Introduction: Tourette's syndrome (TS) is a disorder characterized by repetitive, involuntary movements, and vocalizations known as tics. While there are existing treatment options, there is a growing need for novel pharmacological approaches to manage the symptoms of TS effectively. This study delves into the emerging field of using cannabinoids as a potential treatment for Tourette's syndrome.
Objectives: The primary objectives of this review are to examine the current evidence base for the use of cannabinoids in the treatment of Tourette's syndrome, to assess the biological rationale supporting the use of cannabinoids in managing tic severity, to provide insights into the results of existing clinical trials involving cannabinoids and Tourette's syndrome, and to draw conclusions regarding the potential efficacy and safety of cannabinoid-based treatments for TS.
Methods: Narrative review of the available scientific literature.
Results: There is a strong biological rationale for how cannabinoids could impact tic severity. The endocannabinoid system plays a crucial role in regulating various physiological processes, including motor control and neurotransmitter release. Activation of cannabinoid receptors in the brain may modulate these processes, potentially reducing tics. While limited, two small randomized, placebo-controlled trials of THC have been conducted in TS patients. These trials suggested potential benefits of cannabis-derived agents in reducing tic frequency and severity. Self-report and examiner rating scales demonstrated significant improvements in tic symptoms. The trials indicated that THC treatment did not result in significant adverse effects in TS patients.
Conclusions: The exploration of cannabinoids as a treatment option for Tourette's syndrome is promising but requires further investigation. The biological mechanisms through which cannabinoids may affect tic severity in TS are sound, suggesting their potential as a therapeutic option. Existing trials with THC have shown encouraging results, demonstrating a reduction in tics without significant adverse effects. However, the limited number of trials warrants caution in drawing definitive conclusions. Despite the promising findings, the overall efficacy and safety of cannabinoid-based treatments remain largely unknown. Further trials are essential to address dosing, active ingredients, optimal administration, and potential long-term effects. Clinical use should be approached with caution. While early evidence is encouraging, additional rigorous studies are needed to establish the safety and efficacy of cannabinoid-based treatments for this disorder.

Disclosure of Interest: None Declared

EPV0641

Investigating Epigenetic and Neuroimaging Profiles in Bipolar Disorder and Behavioral Variant Frontotemporal Dementia: An integrated epigenetic-neuroimaging approach

G. Delvecchio[1]*, M. Serpente[1], L. Di Consoli[1], E. Rotondo[1], V. Borraci[1], E. Scola[2], F. M. Triuzi[2], M. Castellani[3], A. Arighi[1], E. Scarpini[1], D. Galimberti[1,4] and P. Brambilla[1,5]

[1]Department of Neurosciences and Mental Health; [2]Department of Neuroradiology; [3]Nuclear Medicine Unit, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico; [4]Department of Biomedical, Surgical and Dental Sciences and [5]Department of Pathophysiology and Transplantation, University of Milan, Milan, Italy
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1296

Introduction: Discriminating between bipolar disorder (BD) and behavioral variant Frontotemporal Dementia (bvFTD) is a clinical challenge as it is still based on clinical judgement, which often leads to misdiagnosis. This challenge is particularly pronounced in cases involving the *C9orf72* hexanucleotide expansion, a genetic factor responsible for a substantial portion of familial FTD cases, as in these patients the development of late psychoses is particularly frequent. Moreover, individuals with *C9orf72* bvFTD are also characterized by behavioral changes that resemble those seen in late-life BD, especially during the early stages of the disease. This raises questions about whether the clinical similarities between BD and bvFTD are rooted in specific alterations within the brain networks involved in cognitive processing or in selective genetic and epigenetic mutations. In light of this, our recently published neuroimaging study has shed light on the presence of distinctive structural and metabolic characteristics in elderly individuals with BD and bvFTD. These findings offer valuable neurobiological insights that may lead to differentiate between bvFTD and elderly BD patients.
Objectives: Building on our previous research, this study further explores the existence of similar epigenetic expression patterns in plasma neural derived extra cellular vesicles (NDEs), such as miRNA and lncRNA, and seeks to correlate these epigenetic data with shared or distinct biological markers obtained through structural Magnetic Resonance Imaging and [18F]-fluorodeoxyglucose (FDG)-Positron Emission Tomography (PET).
Methods: We will plan to conduct statistical analyses on epigenetic and neuroimaging data on *C9orf72* and sporadic bvFTD as well as on late-and early-onset BD patients and on healthy controls. Additionally, A PET study will be also performed on a subpopulation of these patients.
Results: Our hypothesis posits that selective epigenetic modifications may impact the brain's structure and function, in a way that can change the glutamatergic neurotransmission in prefrontal regions, with subsequent indirect effects on subcortical areas.
Conclusions: Our findings will not only help identifying the specific biological signatures of BD and bvFTD, which might have important implications not only in prevention but also in differential diagnosis and treatment, but also offer insights into potential targets for slowing the onset and progression of the structural alterations characterizing these disorders.

Disclosure of Interest: None Declared

EPV0642

Gut Microorganisms, Neuroinflammation and Behavioral Changes

B. T. Adebisi

Functional and Molecular Neurobiology, Institute of Anatomy, Cell Biology, Brain and Neurodegeneration, Osogbo, Nigeria
doi: 10.1192/j.eurpsy.2024.1297

Introduction: Recent clinical and preclinical evidences suggested that neuroinflammation is a key factor which interacts with the neurobiological correlates of major depressive disorder, which are the (i) dysregulation of the hypothalamic-pituitary-adrenal axis, (ii) depletion of brain serotonin and (iii) alteration of neurogenesis in the dentate gyrus of the hippocampus.