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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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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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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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Introduction: Despite evidence for the prenatal onset of abnormal head growth in ASD children, studies on fetal ultrasound data in ASD are limited and controversial.
Objectives: To understand whether people with ASD have abnormal head growth during gestation
Methods: A longitudinal matched case-sibling-control study on prenatal ultrasound biometric measures of ASD children was conducted. Children with ASD were matched to two control groups: (1) typically developed sibling (TDS) and (2) typically developed population (TDP). The cohort comprised 528 children (72.7% males): 174 ASD, 178 TDS, and 176 TDP.
Results: Second-trimester ASD and TDS fetuses had significantly smaller biparietal diameter (BPD) than TDP fetuses (aOR_{zBPD}=0.685, 95%CI=0.527-0.890 and aOR_{zBPD}=0.587, 95%CI=0.459-0.751, respectively). However, these differences became statistically indistinguishable in the third trimester. Head biometric measures were associated with the sex of the fetus, with males having larger heads than females within and across groups. A linear mixed-effect model assessing the effects of sex and group assignment on fetal longitudinal head growth indicated faster BPD growth in TDS vs both ASD and TDP in males (β=0.084 and β=0.100 respectively; p<0.001) but not in females, suggesting an ASD–sex interaction in head growth during gestation. Fetal head shape showed sex-specific characteristics, and head growth was inversely correlated with ASD severity in males and females, thus further supporting the sex effect on the association between fetal head growth and ASD.
Conclusions: Our findings suggest that abnormal fetal head growth is a familial trait of ASD, which is modulated by sex and is associated with the severity of the disorder.

Disclosure: No significant relationships.
Keywords: autism spectrum disorder; Prenatal Ultrasound; Brain Development

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The gut-microbiome-endocannabinoid axis and anhedonia/amotivation: A mediation analysis in a general population cohort

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Introduction: General-population studies investigating the biological correlates of anhedonia/amotivation might be informative for treatment breakthroughs for a number of clinical conditions. Reduced gut-microbial diversity might lead to an anhedonic/amotivational syndrome ("sickness behaviour"). However, how gut-microbial diversity contribute to this clinical phenotype is a key gap in knowledge. We hypothesised the endocannabinoid system would be at play.
Objectives: We tested the hypothesis that the endocannabinoid system mediates the association between gut-microbial diversity and anhedonia/amotivation
Methods: Secondary data analysis on 786 volunteer twins (TwinsUK). Measures of gut-microbiome, faecal endocannabinoid metabolites, and anhedonia/amotivation were collected over five years. To test our hypothesis we used a multilevel mediation model using alpha diversity as predictor, faecal levels of the endocannabinoid palmitoylethanolamide (PEA) as mediator, and anhedonia/amotivation as outcome. Analyses were adjusted for obesity, diet, antidepressants, and sociodemographic covariates.
Results: Mean age was 65.2±7.6; 27% were obese and 4.7% were on antidepressants. Alpha diversity was significantly associated with anhedonia/amotivation (β=-0.37; 95%CI: -0.71 to -0.03; P=0.03). Faecal PEA levels mediated this association: the indirect effect was significant (β=-0.13; 95%CI: -0.24 to -0.01; P=0.03), as was the total effect (β=-0.38; 95%CI: -0.72 to -0.04; P=0.03). The direct effect of alpha diversity on anhedonia/amotivation was attenuated fully
Conclusions: We provided the first evidence showing that the association between gut-microbial features and anhedonia/amotivation is mediated by the endocannabinoid system. These findings shed light on a new therapeutic target in an area of unmet clinical need.

Disclosure: No significant relationships.
Keywords: Microbiome; Cannabis; negative symptoms; mediation

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Effects of substance misuse and family history of substance use disorder on brain structure in patients with attention-deficit/hyperactivity disorder and healthy controls

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Introduction: Literature shows overlapping alterations in brain structure in Attention-deficit/Hyperactivity Disorder (ADHD) and substance use disorder (SUD), suggesting shared pathophysiological mechanisms. It is unclear to what extent family history (trait) effects and/or substance misuse (state) effects explain the observed overlap.
Objectives: Our aim was to examine the effects of (i) SUD family history (FH) and (ii) substance misuse on brain structure in ADHD.
Methods: We compared structural MRI data (cortical thickness; subcortical volumes) between (i) ADHD subjects and controls with or without FH (ADHD-FH+: n=139; ADHD-FH-: n=86; controls-FH+: n=60; controls-FH-: n=74), and (ii) FH-matched ADHD groups with and without substance misuse and controls (ADHD +SM, ADHD-only and controls, n=68 per group). Furthermore, we explored whether FH effects were more pronounced in subjects with SUD in both parents (n=63) compared to subjects with one SUD parent (n=105) and without FH (n=160).
Results: There was no main FH effect on brain structure. ADHD +SM showed decreased CT in inferior frontal gyrus (IFG) compared to controls, while no difference was found between ADHD-only and ADHD+SM or controls. Subjects with SUD in both parents showed decreased thickness of IFG and volume of nucleus accumbens (NAcc), compared to those with one SUD parent.