Memory consolidation facilitated by burst-induced late-phase plasticity

How do alternating periods of learning and rest contribute to memory consolidation? While it is recognized that learning relies on synaptic plasticity triggered by the spiking activity correlation between neurons, the role of rest periods and their biophysical mechanisms remain elusive. In this work, we leverage the interaction between the brain state fluctuations, reflecting changes in neuronal excitability, and memory, relying on synaptic plasticity occurring at different phases. Our approach involves a neural network model capable of transitioning between learning periods characterized by fast low-amplitude oscillations, and rest periods marked by slower large-amplitude oscillations. At the neuronal level, it is characterized by biophysical neurons capable of switching between input-driven tonic firing and the less-explored collective bursting.

In our model, synapses exhibit calcium-based early-phase plasticity, as studied in previous work. Here, we propose a new additional burst-induced late-phase plasticity mechanism. During learning, the early-phase plasticity forms new memories, as traditionally observed. During rest, the early-phase plasticity resets, returning to its baseline set point. It provides a physiological trace to drive the late-phase plasticity facilitating memory consolidation.

Validating our model through a memory task utilizing the MNIST dataset, we demonstrate that switching from tonic to burst, combined with early- and late-phase plasticity enables the network to acquire new information while preserving existing memories. The collective bursting activity during rest, combined with late-phase plasticity, represents the generation of new postsynaptic proteins and morphological synapse changes (termed structural plasticity). We find that substituting rest with an additional learning period impedes memory consolidation, rendering it susceptible to noise.

These findings propose a potential biological mechanism for unsupervised memory consolidation during rest and explain how the brain balances synaptic homeostasis and memory processes. Moreover, they suggest the utility of incorporating rest periods into machine learning models, highlighting the importance of including collective bursting and structural plasticity.

A network model able to switch from input-driven tonic firing (learning) to collective bursting (rest)

The brain manifests distinctive activity patterns during learning and rest, distinguished by a transition at the neuronal level from *tonic* to *burst* firing (Fig. 1A, left-top). In this work, we replicate this neural state transition with a network of conductance-based model neurons performing a classical supervised learning task – MNIST digit classification. During learning, 484 presynaptic neurons represent individual image pixels, and 10 postsynaptic neurons represent the corresponding digit class. Presynaptic neurons spike according to the pixel intensity, while the postsynaptic neuron corresponding to the presented digit class spikes with high frequency, mimicking supervised learning. In the rest period, pre- and postsynaptic neurons exhibit synchronized bursts due to intrinsic ion channel properties. A neuromodulator (NM) projection, acting as an inhibitory input, controls the switch from learning to rest periods (Fig. 1A, right).

Early- and late-phase synaptic plasticity in learning and rest

In biological neurons, synaptic plasticity involves an early phase, marked by increased postsynaptic receptor efficacy and rapid receptor insertion, followed by a late phase dependent on de-novo protein synthesis and morphological changes (Fig. 1B, left-bottom) [Poirazi et Mel, 2001; Lamprechts et LeDoux,2004]. To model the early and late phases of plasticity, we suggest two components of the synaptic weight: the early weight undergoing calcium-based early-phase plasticity (w) [Graupner and Brunel, 2012] and the late weight undergoing late-phase plasticity, as proposed in prior studies [Clopath et al., 2008; Luboeinski et Tetzlaff, 2021]. The resultant effective synaptic weight is the product of the early- and late-weight. However, in this work, we replace the existing late-phase plasticity with our *novel burst-induced late-phase plasticity*. During learning, a high-firing presynaptic neuron (corresponding to a bright pixel) increases its early weight with the concurrently activated output neuron, replicating early-phase plasticity. During rest, we observe each early weight undergoes a burst-

induced reset, returning to baseline. The late weight follows the negative derivative of the early weight dl=-a dwt/dt, where a is a tunable proportionality factor. As the early weight resets and restores postsynaptic receptor efficacy, the late weight increases, modeling new protein generation and morphological changes driven by early synaptic modifications.

The synergistic role of collective bursting and late-phase plasticity facilitates memory consolidation

Our objective is to investigate the impact of rest periods on memory consolidation. At the end of each learning and rest period, we visualize the receptive field (RF) which illustrates the weight matrix associated with the output neuron. Through a repetitive cycle of learning (dark blue) and rest (light blue), we observe the RF consolidation (Fig. 1Bi). This consolidation demonstrates the network adaptability in learning digits and its resilience against noise. Interestingly, substituting rest periods with additional learning reveals a limitation: only the last presented digit is encoded (Fig. 1bii). Moreover, blocking late-phase plasticity—mimicking the effects of protein synthesis inhibitors— reveals the early-weight reset and failure to consolidate memory (Fig. 1biii). Replacing bursting with low-frequency spiking neurons prevents the reset of early weights but also restricts the network from acquiring new memory, which subsequently fades out over time (Fig. 1biv). These alterations make the network notably susceptible to noise.

