($V_{syn-DEP} = -400$ mV) to maintain the bio-realistic phenomenon that the pathways for homosynaptic plasticity strengthen the synapse profoundly (due to the higher number of neurotransmitters released in those specific pathways) than that of heterosynaptic plasticity. Fig. 7(e) shows the rate of the changes in synaptic strengths for both homo and heterosynaptic facilitation ($M_{syn-FAC}$) and depression ($M_{syn-DEP}$) inputs. Observe that both the $M_{syn-FAC}$ and $M_{syn-DEP}$ are increasing monotonically despite the presence of active V_{MFE} over the period of $0s \leq t < 0.8s$. When heterosynaptic facilitation occurs at the neuromemristive synapse, the synaptic strength ($M_{syn-FAC}$) builds up swiftly due to $V_{syn-FAC}$. On the other hand, when heterosynaptic depression happens at the neuromemristive synapse, the synaptic strength ($M_{syn-DEP}$) slowly decreases over the active period of $0.8s$ to $1.2s$

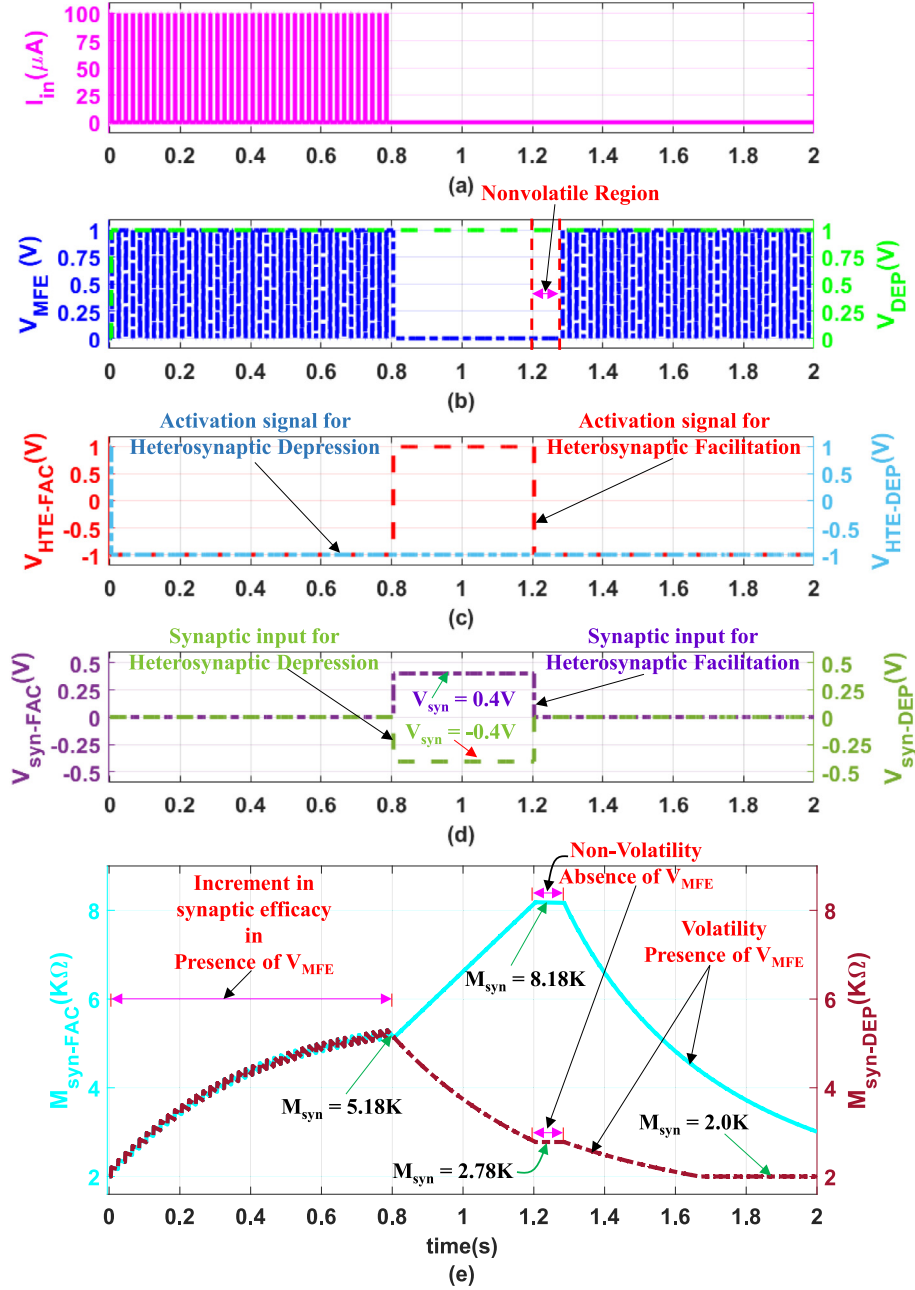


Fig. 7. Heterosynaptic plasticity response of the proposed neuromemristive synapse for Gamma-brainwave (50 Hz). (a) Input stimuli (I_{in}), (b) homosynaptic V_{MFE} and V_{DEP} , (c) control signals for heterosynaptic facilitation ($V_{HTE-FAC}$) and depression ($V_{HTE-DEP}$), (d) synaptic inputs for heterosynaptic facilitation ($V_{syn-FAC}$) and depression ($V_{syn-DEP}$), and (e) rate of change in synaptic strength for facilitation ($M_{syn-FAC}$) and depression ($M_{syn-DEP}$).

because of $V_{syn-DEP}$. Fig. 7 shows that both the $M_{syn-FAC}$ and $M_{syn-DEP}$ keep their synaptic strengths (nonvolatile operation) from 1.2 s to 1.3 s, but they both start to lose strength when the active cycle of V_{MFE} starts up again after 1.4 s. Observe from Fig. 7(e) that the $M_{syn-DEP}$ returns to the initial value $M_{syn-DEP}(0) = 2$ K and holds the initial states despite the active presence of V_{MFE} .

