ABSTRACT BOOK

18th World Congress of Biological Psychiatry & 5th Global Congress of Biological Psychiatry Joint Meeting

Biological Psychiatry in a Changing World: Exploring New Frontiers

27–30 August2026

Novotel Jaipur Convention Centre

Jaipur, India

www.wfsbp-congress.org

Organized by: World Federation of Societies of Biological Psychiatry

Dear Colleagues,

It is our great pleasure, on behalf of the Scientific Programme Committee, to welcome you to the **18th World Congress of Biological Psychiatry**, held jointly with the **5th Global Congress of Biological Psychiatry**, in Jaipur, India, from 27–30 August 2026. Under the theme *"Biological Psychiatry in a Changing World: Exploring New Frontiers,"* the Congress brings together colleagues from across the world at a time of considerable global challenge, but also at a moment of extraordinary scientific opportunity to shape and transform the future of psychiatry.

We are particularly proud of the **richness, breadth and scientific robustness of this year's programme**. Our aim has been to deliver a Congress that reflects the remarkable diversity of contemporary biological psychiatry—spanning fundamental and translational neuroscience, clinical innovation, and the implementation of new knowledge in everyday psychiatric practice. Across four days, delegates can choose from an exceptional variety of formats, including **keynote and special sessions, symposia, pro–con debates, interactive and early-career workshops, free communications, industry-sponsored sessions, poster presentations, and more**.

The scientific scope is equally broad. The programme traverses precision psychiatry and biomarkers, psychopharmacology and novel drug development, neuroimaging and neuromodulation, immunology and inflammation, metabolism and the microbiome, genomics, neurodevelopment, digital psychiatry and artificial intelligence, trauma, addiction, women's mental health, neurodegeneration and the brain–body interface. Importantly, it also tackles some of the questions that are reshaping our field: how we move beyond traditional diagnostic categories; how biomarkers can meaningfully enter clinical practice; how AI can be harnessed responsibly; and how emerging interventions can be translated from discovery to real-world care. Alongside these are highly practical workshops and clinically oriented sessions that ensure that the Congress remains firmly connected to the needs of practising psychiatrists and other mental health professionals.

Our partnership with the **Global Congress of Biological Psychiatry (GCBP)** includes a parallel scientific programme and expands the choice available to delegates. The complementary **GCBP Programme** includes sessions on genomic and pharmacogenomic approaches, neuroinflammation, metabolic psychiatry, sleep, neuromodulation, novel receptor mechanisms, computational psychiatry, and practical clinical skills. Across the programmes, delegates will therefore have an unusually wide range of opportunities to engage with both established leaders and emerging investigators, and with perspectives from different regions of the world.

The setting for this Congress could hardly be more fitting. **Jaipur**, one of India's most celebrated historic cities, provides a vibrant backdrop scientific exchange, meaningful networking, new collaborations, and the forging of lasting friendships. Known as the *Pink City*, Jaipur brings together history, culture, architecture and contemporary India in a way that mirrors our Congress theme: respecting what has come before while looking confidently

towards new frontiers. We hope that, beyond the lecture halls, delegates will take the opportunity to experience the city and the warmth of its hospitality.

We extend my sincere thanks to members of the Scientific Programme Committee, our GCBP colleagues, our professional society partners (CINP, ECNP, WPA) partners, our speakers, chairs, presenters and partners whose collective efforts have made an ambitious programme possible. Above all, we warmly welcome you, our delegates. We hope that these four days will challenge your thinking, stimulate new ideas, foster collaborations across disciplines and continents, and leave you with a renewed sense of excitement about the future of biological psychiatry.

Welcome to WFSBP 2026, welcome to our joint meeting with GCBP, and welcome to Jaipur.

Professor Soraya Seedat
Scientific Programme Committee Chair, President-Elect (WFSBP)
18th World Congress of Biological Psychiatry

Professor Peter Falkai
President, World Federation of Societies of Biological Psychiatry (WFSBP)

Author Index

An author index with page references is provided at the end of this Abstract Book for easy navigation.

WFSBP Global Headquarters

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Congress Venue

Novotel Jaipur
Convention Centre

Nh 12, Tonk Rd
Sitapura Industrial Area
Sitapura, Jaipur
Rajasthan 302022, India

OC
Opening Ceremony

OC-001
The Bipolar Action Network: A Collaborative Organization to Improve Outcomes

Speaker: Andrew Nierenberg, USA

Abstract

Why consider a learning health network for psychiatry? While, as psychiatrists, we do the best we can within the healthcare systems where we work, we generally don't have systems built to help us improve our patients' outcomes. In our systems, in the clinic, we work one-on-one with our patients. Then we use the best available evidence and treatments that arise from basic neuroscience research and clinical trials. But much of this research does not actually inform what we need to do for our patients. Explanatory randomized clinical trials answer the question: Does the intervention change the target outcome? They do not address pragmatic questions: Does the intervention work in clinical practice? Will it work for my patient? What is the probability that my patient will tolerate treatment and respond? Other challenges include the lack of pre-visit planning, systematic diagnosis, guideline-concordant care, measurement-based care, population management and the fact that most clinicians and healthcare systems don't know their patients' outcomes.

To improve the system of care that could yield better outcomes, other fields have developed learning health systems and networks. These are socio-technical organizations that bring together clinicians, researchers, data analysts, patients and their families to define purpose (and how to achieve better outcomes that matter), learn from data, innovate and co-produce solutions. The network uses an idealized design process to define an optimal future system and map what needs to change to achieve it. By developing a culture of humility, curiosity, generosity, and openness, the network can view variations in outcomes as opportunities to learn, share, and improve. Then the sites can use improvement science techniques to change their systems and track progress. The sites track progress by forming a central triple-use registry of patients with measures that matter, which can be used for clinical care, quality improvement, and research. Every clinical encounter adds to the database.

The Bipolar Action Network is developing a learning improvement health network to improve the outcomes of people living with bipolar disorder across all ages. With 9 sites, including one community mental health network, the registry includes more than 2,000 patients. Each site has been trained in quality improvement techniques and has been working on both site-specific and network-wide improvement projects. Care centers have adopted the Clinical Global Impression–Bipolar Version (CGI-BP) as the network's initial clinician-rated severity measure. The PHQ-9 for depressive symptoms is already in routine use at participating sites and was the focus of one of the early quality improvement projects. A broader battery of patient-reported outcomes covering mood symptoms, function, and well-being is being finalized for capture through clinic workflow tools that feed the network registry.

The Bipolar Action Network applies an evidence-based "actor-oriented" organizational architecture that includes three core components: Actors with shared purpose and norms and willingness to contribute; a "commons" for sharing resources and data transparently; and

shared infrastructure, protocols and processes for learning and contribution. It anchors a values-based culture of generosity, contribution, and collaborative learning. And it empowers individuals with lived experience as full partners. The result is a coherent portfolio of processes, tools, and interventions that can be implemented incrementally at participating sites yet collectively produce system-level change.

This presentation will describe the evolution of the Bipolar Action Network and progress to date.

SpS-01
Taking Genetics to Clinical Practice in Mental Health Disorders - Where Do We Stand?

Speaker: Eva Schulte, Germany

Abstract

The past two decades have seen vast improvements in the identification of genetic risk factors for a range of mental health disorders. Yet, this increased knowledge has not translated to clinical care to an equal extent. From recent findings on the genetics of several prominent mental health disorders, this presentation will explore the utility of both common and rare genetic variation in clinical care for mental health disorders.

SpS-02
Development and application of biomarkers for Alzheimer's disease

Speaker: Pedro Rosa-Neto, USA

Abstract

Biomarkers for Alzheimer's disease have revolutionized the understanding, diagnosis, and management of the disease over the past two decades. Alzheimer's disease is characterized by a prolonged preclinical phase during which pathological processes accumulate in the brain years before the onset of cognitive symptoms. Previously, definitive diagnosis depended on postmortem neuropathological examination, greatly limiting opportunities for early intervention and therapeutic development. The emergence of biomarkers transformed the field by enabling the in vivo detection and monitoring of the biological mechanisms underlying Alzheimer's disease. These tools have shifted the conceptualization of Alzheimer's disease from a syndrome defined solely by dementia symptoms to a biologically defined disorder that can be identified long before clinical manifestations appear.

One of the most important advances in the field has been the development of molecular imaging biomarkers using positron emission tomography (PET). Amyloid PET tracers enabled, for the first time, the visualization of amyloid plaques in living individuals and demonstrated that amyloid accumulation begins many years before cognitive decline. These findings established the biological basis for identifying preclinical Alzheimer's disease and significantly improved participant selection for clinical trials. Subsequently, tau PET imaging allowed the mapping of neurofibrillary tangles in vivo and demonstrated strong associations between tau burden, neurodegeneration, and cognitive impairment. Tau imaging has become particularly valuable because the regional distribution of tau pathology closely correlates with disease severity and clinical progression.

