

During the talk she will provide guidance on what should and should not be done for the end users, as well as strengths and limitations of the chatbot intervention.

Disclosure of Interest: None Declared

S0113

Treatment Options for Problematic Internet Use

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Abstract: Research investigating interventions for problematic usage of the Internet (PUI) remains at an early stage but is steadily developing. In terms of therapies assessed through randomized clinical trials, literature suggests that cognitive behavioral therapy may be the most effective intervention but definitive statements as to its benefits need more testing. In relation to pharmacological treatments for PUI, studies have largely examined the efficacy of agents such as antidepressants and stimulants with a potential therapeutic effect of escitalopram, bupropion, methylphenidate, and atomoxetine. Another emerging form of potentially useful treatment involves non-invasive neurostimulation techniques such as transcranial magnetic stimulation and transcranial direct current stimulation. These interventions are thought to mediate their effect in PUI via stimulation of cortical brain cells and modification of their related functions. In summary, although the limited available treatment evidence includes some promising findings, there is a need for higher-quality research to develop best practice guidelines and determine cost-effective options in PUI treatment. The presentation will provide a state-of the-art overview in the field of therapeutics for PUI.

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S0114

Circulating immune cell composition and activation status associate with brain white matter microstructure in bipolar depression

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Abstract: Bipolar disorder (BD) has been consistently associated with alterations in the immune system. Evidence suggests a condition of systemic low-grade inflammation due to decreased adaptive, increased innate immunity, with higher levels of circulating cytokines, higher macrophage/monocyte inflammatory activation patterns, and higher neutrophils to lymphocyte counts; and with a dynamic pattern of premature immunosenescence and partial T

cell defect starting early in adolescence, involving a reduction of naïve T cells and an expansion of memory and senescent T cells. Quantitative analysis of circulating inflammatory markers suggested persistent low-grade inflammation.

A growing literature suggests that the immune system plays a core role in maintaining brain homeostasis, with both adaptive and innate immune support, ensured by cell trafficking across the blood brain barrier, being essential for brain maintenance and repair in healthy conditions, and disrupted in brain disorders including BD. Measured in peripheral blood, these markers of altered immuno-inflammatory setpoints parallel activation of microglia and disruption of white matter (WM) integrity in the brain.

Studies in the field are in its infancy, but findings by our group showed that: circulating Th17 cells correlated with higher FA, while regulatory FOXP3⁺ cells correlated with higher RD and MD, and with lower fMRI neural responses in the right dorsolateral pre-frontal cortex; higher circulating cytokine-producing NK cells were fostered by ongoing lithium treatment and directly correlated with better FA, and inversely with RD and MD, also partially mediating the known benefits from lithium on WM; and activation status and expression of killer proteins by cytotoxic CD8⁺ T cells negatively associated with WM microstructure, thus suggesting that CD8⁺ T cells can leave the blood stream to migrate into the brain and induce an immune-related WM damage in BD.

Implications of these findings for neuroprogression, clinical outcomes, and new treatment strategies of the disorder will be discussed.

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S0115

Proactive psychiatry of addiction: toward the normalization of early prevention

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Abstract: Most substance use disorders (SUDs) emerge in adolescence and young adulthood. Early interventions in young people may reduce the risk and severity of SUD and other mental disorders. However, we are not able to reach our young people early enough for prevention and treatment, and when we do, they often already show a high concentration of comorbid mental disorders and signs of a chronic intermittent course of SUD. Hence, we must reach our addicted young patients at an earlier stage, when symptoms are still mild or transient, or perhaps even before that – when they only show precursors of possible dysfunction.

One of the most prominent precursors of dysfunction is our ability – or lack thereof – to control or "self-regulate" our behaviors, cognitions, and emotions. Many scientists argue that poor self-regulation is perhaps the core determinant of the development of mental health disorders, including addiction. Several prospective general population studies have shown that poor childhood self-control early in life is a strong predictor of many negative outcomes later in life, up to 20 to 30 years later in adulthood. Although correlational in nature, these findings suggest that early childhood interventions that are deliberately aimed at improving self-

regulation may be effective in preventing these negative life outcomes, and that early prevention and intervention targeted at improving self-control may reduce the risk of a broad array of psychiatric and social problems, including addiction. Indeed, several recent large-scale systematic reviews have suggested that self-regulation skills are malleable and can be learned through instruction and practice, and perhaps most so in the early years, roughly around 3 to 6 years, when there is a steep increase in learning curve, when the plasticity of the brain is still high, and when self-regulation skills are still very much in development.

