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Abstract

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STA001

Cannabis and psychosis

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Abstract: This presentation reviews current evidence on the relationship between cannabis use and psychosis with special emphasis on cannabis induced psychosis, schizophrenia and the transition between the two. Cannabis has become increasingly potent over time, with THC concentrations rising dramatically, and this change is accompanied by a growing body of evidence linking cannabis to a range of psychiatric outcomes. Although cannabis is generally considered less harmful than alcohol, it nonetheless contributes to significant mental health risks. Epidemiological data consistently show associations between cannabis use and depression, anxiety, cognitive impairment, and, the primary focus of this lecture, psychotic disorders. Both heavy use and early onset substantially increase these risks, especially among vulnerable individuals.

Acute cannabis-induced psychosis is well documented and typically presents with confusion, hallucinations, emotional instability, depersonalization, and paranoid ideation. The condition can resemble acute schizophrenic psychosis, and symptom patterns are quite similar. While most cases are resolved within days, an acute psychotic episode represents an important clinical warning sign. Some debate persists about whether cannabis can cause persistent psychosis, but accumulating findings indicate that cannabis so consistently triggers schizophrenia in vulnerable individuals, that we mostly look at it as a separate causal factor. Large cohort and genetic studies suggest that the association reflects both shared vulnerability and a direct causal contribution of cannabis. Dose–response relationships have been observed for frequency of use, potency, and age at initiation.

Several candidate genes—including COMT, DRD2, and FAAH—appear to interact with cannabis to increase psychosis risk, but findings remain inconsistent. Individuals with schizophrenia who use cannabis experience poorer clinical outcomes, including reduced medication adherence, more frequent relapses, and increased hospitalizations. Studies show rising treatment-seeking for cannabis-induced psychosis paralleling increased potency of cannabis products.

The transition from cannabis-induced psychosis to schizophrenia is a central clinical concern. Recent meta-analytic evidence indicates that about 20% of people with cannabis-induced psychosis later develop a schizophrenia-spectrum disorder, underscoring the long-term implications. Diagnostic and legal frameworks complicate assessment, as time-based criteria in DSM and ICD attempt to separate substance-induced from primary psychosis, despite substantial clinical overlap. These distinctions have direct consequences for treatment, follow-up, and legal responsibility.

Overall, the evidence strongly supports cannabis as a risk factor for psychosis—particularly for early, frequent, and high-potency use—and highlights the importance of identifying vulnerable individuals and providing early, appropriate intervention.

Disclosure of Interest: None Declared

STA002

Violence risk prediction in psychiatry

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Abstract: Across medicine, accurate prognosis can inform clinical decision-making about management and resource allocation. Several such models have been developed and validated in psychiatry, including those estimating risk of psychosis onset and relapse, and other key adverse outcomes, such as suicide and violence. Reliable risk estimation for these outcomes could inform follow-up intensity, continuity of care, collaborative safety planning, and risk management.

This presentation will provide an overview of prognostic models for violence in people with mental illness. I will outline the predictive performance, acceptability, and impact of new models, which have been translated into clinical risk assessment tools. I will show promising findings for these new models,

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which could be included in clinical guidelines. Further research is required to study whether and how they supplement and augment decision-making in different clinical settings.

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STA003

Unraveling the interaction between stress and genetic risk in psychiatric disorders

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Abstract: Exposure to stress is a ubiquitous aspect of human life, but prolonged or traumatic stress is associated with an increased risk for a wide range of psychiatric disorders. However, not all individuals exposed to such stress develop psychiatric disorders, suggesting that additional factors must mediate risk. Indeed, a growing body of evidence suggests that stress exposure and genetic predisposition interact in complex gene–environment interactions. Much of what is known about these interactions in psychiatric disorders comes from epidemiological studies or from investigations focusing on interactions involving a single gene or a small set of genes. Methodological advances in recent years have made it possible to explore gene–environment interactions using novel approaches that provide insight into their polygenic nature as well as into potential mechanisms through which these interactions act, including epigenetic processes. This session will examine the current state of the field, identify existing limitations, and discuss technological advancements that facilitate focused mechanistic investigations into the interplay between stress and genetic risk.
Specifically, an example will be presented illustrating how novel molecular genetic tools can be used to identify gene–environment interactions and map them onto stress physiology and stress-related neuroimaging phenotypes in a human cohort. This case will serve as an example of how multimodal data—ranging from molecular genetic readouts to human phenotypic measures—can be integrated to better understand stress physiology across multiple levels.

Disclosure of Interest: None Declared

STA004

The therapeutic potential of psychedelic drugs: past, present, and future

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Abstract: The last decade has seen a remarkable resurgence of interest in psychedelic drugs such as psilocybin (from magic mushrooms) LSD and DMT (dimethyl tryptamine – the active ingredient of ayahuasca). This has been driven by the discovery that these psychedelics all act agonists of 5-HT2A receptors plus human imaging studies that reveal this action leads to profound alterations in brain measures of activity particularly in terms of increased entropy of EEG MEG and fMRI signals and reduced within-network, but increased between-network, connectivity. In addition they all can increase synaptic growth and brain plasticity. These findings not only explain the subjective nature of the psychedelic experience but also have implications for the treatment of internalising disorders such as depression addiction anorexia and OCD. Subsequent trials, particularly of psilocybin, in these disorders has revealed significant clinical benefits from even just a single administration. My talk will explore these brain mechanisms and clinical data and discuss the potential place of psychedelic medicine in the future of psychiatry.

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STA005

Efficacy of psychotherapies for mental disorders

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Abstract: Hundreds of randomized controlled trials have examined the effects of psychotherapies on mental disorders. In my presentation I will give an overview of the current knowledge about these effects, based on the Metapsy initiative, a large project with living systematic reviews of psychological treatments of mental disorders. Large meta-analyses of more than 1,000 randomized controlled trials show that psychotherapies are effective for many mental disorders, with substantial symptom reduction, meaningful response rates across age groups and diagnoses, and long-term effects. Cognitive behavioral therapy has been best examined, but there is no evidence in most disorders that it is more effective than other therapies. Although treatments work, population prevalence has not declined, and evidence-based care can only reduce the overall disease burden by roughly 40%. Innovations are therefore necessary, and many are currently developed. Four main domains of innovation will be highlighted: digital interventions, personalized treatments, development of new or improved therapies, and simplification and wider dissemination of existing therapies. However, there are no "silver bullets" that will dramatically change outcomes, and progress can only come from multiple small, incremental advances.

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