trauma-focused treatment normalises activation in brain areas involved in the fear circuit and regions involved in emotion regulation in people with PTSD. Although we assume that working mechanism of personality disorder treatments relies on improving emotion regulation and associated brain regions, there is as of yet little evidence of neurobiological effects of personality treatment on people with PTSD and comorbid PD.
**Objectives:** To 1) study the effect of trauma-focused and/or trauma-focused and personality disorder treatment n brain activation in participants with PTSD and comorbid personality disorders and 2) relate change in brain activation to symptom improvement.
**Methods:** Participants with PTSD and comorbid borderline and/or cluster c personality disorders from the PROSPER-trials (Prediction and Outcome Study for PTSD and personality disorders) were randomized to either trauma-focused treatment (TFT) or TFT with personality disorder treatment (TFT+PT). Brain activation was measured with an emotional face task during functional magnetic resonance imaging scanning before and after treatment. Regions of interest for the analyses were the amygdala, dorsal ACC, insula, ventromedial prefrontal cortex (PFC), ventrolateral PFC and dorsolateral PFC. Bayesian multilevel analyses were conducted to analyze change in brain activation. Clinical measures were clinician-administered PTSD severity, self-rated emotion regulation problems, depression severity and dissociation severity.
**Results:** We included 42 participants with a pre- and posttreatment scan (24 with TFT, 18 TFT+PT). Analyses on the pre-post data are currently being run and will be presented in April.
**Conclusions:** This is one of the first studies to conduct functional MRI analyses on treatment in participants with both PTSD and personality disorders.

**Disclosure of Interest:** None Declared

## O0072

## A Meta-Analysis of fMRI Activation Studies of Ketamine in Healthy Participants

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doi: 10.1192/j.eurpsy.2024.198

**Introduction:** There has been rapidly growing interest in understanding the pharmaceutical and clinical properties of psychedelic and dissociative drugs, with a particular focus on ketamine. This compound, long known for its anesthetic and dissociative properties, has garnered attention due to its potential to rapidly alleviate symptoms of depression, especially in individuals with treatment-resistant depression (TRD) or acute suicidal ideation or behavior. However, while ketamine's psychopharmacological effects are increasingly well-documented, the specific patterns of its neural impact remain a subject of exploration and basic questions remain about its effects on functional activation in both clinical and healthy populations.
**Objectives:** This meta-analysis seeks to contribute to the evolving landscape of neuroscience research on dissociative drugs such as ketamine by comprehensively examining the effects of acute ketamine administration on neural activation, as measured by functional magnetic resonance imaging (fMRI), in healthy participants.
**Methods:** We conducted a meta-analysis of existing fMRI activation studies of ketamine using multilevel kernel density analysis (MKDA). Following a comprehensive PubMed search, we quantitatively synthesized all published primary fMRI whole-brain activation studies of the effects of ketamine in healthy subjects with no overlapping samples (N=18). This approach also incorporated ensemble thresholding ($\alpha$=0.05-0.0001) to minimize cluster-size detection bias and Monte Carlo simulations to correct for multiple comparisons.
**Results:** Our meta-analysis revealed statistically significant ($p<0.05$-0.0001; FWE-corrected) alterations in neural activation in multiple cortical and subcortical regions following the administration of ketamine to healthy participants (N=306).
**Conclusions:** These results offer valuable insights into the functional neuroanatomical effects caused by acute ketamine administration. These findings may also inform development of therapeutic applications of ketamine for various psychiatric and neurological conditions. Future studies should investigate the neural effects of ketamine administration, including both short-term and long-term effects, in clinical populations and their relation to clinical and functional improvements.

**Disclosure of Interest:** None Declared

# Child and Adolescent Psychiatry

## O0073

## A longitudinal study of child and adolescent psychopathology in conditions of the war in Ukraine

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doi: 10.1192/j.eurpsy.2024.199

**Introduction:** According to UNICEF, 2 million children have left the country since the beginning of the war. 2.5 million Ukrainian children are internally displaced persons. Minors often become victims or witnesses of violence.
The events of 2022-2023 are the largest military conflict in the world since World War II. The impact on the mental health of the population is characterized by the variety and mass of traumatizing factors.
Mental trauma causes PTSD, depressive disorders (DD), anxiety disorders (AD), behavioral disorders (CD), attention deficit hyperactivity disorder (ADHD).
**Objectives:** The aim of the study was to determine the prevalence of PTSD and its comorbidities at different stages of experiencing a traumatic experience.

**Methods:** 785 teen`s displaced from the zone of military operations, occupied territories were surveyed. Examinations included: K-SADS-PL, PSC-17, SCARED, CATS. 260 teen`s were examined during - 6, 400 – 6–12 months after traumatization.
**Results:** After 6 months of trauma, PTSD was diagnosed in 9.8%, ADHD – 10.2%, DD-22.3%, AD-30.8%, CD – 15.4%, 28.8%; examined 6 to 12 months after the injury, respectively: 21.9%, 12.6, 33.3%, 11.5%, 18.0%.
**Conclusions:** In war-affected children, PTSD is a risk factor for the subsequent development of comorbid depression, anxiety, conduct disorders, and ADHD. Female sex, secondary traumatization after displacement increase the risk of developing depression, signs of pervasive development and ADHD - the risk of destructive and self-injurious behavior. The prevalence of PTSD, DD, ADHD increases within 6-12 months after the trauma, the sensitivity of children with PTSD to secondary traumatic events increases.