The homeostatic role of heterosynaptic plasticity (i.e., homeostasis) is that when one synapse experiences long-term potentiation (LTP), neighboring synapses go through long-term depression (LTD) to prevent over-excitation or excitotoxicity. To demonstrate homeostasis, the neuromorphic synapse was potentiated over $t < 0.8$ s and $t > 1.8$ s via homosynaptic LTP stimuli. We then depress the proposed synapse over $0.8 \text{ s} \leq t \leq 1.6$ s using heterosyn-

naptic LTD stimuli. For homosynaptic LTP, we provide $I_{stm} = 100 \mu\text{A}$ (PW = 1 ms) with γ -brainwaves ($\gamma = 40$ Hz (PP = 25 ms)) as shown in green in Fig. 8(a). We employed the nonvolatile mode of the neuromemristive synapse to perform homosynaptic potentiation by setting $V_{MFE} = 0$ V and $V_{DEP} = V_H = 1$ V, as shown in Fig. 8(b). Fig. 8(c) shows that the the control signal $V_{HTE} = V_L = -1$ V and synaptic input voltage $V_{syn-DEP} = -400$ mV are facilitating heterosynaptic LTD over a time span $0.8 \text{ s} \leq t \leq 1.6$ s. Observe from magenta I_{in} in Fig. 8(a) that the memristive synapse only exhibiting input stimuli $I_{in} = I_{stm}$ for $V_{HTE} = V_H = 1$ V over the time period $t < 0.8$ s and $t \geq 1.8$ s. The synaptic efficacy (M_{syn}), shown in Fig. 8(d), of the proposed synapse is monotonically increasing up until it reaches its highest point: $M_{syn} \approx 8.5$ K, for the active homosynaptic

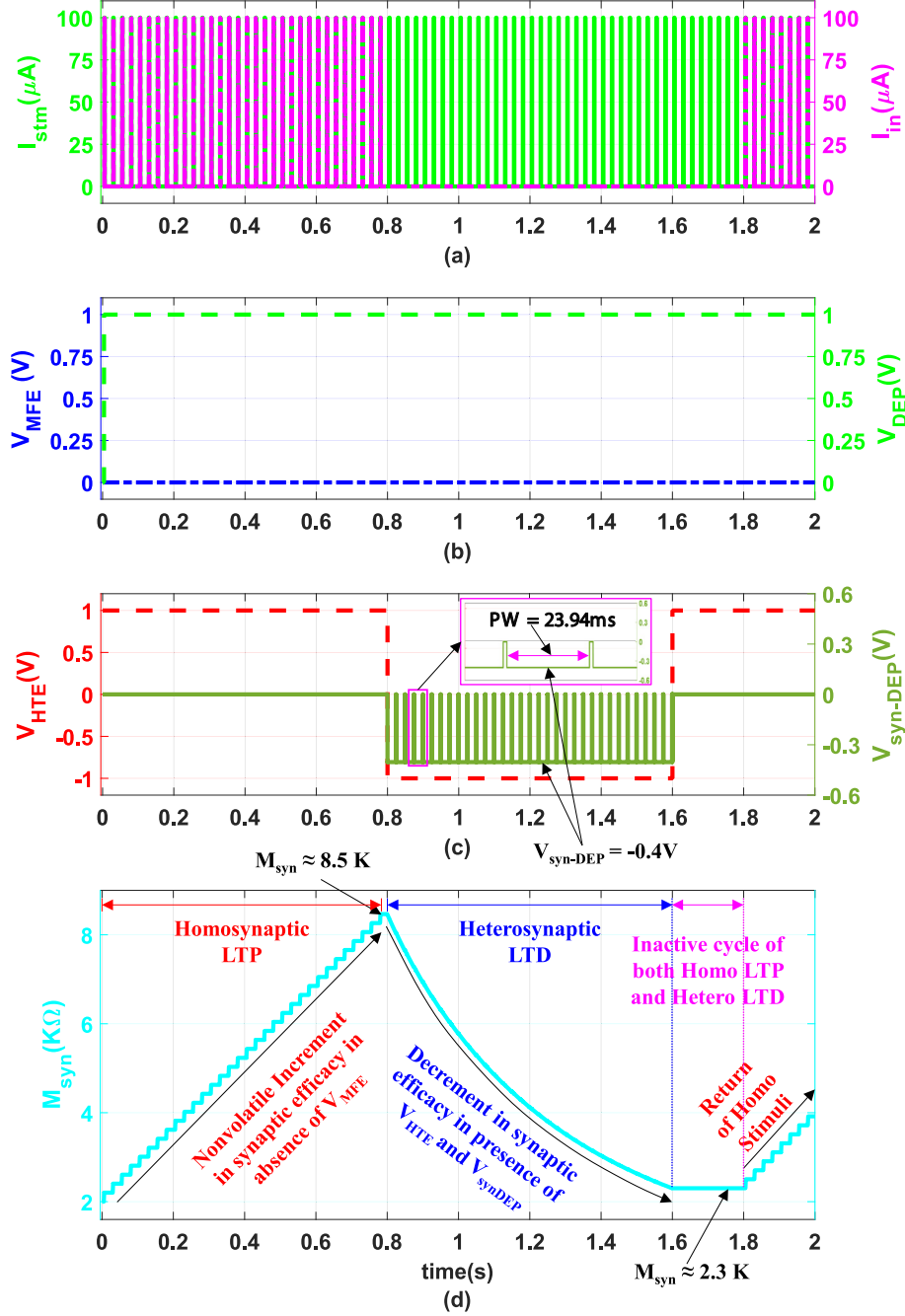


Fig. 8. Response of the proposed neuromemristive synapse on the homeostatic role of the heterosynaptic plasticity for Gamma-brainwave (50 Hz). (a) External stimulations (I_{stm}) and input stimuli (I_{in}), (b) homosynaptic V_{MFE} and V_{DEP} , (c) heterosynaptic control signals (V_{HTE}) and synaptic inputs for heterosynaptic depression ($V_{syn-DEP}$), (d) rate of change in synaptic strength (M_{syn}).

sequential I_{in} stimuli over $t \leq 0.8$ s and $t \leq 1.8$ s. On the other hand, M_{syn} is gradually decreasing and approaching the initial state $M_{syn} \approx 2.3$ K due to active heterosynaptic LTD generated by $V_{HTE} = V_L = -1V$ and $V_{syn-DEP} = -400$ mV for $0.8 \leq t \leq 1.6$ s. Fig. 8 indicates that the proposed neuromemristive synapse is capable of performing both homosynaptic LTP and heterosynaptic LTD when given the appropriate control and stimulating signals. It also proves that the neuromemristive synapse can maintain homeostasis (i.e., homeostatic regulation) by demonstrating heterosynaptic LTD, while the nearby artificial synapse may experience homosynaptic LTP. Such occurrences are necessary for maintaining neural or neuronal network stability and preventing excitotoxicity [53,54].

