

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 ($A\beta$) 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 μ l $A\beta$ 1-42 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), $A\beta$ (icv $A\beta$ +po saline), and $A\beta$ +NR (icv $A\beta$ +po NR). After the injection, to reduce $A\beta$ 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 $A\beta$ group had the shortest latency to enter the dark area. However, the $A\beta$ +NR group exhibited a prolonged latency compared to the $A\beta$ group ($F(3,2)=5.5$; $p < 0.05$). $A\beta$ 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 ($A\beta$ +NR), $A\beta$ -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 $A\beta$ +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 $A\beta$ 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