**Disclosure of Interest:** None Declared

## O0076

## The association between glucose 6-phosphate dehydrogenase (G6PD) deficiency and attention deficit/hyperactivity disorder (ADHD)

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doi: 10.1192/j.eurpsy.2024.200

**Introduction:** Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an X-linked genetic enzymopathy that impacts 4.9% of the population, with greater prevalence among Mediterranean, East Asian, and African populations. G6PD deficiency results in levels of nicotinamide-adenine dinucleotide phosphate (NADPH) and glutathione (GSH) that are insufficient for maintaining the balance of oxidation-reduction in the body. This results in elevated production of reactive oxygen species (ROS), oxidative stress on proteins and lipids, damage to DNA, and potential activation of chemokine and cytokine pathways by astrocytes and microglia. We propose that these direct and indirect effects of G6PD deficiency are associated with development of ADHD.
**Objectives:** This study investigated the association between G6PD deficiency and Attention Deficit/Hyperactivity Disorder (ADHD).
**Methods:** The study involved 7,473 G6PD-deficient patients and 29,892 matched case-controls (selected at a 1:4 ratio) from a cohort of 1,031,354 within the Leumit Health Services database. Clinical characteristics were analyzed using Fisher's Exact Tests for categorical variables and Mann-Whitney U tests for continuous variables.
**Results:** The average age of patients was 29.2 ± 22.3 years, with 68.7% being male. The mean follow-up duration was 14.3 ± 6.2 years. Individuals with G6PD deficiency showed a significant 16% higher risk of being diagnosed with ADHD (Odds Ratio (OR) = 1.16 [95% CI, 1.08-1.25], $p < 0.001$) on follow up. Furthermore, G6PD deficiency was associated with a 30% greater likelihood of seeking care from adult neurologists (OR = 1.30 [95% CI, 1.22-1.38], $p < 0.001$) and a 12% higher probability of consulting adult psychiatrists (OR = 1.12 [95% CI, 1.01-1.24], $p = 0.048$). The use of stimulant medications among G6PD deficient individuals was 17% higher for methylphenidate class drugs (OR = 1.17 [95% CI, 1.08, 1.27], $p < 0.001$), and use of amphetamines elevated by 16% (OR = 1.16 [95% CI, 1.03, 1.37], $p = 0.047$).
**Conclusions:** This study establishes a significant association between G6PD deficiency and an increased risk of ADHD diagnoses. These findings suggest potential opportunities for the development of culturally sensitive interventions.

**Disclosure of Interest:** B. Krone Consultant of: HIPPO T&C, Signant Health, J. Newcorn: None Declared, I. Manor: None Declared, E. Merzon: None Declared

## O0077

## The bifactor model of the Hungarian self-report version of the Strengths and Weaknesses of ADHD and Normal Behaviors scale

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doi: 10.1192/j.eurpsy.2024.201

**Introduction:** Attention Deficit/Hyperactivity Disorder (ADHD) is one of the most common neuropsychiatric conditions, maintaining its presence well into adolescence and adulthood, resulting in impaired functioning. Evaluating ADHD symptoms through self-reporting plays a crucial role in assessing individuals within these age groups. The novel self-report version of the Strengths and Weaknesses of ADHD and Normal Behaviors (SWAN) scale offers a comprehensive assessment of behaviour, extending beyond just focusing on the typical signs and symptoms of ADHD, thus providing a more holistic perspective.
**Objectives:** Our goal was to assess the factorial validity of the Hungarian version of the SWAN self-report by comparing a two-factor model with bifactor models with a general and 1) two specific factors (inattention, hyperactivity/impulsivity), 2) three specific factors (inattention, motor hyperactivity/impulsivity, verbal hyperactivity/impulsivity) in a community sample.
**Methods:** Data from 717 adolescents and young adults (mean age = 20.0 years, SD = 3.10, range: 14 - 25 years, female: N = 664, 92.6%) were analysed. Participants completed an online questionnaire including the SWAN scale after giving informed consent. Confirmatory factor analyses were conducted based on the maximum likelihood estimator (ML).
**Results:** The bifactor model with a general and three specific factors demonstrated the best fit to our data (CFI = .933, RMSEA = .064 [90% CI: .058 – .071], SRMR = .038). While the overall composite reliability was excellent ($\omega = .91$), the reliability of the specific verbal hyperactivity/impulsivity factor fell below acceptable ($\omega_h = .40$).
**Conclusions:** In line with previous studies, the fit indices of the bifactor models were superior to the non-hierarchical two-factor model. Our results support the existence of a strong general factor but suggest uncertainty in the capacity of the specific factors to consistently explain the distinct variance in observed variables,