Modular input-specific role of heterosynaptic plasticity facilitates synaptic competition by allowing increased activity or allocating specific pathways to strengthen the synapse. In order to illustrate the modular input-specificity of heterosynaptic plasticity, we first applied homosynaptic short-term plasticity (STF and STD) to the neuromorphic synapse over a time span of 0 s to 0.6 s. For $t < 0.2$ s, we give homosynaptic STF stimuli $I_{in} = 100$ μA ($PW = 1$ ms) along with γ -brainwaves ($\gamma = 50$ Hz ($PP = 20$ ms)), which can be seen in magenta in Fig. 9(a). We used the neuromemristive synapse in a nonvolatile configuration for homosynaptic STF ($V_{MFE} = 0$ over $t \leq 1.6$ s) and depressed it with $V_{DEP} = -1V$ for homosynaptic STD over $0.2 \leq t < 2$ s, as shown in Fig. 9(b). From

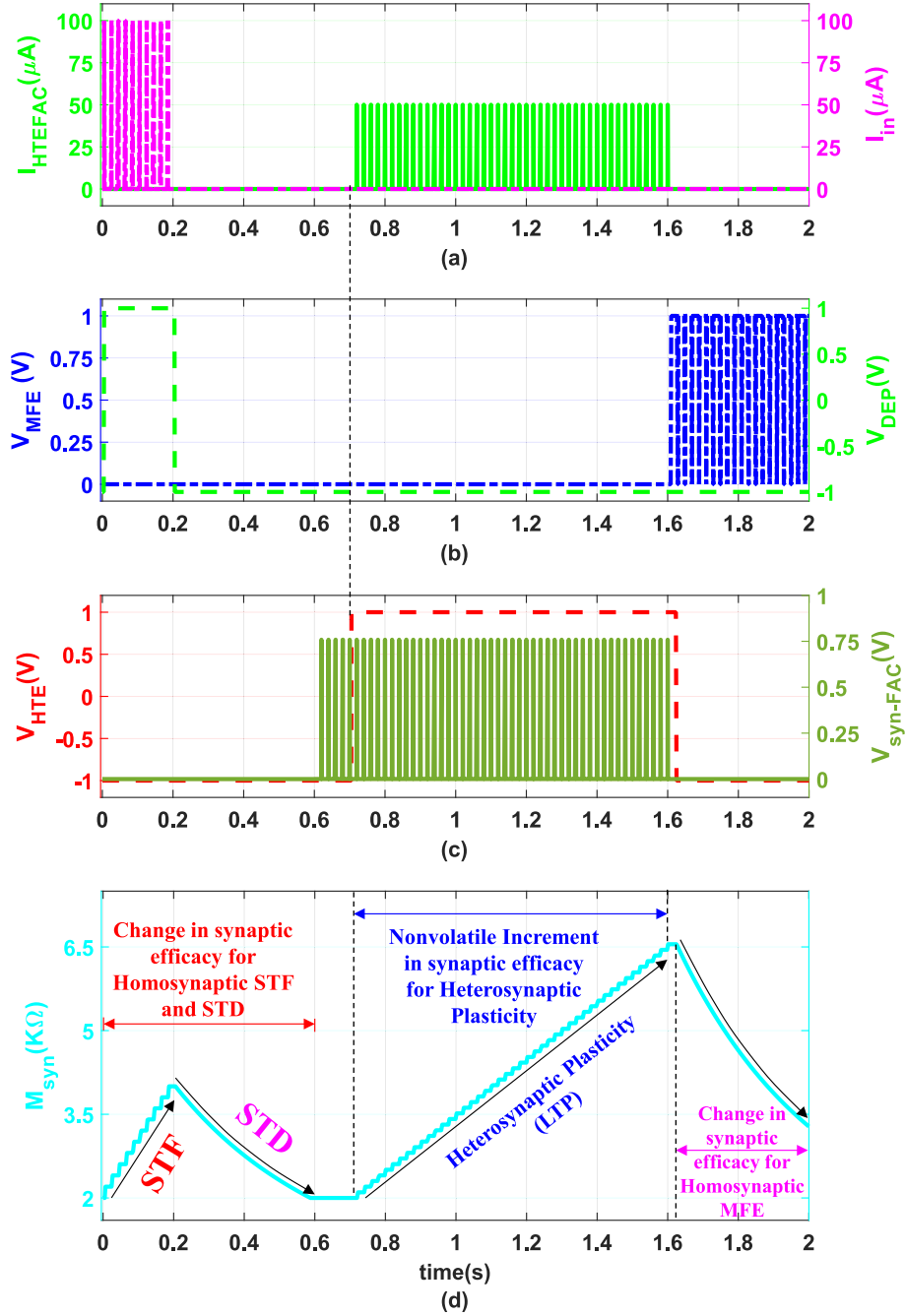


Fig. 9. Modular input-specific plasticity response of the heterosynaptic plasticity of the proposed neuromemristive synapse for Gamma-brainwave (50 Hz). (a) Input stimuli (I_{in}) and heterosynaptic facilitation stimuli (I_{HTEFAC}), (b) homosynaptic V_{MFE} and V_{DEP} , (c) heterosynaptic control signals (V_{HTE}) and synaptic inputs for heterosynaptic facilitation ($V_{syn-FAC}$), (d) rate of change in synaptic strength (M_{syn}).

