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

O179

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

A. Minichino*, M. Jackson, P. Burnet and B. Lennox

Psychiatry, University of Oxford, Oxford, United Kingdom

*Corresponding author.
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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

O180

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

M. Novi[1]*, M. Paraskevopoulou[2], D. Van Rooij[3], A. Schene[4], J. Buitelaar[3] and A. Schellekens[4]

[1]Department Of Clinical And Experimental Medicine, University of Pisa, Pisa, Italy; [2]Department Of Psychiatry, Radboud University Medical Center, Nijmegen, Netherlands; [3]Department Of Cognitive Neuroscience, donders Institute For Brain, Cognition, And Behaviour, Radboud University Medical Center, Nijmegen, Netherlands and [4]Department Of Psychiatry, Donders Institute For Brain, Cognition And Behaviour, Radboud University Medical Center, Nijmegen, Netherlands

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.366

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.

Conclusions: Substance misuse in ADHD might result in smaller IFG, which is in line with findings in SUD-literature. A contribution of premorbid alterations, due to FH, could not be ruled out, particularly for IFG thickness. Future studies should further investigate the potential role of these regions in treatment and prevention strategies.

Disclosure: No significant relationships.
Keywords: Attention-deficit/hyperactivity disorder; Substance Use Disorder; Cortical thickness; Subcortical volumes

O181

Perceptual processing links autism and synesthesia: A twin study

J. Neufeld[1]*, T. Van Leeuwen[2], L. Wilsson[1], H. Norrman[1], M. Dingemanse[2,3] and S. Bölte[1,4,5]

[1]Center Of Neurodevelopmental Disorders At Karolinska Institutet (kind), Karolinska Institutet, Stockholm, Sweden; [2]Donders Institute For Brain, Cognition And Behaviour, Radboud University, Nijmegen, Netherlands; [3]Centre For Language Studies, Radboud University, Nijmegen, Netherlands; [4]Child And Adolescent Psychiatry, Stockholm Health Care Services, Region Stockholm, Stockholm, Sweden and [5]Curtin Autism Research Group, Essential Partner Autism Crc, School Of Occupational Therapy, Social Work And Speech Pathology, Curtin University, Perth, Australia

*Corresponding author.
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Introduction: Synesthesia is a non-pathological condition where sensory stimuli (e.g. letters or sounds) lead to additional sensations (e.g. color). It occurs more commonly in individuals diagnosed with Autism Spectrum Condition (ASC) and is associated with increased autistic traits and autism-related perceptual processing characteristics, including a more detail-focused attentional style and altered sensory sensitivity. In addition, autistic traits correlate with the degree of synesthesia (consistency of color choices on an objective synesthesia test) in non-synesthetes.
Objectives: We aimed to investigate whether the degree of synesthesia for graphemes is associated with autistic traits and perceptual processing alterations within twin pairs, where all factors shared by twins (e.g. age, family background, and 50-100% genetics) are implicitly controlled for.
Methods: We investigated a predominantly non-synesthetic twin sample, enriched for ASC and other neurodevelopmental disorders (n=65, 14-34 years, 60% female), modelling the linear relationships between the degree of synesthesia and autistic traits, sensory sensitivity, and visual perception, both within-twin pairs (22 pairs) and across the entire cohort.
Results: A higher degree of synesthesia was associated with increased autistic traits only within the attention to details domain, with sensory hyper-, but not hypo-sensitivity and with being better in identifying fragmented images. These associations were stronger within-twin pairs compared to across the sample.
Conclusions: Consistent with previous findings, the results support an association between the degree of synesthesia and autistic traits and autism-related perceptual features, however restricted to specific domains. Further, the results indicate that a twin design can be more sensitive for detecting these associations.

Disclosure: No significant relationships.
Keywords: autism spectrum condition; synesthesia; twin design; sensory processing

O182

The effects of recreational use of marijuana in adolescent brain health: A review

G. Kurnijuanto* and T. Kantohe

Faculty Of Medicine, Sam Ratulangi University, Manado, Indonesia

*Corresponding author.
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Introduction: Marijuana is widely used among people, recreationally and medically. However, recent studies have shown that Marijuana has negative effects on brain structures and functions.
Objectives: To discuss the effects of Marijuana use on brain development in adolescence.
Methods: The method that is used in this study is literature review, through analyzing and summarizing the data that were collected from PubMed, epidemiology articles from BNN and CDC, and other online journals to understand the effects of Marijuana on the brain development in adolescence. There were 25499 articles that were filtered and screened resulting in 10 articles that were used as data of this literature review.
Results: Marijuana effects on the brain are divided into structural changes and functional changes. Structural changes are seen in the brain hemispheres, amygdala, hippocampus, and nucleus accumbens. While functional changes are seen in behavioral and cognitive changes in everyday life and even psychotic disorders.
Conclusions: Marijuana use has shown negative effects on the human body, organs that are rich in cannabinoid receptors, especially the Brain. Therefore, Marijuana use among adolescents may disrupt their developing brain, and cause adolescents to have structural and functional changes in the brain.

Disclosure: No significant relationships.
Keywords: Marijuana; adolescent; Brain

O183

Predictive biomarkers for negative symptoms in schizophrenia

N. Cakici*, L. De Haan and N. Van Beveren

Department Of Psychiatry And Amsterdam Neuroscience, Academic Medical Center, Amsterdam, Netherlands

*Corresponding author.
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Introduction: Increasing evidence shows that impaired neuroplasticity and high inflammation play a crucial role in the pathophysiology of schizophrenia. Prospective studies demonstrated that patients with high inflammation usually have a poor treatment response and clinical practice learns that negative symptoms are challenging to treat. The predictive value of biomarkers for negative symptoms in patients with schizophrenia has sparsely been explored.
Objectives: Here, we investigated whether biomarkers are associated with negative symptoms at baseline, and whether biomarkers could predict negative symptoms after six years in patients with schizophrenia.