Conclusion

This uniquity in this work lies in combining neuronal activity switches (tonic and burst firing, associated with learning and rest) with early- and late-phase plasticity models. We propose a model of burst-induced late-phase synaptic plasticity, exploiting the early weight as an eligibility trace. It provides insights into biological mechanisms during rest and offers a versatile tool for broader networks or diverse memory tasks. Importantly, this model hints at potential enhancements for learning algorithms without necessitating replay or recall mechanisms.

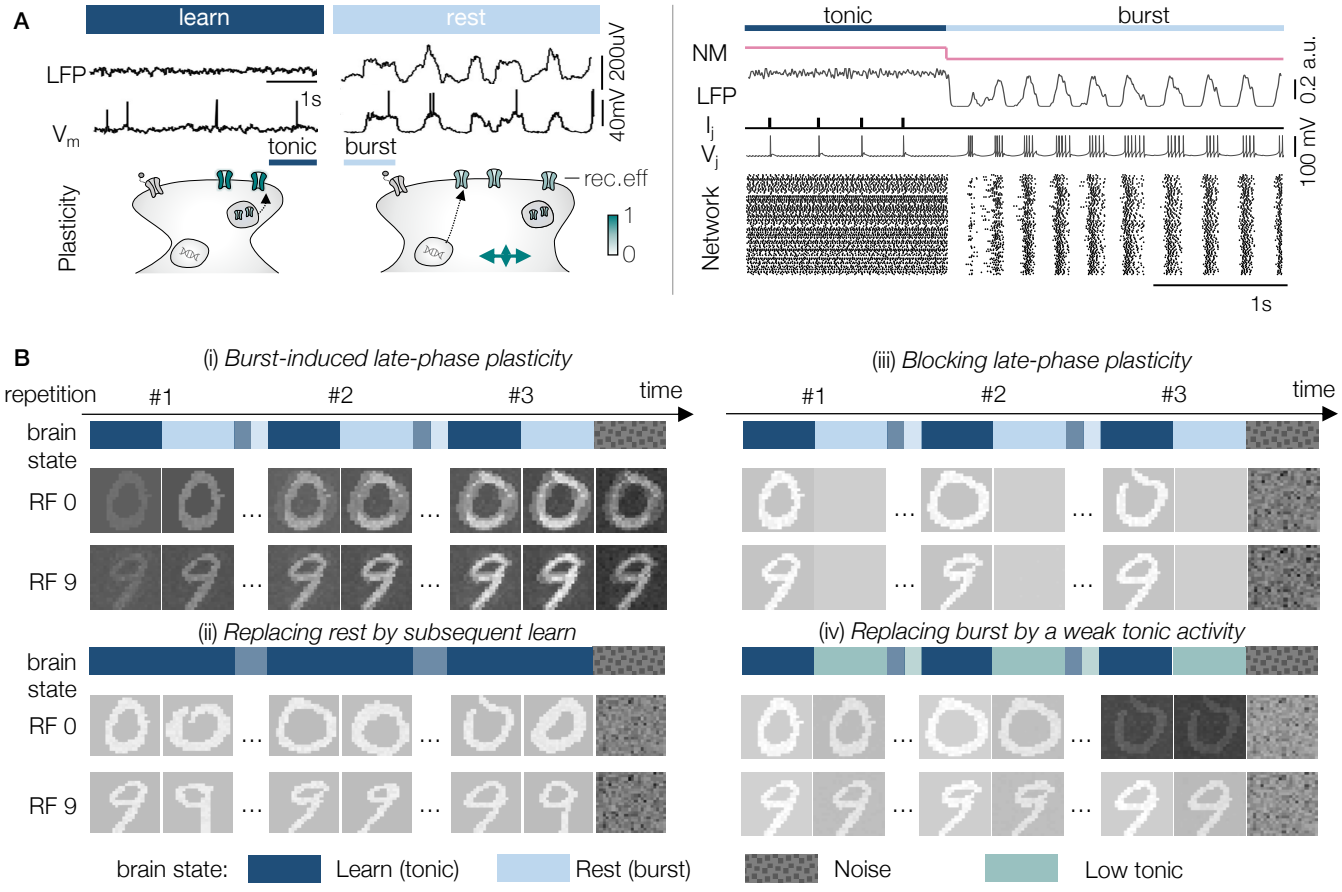

Fig 1. A. Switches in brain states with the associated neural activity and plasticity mechanisms (left) modeled in a neural network (right). **B.** Receptive fields (RF) for the output neurons associated with the digits 0 and 9 with different combinations of neural activity and plasticity mechanisms.