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Multimodal magnetic resonance spectroscopy and surface-based morphometry study of individuals at ultra-high-risk for psychosis

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doi: 10.1192/j.eurpsy.2021.362

Introduction: Studies examining gamma-aminobutyric acid (GABA) or glutamate in ultra-high risk for psychosis (UHR) have shown conflicting results, and a number of multimodal studies examining associations between metabolite and structural characteristics is very limited.

Objectives: We aimed to investigate potential associations between GABA and glutamate levels and cortical thickness in the frontal lobe in UHR individuals and healthy controls (HC).

Methods: 20 male UHR individuals and 19 healthy controls (HC) underwent structural MRI and MR spectroscopy at 3T Philips scanner. T1-weighted images were processed via FreeSurfer 6.0 to quantify cortical thickness for selected frontal regions labeled according to Desikan atlas. MEGA-PRESS acquisitions were analyzed with jMRUI (ver. 5.1 Alpha), levels of GABA and glutamate were calculated as ratios to creatine + phosphocreatine.

Results: The study revealed: 1) GABA/Cr ratios reduction in the left frontal lobe ($p=0.001$) which was not attributable to antipsychotic medication; 2) cortical thickness reductions in the left pars orbitalis ($p=0.005$) (the anterior part of the inferior frontal gyrus) in the UHR individuals compared to HC. No significant correlations between GABA/Cr ratios and cortical thickness were identified in both groups.

Conclusions: The findings indicate that the UHR state is associated with altered GABA levels and cortical thickness reductions in the prefrontal cortex. The results also show that GABA levels are not directly related to cortical abnormalities, suggesting that altered metabolite levels may be associated with a complex system of structural and functional impairments, rather than directly correlating with structural changes in separate cortical regions. The work was supported by RFBR grant 19-29-10040.

Disclosure: No significant relationships.

Keywords: Magnetic resonance spectroscopy; GABA; Cortical thickness; Ultra-high risk of psychosis

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Cortical thickness abnormalities in long-term remitted cushing's disease

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doi: 10.1192/j.eurpsy.2021.363

Introduction: Remitted Cushing's disease (RCD)-patients commonly continue to present persistent psychological and cognitive deficits, and alterations in brain function and structure. Assessing cortical thickness and surface area of RCD-patients may offer further insight into the neuroanatomical substrates of Cushing's disease.

Objectives: To assess cortical thickness and surface area in RCD-patients in comparison to healthy controls (HCs).

Methods: Structural 3T MRI's were obtained from 25 long-term RCD-patients, and 25 age-, gender-, and education-matched HCs. T1-weighted images were segmented to extract mean cortical thickness and surface area values of 68 cortical gray matter regions. Paired sample t-tests explored differences between the anterior cingulate cortex (ACC; region of interest), and the whole brain. Validated scales assessed psychiatric symptomatology, self-reported cognitive functioning, and disease severity.

Results: After correction for multiple comparisons, ROI analyses indicated that RCD-patients showed reduced cortical thickness of the left caudal ACC and the right rostral ACC compared to HCs. Whole-brain analyses indicated thinner cortices of the left caudal ACC, left cuneus, left posterior cingulate cortex, right rostral ACC, and bilateral precuneus compared to HCs. No cortical surface area differences were identified. Cortical thickness of the left caudal ACC was inversely associated with anxiety symptoms and disease duration.

Conclusions: In six of 68 regions examined, RCD patients had reduced cortical thickness in comparison to HCs. Cortical thickness of the left caudal ACC was inversely associated with disease duration, suggesting that prolonged and excessive exposure to glucocorticoids may be related to cortical thinning of brain structures involved in emotional and cognitive processing.

Disclosure: No significant relationships.

Keywords: Neuroimaging; Cushing's Disease; endocrinology; Psychiatric symptomatology

Neuroscience in psychiatry

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Association between abnormal fetal head growth and autism spectrum disorder

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doi: 10.1192/j.eurpsy.2021.364