The dynamics of neural circuits during transcranial magnetic stimulation

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Venue
Lecture room 104, Project Research Building[DO4] 1F , Graduate School of Life Sciences( Katahira Campus)

Repetitive transcranial magnetic stimulation (rTMS), a non-invasive brain stimulation method, has gained considerable interest in experimental and clinical neuroscience for its capability of activating cortical neuronal populations and inducing plasticity. Our current understanding of the physiological mechanisms of rTMS is limited. A rough insight is provided by histological studies of rTMS treated rats. Depending on the used rTMS stimulation protocol, the protein expression of calcium-binding-proteins, which are expressed predominantly in inhibitory cells, is changed. Intermittent Theta Burst Stimulation (iTBS) leads to a decrease in the number of parvalbumin-positive cells, whereas the continuous theta burst stimulation (cTBS) and 1 Hz reduce the number of calbindin-positive neurons. These changes occur in both anesthetized and awake behaving animals. Learning processes can counteract the rTMS-induced decrease in protein expression and behavioral tasks show that the rTMS treated animals can learn faster. These results suggest that different rTMS protocols may affect specific aspects of inhibition and network activity and function. This is in agreement with the human TMS EEG experiments which show that GABAergic inhibitory transmission has a strong modulation of cortical excitability and connectivity. However, direct experimental evidence for the effect of rTMS on inhibition is still lacking since even the neural effects induced by a single-TMS pulse (spTMS) remain poorly understood.

The strong electromagnetic field induced by the TMS so far has prevented the study of the direct TMS-evoked electrical activity at the neuronal level. I will present a novel method that provides direct in vivo access to TMS-evoked neuronal activities in laboratory rodents by removing the induced electrical artifacts. Exploiting this method, we investigated the time-dependent responses of motor cortex neurons to spTMS, as it is routinely used in humans. We found high-frequency spiking within the first 6 ms which depended critically on the TMS coil orientation (medio-lateral vs posterior-anterior orientation), and a multiphasic spike–rhythm alternating between excitation and inhibition 6–300 ms after the stimulation pulse. The observed activation patterns show strong similarities with the ones recorded at the level of the spinal cord and of muscles during TMS in humans. Our results provide a new level of insight into the physiological basis of TMS-induced neural activity which will be helpful for future improvements of this non-invasive method for the treatment of the human brain.

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