EPV0625

Neurological and neuropsychiatric comorbidities occurring in fatty liver diseases

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Introduction: The most common liver diseases associated with the fat accumulation in the hepatic tissue are metabolic associated fatty liver disease (MAFLD), non-alcoholic fatty liver disease (NAFLD), and non-alcoholic steatohepatitis (NASH). Many studies previously reported several key mechanisms tying hepatic injury to extrahepatic manifestations. In this way, the co-occurrence of cognitive decline, mood, and affective changes could suggest the existence of a strong neurological component predisposing to neuropsychiatric comorbidities.
Objectives: In this study, we aimed to describe the neurological and neuropsychiatric comorbidities of MALFD, NAFLD, and NASH.
Methods: The main scientific databases were screened for English-written studies using the following key words: ”cognitive decline”, ”neuronal loss”, ”affective disorders”, ”anxiety”, ”depression”, ”MAFLD”, ”NAFLD”, ”NASH”. Exclusion criteria: (1) studies not focussing on fatty liver diseases; (2) not describing comorbid conditions; (3) not providing correlative analysis of disease co-occurrence or mechanistic associations.
Results: Hepatic encephalopathy (HE) is the main NAFLD/NASH extrahepatic manifestation commonly characterized by impaired cognition, rapid mood swings, depressive and anxious behaviours, and defective sleep. It is currently reported that more than 70% of the cirrhotic patients develop HE. Cognitive impairments and brain tissue reduction were found in NAFLD patients, while MAFLD patients’ cognitive dysfunctions (mild cognitive impairment and hippocampal-dependent memory impairment) were not associated with the presence of metabolic syndrome. Similarly Alzheimer’s disease (AD) was not described as comorbid in MAFLD. By contrast, since NAFLD and NASH are often characterized by insulin resistance and dyslipidaemia – significant triggers of dementia. By far the most prevalent neuropsychiatric comorbidity in NAFLD and NASH is the major depressive syndrome, diagnosed in almost 30% of the cases. Also, a correlation between the anxiety manifestation and the progression from NAFLD to NASH was described. In this context, as a response to the vast evidence that connect liver dysfunction to cognitive impairments, the liver-brain axis function was hypothesized.
Conclusions: MAFLD, NAFLD, and NASH are frequently associated with cognitive decline. The main NASH neurological comorbidity is hepatic encephalopathy, but it could also be seen in NAFLD. While Alzheimer’s disease occurs in NAFLD and NASH, more studies are needed to explain the severity-dependent association. Depression and anxiety were also reported in NAFLD and NASH.
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EPV0626

Tobacco and mental health- Deciphering the vulnerability to nicotine in patients with schizophrenia as a function of their dopamine neurotransmission genes: a clinical and preclinical study

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Introduction: Schizophrenia (SZ) is a severe and frequent mental disorder that has multifactorial origins (genetic but also environmental vulnerability, gene environment interactions). Our team has shown that in France smoking is common among these patients and often begins before the onset of symptoms. This calls into question the hypothesis of self-medication of psychotic symptoms and cognitive disorders by tobacco consumption, and raises the question of specific interactions between nicotine consumption and the genes of the dopaminergic and cholinergic nicotinic systems, in particular with regard to adolescence, period of neurodevelopmental vulnerability.
Objectives: to study (i) the interactions between the DA system and exogenous nicotine in a pre-clinical mouse model (transgenic for DAT) (ii) the impact of smoking on the psychotic and clinical phenotype in a national cohort of SZ patients (iii) interactions between tobacco consumption and several genetic polymorphisms of the dopaminergic and nicotinic system in SZ population. Hypothesis: Disruption of the balance between DA and nicotine systems by nicotine consumption in adolescence may be a key neurobiological mechanism for the emergence of SZ disorders.
Methods: The characterization of the model is behavioral (anxiety, memory, social interactions, locomotion, motor coordination) but also biochemical. For the clinical approach, we exploit the clinical / cognitive / genetic data of a national cohort (Fondamental foundation) and another smaller and local cohort.
Results: We demonstrate, for the clinical study, that some clinical and cognitive characteristics are associated with tobacco use in schizophrenia patients, with more cognitive distubrances in smokers, against the self medication hypothesis. Genes envionment interactions also demonstrate associations with genes involved in dopaminergic system. Regarding the preclinical study, we show a gene environment interaction, as heterozygote mice for the DAT

