patients. In both IBD and depression, there is evidence of disruptions in circulating miRNAs.
Objectives: One facet of the ongoing project titled "The brain-gut axis linking inflammatory bowel disease with anxiety and depression: the inflammation-microbiome network" (CRP/ROU21-01) involves the exploration of circulating miRNA profiles in various patient groups.
Methods: These groups encompass IBD patients with symptoms of anxiety and/or depression (IBD+A&D+), patients lacking anxiety and depression symptoms (IBD+A&D-), a cohort of individuals without IBD but experiencing depressive and anxiety symptoms (IBD-A&D+), and a control group (IBD-A&D-). Thus far, our investigation has entailed screening a comprehensive panel of 179 miRNAs in the plasma of six IBD patients and 12 non-IBD patients (CTRL) to identify a subset of highly dysregulated miRNAs. MiRNA isolation was achieved using the miRNeasy Serum/Plasma Kit, and miRNA expression levels were assessed via quantitative reverse transcription-polymerase chain reaction (qRT-PCR) utilizing the Human serum/plasma focus, MIRCURY LNA miRNA Focus PCR panel (Qiagen).
Results: Our statistical analysis revealed significant differential expression in 45 miRNAs ($p<0.05$). Specifically, we identified 29 miRNAs with elevated expression and seven miRNAs with reduced expression. Among these dysregulated miRNAs, 15 (miR-223-3p, miR-143-3p, let-7f-5p, miR-30b-5p, miR-26a-5p, let-7a-5p, miR-339-5p, let-7d-5p, miR-221-3p, miR-191-5p, let-7g-5p, miR-24-3p, miR-107, miR-26b-5p, miR-320b) were associated with depression and/or anxiety and were previously identified as dysregulated in the plasma of patients in other studies. These miRNAs will soon undergo evaluation in the plasma of IBD-A&D+ and IBD+A&D+ patients.
Conclusions: These initial findings provide us with a panel of circulating miRNAs that warrant further investigation in the aforementioned patient groups. The miRNA profile we obtained may either be unique to IBD or linked to the intricate phenotypes of IBD occurring concurrently with anxiety and depression. A more profound comprehension of these mechanisms will aid in the development of enhanced
diagnostic tools and disease monitoring strategies, as well as the exploration of innovative therapeutic approaches.

Disclosure of Interest: None Declared

Neuroimaging

O0068

Longitudinal amygdala resting state functional connectivity develops differently in adolescents with internalising disorders compared to healthy peers

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Introduction: Longitudinal neuroimaging studies focused on adolescents with internalising psychopathology (i.e. with clinical anxiety and/or depression) are scarce, even though anxiety and depression are highly prevalent mental illnesses in adolescence. Often linked to comorbidity with anxiety disorders, a large proportion of depressed adolescents displays more severe symptoms and poorer response to treatment. Previous longitudinal resting-state fMRI (RS-fMRI) studies of intrinsic functional connectivity (iFC) in depressed adolescents point to dysregulation of underlying neural networks such as the corticolimbic network, including among others the amygdala and frontal regions, which are involved in emotion processing and regulation.
Objectives: This naturalistic study investigates longitudinal changes in resting-state iFC in adolescents with internalising disorders, compared with healthy peers.
Methods: 23 treatment naïve adolescent patients with clinical depression and comorbid anxiety (INT) and 24 healthy controls (HC) participated in RS-fMRI scans at baseline and after three months. Questionnaires measuring anxiety and depression were completed at both timepoints. Imaging analyses were conducted using independent component analysis (ICA) to extract 7 networks, being the default mode, frontoparietal (bilateral), affective, salience, executive control and dorsal attention network. Additional iFC of amygdala subregions, being laterobasal (LB) and centromedial (CM), was investigated using seed-based analyses. To investigate changes over time between groups, voxelwise analyses were conducted using FSL's PALM.
Results: No significant results within ICA defined networks were found. iFC between the left LB amygdala and left frontal pole significantly increased over time in patients and decreased in HC. iFC between the right LB amygdala and right pre- and post-central gyrus also significantly increased over time in patients and decreased in HC, and was significantly associated with reduction in depressive symptoms within patients.
Conclusions: This study provides initial evidence that iFC between the laterobasal amygdala and frontal regions develops differently over time in adolescents with internalising disorders compared to healthy peers and that it is associated with reduction in depressive symptoms.