Fig. 9(a), it can be seen that the neuromemristive synapse is being stimulated by heterosynaptic LTP stimuli $I_{HTEFAC} = 50 \mu A$ (PW = 1 ms) with $\gamma = 50$ Hz (PP = 20 ms) over $0.7 s \leq t < 1.6 s$. These stimuli are generated by $V_{syn-FAC} = V_H = 0.75$ V (PW = 1 ms) with $\gamma = 50$ Hz (PP = 20 ms) over $0.6 s \leq t < 1.6 s$, as shown in Fig. 9(c). The heterosynaptic control signal $V_{HTE} = V_H = 1$ V over $0.7 s \leq t \leq 1.65$ s and for the rest of the time span $V_{HTE} = V_H = -1$ V, as shown in Fig. 9(c). Observe from Figs. 9(a) and (c) that the proposed synapse generates I_{HTEFAC} (green in Fig. 9(a)) only while $V_{HTE} = V_H = 1$ V (red in Fig. 9(c)). Fig. 9(d) shows that the artificial synaptic strength (M_{syn}) is swiftly increasing for both homosynaptic STF ($t < 0.2$ s) and heterosynaptic LTP ($0.7 s - t \leq 1.65$ s). On the other hand, it is steadily decreasing for homosynaptic STD ($0.2 s \leq t \leq 0.6$ s)

and gradually decreases for homosynaptic MFE ($t \geq 1.65$ s). Fig. 9 shows that we can modulate the potentiation or depression activity of the specific pathways to strengthen or weaken the proposed neuromemristive synapse. It also proves that the proposed synapse qualitatively imitates the synaptic pruning mechanism of bio-synapses as it refines the connectivity of the specific pathways and facilitates selective and effective information processing.

To implement the associative role of the heterosynaptic plasticity, we provide a homosynaptic stimuli $I_{in} = 75 \mu A$ (PW = 1 ms) with $\beta = 25$ Hz (PP = 20 ms) and a heterosynaptic stimuli $I_{HTEFAC} = 40 \mu A$ (PW = 1 ms) with $\beta = 33$ Hz (PP = 30.3 ms) as shown in Fig. 10(a). For this homosynaptic simulation, we didn't consider the memory fading effect (i.e., $V_{MFE} = 0$ V) as we want the neu-

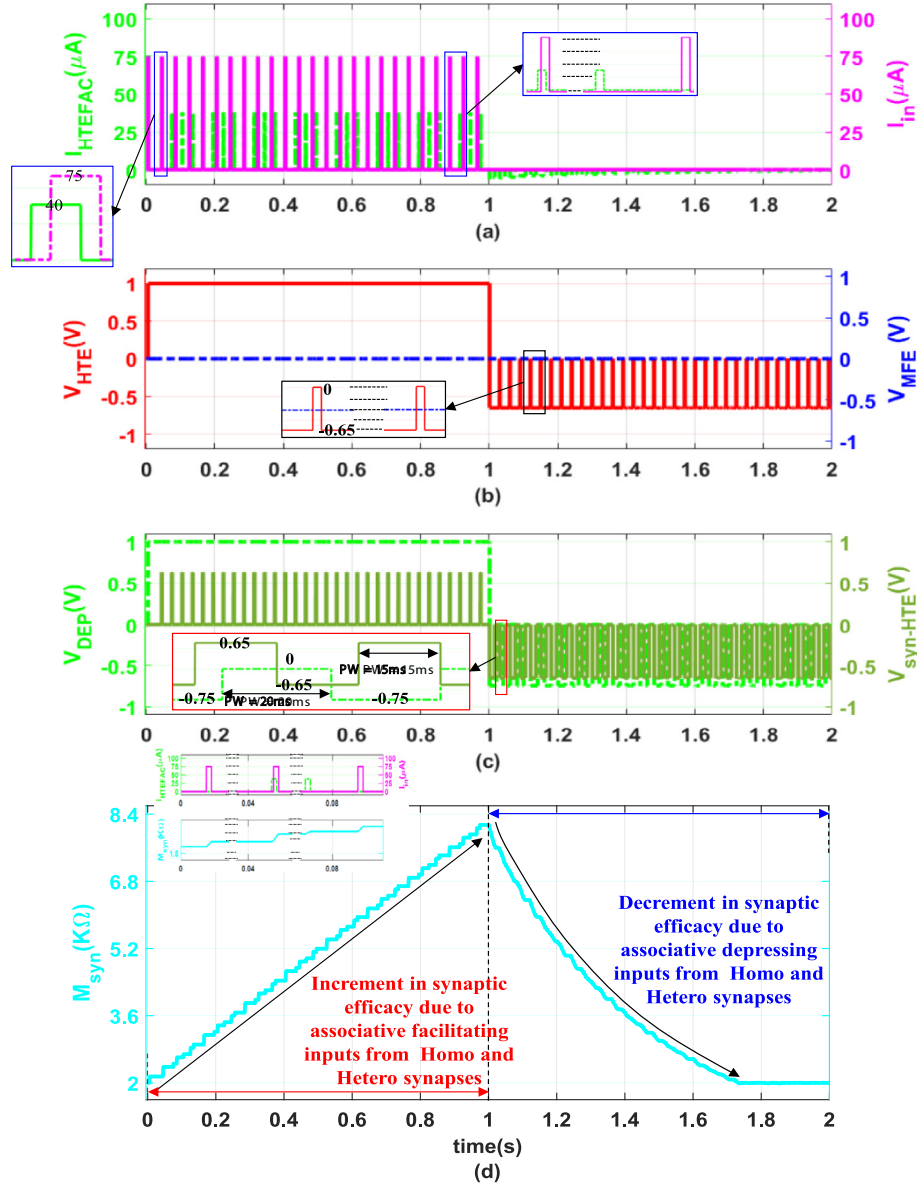


Fig. 10. Response of the proposed neuromemristive synapse for associative role of the heterosynaptic plasticity for Beta-brainwave (25 Hz for Homosynaptic and 33 Hz for Heterosynaptic synapse). (a) External stimulations (I_{stm}) and input stimuli (I_{in}), (b) homosynaptic V_{MFE} and heterosynaptic control signals (V_{HTE}) (c) homosynaptic depression signal (V_{DEP}) and synaptic inputs for heterosynaptic synapse ($V_{syn-HTE}$), (d) rate of change in synaptic strength (M_{syn}).