gene exposed to Nicotine durig a critical neurodevelopmental window (adolescence) show a loss of familiarity in the social memory test and a loss of cognitive flexibility at the set shifting test, similar to what is found in schizophrenia.
Conclusions: TBetter characterization of patients with schizophrenia is necessary to better understand the physiopathology of this disease and explore new personalized preventive and therapeutical targets. We show that tobacco is associated with cognitive disturbances in schizophrenia patients, against selfmedication hypothesis; and in our animal model, that nicotine exposition during the adolescence combined to a moderate cortical and limbic hyperdopaminergia, is associated with persistent cognitive and social deficits, in favor of a gene environment interaction.

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EPV0627

Diagnostic Challenges of Functional Cognitive Disorders

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Introduction: Functional cognitive disorders (FCD) denotes a complaint about memory function or, less commonly, another cognitive process, in the absence of relevant neuropathology and with evidence of inconsistency between symptoms reported and signs identified at assessment.
Increasing numbers of people with FCD are being identified.
Most are discharged back to primary care without a diagnosis or are given the label of mild cognitive impairment, which is not a synonym for FCD.
Objectives: There is a multitude of terms in the clinic and in the research to describe this kind of complaints. Some terms seem to minimize and normalize the state, whereas others posit an underlying cause.
Given this lack of order, it is a challenge to diagnose it and give it the proper clinical guidance.
This literature-based review aims to fill this gap.
Methods: Data was obtained through an internet-based literature search, using the databases PubMed, Cochrane Library and NCBI. The World Health Organization was also utilized. Nine articles from the last two years were included.
Results: It is listed a nosology in six categories and a selection of clinical features which may help with the discrimination of functional and neurological disease causes of memory disorders.
Conclusions: Patients presenting with complaints about memory function require standard psychiatric and neurological history and examination.
It should be emphasized that these conditions are not diagnoses of exclusion but have positive symptoms and signs that should become well-known.
Nevertheless, we remain uncertain about prognosis and treatments, both psychological and pharmacological. Its development would reduce the burden of patients in the healthcare systems.

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EPV0628

The Effects of Serotonergic Psychedelics on Neural Activity: A Meta-Analysis of Task-Based Functional Neuroimaging Studies

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Introduction: Curiosity toward the effects of psychedelic drugs on neural activation has increased due to their potential therapeutic benefits, particularly serotonergic psychedelics that act as 5-HT2A receptor agonists such as LSD, psilocybin, and MDMA. However, the pattern of their effects on neural activity in various brain regions in both clinical and healthy populations is still not well understood, and primary studies addressing this issue have sometimes generated inconsistent results.
Objectives: The present meta-analysis aims to advance our understanding of the most widely used serotonergic psychedelics – LSD, psilocybin, and MDMA – by examining their effects on the functional activation throughout the whole brain among both clinical and healthy participants.
Methods: We conducted this meta-analysis by applying multilevel kernel density analysis (MKDA) with ensemble thresholding to quantitatively combine existing functional magnetic resonance imaging (fMRI) studies that examined whole-brain functional activation of clinical or healthy participants who were administered a serotonergic psychedelic.
Results: Serotonergic psychedelics, including LSD, psilocybin, and MDMA, exhibited significant effects (α=0.05) on neural activation in several regions throughout the cerebral cortex and basal ganglia, including effects that may be common across and unique within each drug.
Conclusions: These observed effects of serotonergic psychedelics on neural activity advance our understanding of the functional neuroanatomy associated with their administration and may inform future studies of both their adverse and therapeutic effects, including emerging clinical applications for the treatment of several psychiatric disorders.

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EPV0630

Neuropsychological diagnosis of impaired mental functions in alcoholic disease of the second stage

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