Structural and functional neuroimaging biomarkers also play an essential role in disease characterization. Magnetic resonance imaging (MRI) is widely used to evaluate cortical atrophy, hippocampal volume loss, and cerebrovascular abnormalities associated with Alzheimer's disease. Functional imaging techniques such as fluorodeoxyglucose PET provide important information regarding cerebral metabolic dysfunction and neuronal injury. Together, these imaging modalities contribute to disease staging, prognosis, and longitudinal monitoring.

In parallel, increasing attention has been directed toward biomarkers of neuroinflammation and glial activation. Research has demonstrated that microglia and astrocytes actively contribute to disease progression through immune signaling, inflammatory cascades, and interactions with amyloid and tau pathology. Imaging and fluid biomarkers targeting inflammatory pathways have therefore become important tools for understanding disease mechanisms and identifying novel therapeutic targets. These findings contributed to the recognition that Alzheimer's disease is not solely a disorder of protein aggregation but also involves complex vascular, immune, and metabolic alterations.

The integration of biomarkers has also transformed clinical trial design and therapeutic development. Biomarker-guided recruitment improves diagnostic precision and enriches clinical trials with individuals who truly harbor Alzheimer's pathology, thereby reducing heterogeneity and improving statistical power. Biomarkers are increasingly used to assess

target engagement, monitor disease progression, and evaluate therapeutic response, particularly with the emergence of anti-amyloid and anti-tau therapies.

SpS-04
Global Guidelines, AI, and Psychiatry - Joint WFSBP, CINP, WPA, and GCBP Symposium

Chair: Joseph Zohar, Israel

Co-Authors:

Peter Falkai

Abstract

Introduction:

Artificial intelligence (AI) is rapidly transforming medicine, includingpsychiatry. This joint symposium aims to explore how AI can be integrated into psychiatric practicefrom multiple perspectives, with representatives from the three organizations: GCBP, WFSBP, andCINP. The symposium will focus on opportunities, challenges and solutions while comparing classicalapproaches to integration of AI in the process.

The first talk, by Prof. Joseph Zohar (CINP), will address theintersection of global guidelines and AI. Rapid advances in AI are reshaping how psychiatric guidelinesare developed and implemented. The Global Guidelines (GG) initiative aims to build a framework thatis both locally sensitive and personalized, with recommendations adapting to available services,medications, and cultural context. Clinicians will be able to enter a brief clinical vignette -along withpast history, genetic background, and other relevant features - and the GG system will suggest tailorednext steps. To maximize accessibility, GG will be smartphone-based and free of charge, supportingbroad uptake across diverse clinical settings. As a digital platform, it can be updated annually, helpingensure the guidance remains current. Responsible integration of AI in psychiatry should be anchoredin four non-negotiable principles: • Explainable AI, to maintain transparency and trust; • Shareddecision-making, to preserve patient autonomy; • Integration with electronic health records, to ensure continuity and accountability; • Harm prevention, through multilayered safety controls. Thesefoundations will guide the ethical implementation of AI-supported clinical tools. The second talk, byProf. Peter Falkai / Prof. Kristina Adorjan (WFSBP), will present work in progress within the GlobalGuidelines Initiative, focusing on dual disorders: schizophrenia and substance use disorders. Thespeakers will summarize progress to date, highlight the challenges of developing guidelines intendedfor global use, review the first year of work, and outline a blueprint for the next phases of development.Dr. Sandeep Vohra (GCBP) will provide an updated overview of the current state of new technologies,including AI, and explore how these tools may further reshape psychiatric practice—ranging from earlydetection to personalized treatment pathways. He will also discuss an ecological model that may beparticularly relevant for countries with limited resources. The session will conclude with ample time forQ&A, with questions addressed by all three speakers.

SpS-04
Global Guidelines, AI, and Psychiatry - Joint WFSBP, CINP, WPA, and GCBP Symposium

SpS-04-002
Global Guidelines for Dual Disorders: Schizophrenia and Substance Use Disorders

Speaker: Peter Falkai, Germany

Abstract

Objective:
Global Guidelines, AI, and Psychiatry: Joint WFSBP, CINP, WPA, and GCBP symposium

Methods:
Currently, many mental health guidelines are not sufficiently tailored to local contexts, lacking sensitivity to the availability of services, medications, cultural differences, and languages. These guidelines are often accessible only through journals or books, limiting their reach, and they are typically updated every few years, resulting in delays in reflecting cutting-edge information. Furthermore, current guidelines prioritize hierarchical levels of evidence rather than aligning with precision medicine principles.

Vision:

We propose the creation of Global Guidelines (GG) in collaboration with the CINP, WPA, and WFSBP, designed to address these limitations:

1. Local Sensitivity: The GG will consider the local availability of medications, services, cultural issues, and languages. This ensures that recommendations are relevant to the region where they are accessed.

2. Global Availability: The guidelines will be made available as a free-of-charge app, ensuring widespread accessibility through smartphones.

3. Annual Updates: The app will facilitate updates on an annual basis. A dedicated taskforce will meet biannually to ensure the guidelines reflect the latest evidence and advancements.

4. Incorporation of AI and Search Engine: Modern technology will be leveraged to include AI and a search engine within the app, enhancing usability and precision.

5. Patient-Focused Approach: Clinicians will be able (via GG agents) to focus their questions on specific patient (including age, gender, past history, etc.) and get evidence-based clinical steps tailored to the patient's needs, covering pharmacology, CBT, family intervention, rehabilitation, and more.

Responsible AI:

We propose that the GG-AI will be build on four system capabilities: Explainable AI to ensure transparency and trust; Shared Decision-Making to protect patient autonomy; Electronic Health

Record integration to secure continuity and accountability; and Harm Prevention to embed multilayered safety controls.

SpS-06
Brain Dimensions of Depression: From Biomarkers to Home-Based Neuromodulation

Speaker: Cynthia Fu, United Kingdom

Abstract

Major depressive disorder is highly heterogeneous, and current treatments are largely selected through trial and error. This presentation will explore how neuroimaging, machine learning and large-scale international collaborations are beginning to reveal neurobiological dimensions of depression. Findings from the COORDINATE-MDD consortium have identified brain-based dimensions that may help stratify patients and guide treatment selection. At the same time, home-based transcranial direct current stimulation (tDCS) is emerging as a promising, scalable and mechanistically informed treatment for depression. This talk will examine how these advances may help enable the development of precision psychiatry in depression.

SpS-07
Advances in Psychosis: Real Advances and Chimeras Over the Past Half-Century

Speaker: Robin M. Murray, United Kingdom

Abstract

In 1976 Eve Johnstone published a paper that showed that people with schizophrenia had increased ventricular size and cognitive impairment. This was widely taken as confirming that schizophrenia was an adult onset neurodegenerative disorder. A decade later we suggested that the changes present at onset of illness were in part neurodevelopmental. It took until 2008 before we began realised that the changes which followed onset were largely a consequence of the life experience associated with schizophrenia including antipsychotic medication. Molecular genetics was applied to psychosis from the 1980s but it was 20 years before this began to bear fruit. Now we can measure predisposition to the main psychiatric disorders, and shown that there is a huge overlap; thus two thirds of the genes predisposing to schizophrenia also predispose to bipolar disorder, thus invalidating much of the effort devoted to subclassifying psychosis. 5 decades of imaging studies have taught us very little with the exception of PET studies which have shown that the final common pathway underlying psychosis (both schizophrenia and mania) in most patients is increased striatal dopamine. Current treatment of psychosis relies heavily on blocking the D2 receptor but rarely addresses the risk factors that have contributed to the onset and persistence of the psychosis. A better way to optimize research into, and personalise treatment of, psychosis is to attend to the causal risk factors. Epidemiological studies have shown us that, acting on a background of polygenic susceptibility, the major types of environmental risk factors comprise neurodevelopmental hazards, drug abuse and social adversity. Unfortunately, research lumps together patients exposed to these different risk factors, and as a result, the neuropsychological or imaging characteristics associated with a particular environmental risk factor are obscured by being merged with patients with whom they have little in common aetiologically. A better strategy is to identify patients whose psychosis has resulted predominantly from exposure to one particular risk factor and then establish the pathway and characteristics associated with this risk. Thus, patients with neurodevelopmental impairment show an excess of premorbid abnormalities, prominent negative symptoms, cognitive and brain structural abnormalities, and poorer outcome. Secondly, those patients whose psychosis results from drug abuse have largely normal premorbid social function and cognition and prominent positive symptoms. Thirdly, patients who have been exposed to social adversity often present with PTSD symptoms or anxiety and depression as part of their psychosis. Appropriate interventions should be offered, in addition to antipsychotics, to patients in accord with their exposure to the different risk factors. For example, the patient who had been subject to child abuse should be offered trauma-based therapy, and antidepressants as appropriate, while the patient who continues to abuse cannabis should be offered CBT/motivational therapy to decrease his/her drug consumption. Those patients who have been exposed to several risk factors deserve to have attention directed to each of these in turn. Finally this approach leads to an obvious approach to prevention by focussing on diminishing exposure to the various risk factors.