romemristive synapse to be operated in nonvolatile mode. A bipolar signal was provided as a heterosynaptic plasticity controlling signal with $V_{HTE} = V_H = 1$ V for $t < 1$ s and $V_{HTE} = V_L = -0.65$ V for $t \geq 1$ s with a duty ratio of 49.5 % as shown in Fig. 10(b). Fig. 10(c) illustrates the homosynaptic depression (V_{DEP}) and heterosynaptic synaptic signal ($V_{syn-HTE}$) during both facilitation ($t < 1$) and depression ($t \geq 1$) cycles. Fig. 10(d) shows that the synaptic efficacy (M_{syn}) of the neuromemristive synapse was potentiated over the time span $t < 1$ s due to the combinational homosynaptic ($I_{in} > 0$) and heterosynaptic ($I_{HTEFAC} > 0$) facilitation stimuli where $V_{DEP} > 0$, $V_{HTE} > 0$, and $V_{syn-HTE} > 0$. However, the synaptic efficacy (M_{syn}) neuromemristive synapse was depressed within $1 \leq t \leq 2$ s due to the combinational homosynaptic ($V_{DEP} < 0$) and heterosynaptic ($V_{syn-HTE} < 0$) depression stimuli where $V_{HTE} < 0$. It is interesting to note that both homo and hetero synaptic facilitation stimuli helped M_{syn} build up during the potentiation phase, while both homo and hetero synaptic depression stimuli weakened M_{syn} . Therefore, we can say that the suggested neu-

romemristive synapse can continue associative learning as it turns on both homosynaptic and heterosynaptic pathways at the same time and makes one synapse stronger. In contrast, during the depression phase, the system modifies the surrounding synaptic environment by depressing the neuromemristive synapse, while another neuromemristive synapse undergoes long-term projection (LTP) within a neuronal or neural network.

Strong stimulation

To investigate the response of the proposed neuromemristive synapse to strong stimulating phenomena, the same amplitude of $I_{in} = 120 \mu A$ (PW = 1 ms) at two different brainwaves, $\alpha = 10$ Hz (PP = 100 ms) and $\beta = 25$ Hz (PP = 40 ms) was provided, as shown in Figs. 11 (a) and (e), respectively. Observe that the stimulating inputs in Figs. 11(a) and 4(i), as well as Figs. 11(e) and 4(m), are indifferent, except for the pulse amplitude. V_{MFE} signals in Figs. 11 (a) and 4(i), as well as Figs. 11(e) and 4(m), remain the same.

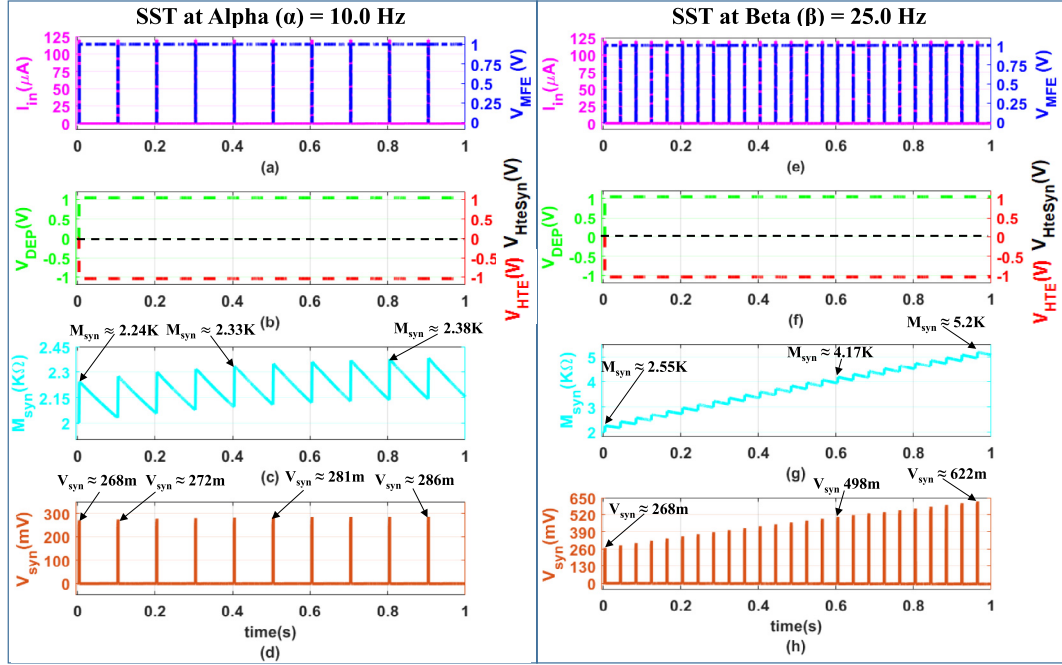


Fig. 11. Strong stimulation response of the proposed neuromemristive synapse. Strong stimulation stimuli I_{in} and 1's complimentary memory fading effect signal V_{MFE} signals (a) for Alpha-brainwaves (10 Hz) and (e) for Beta-brainwaves (25 Hz). Control signals for homosynaptic depression and heterosynaptic mode operations (b) for Alpha-brainwaves (10 Hz) and (f) for Beta-brainwaves (25 Hz). Rate of change of the synaptic strength (M_{syn}) (c) for Alpha-brainwaves (10 Hz) and (g) for Beta-brainwaves (25 Hz). Synaptic voltage (V_{syn}) of the proposed artificial synapse (d) for Alpha-brainwaves (10 Hz) and (h) for Beta-brainwaves (25 Hz).

