The endogenous nature of bursting leads to homeostatic reset in synaptic weights: a key player to regularize network connectivity during sleep.

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Learning and memory rely on the ability of neurons to form new connections, a property called synaptic plasticity. Synaptic connections can be strengthened or weakened via plasticity rules sensitive to neuronal firing. Simultaneously, brain information processing is shaped by fluctuations in neuronal activities, defining brain states. A well-known example of brain state switches is the transition from wakefulness to sleep. It is characterized by a change in population rhythm from active to oscillatory state, while at the cellular level neurons switch from tonic to burst. Altogether, it raises the question of how changes in neuronal activity affect memory formation and more precisely how switches from tonic to burst impact synaptic plasticity.

To investigate this question, we used a cortical network built with conductance-based neuron models able to switch between tonic and burst (Fig 1A). The synaptic connections within the network are plastic. They are driven either by phenomenological rules, such as pair-based [Pfister,2006] or calcium-based rules [Graupner,2016]. These rules are fitted on experimental data [Sjostrom,2001]. We showed that a switch to burst reminiscent of sleep leads to a *homeostatic reset* of synaptic weights, meaning that all weights converge towards a basal value (Fig 1B).

Here, we developed analytical analyses to understand the mechanisms underlying this reset and predict its value. For phenomenological plasticity rules, potentiation and depression balance leading to a converging point for the synaptic weight. The burst induces a homogeneous spike train correlation between pre and postsynaptic firing activity thanks to the stationarity during sleep (Fig 1C, left). By contrast, in wakefulness, the correlation is highly heterogeneous. It comes from the variability in spiking activity used for the quick processing of incoming information such that no equilibrium is reached (Fig 1C, right). A similar analysis is derived for calcium-based rules. The burst of action potential drives homeostatic fluctuations in calcium activity. Once again, the burst generates a balance between potentiation and depression unreached during wakefulness. Altogether, the mechanisms of the synaptic reset are *rooted* in the *endogenous* nature of the sleep-like bursting activity (Fig 1C-D).

Additionally, we show that the homeostatic reset is robust to neuronal variability and network heterogeneity. The sleep-dependent reset could play a central role in sleep homeostasis and sleep-dependent memory consolidation.

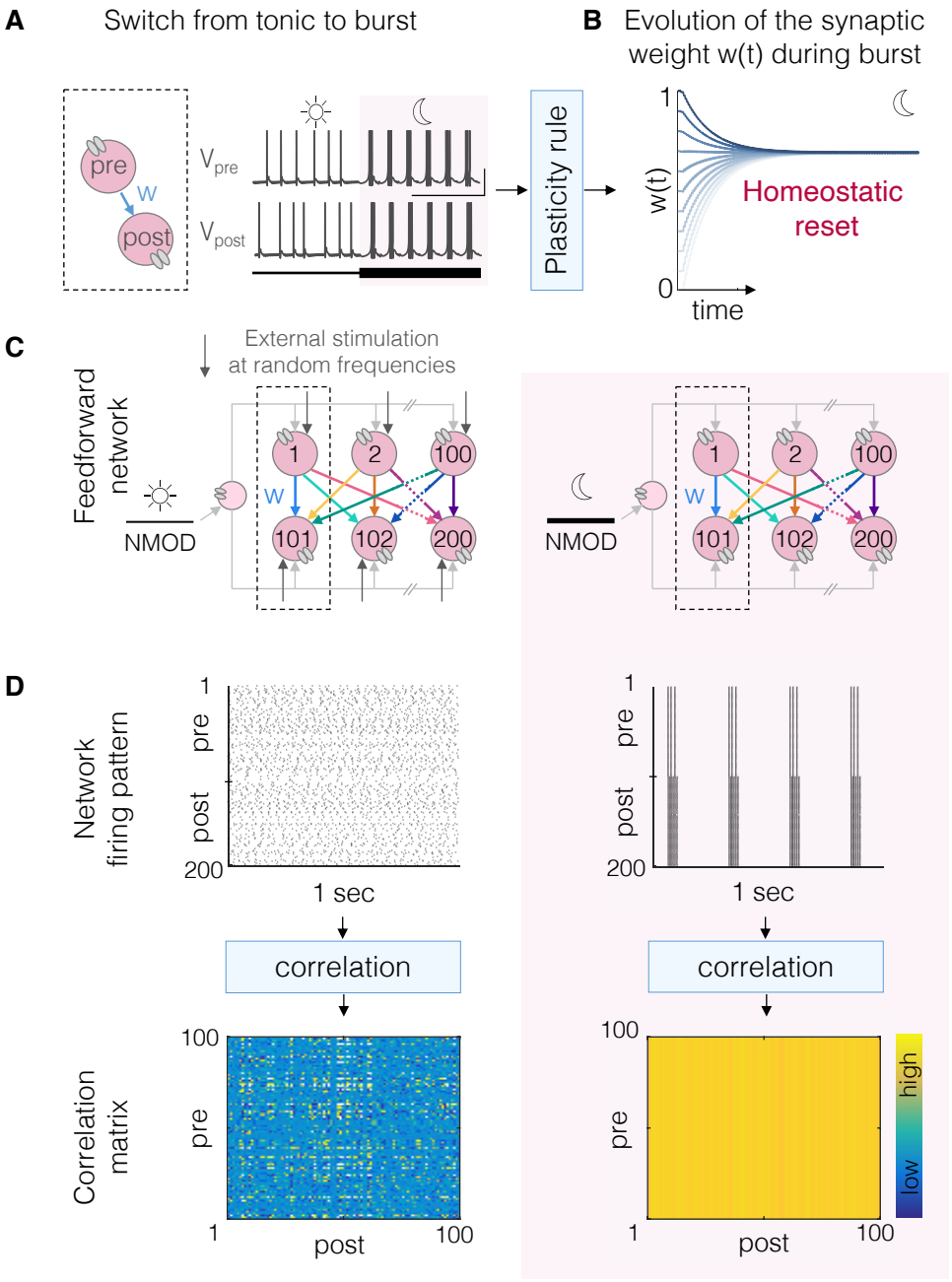

Fig 1A. Switch from tonic to burst in a 2-cell circuit. **B.** The synaptic strength converges towards a basal value during burst. **C.** A 2-layer network with 100 pre- to 100 postsynaptic neurons. **D.** Network activity during wakefulness (sleep) leads to a heterogenous (homogeneous) correlation matrix.