SpS-08
Beyond DSM-5: What Comes Next—and Why It Matters' or 'The Future of Diagnosis: Inside the Next DSM

Speaker: Nitin Gogtay, USA

Abstract

Psychiatry stands at the threshold of a diagnostic transformation. For decades, categorical, symptom-based systems have organized clinical practice and research. Yet converging evidence from neuroscience, genetics, developmental science, and systems biology increasingly suggests that traditional diagnostic boundaries may not map onto underlying pathophysiology—and that psychiatric nosology must also evolve toward a more holistic and person-centered framework. The next DSM presents an opportunity not merely to revise existing categories, but to reconsider the architecture of psychiatric diagnosis itself.

Dr. Nitin Gogtay will present the proposed next-generation diagnostic formulation in which the DSM—to be reconceptualized as the Diagnostic and Scientific Manual—moves beyond static categories toward a multidimensional and dynamic model. This approach incorporates socio-cultural and environmental determinants, developmental context, quality of life and functioning, transdiagnostic domains, longitudinal trajectory, and illness severity. Within this evolving framework, emerging evidence regarding candidate biomarkers and biological factors is envisioned as a critical component in informing psychiatric nosology.

Dr. Anissa Abi-Dargham will present the work of the Biomarkers and Biological Factors Subcommittee, examining how validated neurobiological signatures, including imaging-based circuit measures, molecular markers, genetic findings, and other systems-level factors, may inform future diagnostic architecture. She will address both the promise and current limitations of biomarkers, emphasizing standards for validation, replication, and translational relevance, while outlining a roadmap toward biologically anchored dimensions of psychopathology.

Together, the speakers will argue that the next DSM will adopt a holistic, forward-looking, extensible, and modular approach to psychiatric nosology—one that more closely aligns diagnosis with underlying mechanisms and helps advance the goals of precision psychiatry.

SpS-08
Beyond DSM-5: What Comes Next—and Why It Matters' or 'The Future of Diagnosis: Inside the Next DSM

SpS-08-001
A Framework for Incorporating Biomarkers into the Next DSM

Speaker: Anissa Abi-Dargham, USA

Abstract

Psychiatry is undergoing a pivotal transition as biological and behavioral markers evolve from exploratory research tools toward potential elements of clinical decision-making. In many areas of medicine, biomarkers—objective indicators of biological states or responses—have transformed diagnosis, prognosis, risk stratification, and personalized treatment. Yet their integration into mental health care has lagged, despite extensive research across genetics, molecular pathways, inflammation, neuroimaging, electrophysiology, cognition, social function, behavior, and digital phenotyping. Emerging multimodal "biosignatures" that synthesize information across domains show particular promise, but no biomarker is currently used routinely in psychiatric practice. Translation has been hindered by the intrinsic complexity of mental disorders, characterized by heterogeneous presentations, modest effect sizes, overlapping and pleiotropic biological pathways, and multiple mechanisms producing similar syndromes. These features complicate validation and obscure demonstrations of clinical utility. Structural challenges further impede progress: evidence remains fragmented across disciplines, reporting is inconsistent, contexts of use are rarely specified, and standardization is limited. As a result, many candidate biomarkers with potential clinical value do not yet meet essential requirements such as reproducibility, normative referencing, external validation, and incremental benefit beyond standard clinical assessment. The main barrier is that the field lacks a unified approach with common definitions and agreed upon validation standards to organize evidence and judge when a tool is ready for clinical use.

Against this backdrop, the American Psychiatric Association (APA) has initiated planning for a future iteration of the Diagnostic and Statistical Manual of Mental Disorders (DSM). The current DSM relies primarily on symptom-based criteria and does not incorporate biological mechanisms. Recognizing this gap—along with the need for rigorously validated biomarkers—the APA convened a Subcommittee on Candidate Biomarkers and Biological Factors to develop a framework for integrating biomarkers into a future DSM. The subcommittee has been meeting regularly, surveying the landscape of biomarker science, and constructing an evaluation system for assessing validation standards. This system will guide the identification of biomarkers and biological factors suitable for inclusion in the manual.

The proposed framework adopts a tiered, evidence-based approach in which emerging biomarkers are introduced within a designated "emerging category" and updated regularly as evidence accumulates, effectively transforming the DSM into a "living document." The process is applicable to both categorical and transdiagnostic constructs; aligning evidence across these approaches is expected to reveal convergent biological pathways shared across disorders.

This roadmap seeks to establish a coordinated pathway for translating biomarker discoveries into clinically actionable tools that can enhance diagnosis, guide treatment selection, support monitoring, and ultimately advance personalized psychiatric care.

SpS-09
Neurodevelopmental origins of mental illness: Data from Indian longitudinal studies

Speaker: Vivek Benegal, India

Abstract

Objective:
The Consortium on Vulnerability to Externalizing Disorders and Addictions (c-VEDA; N≈9,000) and the Pathways to Resilience and Mental Health (PARAM; N≈8,000) studies constitute two of the largest longitudinal developmental mental health cohorts from India. Together, they provide a unique platform to investigate trajectories of risk, resilience, and psychopathology across childhood, adolescence, and young adulthood in a low- and middle-income country setting.

Methods:
Both programmes employ multisite longitudinal designs and integrate deep phenotyping spanning psychiatric assessments, cognition, neuroimaging, genomics, epigenetics, environmental exposures, biospecimens, and longitudinal follow-up. The cohorts capture substantial social, cultural, geographic, and environmental diversity, enabling examination of how biological and contextual factors interact across development to influence mental health outcomes.

Results / Discussion:
Research from these cohorts demonstrates that childhood adversity, socioeconomic disadvantage, family dysfunction, environmental toxicants, and cumulative developmental risks are major determinants of psychopathology and substance use vulnerability. Neurodevelopmental studies have characterized trajectories of cognitive maturation, social cognition, brain structure, and functional connectivity, while identifying neural correlates of externalizing behaviours, internalizing symptoms, substance use, and transdiagnostic psychopathology. The programmes have generated population-specific neuroimaging resources, including Indian brain templates, normative developmental models, and contributions to international brain charting efforts. Genetic and biological investigations have examined polygenic risk, cross-ancestry psychiatric genetics, gene-environment interactions, DNA methylation, and emerging peripheral biomarkers associated with brain development and mental health outcomes. Methodological innovations include harmonized multimodal data acquisition, quality control frameworks, federated analytics, and participation in global collaborations such as ENIGMA and the Lifespan Brain Chart Consortium.

Conclusion:
The combined c-VEDA and PARAM platforms provide an unprecedented opportunity to understand developmental mental health in diverse populations. Their findings support a multilevel framework in which social adversity, environmental exposures, biological vulnerability, cognition, and brain development interact dynamically to shape risk and resilience. These cohorts represent major resources for global mental health research and for developing prevention-oriented approaches to improve youth mental health outcomes.

SpS-10

Medication strategies for managing difficult-to-treat depression in young people: Absence of evidence or evidence of absence?

Speaker: Christopher Davey, Australia

Abstract

Background:

Depression commonly has its onset in the years from puberty to early adulthood, during a developmental period that has become the focus of youth mental health services. Depression in many young people does not respond to psychotherapies or first-line antidepressant medications, leaving clinicians to consider strategies that might address it. Most of these are inferred from the management of difficult-to-treat depression (DTD) in adults, with few studies examining the treatments in young people. Despite this, there is widespread use of augmentation agents for DTD in young people; especially of atypical antipsychotic medications. Low-dose ketamine has emerged as a potential treatment, providing one of the few treatments that have been subjected to randomised clinical trials (RCTs).

