Automatic modulation of critical brain dynamics

Several lines of electrophysiological, blood-oxygenation-level dependent signal imaging and behavioral evidence show that many features of central-nervous system activity are scale-free. The scale-free dynamics is relevant because it is a signature characteristic of complex systems poised at criticality. Operating at a critical state endows the system maximal dynamic range and optimal information storage and transmission capacity. The scale-free dynamics of a near-critical complex system can be quantitatively described by the corresponding power-law scaling exponents of long-range temporal correlations (LRTCs). Discovering means to influence brain criticality would be important for both examining its functional role in cognition and for developing novel therapeutic approaches for brain disorders associated with abnormal LRTCs.

It has been suggested that the net balance between excitation and inhibition is the control parameter that tunes the brains to operate in the critical regime and to avoid the sub-critical and super-critical states that are associated with aberrant levels of neuronal inhibition and excitation, respectively. We advance a closed-loop neurofeedback stimulation paradigm that may modulate the excitation/inhibition (E/I) balance and the LRTCs. Changes in neuronal activity caused by neurofeedback training are likely associated with systematic shifts in the cortical E/I balance as shown, *e.g.*, by cortico-spinal excitability measurements. In particular, voluntary suppression of *?* -activity increases the cortico-spinal excitability and decreases the intra-cortical inhibition which shows that cortical E/I balance can be directly modulated via neurofeedback. However, to achieve such E/I shifts during cognitive tasks or in clinical applications, automatic means for E/I modulation would be preferable over voluntary and cognitive-effort demanding methods. Closed-loop stimulation is a neurofeedback paradigm that combines the endo- and exogenous approaches so that specific aspects of neuronal activity as used to trigger sensory stimuli that then reciprocally influence the ongoing neuronal activity.

In this study, we developed electroencephalography (EEG) based closed-loop stimulation paradigm where high-amplitude *?*-waves trigger visual flash stimuli during an eyes-closed resting condition. The stimulation threshold allows intrinsic neuroregulation to control the stimulation rate through *?*-wave amplitude adjustment. Given the direct link of *?*-oscillations and excitability, such adjustments are associated with shifts in the E/I balance. Moreover, because the subjects are not informed about the connection between ongoing neuronal activity and the visual stimuli, the paradigm assesses specifically the effects of endogenous adaptive. We hence hypothesized that the closed-loop stimulation changes LRTCs, which would imply a change in the operating point of brain dynamics along the sub-/super-critical axis. We tested the hypothesis by measuring the scaling exponents of EEG LRTCs during the neurofeedback (closed loop) and sham (disconnected loop) conditions.

Our results show that closed-loop stimulation induces changes in LRTCs and hence in critical dynamics without changes in the global power of neuronal oscillations. Importantly, the comparison with the sham condition showed that while both conditions were characterized by overall

suppressed LRTCs, which is attributable to the sensory stimuli disrupting endogenous dynamics, the effect of closed-loop stimulation was to enhance the LRTCs. The changes in the LRTCs but not in the power of ?-oscillations during the stimulation suggest that the brains prevent the global cortical excitation by preserving E/I balance.

The proposed approach demonstrated the possibility for automatic modulation of LRTCs. The effect size of automatic modulation was smaller than that attributable to the disruptive effects of the visual stimuli per se, but prolonged stimulation as well as using more subtle, lower intensity stimuli could both increase the effect and decrease the stimulation related disruption of cortical dynamics. This approach thus opens novel avenues for both examining the functional roles of brain criticality in healthy subjects and for developing novel therapeutic approaches for brain disorders associated with abnormal LRTCs.

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