

Neuronal circuitry AND rTMS in depression

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In the pathophysiology of depression, research is increasingly focused on deficits in structure and function of specific brain areas and of neural networks. One of those circuits is the limbic-cortico-striatal-pallidal-thalamic network. In depression, the activity of the amygdala, thalamus and ventral limbic cortical regions is increased, whereas the activity of dorsal prefrontal cortical regions is decreased. The dorsolateral prefrontal cortex (DLPFC) is thought to evaluate impulsive, emotional reactions on affective stimuli and place them in a context. The decrease in DLPFC function in depression may play a pathophysiological factor in the development of depressed mood.

Clinically effective treatment with psychotherapy or medication appears to (partly) normalize these abnormal activations. In the last decade, repetitive transcranial magnetic stimulation (rTMS) has been investigated in many clinical trials as a new therapy for depression. This is because rTMS can inhibit or stimulate cortical brain regions beyond the period of actual stimulation. Stimulation with low frequency (< 1 Hz) decreases cortical activity, whereas stimulation with high frequency (5-20 Hz) increases cortical activity. Those changes occur both locally and at a distance in functionally connected brain areas. Theoretically, this renders rTMS capable of modifying and normalizing dysfunctioning networks. Until now, in most trials high-frequency rTMS has been applied over the left DLPFC, in order to restore the normal activity in the hypoactive prefrontal cortex. The effect on clinical symptoms has been investigated. In this talk I will describe and discuss the controlled clinical trials that have applied rTMS to the left DLPFC to treat depression. The statistically significant therapeutic effects in these, small sized, trials support a role of the DLPFC in mood regulation. However, the clinical implications still seem limited.

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Speaker session 39