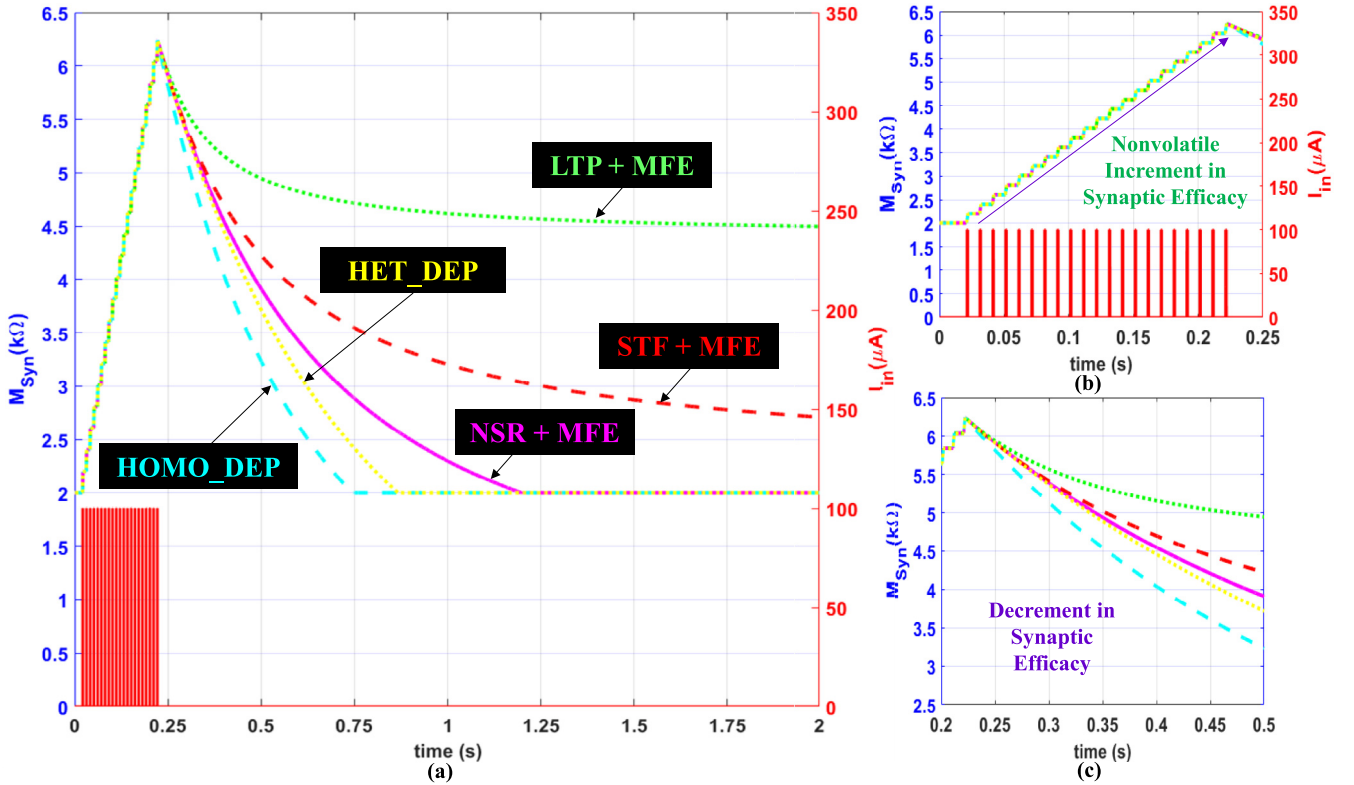


Fig. 12. Comparison of the synaptic weakening (M_{syn}) of proposed neuromemristive synapse for different plasticity modes. Observe that the synaptic efficacy of the proposed neuromemristive synapse weakens faster for homosynaptic depression than heterosynaptic depression and other synaptic operations, which act similarly to the biological synapse.

Figs. 11(b) and (f) show the homosynaptic LTD (V_{DEP}) and the inactive heterosynaptic plasticity ($V_{HTE} = V_L = -1V$ and $V_{HteSyn} = V_0 = 0V$) signals. Fig. 11(c) shows that the neuromemristive synapse (M_{syn}) builds up its efficacy at neutral frequency ($\alpha = 10$ Hz) for strong stimulation, whereas it did not modify its efficacy for regular stimulation in Fig. 4(k). Observe from Fig. 11(g) that the neuromemristive synapse accumulates higher synaptic strength (M_{syn}) for strong stimuli than in Fig. 4(o) at $\beta = 25$ Hz. Figs. 11(d) and (h) show a higher production of synaptic voltage (V_{syn}) compared to Figs. 4(l) and (p). Strong stimulation enhances the synaptic strength of the artificial synapse more than regular stimulation, thereby increasing the likelihood of postsynaptic firings. Therefore, we can conclude that the neuro-memristive synapse effectively imitates the strong stimulation of the biosynapse.

Comparison of synaptic weakening process for different plasticity modes

Section 2 emphasizes notable disparities in the synaptic weakening rate of a specific biosynapse during homosynaptic depression and homosynaptic reuptake, in contrast to heterosynaptic depression. The rate of synaptic weakening over time is dependent upon prior stimulations, similar to LTP, STF, or NSR. A biosynapse exhibiting LTP requires significantly more time to weaken its efficacy than that of STF and NSR exhibiting synapses. Therefore, to demonstrate the synaptic weakening attributes of the proposed neuromemristive synapse, the same input stimuli ($I_{in} = 100\mu A$, $PW = 1$ ms, and $PP = 10$ ms ($\gamma = 100$ Hz)) was provided to the different synaptic modes, as shown in Fig. 12. The neuromemristive synapse monotonically increases its synaptic efficacy (M_{syn}) in the active cycle of I_{in} for the synaptic modes of homosynaptic NSR, STF, and LTP, homosynaptic depression (STD or LTD), and heterosynaptic depression, as shown in Fig. 12(b). Observe that the nonvolatile increment of the M_{syn} is exactly the same ($0 \leq t \leq 0.22$ s) for all the aforementioned synaptic modes. The proposed synapse initiates the synaptic weakening process at the same time ($t > 0.22$ s), as shown in Fig. 12(c). The homosynaptic NSR, STF, and LTP-exhibiting synapses initiate weakening only through the active MFE route, and both homosynaptic and heterosynaptic depressions weaken through their specified routes. Fig. 12 shows that the artificial synapse acts like the biological one and swiftly weakens its efficacy in homosynaptic depression compared to heterosynaptic depression. Moreover, it weakens faster than that of NSR, STF, and LTP exhibiting synapse with active V_{MFE} . Similar to biosynapse, the synaptic strength of the proposed LTP exhibiting artificial synapse decreases more gradually than that

of STF and NSR. Therefore, we can conclude that the synaptic weakening response of the proposed neuromemristive synapse is qualitatively equivalent to the attributes of a biological synapse.

