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White matter changes following electroconvulsive therapy for depression: a mega-analysis

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Introduction: Electroconvulsive therapy (ECT) is proposed to exert an effect on white matter (WM) microstructure, but the limited power of previous studies made it difficult to highlight consistent patterns of change in diffusion metrics.

Objectives: We initiated a multi-site mega-analysis and sought to address whether changes in WM microstructure occur following ECT.

Methods: To this end, diffusion tensor imaging (DTI) data (n=58) from 4 different sites were harmonized before pooling them by using ComBat, a batch-effect correction tool that removes inter-site technical variability, preserves inter-site biological variability and maximizes statistical power. Downstream statistical analyses aimed to quantify changes in Fractional anisotropy (FA), Mean Diffusivity (MD), Radial Diffusivity (RD) and Axial Diffusivity (AD), by employing whole-brain, tract-based spatial statistics (TBSS).

Results: ECT increases FA in the right splenium of the corpus callosum and the left cortico-spinal tract. Both the left superior longitudinal fasciculus and the right inferior fronto-occipital fasciculus showed increases in AD. Increases in MD and RD could be observed in overlapping white matter structures of both hemispheres. Finally, responders showed significantly smaller FA values in the left forceps major and smaller AD values in the right uncinate fasciculus compared with non-responders.

Conclusions: This is the first and largest multi-site mega-analysis to demonstrate that ECT normalizes altered WM microstructure in important brain circuits that are implicated in the pathophysiology of depression. Furthermore, responders appear to present a more decreased WM integrity at baseline, which if replicated could serve as a biomarker for ECT response.

Disclosure: No significant relationships.

Keywords: Diffusion Tensor Imaging; Electroconvulsive therapy; Tract-Based Spatial Statistics; Depression

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N-Methyl-D-Aspartate Receptor availability in First-Episode Psychosis: a multi-modal PET-MR brain imaging study

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Introduction: N-Methyl-D-Aspartate Receptor (NMDAR) hypofunction is hypothesised to underlie psychosis but this has not been tested early in illness.

Objectives: Our aim was to determine if NMDAR availability was lower in patients with first episode psychosis compared to healthy controls.

Methods: To address this, we studied 40 volunteers (21 patients with first episode psychosis and 19 matched healthy controls) using PET imaging with an NMDAR selective ligand, [¹⁸F]GE179, that binds to the ketamine binding site to index its distribution volume ratio (DVR) and volume of distribution (V_T). Striatal glutamatergic indices (glutamate and Glx) were measured simultaneously using magnetic resonance spectroscopy imaging (¹H-MRS).

Results: Hippocampal DVR, but not V_T, was significantly lower in patients relative to controls (p=0.02, Cohen's d=0.81; p=0.15, Cohen's d=0.49), and negatively associated with total (rho=-0.47, p=0.04), depressive (rho=-0.67, p=0.002), and general symptom severity (rho=-0.74, p<0.001). Exploratory analyses found no significant differences in other brain regions (anterior cingulate cortex, thalamus, striatum and temporal cortex). We found an inverse relationship between hippocampal NMDAR availability and striatal glutamate levels in people with first-episode psychosis (rho = -0.74, p <0.001) but not in healthy controls (rho = -0.22, p = 0.44).

Conclusions: These findings are consistent with the NMDAR hypofunction hypothesis and identify the hippocampus as a key locus for relative NMDAR hypofunction, although further studies should test specificity and causality.

Disclosure: No significant relationships.

Keywords: Psychosis; Neuroimaging; NMDAR; Glutamate

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The thalamus and its subregions – a gateway to obsessive-compulsive disorder

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Introduction: Higher thalamic volume has been found in children with obsessive-compulsive disorder (OCD) and children with clinical-level symptoms within the general population (Boedhoe