Disclosure of Interest: None Declared

O0069

Abnormal Neural Activation in Attention-Deficit/Hyperactivity Disorder: A Meta-Analysis of Functional Magnetic Resonance Imaging Studies

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Introduction: Attention-deficit/hyperactivity disorder (ADHD) is a highly prevalent psychiatric condition that frequently originates in early development and is associated with a variety of functional impairments. Despite a large functional neuroimaging literature on ADHD, our understanding of the neural basis of this disorder remains limited, and existing primary studies on the topic include somewhat divergent results.
Objectives: The present meta-analysis aims to advance our understanding of the neural basis of ADHD by identifying the most statistically robust patterns of abnormal neural activation throughout the whole-brain in individuals diagnosed with ADHD compared to age-matched healthy controls.
Methods: We conducted a meta-analysis of task-based functional magnetic resonance imaging (fMRI) activation studies of ADHD. This included, according to PRISMA guidelines, a comprehensive PubMed search and predetermined inclusion criteria as well as two independent coding teams who evaluated studies and included all task-based, whole-brain, fMRI activation studies that compared participants diagnosed with ADHD to age-matched healthy controls. We then performed multilevel kernel density analysis (MKDA) a well-established, whole-brain, voxelwise approach that quantitatively combines existing primary fMRI studies, with ensemble thresholding (p<0.05-0.0001) and multiple comparisons correction.
Results: Participants diagnosed with ADHD (N=1,550), relative to age-matched healthy controls (N=1,340), exhibited statistically significant (p<0.05-0.0001; FWE-corrected) patterns of abnormal activation in multiple brains of the cerebral cortex and basal ganglia across a variety of cognitive control tasks.
Conclusions: This study advances our understanding of the neural basis of ADHD and may aid in the development of new brain-based clinical interventions as well as diagnostic tools and treatment matching protocols for patients with ADHD. Future studies should also investigate the similarities and differences in neural signatures between ADHD and other highly comorbid psychiatric disorders.

Disclosure of Interest: None Declared

Neuroscience in Psychiatry

O0070

Nicotinamide Riboside Attenuates Memory Impairment and Depressive-like Behavior in an Alzheimer's Disease Animal Model

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Introduction: Depression in Alzheimer's disease (AD) differs from major depression in terms of clinical features and treatment. Antidepressants do not provide the expected benefits in depressive symptoms accompanying cognitive decline in AD, suggesting distinct mechanisms. Emerging research suggest that compromised mitophagy, the selective removal of damaged mitochondria, may contribute to the pathogenesis of AD. However boosting nicotinamide adenine dinucleotide (NAD+) to induce mitophagy reduces amyloid β (Aβ) aggregation and enhances cognitive function in AD models (Kerr *et al.*,Trends Neurosci 2017;40:151-66). Nevertheless, data on NAD's impact on depression in AD remains limited.
Objectives: This study aimed to examine the impact of the NAD+ precursor nicotinamide riboside (NR) on cognitive and neuropsychiatric symptoms in a AD rat model.
Methods: To induce the AD, a single dose of 5 µl Aβ1-42 was injected into each lateral ventricle of rats (day 0), while the control group received an intracerebroventricular (icv) saline (0.9%NaCl). Four experimental groups were designed: control (icv saline+po saline), NR (icv saline+po NR), Aβ (icv Aβ+po saline), and Aβ+NR (icv Aβ+po NR).After the injection, to reduce Aβ clearance (Kang *et al.* Science. 2009;32 1005-7.) rats were subjected to 96 hours of sleep deprivation.Starting from day 6, rats were given either 700 mg/kg oral NR or saline, and handling test scores were recorded daily.The procedures were repeated daily until the rats were sacrificed on day 28.Behavioral experiments were randomly conducted at the end, and statistical analysis was performed using repeated measures ANOVA, followed by the Tukey post hoc test.
Results: Passive avoidance test results showed that the Aβ group had the shortest latency to enter the dark area. However, the Aβ+NR group exhibited a prolonged latency compared to the Aβ group ($F(3,2)=5.5;p<0.05$). Aβ injection induced depressive-like behavior in rats, as indicated by the forced swim test (FST) for behavioral despair and the sucrose preference test (SPT) for anhedonia. In AD rats treated with NR (Aβ+NR), Aβ-induced depressive-like behavior was reduced, with lower FST immobility scores ($F(3,2)=6.2;p<0.05$) and increased sucrose preference in the SPT ($F(3,2)=7.5;p<0.05$). There were no significant differences in anxiety-like behaviors among the groups, assessed by the time spent in the open arm in the elevated plus maze test ($F(3,2)=1.9;p>0.05$). During the 28-day monitoring period, the Aβ+NR group of rats exhibited a more rapid decrease in aggression levels compared to the other groups in the handling test. This decrease was significant between days 7 and 10 compared to the Aβ group ($F(48,5)=1.5;p<0.05$).
Conclusions: NR improved memory, reduced depressive behavior, and lowered aggression in AD rats. This suggests that NAD+ precursor NR effectively treats cognitive decline and neuropsychiatric symptoms in an AD model.

Disclosure of Interest: None Declared

O0071

Treatment effect of trauma-focused treatment and/or integrated trauma-focused and personality disorder treatment on brain activation during an emotional face task

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Introduction: Post-traumatic stress disorder (PTSD) and personality disorders are highly comorbid. There is some evidence that