EPV0640

Short-term olfactory deprivation reorganizes brain activity

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Introduction: Olfactory loss (including short-term) initiates neural reorganization processes in the brain, but the central mechanisms of this largely remain unknown.
Objectives: We aim to conduct a neurophysiological study of neocortical mechanisms for the effects of olfactory sensation on functional activity of human brain under temporary obstruction of nasal breathing.
Methods: 123 healthy volunteers (76 female and 47 male students aged 18 to 23 years)participated in this study.EEG was registered during the rest state (5 min), olfactory blockage (5 min), under odor stimulation with the lemon essential oil (5 min) and renewal of nasal breathing (5 min). We estimated the spectral power density and the levels of coherence of all the frequencies from 0.2 to 35 Hz.
Results: The onset of orthonasal olfactory sensory blockage was accompanied by an increase in the power of processes of local synchronization of beta frequency in the caudal regions of the brain, with simultaneous enhancement of the coherence of the theta band in parietal-occipital zones and a certain enhancement of the interfrontal and intrahemispherical left-side and right-side interactions in the beta2-subband. Thus, the sudden cessation of olfactory detection led to activation of thalamo-cortical loops and top-down control systems (search for the olfactory signal): that is an active orienting process (triangle of increase in F4-T4-P4 connections) with emotional coloring (the caudal localization of the coherence changes in the theta band). The prolongation of the nasal blockage, despite the possibility of activation of the retronasal route of odor perception and odorized air, was accompanied by a definite inhibition of distant interactions in the posterior regions of the brain in the theta-band and a significant decrease in right-brain long-distant and parietooccipital beta2-subband functional connectivity. Restoration of nasal breathing and olfactory perception is accompanied by sufficiently powerful activation of interhemispheric long and short distance information interactions in the theta1,2 and beta1 frequency bands.
Conclusions: Our data indicated that cessation and restoration of olfactory perception lead to an increase cognitive activity, the development of memory processes, the current sensory and cognitive-emotional control of behavioral reactions, focusing attention, assessment of the significant stimulus.

Disclosure of Interest: None Declared

EPV0637

Neuropsychiatric manifestations inaugurating Biermer's disease

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Introduction: Vitamin B12 deficiency gives rise to a wide spectrum of hematological, gastrointestinal, psychiatric, and neurological disorders. Notable among the neuropsychiatric symptoms are mood disturbances, cognitive decline, and psychotic manifestations.
Objectives: We present a case of a woman with neuropsychiatric symptoms linked to vitamin B12 deficiency to highlight certain organic aetiologies with psychiatric symptoms in the foreground.
Methods: We discussed through a clinical case and a literature review, the relationship between neuropsychiatric symptoms and vitamine B12 deficiency in the context of biermer's disease.
Results: We presented a patient aged 51-years-old without neurological or psychiatric history, she was hospitalised in a psychiatry department for behavioral disturbances, hetero-aggression, and incoherent speech. The psychiatric examination revealed distant contact, inappropriate affects, disorganized speech with persecutory delusions, memory problems, and poor insight. Neurological et physical examinations were normal, and cerebral magnetic resonance imaging (MRI) showed no abnormalities. First, haloperidol 25mg was prescribed, however, there was only partial improvement. Complete blood counts revealed macrocytic anemia (Hemoglobin: 8 g/dL, mean corpuscular volume: 106 fL). Her serum B12 assay was 48.19 pmol/L.Given these results we proceed to a Fundic biopsy, performed by fibroscopy, that revealed fundic atrophy and intestinal metaplasia compatible with Biermer's disease. Vitamin B12 replacement therapy began with hydroxocobalamin at 1000 µg/day intramuscularly for 15 days, followed by 1000 µg every 15 days for one month. Subsequently, there was a remarkable improvement in psychotic symptoms and cognitive function. Follow-up assessments demonstrated a return to baseline functioning.
Conclusions: This case, coupled with prior studies, emphasizes the importance of considering vitamin B12 deficiency in the differential diagnosis of neuropsychiatric symptoms. Therefore, prompt diagnosis and treatment of vitamin B12 deficiency are imperative in preventing potential irreversible neurological damage.

Disclosure of Interest: None Declared

EPV0640

Exploring the Potential of Cannabinoids in the Treatment of Tourette's Syndrome

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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

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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

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Gut Microorganisms, Neuroinflammation and Behavioral Changes

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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.