Methods:
In this presentation I will provide an overview of the field of youth depression, including the epidemiology and course of illness in this period, and the new services that are managing mental illnesses in young people in Australia and internationally. I will present the evidence for different medication strategies for managing DTD in youth, with some focus on recent trials of ketamine. This will include the results of our own recently completed Study of Ketamine for Youth Depression (SKY-D) trial, which enrolled 110 16- to 25-year-olds who were treated with weekly subcutaneous ketamine or active placebo for 4 weeks (presented here for the first time).

Results / Discussion:
There are no RCTs examining oral augmentation agents for DTD in young people, although some trials for bipolar depression. There have been four RCTS of ketamine (or esketamine) for depression, including our SKY-D trial. Some have shown acute benefit, but the studies that have examined repeated dosing have not demonstrated effectiveness. In our SKY-D trial, ketamine produced significantly greater improvement in depressive symptoms 24 hours after each administration, but there were no between-group differences after 4 weeks of treatment.

Conclusion:
Depression causes enormous burden in young people, and those with DTD are more likely to show impairments throughout adult life. Effective treatment early in the course is therefore vital. First-line treatments with psychotherapies and antidepressant medications are helpful for many young people, but for those who don't respond there are few evidence based strategies. While there is mostly an absence of evidence for the treatments, there is emerging evidence of absence for the effectiveness of repeated dosing of ketamine. We need a greater focus on developing effective treatment strategies for young people with DTD to alleviate the difficulties that will often endure for many years.

PC-01
Cannabis use in adolescence negatively impacts emerging adults' neurodevelopmental and cognitive abilities

PC-01-002
Cannabis, the Good, the Bad and the Psychotic

Proponent: Robin M. Murray, United Kingdom

Abstract

Psychosis: Heavy cannabis use, particularly during teen-age years, is associated with increased risk of subsequent psychosis. Our work shows a) a dose-response relationship between the level of use and the risk of later psychosis; b) continued use by those with established psychosis is associated with a worse outcome, c) administration of tetrahydrocannabinol (THC), the psychoactive ingredient of cannabis, induces transient psychosis in normal subjects, d) polygenic risk for schizophrenia and cannabis use have independent effects on risk of psychosis combining in some people to push the individual into psychosis. Our European and Global South studies demonstrate that daily use of high potency cannabis has a significant impact on the incidence of psychosis. Now it is becoming clear from a number of countries and US States that increased use of high potency cannabis, often following legal changes, is associated with an increase in new cases of psychosis.

Traditional services have resulted in those with cannabis-induced psychosis falling between psychosis and addiction services with the former treating the psychosis but not the dependence, and the latter usually rejecting the patients because of their psychosis. We have established a cannabis and psychosis clinic dedicated to their treatment. In addition to treatment of psychosis it provides individual therapy (CBT/motivational approaches) as well as weekly on-line group, and where appropriate Sativex to cover cannabis withdrawal. The results have been surprisingly good with 80% stopping their cannabis use with resultant decrease in psychotic symptoms and 75% returning to work or studies.

Other Psychiatric Disorders: There has been much less research into the occurrence of other psychiatric disorders following heavy cannabis use. However evidence has accumulated in recent years that use of cannabis in adolescence is associated with an increased risk of depression and suicide. In addition, if people who are using so called “medicinal cannabis” are suffering from anxiety, PTSD, or depression, these symptoms decrease if they can be persuaded to reduce or stop their cannabis use. Most recently, reports have been appearing that heavy cannabis use is associated with a risk of violence, and indeed many bizarre homicides have turned out to be perpetrated by heavy cannabis users.

Cognition: Not surprisingly, while cannabis users are intoxicated they show impaired cognition, especially of memory. What is less known is that contrary to alcohol, cannabis stays in the body for much longer, and indeed can be detected in urine for up to one month. As a result, young people who binge on cannabis at the weekend may show impaired performance at school or university the next week. Many studies show that teenagers who use cannabis heavily are less likely to pass school or university exams and are less successful in work. There is some evidence that people who heavily use cannabis for decades may show IQ decline which does not reverse fully after stopping cannabis – however, this latter remains controversial.

C-01

Cannabis use in adolescence negatively impacts emerging adults' neurodevelopmental and cognitive abilities

PC-01-002

Cannabis use in adolescence negatively impacts emerging adults' neurodevelopmental and cognitive abilities (Opponent)

Proponent: Debasish Basu, India

Abstract

The key questions, both from a scientific and a public health perspective in this important debate are: Which cannabis? How strong cannabis? How much cannabis? How frequent cannabis? And, importantly, cannabis in the context of what all factors - genetic, developmental, environmental, other mental health conditions? When we try to assiduously partition these factors and fine-grain the analyses, the answer becomes far more nebulous, uncertain, and context-dependent. We simply do not have strong evidence enough that all cannabis - even used in adolescence though that's certainly a critical risk period - is bad for the emerging adults' neurodevelopmental and cognitive abilities. The context matters.

PC-02
Immuno-metabolic depression: Is it prime time to adopt immuno-metabolic modulating pharmacological and lifestyle interventions?

PC-02-001
The Case for Precision: Why Immunometabolic Depression is Ready for Clinical Implementation (Proponent)

Proponent: Jane A. Foster, USA

Abstract

Depression is the leading cause of disability world-wide. Unlike other medical disorders, there are no biologically based clinical tests to guide treatment selection. Current clinical guidelines rely on clinical symptoms, symptom severity, quality of life, number of past episodes, previous treatment response, and known risk factors (PMID: 38711351). Unfortunately, only one third of patients achieve remission (PMID: 27717250). The importance of the peripheral immune system to depression has been studied for 30 years and it is time to leverage this knowledge to advance precision medicine approaches in depression.

Immunometabolic depression (IMD) is characterized by low-grade systemic inflammation, metabolic dysfunction, and the presence of atypical depressive symptoms. Based on an extended body of literature, it is evident that IMD represents a distinct and identifiable depression subtype. Individuals with key IMD features show reduced response to standard antidepressant treatments suggesting that these individuals would benefit from an alternative treatment approach. Promising approaches including dietary interventions, microbiome-targeted approaches such as probiotics and prebiotics, as well as wellness approaches that integrate mindfulness and exercise. Notably, this depression subgroup is linked to cardiometabolic comorbidity and targeting their immunometabolic dysfunction has the potential to improve both mental and physical health.

While a validated, standard biomarker is not yet available for IMD – the key biological and clinical features are known, and ready for integration into the existing clinical guidelines for treatment of depression. During this debate, the key biological and clinical features that can be leveraged to better recognize and treat individuals with IMD will be presented. In addition, the current evidence for this approach and the gaps in the literature will be considered.

PC-02
Immuno-metabolic depression: Is it prime time to adopt immuno-metabolic modulating pharmacological and lifestyle interventions?

PC-02-002
Immuno-metabolic depression: Is it prime time to adopt immuno-metabolic modulating pharmacological and lifestyle interventions? (Opponent)

Opponent: Gin S. Malhi, Australia

Topic:
2) Schizophrenia

Abstract

I will be cautioning against this proposal. Amongst other arguments I will be raising the following points. First, it is unclear which depression this approach is referring to and what evidence exists to support a specific and targetable immuno-metabolic dysfunction that underpins depression. Specifically, are the putative mechanisms by which immuno-metabolic changes bring about depression or maintain this mental state sufficiently well-known and have the steps and linkages been mapped to an extent that modulation can be achieved predictably and reliably to effect change in mood or is it merely an association. Second, lifestyle interventions are inherently broad and so gaining specificity of action is inherently challenging. In the gut microbiome for example the level of complexity it has been argued is on par with neural connections within the brain. Given this is the case how can immuno-metabolic modulation be attained and with the fidelity needed to effect change in mood and alleviate depression. Lastly, it must be made clear whether the aim is to prevent depression, treat depression or maintain wellness once remission of depressive symptoms has been achieved.

PC-03
Are GLP-1 receptor agonists the new frontier in the treatment of psychiatric disorders?

PC-03-001
Are GLP-1 receptor agonists the new frontier in the treatment of psychiatric disorders? (Proponent)

Proponent: Dan Siskind, Australia

Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have rapidly entered the public consciousness, propelled by celebrity endorsements of agents such as Ozempic and narratives promising effortless weight loss, youth, and vitality. This hype has fuelled unrealistic expectations and, at times, obscured the underlying evidence base. Yet for people living with severe mental illness, GLP-1RAs represent more than a cultural phenomenon—they offer a potential therapeutic advance. Antipsychotic medications are associated with substantial metabolic adverse effects, contributing to obesity, cardiometabolic disease, and premature mortality. In this context, GLP-1RAs provide an effective strategy to mitigate weight gain and improve metabolic health in this highly vulnerable population. Beyond metabolic benefits, GLP-1RAs have also been proposed to improve cognition and other neuropsychiatric outcomes; however, emerging data suggest that these effects may be more limited and nuanced than initially anticipated. In this debate, Professor Siskind will examine whether GLP-1RAs truly represent a new frontier in psychiatric treatment, critically appraising the evidence, separating hype from reality, and considering their appropriate role in the care of people with severe mental illness.

