

Introduction: Adolescence is a period marked by highest vulnerability to the onset of depression, with profound implications for adult health. Neuroimaging studies have revealed considerable atrophy in brain structure in these patients with depression. Of particular importance are regions responsible for cognitive control, reward, and self-referential processing. However, the causal structural networks underpinning brain region atrophies in adolescents with depression remain unclear.

Objectives: This study aimed to investigate the temporal course and causal relationships of gray matter atrophy within the brains of adolescents with depression.

Methods: We analyzed T1-weighted structural images using voxel-based morphometry in first-episode adolescent patients with depression ($n=80$, 22 males; age = 15.57 ± 1.78) and age, gender matched healthy controls ($n=82$, 25 males; age = 16.11 ± 2.76) to identify the disease stage-specific gray matter abnormalities. Then, with granger causality analysis, we arranged the patients' illness duration chronologically to construct the causal structural covariance networks that investigated the causal relationships of those atypical structures.

Results: Compared to controls, smaller volumes in ventral medial prefrontal cortex (vmPFC), dorsal anterior cingulate cortex (dACC), middle cingulate cortex (MCC) and insula areas were identified in patients with less than 1 year illness duration, and further progressed to the subgenual ACC, regions of default, frontoparietal networks in longer duration. Causal network results revealed that dACC, vmPFC, MCC and insula were prominent nodes projecting exerted positive causal effects to regions of the default mode and frontoparietal networks. The dACC, vmPFC and insula also had positive projections to the reward network, which included mainly the thalamus, caudate and putamen, while MCC also exerted a positive causal effect on the insula and thalamus.

Conclusions: These findings revealed the progression of structural atrophy in adolescent patients with depression and demonstrated the causal relationships between regions involving cognitive control, reward and self-referential processes.

Disclosure of Interest: None Declared

Eating Disorders

EPP0220

Prevalence And Risk Factors Of Eating Disorders In The Tunisian General Population

M. Turki, A. Hadj Ali*, G. Chakchouk, S. Ellouze, N. Halouani and J. Aloulou

Psychiatric department B, Hedi Chaker University Hospital, Sfax, Tunisia

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.414

Introduction: Eating disorders (ED) negatively affect physical, mental, and social well-being. The exact psychopathology of ED is still unknown, with research suggesting the interplay of a combination of factors.

Objectives: The aim of our study was to estimate the prevalence of ED in the Tunisian general population, and to identify associated risk factors.

Methods: We conducted a cross-sectional, descriptive and analytical study among Facebook group members, using an online

questionnaire, over the period from February 17, 2023 to May 26, 2023. All respondents over the age of 18 were included in the study. All participants filled a socio-demographic questionnaire. The Eating Attitudes Test (EAT-26) was used to screen for those at risk of eating disorders.

Results: A total of 528 responses were included in the study. The mean age of the sample was 33.3 ± 11.95 years. The subjects were unmarried in 63.4% of cases, of low socio-economic level in 19.5%, with a university education in 75.2% and with a regular occupation in 56.1% of cases.

The mean EAT-26 score was 12.36 ± 10.34 . according to this scale, 12.3% of our population were at high risk of developing an ED.

In a multivariate analysis, the female gender ($p = 0.006$), the low economic status ($p = 0.012$), a psychiatric comorbidity ($p < 0.001$), and physical activity ($p = 0.037$) were strongly associated with ED.

Conclusions: This study highlighted the magnitude of the risk of disordered eating attitudes in the Tunisian population and the need for programs to prevent and control these disorders.

Disclosure of Interest: None Declared

EPP0221

Evaluating the role of autistic traits and sensory sensitivity in eating disorders and autistic-like eating behaviours

G. Ingrosso^{1*}, V. Nistico^{1,2,3}, F. Lombardi¹, B. Morlacchi¹, A. C. Cigognini¹, R. Faggioli⁴, A. Mottaran^{4,5}, M. Tramontano^{4,5}, L. Ranzini^{4,5}, C. A. Redaelli^{4,5}, S. Anselmetti^{4,5}, S. Bertelli^{4,5}, O. Gambini^{1,3,4} and B. Demartini^{3,3,4,4}

¹Health Science Department, University of Milan; ²Department of Psychology, University of Milan Bicocca; ³Aldo Ravelli Research Center for Neurotechnology and Experimental Brain Therapeutics, University of Milan; ⁴UO Psichiatria 51 e 52, ASST Santi Paolo e Carlo, Presidio San Paolo and ⁵NutriMente Onlus, Milan, Italy

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.415

Introduction: In recent decades, there has been extensive research on the association between Autism Spectrum Disorders (ASD) and Eating Disorders (ED), as well as the existence of sensory sensitivity alterations in both diagnostic groups.

Objectives: The present study aimed to examine the presence of autistic traits in a sample of adult women diagnosed with different ED, and the concurrent role of autistic traits and sensory sensitivity in both their eating disorder symptomatology and their autism-related eating behaviours.

Methods: Seventy-five women with different ED completed the Eating Attitude Test (EAT-26), the Autism Quotient (AQ), the Ritvo Autism Asperger Diagnostic Scale-Revised (RAADS-R), the Sensory Perception Quotient - Short Form 35 item (SPQ-SF35) and the Swedish Eating Assessment for Autism Spectrum Disorders (SWEAA), which investigates specific eating behaviour related to autism.

Results: 12% of the sample scored above the cut-off at both the AQ and the RAADS-R, while 68% scored above the cut-off at the RAADS-R only. We found an association between: i) hypersensitivity in the taste domain and ED severity and autistic-like eating behaviours; ii) hypersensitivity in the vision domain and