O0035

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

J.-B. Belge[1]*, P. Mulders[2], L. Van Diermen[1], A. Dols[3], M. Oudega[3], I. Tendolkar[2] and P. Van Eijndhoven[2]

[1]University of Antwerp, Collaborative Antwerp Psychiatric Research Institute (capri), Duffel, Belgium; [2]Radboud University Medical Center, Donders Institute For Brain, Cognition And Behavior, Centre For Neuroscience, Nijmegen, Netherlands and [3]Amsterdam University Medical Center, Amsterdam Neuroscience, Amsterdam, Netherlands
*Corresponding author.
doi: 10.1192/j.eurpsy.2022.237

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

O0036

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

K. Beck[1]*, A. Arumuham[1], S. Brugger[2], R. Mccutcheon[1], M. Veronese[1], S. Kaar[1], T. Pillinger[1], J. Stone[3] and O. Howes[1]

[1]Institution of Psychiatry, Psychology & Neuroscience, Department Of Psychosis Studies, United Kingdom, United Kingdom; [2]Cardiff University Brain Research Imaging Centre (CUBRIC), School Of Psychology, HQ, United Kingdom and [3]Brighton and Sussex Medical School, University Of Sussex, Brighton, United Kingdom
*Corresponding author.
doi: 10.1192/j.eurpsy.2022.238

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, [^{18}F]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 (^{1}H-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

O0037

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

C. Weeland[1]*, C. Vriend[1], Y. Van Der Werf[1], C. Huyser[2], M. Hillegers[3], H. Tiemeier[4], T. White[5], N. De Joode[1], P. Thompson[6], D. Stein[7], O. Van Den Heuvel[1] and S. Kasprzak[1]

[1]Amsterdam UMC, Department Of Anatomy And Neurosciences; Department Of Psychiatry, Amsterdam, Netherlands; [2]Academic Center for Child and Adolescent Psychiatry, De Bascule, Amsterdam, Netherlands; [3]Erasmus Medical Center, Department Of Child And Adolescent Psychiatry, Rotterdam, Netherlands; [4]Harvard TH Chan School of Public Health, -, Boston, United States of America; [5]Erasmus MC, Child And Adolescent Psychiatry And Psychology, Rotterdam, Netherlands; [6]eImaging Genetics Center, Stevens Institute for Neuroimaging & Informatics, Keck School of Medicine, University of Southern California, -, Los Angeles, United States of America and [7]University of Cape Town, Dept Of Psychiatry & Neuroscience Institute, Cape Town, South Africa
*Corresponding author.
doi: 10.1192/j.eurpsy.2022.239

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

https://doi.org/10.1192/j.eurpsy.2022.237 Published online by Cambridge University Press