PC-03
Are GLP-1 receptor agonists the new frontier in the treatment of psychiatric disorders?

PC-03-002
Are GLP-1 receptor agonists the new frontier in the treatment of psychiatric disorders? – Nah, not so much (Opponent)

Opponent: Andreas Reif, Germany

Topic:
2) Schizophrenia

Abstract

GLP-1 receptor agonists (lead compound: semaglutide), originally developed as quite effective treatments for type II diabetes, have equally revolutionized obesity treatment as well as the stock market for pharmaceutical industries -semaglutide made NovoNordisk for a considerable time the most valuable European company. Following the discovery of the weight-loosing effects of GLP-1 agonists, a plethora of other positive effects were described -seemingly every organ was suggested to benefit from this treatment principle, which also well aligned with the longevity movement. Unsurprisingly, also mental disorders soon were added to this catalogue: beyond long-hanging fruits such as antipsychotic-induced weight gain (why should GLP-1 agonism not be effective there as well?), a range of other phenotypes from depression to various substance use disorders and even dementia were proposed to benefit from GLP-1 agonists. This led to hundreds of reviews, position papers and similar opinion pieces (and some career moves, perhaps), but to surprisingly few randomized controlled trials (therefore, this part of the talk will be rather short) despite the fact that semaglutide was approved 8 years ago. The strikingly low ratio of secondary to primary literature is a key index for hype, and indeed, GLP-1 agonists are on top of the hype cycle when it comes to the treatment of mental disorders – we have seen similar phenomena e.g. for psychedelics. Certainly these compounds will be beneficial for some patients, and some indications, yet they are not a panacea; at least there is neither preclinical nor clinical evidence for this. It is therefore important to exactly identify patients who will profit from this drug class the most – in the sense of precision psychiatry – in order not to discard medication which is, in principle, effective, but not for all patients. Curbed enthusiasm, expectation management and realistic, data driven assessment might therefore really advance the field, but not overblown hype.

S-01

The management of Obsessive-Compulsive Disorder (OCD) with complex comorbidities: Strategies from the new 2025 CANMAT/ICOCS OCD treatment guidelines

Chair: Michael Van Ameringen, Canada

Abstract

Objectives:
BACKGROUND AND OBJECTIVES: Once neglected, obsessive-compulsive disorder (OCD) is now recognized as a common, highly disabling early-onset brain disorder, affecting 2% of the population at some point in their lifetime. The burden associated with OCD is broad, with individuals often experiencing distress, impaired functioning, social isolation, and poor quality of life. Comorbidity is highly associated with OCD, with rates upwards of 71%, resulting in a more disabling, chronic, and treatment resistant presentation, whilst adding complexity to the treatment course. Clinical and translational research in OCD is expanding and has contributed to significant advances in the understanding of this complex disorder. The Canadian Network for Mood and Anxiety Treatments (CANMAT) has long published evidence-based guidelines for managing Major Depressive Disorder and Bipolar Disorder. CANMAT guidelines include recommendations accompanied by the level of evidence available to support each graded line of treatment. In addition, recommendations are ranked based on expert consensus from the CANMAT editorial group on clinical outcomes including safety, tolerability, and feasibility, where possible.

Structured rationale:
Although several evidence-based clinical guidelines for the management of OCD have been published, recent feedback from topic experts and stakeholders identified the need for an update. A group of international OCD experts from CANMAT in association with the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) compiled a comprehensive, updated set of evidence-based treatment guidelines for OCD across the lifespan, based upon current research findings. RESULTS AND CONCLUSIONS: This symposium will present the key findings from the newly published 2025 CANMAT OCD Guidelines with a focus on OCD with its common comorbidities. The first speaker will review and highlight the principles of management of OCD, review the evidence for the first-line treatment recommendations for both pharmacotherapy and psychotherapy and review the evidence and recommendations for treatment-resistant OCD, including pharmacological, non-pharmacological and neuromodulation treatments. The second speaker will discuss the treatment of OCD with comorbid Major Depression. The third speaker will review the treatment recommendations for OCD with comorbid Bipolar Disorder. The fourth speaker will present the treatment recommendations for OCD with anxiety, depression and related disorders during the perinatal period, including pregnancy and breastfeeding.

S-01
The management of Obsessive-Compulsive Disorder (OCD) with complex comorbidities: Strategies from the new 2025 CANMAT/ICOCS OCD treatment guidelines

S-01-001
Introducing the Canadian Network for Mood and Anxiety Treatments (CANMAT) and International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) 2025 International Guidelines for the Management of Patients with Obsessive-Compulsive Disorder(OCD)

Speaker: Michael Van Ameringen, Canada

Topic:
7) Mood disorders

Abstract

Abstract Title:
Introducing the Canadian Network for Mood and Anxiety Treatments (CANMAT) and International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) 2025 International Guidelines for the Management of Patients with Obsessive-Compulsive Disorder (OCD)

Abstract:
The International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) is a global network of expert clinicians and researchers, whose principal objective is to support and stimulate the study and treatment of OCD spectrum disorders. The ICOCS, in concert with the Canadian Network for Mood and Anxiety Treatment (CANMAT) assembled a group of international OCD experts to compile an updated set of treatment guidelines for OCD based upon current research findings. The efficacy and acceptability of available treatments of OCD was examined across the lifespan. The recommendations in these guidelines used the CANMAT framework which identifies the Level of Evidence (based upon the type of study – open label, randomized controlled trial, meta-analysis etc.), as well as Treatment Ranking (first line, second line etc). This presentation will introduce the framework of these new treatment guidelines and discuss principles of management for OCD. First-line treatment recommendations for both pharmacotherapy and psychotherapy will be discussed as well as recommendations for the management of treatment-resistant OCD, including pharmacological, non-pharmacological and neuromodulation treatments.

S-01
The management of Obsessive-Compulsive Disorder (OCD) with complex comorbidities: Strategies from the new 2025 CANMAT/ICOCS OCD treatment guidelines

S-01-003
New updates for the treatment of OCD with comorbid Bipolar Disorder

Speaker: Ayal Schaffer, Canada

Abstract

Abstract Title:
New Updates for the Treatment of OCD with Comorbid Bipolar Disorder

Abstract:
OCD is a relatively common yet understudied comorbidity for patients with BD. With potential onset early or later in the illness course, periodic screening is warranted, especially given impacts on illness presentation and clinical management. This presentation will focus on the latest evidence regarding epidemiology, clinical presentation, evidence-based treatments, and new clinical guidelines for the management of this complex presentation.

S-02
From rapid-acting agents to precision neuromodulation: new directions in depression treatment

Chair: Steven Marwaha, United Kingdom

Co-Authors:

Muralidharan Kesavan

Topic:
7) Mood disorders

Abstract

Objectives:
The aim of this symposium is to:

Integrate evidence on rapid-acting biological treatments, biomarker-guided neuromodulation, and culturally informed augmentation strategies in order to advance a more personalised and globally relevant approach to improving outcomes in major depression.

The objectives of this symposium are to:

• Present emerging evidence for rapid-acting treatments for major depression, including findings from a recent systematic review and meta-analysis of nitrous oxide (N2O) as a novel treatment.

• Highlight biomarker-informed neuromodulation approaches, including EEG-guided frameworks to optimise ECT, tACS and TMS treatment selection and dosing.

• Showcase augmentation strategies for treatment-resistant depression (TRD), with particular insights from Indian clinical practice integrating pharmacotherapy, TMS, and yoga-based interventions.

• Synthesise these complementary perspectives to advance understanding of how emerging biological treatments, precision neuromodulation, and culturally informed augmentation approaches can together improve outcomes for patients with depression.

• Stimulate interdisciplinary discussion on pathways towards personalised, scalable, and sustainable interventions across different healthcare settings.

Structured rationale:
Major depression remains a leading global cause of disability, and many patients do not achieve adequate symptom relief from standard treatments such as antidepressants. There is a need for interventions that act more rapidly, can be personalised to individual neurobiological profiles, and are adaptable across diverse clinical settings. This symposium brings together current

advances in pharmacological, neuromodulatory, and augmentation approaches to inform the next steps in improving treatment outcomes for those with depression.