Power dissipation of the proposed neuromemristive synapse

Table 5 illustrates the maximum power dissipation per cycle of the proposed neuromemristive synapse and the synapses presented in [21] and [22] across several synaptic operation modes. The neuromemristive synapse exhibits the highest power consumption in the heterosynaptic modular input-specific function, whereas the homosynaptic non-plastic synaptic response (NSR) consumes the least power. Factors such as the amplitude, frequency, and pulse width of both homosynaptic and heterosynaptic stimulations significantly influence the power dissipation. The proposed synapse demonstrates reduced power dissipation during long-term plasticity and strong stimulation ($\beta = 25$ Hz) compared to the power dissipation reported in [21] and [22] under similar input conditions as shown in Table 5. Table 5 also shows that the proposed synapse exhibits significantly higher power dissipation during short-term plasticity. Such higher power dissipation occurs due to variations in homosynaptic stimulation patterns compared to those in [21] and [22]. The power consumption of the proposed synapse is qualitatively comparable to that of biological synapses, which requires substantial energy to process information for homosynaptic long-term synaptic plasticity, strong stimulations, and heterosynaptic plasticity functions.

Synaptic performance, limitations, and future perspectives

The proposed neuromemristive synapse exhibits robustness due to its bio-realistic operation, energy efficiency, and CMOS compatibility, which helps overcome the mechanical instability and stochastic switching of alternative technologies. The proposed neuromemristive synapse demonstrates robustness due to its bio-realistic operation, energy efficiency, and CMOS compatibility. These attributes allow it to overcome the mechanical instability and stochastic switching prevalent in alternative technologies mentioned in the introduction section. Compared to other state-of-the-art existing synapses, our proposed synapse demonstrates high specificity. It mimics distinct biological mechanisms, including both homo- and heterosynaptic plasticity (STF, STD, LTP, LTD), reuptake processes, and input-specific associativity properly, which other synapses lack. We utilize a single memristor and a controlled capacitor to design the proposed synapse, which highlights the scalability due to the inherent miniaturization potential

Table 5

Power dissipation of the proposed neuromemristive, ref [21], and ref [22] synapses in a cycle for different synaptic modes of operations.

Synapse Type	Synaptic Response	Power Dissipations of Proposed Neuromemristive Synapse (μW)	Power Dissipations of Ref [21] Synapse (μW)	Power Dissipations of Ref [22] Synapse (μW)
Homosynaptic	Nonplastic Synaptic Response (NSR)	113.4	283.673	196.553
	Synaptic Response for $\beta = 25$ Hz	189.15	–	–
	Short-term Plasticity (STF + STD)	280.612	137.85 (STF+MFE)	131.994
	Long-term Plasticity (LTP + LTD)	444.659	694.43 (LTP+MFE)	452.95
	Strong Stimulation (SST)	416.574 ($I_{in} = 120\mu A$)	643.244 ($I_{in} = 130\mu A$)	424.921 ($I_{in} = 130.2 \mu A$)
Heterosynaptic	Heterosynaptic Facilitation	465.59	–	–
	Heterosynaptic Depression	409.005	–	–
	Homeostasis role	451.325	–	–
	Modular Input-specific role	670.555	–	–
	Associative role	642.585	–	–

of memristive devices, their low power consumption, and their area-efficient architecture. In addition, the proposed synapse allows for post-fabrication calibration. The calibration can be achieved by initializing the memristor within a linear region (2 K–14 K), adjusting transistor bias, and controlling the system input. Specifically, we can control the resting membrane (V_{REST}) and hyperpolarize membrane (V_{HYP}) potentials to calibrate the transistors, as these voltages control the discharge rate for the memory fading effect, homosynaptic depression, heterosynaptic depression, and partial discharging blocks. Therefore, the proposed synapse might be suitable for large-scale integration in spiking neural networks (SNNs) and a viable platform for brain-like intelligence.

Despite the aforementioned promising results, one of the limitations of this study is the absence of physical fabrication data, since we confined the validation to simulation environments. SPICE simulations provide critical insights into functional behavior; however, they are unable to illustrate the consequences of process variation and reliability issues that are inherent in physical hardware. Moreover, we present a successful operation of a single synapse without system-level integration, which is insufficient to guarantee performance in a large-scale neuromorphic system. Therefore, our proposed synapse still needs to prove its path to large-scale manufacturing and integration into functional neuromorphic computing systems. Our near future work will therefore focus on fabricating the synapse in a standard CMOS process and characterizing its performance, variability, and endurance. The subsequent objective is to demonstrate the operation of small-scale spiking neural networks (SNNs) with Hebbian, anti-Hebbian, and associative learning using these neuromemristive synapses and neurons to validate their collective learning capabilities and scalability. Additionally, we want to fabricate an industrial design of the proposed neuromemristive synapse and utilize it in designing human brain-like intelligence in future.

Conclusion

The proposed neuromemristive synapse mimics the bio-realistic characteristics of a synapse and its plasticity according to the brainwaves of various brain states. It imitates the homosynaptic short- and long- term plasticity (STF, STD, LTP, and LTD) as well as the LTP, LTD, homeostatic, and modular input-specific roles of heterosynaptic plasticity. The proposed synapse also exhibits reuptake and diffusion mechanisms, which involve the removal of neurotransmitters from the synaptic cleft. Furthermore, the proposed synapse demonstrates bio-realistic behaviors of a neuron, such as strong stimulation, an exponentially decaying conductance trace, and voltage-dependent synaptic responses. Various SPICE simulations verified the mimicking features of the proposed artificial synapse in relation to biological phenomena.

Unlike multiple memory elements-based prior artificial synapses, the proposed neuromorphic circuit was implemented in a circuit with off-the-shelf devices and can be utilized in both volatile and nonvolatile configurations. It is not simply another synaptic model, relying solely on mathematical or macro-models of the memristor. We can implement the proposed power- and area- efficient bio-inspired synapse in miniature CMOS ICs because the mem-conductive neuromorphic architectures overcome the limitations of graphene, liquid crystal carbon nanotube composites, subthreshold analog- and transistor-based CMOS neural cir-

cuits, and conventional Von-Neumann architectures. In addition, academics can also utilize it to analyze the synaptic functionalities of neurons, replacing the need for in vivo or in vitro analysis.