This presentation provides an overview of the rationale and study findings of early prevention of substance use disorders and other mental health disorders. In terms of broad prevention, much can be gained by widespread, consistent implementation and normalization of early prevention at the pre- and elementary school level.

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S0116

Peripheral inflammation relationships with cognitive deficits and genetic factors in psychosis

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Abstract: Elevated peripheral inflammation is common in psychosis. Impairments in general cognition were linked to elevated C-reactive protein (CRP) and other inflammatory markers in patients with psychotic disorders. Whether there is a subgroup of persons with elevated peripheral inflammation demonstrating deficits in specific cognitive domains remains unclear. While molecular underpinnings of altered inflammation in psychosis are hypothesized, genetic contributions to relationships of psychosis, inflammation, and cognition have not been clarified. Thirteen peripheral inflammatory markers and 17 neurobehavioral tasks were quantified in a subset of participants (129 psychosis, 55 healthy controls-HCs) from the Bipolar-Schizophrenia Network on Intermediate Phenotypes (B-SNIP) consortium. Principal component analysis resulted in 5 inflammation factors across inflammatory markers. Three latent cognitive domains (Visual Sensorimotor, General Cognitive Ability, and Inhibitory Control) were characterized based on the neurobehavioral battery. Hierarchical clustering identified a psychosis subgroup with elevated inflammation and worse cognitive performance. Genetic predispositions to schizophrenia and cognition were explored in relation to inflammation. Among persons with psychosis, higher inflammation indices were associated with impairments of Inhibitory Control and Visual Sensorimotor function. Greater deficits in Inhibitory Control were observed in a high inflammation patient subgroup. Consistent with previous studies, global genetic correlations of schizophrenia, CRP, and cognition were observed. Significant bivariate local genetic correlations of CRP with schizophrenia or cognition across 22 loci with several genes in 1 locus on chromosome 3 suggested pleiotropic mechanisms for inflammatory relationships with cognition and psychosis. Specific neurobehavioral domains may be more sensitive to inflammation dysregulation in psychosis as compared to general cognitive function, particularly performance on tasks requiring ongoing behavioral monitoring and control. These, along

with evidence of genetic correlations of CRP, psychosis, and cognition, provide further supporting evidence that inflammation dysregulation is an important underlying mechanistic contributor to the disruption of cognition in psychosis. Targeting this dysregulation may be an avenue for novel therapeutics to improve cognitive outcomes in these patients.

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S0117

Bullying prevention as a preventive strategy for mental health

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Abstract: Bullying constitutes a major public health concern, on account of its high prevalence rates and its association with a wide range of adverse health outcomes across the lifespan, including increased incidence of mental disorders such as depression, anxiety, and psychotic disorders. Previous research suggests effectiveness of school-based programmes in reducing bullying prevalence and improving mental health outcomes in children and adolescents. Despite the fact that some subpopulations such as young people with special educational needs are at increased risk for both bullying victimisation and mental health difficulties, there is little information on the effectiveness of universal school-based programmes in these high-risk populations. We will review available evidence of the effectiveness of school-based anti-bullying interventions as a tool to improve youth mental health, including results from a cluster-randomised clinical trial conducted in 20 publicly funded schools in Madrid to test the effectiveness of a 12-week web-enabled, user-friendly, school-based, preventive programme incorporating universal and targeted components (LINKlusive; ISRCTN15719015) and discuss the potential implications, challenges, and unmet needs of such approaches.

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S0118

A machine learning approach on whole blood immunomarkers to identify an inflammation associated psychosis onset subgroups

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Abstract: Psychosis onset is a transdiagnostic event that leads to a range of psychiatric disorders, which are currently diagnosed through clinical observation. Since several years, the role of immune system in the pathophysiology of psychosis has been well-recognized, showing differences from the onset to chronic