First, the session begins by presenting evidence on rapid-acting biological treatments, drawing on findings from a recent systematic review and meta-analysis of nitrous oxide (N2O). This presentation will summarise what is known about N2O's antidepressant efficacy, rapid time course of action, and safety profile in depressive disorders. It will also outline key gaps in the evidence base, including the durability of response and optimal dosing strategies.

Second, the symposium turns to precision neuromodulation, presenting an EEG-guided framework for tailoring ECT, tACS and TMS protocols. This work demonstrates how neurophysiological markers can be used to stratify patients and inform stimulation parameters with the aim of improving treatment (tACS and TMS) response. It also aims to highlight the role of biological measures in guiding intervention selection.

Third, the session broadens to a global and applied clinical perspective, drawing on the Indian experience of combining pharmacotherapies, TMS, and yoga-based interventions for augmentation in individuals with treatment-resistant depression. This talk will illustrate how multimodal strategies can be implemented in real-world practice and adapted to global healthcare systems.

Together, these presentations provide an overview of advances in the current evidence, biomarkers, and combined treatment approaches in depression.

S-02
From rapid-acting agents to precision neuromodulation: new directions in depression treatment

S-02-001
Evaluating Nitrous Oxide as a Rapid-Acting Antidepressant: Efficacy, Time Course, and Safety

Speaker: Kiranpreet Gill, United Kingdom

Abstract

Abstract Title:
Nitrous Oxide as a Rapid-Acting Treatment for Depression: Evidence, Gaps, and Pathways to Personalised Clinical Application

Abstract:
Background: Depression affects over 300 million people globally and remains a leading cause of disability. Standard antidepressants fail 30-50% of patients and require weeks to exert clinical effects, creating an urgent need for rapidly acting, mechanistically novel interventions. Nitrous oxide (N2O), a partial NMDA receptor antagonist, modulates glutamatergic signalling and opioid systems implicated in depression pathophysiology. Early-phase trials demonstrate rapid antidepressant effects.

Methods: Systematic review and meta-analysis of clinical trials evaluating N2O for major depressive disorder, treatment-resistant depression, and bipolar depression. Seven completed RCTs (n=247), four published protocol papers, and eight active/registered ClinicalTrials.gov interventional studies were identified. Pooled analyses examined depression symptom scores and AEs at standardised timepoints. Evidence mapping characterised trial design, populations, and outcomes across completed and ongoing studies.

Results: Single-session N2O inhalation (25-50%) significantly reduced depressive symptoms at 2 hours (pooled MD −2.74, 95% CI: −4.72 to −0.76; p=0.007) and 24 hours (MD −3.32, 95% CI: −5.09 to −1.55; p<0.0001) post-administration. Repeated-dosing protocols showed sustained benefit with response rates of 75-92% and remission rates up to 75%. AEs were mild and dose dependent. Research remains concentrated in early-phase, single-session designs in adult MDD/TRD populations; substantial gaps persist regarding long-term durability, optimal dosing, biomarker-guided selection, and outcome standardisation.

Conclusions: N2O demonstrates rapid antidepressant effects with favourable tolerability. Large-scale, methodologically rigorous trials are needed to clarify sustained efficacy with repeated dosing, identify predictive biomarkers, and evaluate diverse populations. Integrating N2O within precision psychiatry frameworks offers promise for advancing personalised depression care.

S-02
From rapid-acting agents to precision neuromodulation: new directions in depression treatment

S-02-003
Augmentation treatments for major depression: The Indian experience

Speaker: Muralidharan Kesavan, India

Abstract

Abstract Title:
Augmentation treatments for major depression: the Indian experience

Abstract:
The session broadens to a global and applied clinical perspective, drawing on the Indian experience of combining pharmacotherapies, TMS, and yoga-based interventions for augmentation in individuals with treatment-resistant depression. This talk will illustrate how multimodal strategies can be implemented in real-world practice and adapted to global healthcare systems.

S-03
Rewriting Geriatric Depression: Neurobiology, Biomarkers, and Brain-Based Interventions

Chair: Debanjan Banerjee, India

Co-Authors:

Gautam Saha

Topic:
7) Mood disorders, 4) Dementia

Abstract

Objectives:
1. To delineate key neurobiological mechanisms underlying geriatric depression, with a focus on inflammation and brain aging.

2. To assess the clinical utility of biological and imaging biomarkers for diagnosis, prognosis, and treatment stratification in late-life depression.

3. To summarize contemporary pharmacological advances targeting multimodal and mechanism-based pathways in geriatric depression.

4. To define the evidence-based role of non-invasive brain stimulation in treatment-resistant and complex late-life depression.

Structured rationale:
Geriatric depression is increasingly recognised as a biologically heterogeneous condition shaped by brain aging, neuroinflammation, vascular and neurodegenerative changes, and altered neural circuitry. Traditional monoaminergic models inadequately explain its clinical complexity, variable treatment response, and frequent overlap with cognitive impairment.

This symposium integrates contemporary evidence on inflammatory mechanisms and imaging biomarkers with advances in pharmacological and neuromodulatory treatments, offering a coherent, brain-based framework to refine diagnosis, personalise interventions, and improve outcomes in late-life depression.

S-04
Biological markers and eating disorders: new discoveries and scientific consensus

Chair: Hubertus Himmerich, United Kingdom

Co-Authors:

Sarah Cohen-Woods

Topic:
10) Other

Abstract

Objectives:
Submitted on behalf of the WFSBP Task Force on Eating Disorders, this symposium summarizes recent research on biological markers and presents key aspects of the forthcoming WFSBP consensus statement on candidate biomarkers for anorexia nervosa, submitted to the World Journal of Biological Psychiatry.

Structured rationale:
Sarah Cohen-Woods, Professor at Flinders University (Australia), will present novel data on the genetics and epigenetics of eating disorders and disordered eating across the lifespan.

Marta Tyszkiewicz-Nwafor, child and adolescent psychiatrist and researcher at Poznan University of Medical Sciences, will discuss the role of adipose tissue–derived hormones and gastrointestinal peptides within the brain–gut–metabolic axis, focusing on their relevance as biomarkers of starvation, biological adaptation, and treatment response.

Wafa Alharbi, Lecturer at Taibah University (Saudi Arabia) and PhD student at King's College London (UK), will present a systematic review of biomarker outcomes regarding virtual reality (VR) interventions for obesity and eating disorders.

Hubertus Himmerich, Reader in Eating Disorders at King's College London and Vice Chair of the WFSBP Task Force on Eating Disorders, will introduce the upcoming consensus paper on candidate biomarkers for anorexia nervosa, developed by the Task Force over the past three years.

S-04
Biological markers and eating disorders: new discoveries and scientific consensus

S-04-002
The role of adipose tissue–derived hormones and gastrointestinal peptides within the brain–gut–metabolic axis

Speaker: Marta Tyszkiewicz-Nwafor, Poland

Abstract

Abstract Title:
Adipose- and Gut-Derived Signals in Anorexia Nervosa: Biomarkers of Starvation Adaptation and Treatment Response

Abstract:
Anorexia nervosa is increasingly conceptualized as a metabo-psychiatric disorder in which chronic energy deficiency induces systemic endocrine, metabolic and brain-related adaptations. This presentation will focus on adipose tissue-derived hormones and gastrointestinal peptides as interacting signals within the brain-gut-metabolic axis, with particular attention to leptin, adiponectin, ghrelin, PYY, GLP-1 and emerging adipokines as candidate biomarkers of starvation state, biological adaptation and response to nutritional rehabilitation.

Rather than treating these molecules solely as appetite regulators, the talk will discuss their role in linking energy availability with neuroendocrine adaptation, reward processing, interoception, cognitive functioning and treatment response. Findings from our longitudinal research in adolescent anorexia nervosa will be used to illustrate how peripheral metabolic and gut-derived markers may interact with neurocognitive outcomes, depressive symptoms and the clinical trajectory of nutritional rehabilitation.

Evidence will be evaluated against the framework of the WFSBP consensus statement on candidate biomarkers for anorexia nervosa, with emphasis on context of use, the distinction between state and trait markers, and the limitations of diagnostic specificity and prognostic validation. Future directions will focus on moving from single biomarkers toward integrated biological signatures — combining adipokine and gastrointestinal peptide profiles with metabolomics, microbiome-related pathways and deep clinical phenotyping — across the spectrum of restrictive eating phenotypes.