Data availability

We will make data from simulations and circuit implementations available upon request.

Declaration of generative AI in scientific writing

We wrote and revised the manuscript by ourselves. We used QuillBot Premium only as a grammatical checker. We accepted some of QuillBot's suggestions for clarity in the sentences.

CRediT authorship contribution statement

Zubaer Ibna Mannan: Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing. **Sami Azam:** Formal analysis, Funding acquisition, Investigation, Project administration, Resources, Supervision, Validation, Visualization, Writing – review & editing. **Ram Kaji Budhathoki:** Formal analysis, Investigation, Resources, Validation, Visualization, Writing – review & editing. **Nur Alam:** Formal analysis, Investigation, Resources, Validation, Visualization, Writing – review & editing. **Hyongsuk Kim:** Funding acquisition, Investigation, Project administration, Resources, Supervision, Visualization, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix

A modified memristor emulator [68], illustrated in Fig. A1(a), was employed to design the proposed neuromemristive synapse due to the unavailability of commercial memristors. The emulator was operated within the linear resistance range (2 k Ω – 14 k Ω) to mitigate undesired effects stemming from nonlinear ionic dopant drift at boundary conditions [69,70], as illustrated in Fig. A1(b). The memristive fingerprints [71] of zero-crossing and frequency-dependent pinched hysteresis loops of the modified memristor emulator ($M(0) = 2$ K, $C_T = 2\mu\text{F}$, and $R_T = 4$ K) for bipolar sinusoidal and pulse waves are shown in Figs. A1 (c) and (d), respectively. The pinched hysteresis loop lobe area decreases with increasing frequency, converging to a single-valued straight line for $f \geq 100$ Hz. This behavior confirms that the emulator exhibits the defining characteristics of a memristor [71].

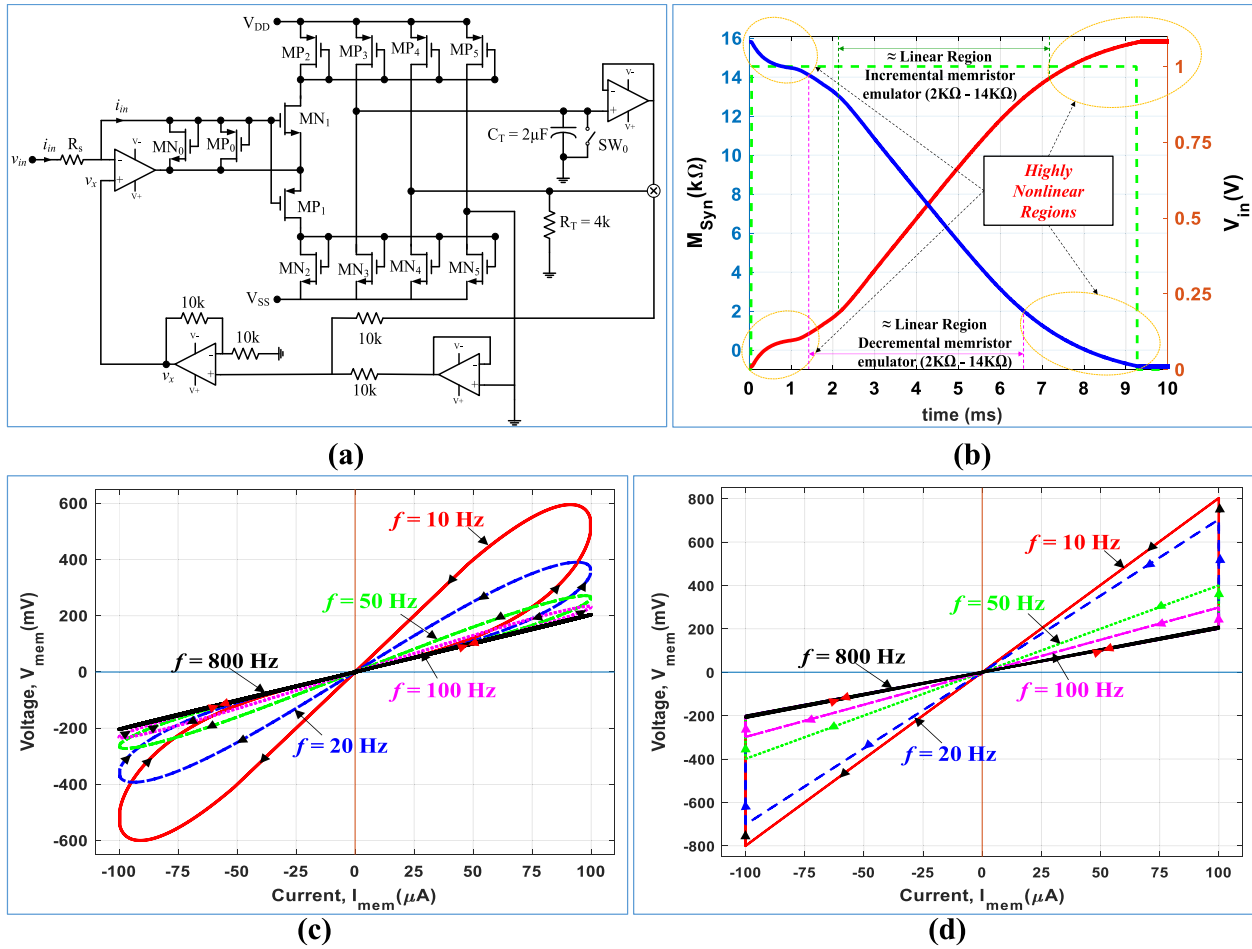


Fig. A1. (a) Circuit diagram of the incremental memristor emulator that used to design the neuro-memristive synapse. (b) Memristance vs. time relationship of the incremental and decremental memristor emulator. Frequency-dependent pinched hysteresis loops for zero-mean periodic stimulation of $I_{in} = 100 \mu A$ for (a) bi-polar sinusoidal input, and (b) pulse input with frequencies $f = 10$ Hz, 20 Hz, 50 Hz, 100 Hz, and 800 Hz.

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