S-04
Biological markers and eating disorders: new discoveries and scientific consensus

S-04-003
Virtual Reality Interventions in Obesity and Eating Disorders: A Systematic Review of Biomarker Outcomes

Speaker: Wafa Alharbi, United Kingdom

Abstract

Abstract Title:
Virtual Reality Interventions in Obesity and Eating Disorders: Biomarker Evidence and the OASIS Project

Abstract:
Virtual reality (VR) is increasingly being explored as an innovative intervention for obesity and eating disorders, yet its effects on biological markers remain underexamined. This presentation will summarize findings from a systematic review of VR-based interventions in obesity and eating disorders, focusing on biomarker, clinical, and behavioural outcomes across anthropometric, physiological, metabolic, endocrine, and neurocognitive domains. Current evidence suggests that VR interventions may have beneficial effects on weight-related outcomes in obesity and may offer a promising adjunctive approach for exposure, body image, anxiety reduction, and treatment engagement in eating disorders. However, biomarker evidence remains heterogeneous and limited, particularly in anorexia nervosa and other eating disorder populations. The presentation will also introduce the OASIS project, a patient-centred VR intervention being developed for adults with anorexia nervosa, as an example of how emerging evidence can inform future feasibility work and digital innovation in eating disorder treatment. Future directions for biomarker-informed VR research will be discussed.

S-04
Biological markers and eating disorders: new discoveries and scientific consensus

S-04-004
The World Federation of Societies of Biological Psychiatry (WFSBP) consensus statement on candidate biomarkers for anorexia nervosa

Speaker: Hubertus Himmerich, United Kingdom

Abstract

Abstract Title:
The World Federation of Societies of Biological Psychiatry (WFSBP) consensus statement on candidate biomarkers for anorexia nervosa

Abstract:
Objectives: This World Federation of Societies of Biological Psychiatry (WFSBP) consensus paper aims to summarise and evaluate the published study results on objectively measurable biological markers associated with anorexia nervosa (AN).

Methods: The relevant literature was reviewed by the WFSBP Task Forces on Eating Disorders and on Biological Markers, and a consensus regarding the significance of the published evidence was reached.

Results: Candidate biological markers that have been associated with AN include clinical (e.g. body weight), molecular (e.g. genetic, epigenetic, hormonal, immunological, metabolomic), cellular (e.g. leukocytes), neuroimaging (e.g. structure, function, connectivity), digital, cardiac and neurophysiological parameters. Some clinical and laboratory parameters are risk markers in clinical practice. Biological markers have pathophysiological relevance in understanding the biological and metabolic pathophysiology of AN and its physical health consequences. Few studies have examined pharmacogenetics or therapeutic drug monitoring as tools to monitor and guide the treatment of AN.

Conclusions: Biological markers will hopefully soon enable clinicians to intervene earlier in a more targeted manner to mitigate treatment resistance. However, the current scientific basis for most biological markers are group comparisons only. Studies on sensitivity, specificity and the prognostic value of these markers are lacking.

Reference: Himmerich et al.: World Federation of Societies of Biological Psychiatry (WFSBP) consensus statement on candidate biomarkers for anorexia nervosa. World J Biol Psychiatry. 2026;27(4):257-348. https://doi.org/10.1080/15622975.2026.2626934.

S-05
Brain-body convergence for clinical impact in schizophrenia: visceral adiposity, semaglutide, and mpfc alpha-tacs

Chair: Dan Siskind, Australia

Topic:
2) Schizophrenia

Abstract

Objectives:
1. Describe the long-term metabolic trajectories following cessation of semaglutide in clozapine-treated patients and identify implications for treatment duration and maintenance strategies.

2. Explain the mechanistic link between visceral adipose tissue and accelerated biological brain aging in schizophrenia spectrum disorders, and evaluate VAT as a potential clinical biomarker.

3. Summarize how alpha-tACS targeting the mPFC modulates cortical inhibition and functional connectivity, and assess its potential clinical utility for persistent psychotic symptoms.

4. Integrate metabolic, neurobiological, and circuit-level mechanisms into a cohesive framework for improving clinical outcomes in schizophrenia.

Structured rationale:
Individuals with schizophrenia spectrum disorders (SSDs) experience a disproportionate cardiometabolic burden, with cascading effects on brain structure, function, and long term outcomes. This symposium integrates evidence across three complementary studies to illuminate a mechanism-to-clinic care arc: (1) the durability of GLP 1 receptor agonist effects on weight and metabolic risk after treatment cessation in clozapine treated patients; (2) the unique association between visceral adipose tissue (VAT) and accelerated biological brain aging; and (3) circuit level modulation via medial prefrontal cortex (mPFC) alpha tACS leading to symptom improvement with electrophysiological and connectivity readouts. Together, these talks advance a convergent model linking metabolic dysregulation, brain aging phenotypes, and network level interventions with clear implications for clinical decision making.

Dr. Dan Siskind will present data from The COaST randomized, multi centre, participant and investigator blinded trial comparing weekly semaglutide (titrated to 2.0 mg) versus placebo over 36 weeks in adults with schizophrenia/schizoaffective disorder on clozapine. A 12 month, medication free follow up examines trajectories of weight, BMI, waist circumference, glycaemic indices, and other metabolic parameters from both trial baseline and endpoint to follow up. Planned analyses focus on weight regain, persistence of metabolic improvements, inter individual variability, and associations with ongoing standard metabolic care. As the first longitudinal dataset of post semaglutide outcomes in this high risk group, findings will inform treatment duration, maintenance strategies, and cardiometabolic risk reduction.

Dr. Mahavir Agarwal will describe a cross sectional sample (N=94) with abdominal and brain MRI, normative modeling (CentileBrain) yielded a global brain biological age; BrainAGE was defined as biological minus chronological age. Hierarchical regressions showed that common metabolic indicators (anthropometrics, mean arterial pressure, triglycerides, HDL, insulin resistance, HbA1c) explained 30% of BrainAGE variance (R^2=.30), while %VAT added an independent 8% (ΔR^2=.08) and emerged as the largest predictor; the final model accounted for 38% of variance. These data position VAT—a modifiable, metabolically active depot—as a robust correlate of advanced brain aging in SSDs, beyond standard metabolic markers, with implications for targeting adiposity to protect brain health and cognition.

Dr. Sreeraj VS will present data from a study involving five day, twice daily, 10 Hz tACS protocol (electrodes at AFz/CPz) among 25 patients with persistent psychotic symptoms despite antipsychotic therapy, clinically meaningful improvements were observed in positive and negative symptoms (moderate–large rank biserial effect sizes). Resting state fMRI revealed altered connectivity between supramarginal/angular gyrus and mPFC (FDR corrected p=0.031). TMS EEG paradigms indicated enhanced GABA(B) mediated cortical inhibition (LICI), including improved P180 suppression and latency changes; reduction in LICI N100 latency correlated with negative symptom improvement (r=0.58, p=0.01). These convergent measures support a mechanistic model in which mPFC targeted alpha tACS restores network efficiency and inhibition within the default mode circuitry.

Collectively, the studies propose a testable translational pathway: visceral adiposity may accelerate brain aging in SSDs, providing a mechanistic substrate that could degrade network function; GLP 1RA based strategies (and their maintenance/cessation trajectories) address upstream metabolic risk; and mPFC alpha tACS offers a downstream circuit level intervention with measurable inhibitory and connectivity signatures linked to symptom change.

S-05
Brain-body convergence for clinical impact in schizophrenia: visceral adiposity, semaglutide, and mpfc alpha-tacs

S-05-001
Metabolic Outcomes 12 Months After Cessation of Semaglutide for Clozapine-Associated Obesity: Follow-up Data from the COaST Randomised Controlled Trial

Speaker: Dan Siskind, Australia

Abstract

Abstract Title:
Outcomes 12 Months After Cessation of Semaglutide for Clozapine-Associated Obesity: Follow-up Data from the COaST Randomised Controlled Trial

Abstract:
Background:

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) produce substantial short-term weight loss in people with schizophrenia receiving clozapine. However, the durability of these effects following treatment cessation remains unclear. Previous follow-up studies of GLP-1RAs in clozapine-treated populations have demonstrated post-treatment weight regain, highlighting the importance of longer-term metabolic evaluation. We examined 12-month post-endpoint metabolic outcomes following semaglutide treatment in the COaST trial.

Methods:

COaST was a 36-week, multi-centre, participant- and investigator-blinded randomised controlled trial comparing weekly subcutaneous semaglutide (titrated to 2.0 mg) with placebo in adults with schizophrenia or schizoaffective disorder prescribed clozapine. Following completion of the intervention phase, participants were followed for 12 months after trial endpoint, during which no study medication was provided. Outcomes included change in weight, body mass index (BMI), waist circumference, glycaemic measures, and other metabolic parameters. Changes were examined from trial baseline to 12-month follow-up, and from trial endpoint to 12-month follow-up.

Results:

Analyses will evaluate trajectories of weight and metabolic outcomes following cessation of semaglutide, including the extent of weight regain, persistence of metabolic improvements, and inter-individual variability. Secondary analyses will explore associations with ongoing standard metabolic treatments and clinical characteristics.

Conclusions:

This study will provide the first longitudinal data examining metabolic outcomes 12 months after cessation of semaglutide in people with schizophrenia receiving clozapine. These findings will

inform clinical decisions regarding treatment duration, maintenance strategies, and long-term cardiometabolic risk reduction in this high-risk population.

S-05
Brain-body convergence for clinical impact in schizophrenia: visceral adiposity, semaglutide, and mpfc alpha-tacs

S-05-003
Modulating alpha-oscillations of mPFC for persisting psychotic symptoms in schizophrenia: mechanistic insights from cortical reactivity and connectivity

Speaker: Sreeraj Vanteemar, India

Abstract

Abstract Title:
Modulating alpha-oscillations of mPFC for persisting psychotic symptoms in schizophrenia: mechanistic insights from cortical reactivity and connectivity

Abstract:
A five-day, twice daily, 10 Hz tACS protocol (electrodes at AFz/CPz) among 25 patients with persistent psychotic symptoms despite antipsychotic therapy, clinically meaningful improvements were observed in positive and negative symptoms (moderate to large rank biserial effect sizes). Resting state fMRI revealed altered connectivity between supramarginal/angular gyrus and mPFC (FDR corrected p=0.031). TMS EEG paradigms indicated enhanced GABA(B) mediated cortical inhibition (LICI), including improved P180 suppression and latency changes; reduction in LICI N100 latency correlated with negative symptom improvement (r=0.58, p=0.01). These convergent measures support a mechanistic model in which mPFC targeted alpha tACS restores network efficiency and inhibition within the default mode circuitry.

S-06
T4: challenges of translating biological therapies into the clinic

Chair: Andrew Nierenberg, USA

Co-Authors:

Michael Berk

Topic:
10) Other

Abstract

Objectives:
The objectives of this symposium are to explore the challenges of translating research into clinical practice and opportunities and methods to improve the process.

Structured rationale:
The rationale for the symposium is that we face a crisis of innovation in psychiatric treatment, with a lag of many years from a biological insight to improving outcomes in the clinic.

S-06
T4: challenges of translating biological therapies into the clinic

S-06-003
From stem cells on the bench to clinical trials to the bedside

Speaker: Jee Hyun Kim, Australia

Abstract

Abstract Title:
A platform for novel therapy development: The discovery of trimetazidine to treat bipolar depression

Abstract:
Psychiatric disorders are a major cause of disability across the world. Treatment is what lived experience consumers and carers seek, and is the most effective way to prevent suicide and reduce stigma, comorbidity, as well as economic and societal costs. Existing pharmacotherapies are however often inadequate. Yet, we have seen few truly novel pharmacotherapies emerge in recent years, largely due to the absence of a known pathophysiology for each disorder. Here we describe the successful platform we have adopted that enables targeted drug repurposing. In this path, patient-derived stem cells are used to detect transcriptomic targets to identify existing agents that address these targets, which are then validated in non-human animal models and pharmacoepidemiological studies. These findings can be confirmed in clinical trials. This unbiased approach bypasses the lack of known pathophysiology. Validation steps bring greater scientific rigor and mechanistic insights to drug repurposing to allow only drug candidates with the strongest mechanistic evidence to be clinically trialled.

S-07
Modified electro conclusive therapy: recent advances

Chair: Anjali Yadav, India

Co-Authors:

Sachin Pursnani

Topic:
10) Other

Abstract

Objectives:
To shed light on technolgical advancements on ect

Structured rationale:
Modified electroconvulsive therapy (ECT) continues to be a vital intervention in modern psychiatric practice. Ongoing advances in stimulus delivery, anesthetic techniques, seizure monitoring, and cognitive-sparing strategies have significantly enhanced its safety, efficacy, and patient acceptance. With evolving evidence and updated clinical guidelines, it is essential for clinicians to remain informed about these developments. This symposium aims to provide an updated overview of recent advances in modified ECT, promote evidence-based practice, and facilitate informed clinical decision-making in contemporary psychiatric care.

S-08
Dual Disorders in Transition: Advances in Guidelines, Integrated Systems of Care, and International Perspectives

Chair: Christian Schütz, Canada

Co-Authors:

Yatan Pal Singh Balhara

Abstract

Objectives:
Review recent developments in international guidelines for the assessment and treatment of dual disorders.

Understand key components of comprehensive and integrated systems of care for individuals with severe dual disorders.

Examine recent advances, challenges, and opportunities in dual disorder care and research in India.

Identify priorities for future international collaboration in dual disorders research, policy, and clinical practice.

Structured rationale:
Background: Dual disorders are highly prevalent and associated with substantial morbidity, mortality, and healthcare utilization. Advances in biological psychiatry increasingly demonstrate shared neurobiological mechanisms across substance use and other mental disorders, supporting integrated and transdiagnostic approaches to assessment and treatment.

Relevance: Recent developments in evidence-based guidelines, comprehensive models of care, and international initiatives provide new opportunities to improve outcomes for individuals with dual disorders. Sharing experiences across healthcare systems can accelerate the adoption of effective practices.

Objectives: To review current developments in dual disorder guidelines, describe the implementation of a comprehensive integrated care system, and examine recent advances and challenges in India.

Expected Impact: Participants will gain practical and strategic insights into evidence-based treatment, service development, and international approaches that can inform clinical practice, policy, education, and research.

S-09
Silent Clues Unveiled: Video Mastery of Psychiatric Observational Signs

Chair: Shobit Garg, India

Co-Authors:

Gautam Saha

Topic:
10) Other

Abstract

Objectives:
1. Identify and interpret subtle observational psychiatric signs through video examples, distinguishing them from verbal symptoms.

2. Correlate specific visual signs (e.g., psychomotor changes, catatonia, soft neurological signs) with relevant neurobiological pathways and disorders.

3. Apply video-enhanced observational techniques to improve diagnostic precision, early intervention, and treatment monitoring in everyday practice.

4. Discuss ethical considerations, consent protocols, and best practices for using clinical videos in education and research.

5. Recognize the value of observational skills in integrating with biological psychiatry approaches for holistic patient care.

Structured rationale:
Clinical psychiatry remains an art as much as a science, where keen observational skills often uncover diagnostic clues that self-reported symptoms alone cannot reveal. This video-centric symposium will immerse participants in the visual world of subtle psychiatric signs—those detectable through careful, trained observation of appearance, posture, movement, facial expressions, and behavior—bridging classical descriptive psychopathology with modern biological psychiatry. Using high-quality, ethically obtained and fully anonymized video footage of real patients (with institutional consent and privacy safeguards), the session will demonstrate key observable markers such as psychomotor slowing/agitation in depression, negative symptoms and mannerisms in schizophrenia, catatonic postures and negativism, neurological soft signs (e.g., dysdiadochokinesia, primitive reflexes), abnormal eye contact/gaze patterns in autism spectrum or social anxiety, stereotypies/tics in neurodevelopmental disorders, prodromal behavioral shifts in emerging psychoses, and a dedicated segment on videos of abnormal movements (including tics, stereotypies, mannerisms, dyskinesias, choreiform movements, tremors, and catatonic motor phenomena). Presenters will explicitly link these visual signs to underlying biological mechanisms, including dopaminergic/glutamatergic imbalances, frontal-subcortical dysfunction, neuroinflammation, and genetic/neuroimaging

correlates. In an era of increasing reliance on questionnaires and digital tools, this symposium revives and modernizes the foundational skill of "seeing" psychopathology, offering practical, immediately applicable training to enhance early detection, differential diagnosis, and biologically targeted interventions. By focusing exclusively on video demonstrations rather than static slides, attendees will gain hands-on observational expertise, address common training gaps in visual pattern recognition, and appreciate how these "silent" signs complement biomarkers in precision psychiatry. This timely, interactive format promises high engagement and direct clinical impact for psychiatrists, residents, and allied mental health professionals. This video-centric symposium, featuring experts from India and Bangladesh, will immerse participants "with cross-cultural perspectives on behavioural signs in South Asian populations". The addition of regional diversity enhances relevance to global biological psychiatry while addressing training gaps in observational skills.

S-10
Advancing Women's Mental Health: Hormones, Perinatal Care, and Education

Chair: Florence Thibaut, France

Co-Authors:

Harish Thippeswamy

Topic:
5) Women's mental health, 10) Other

Abstract

Objectives:
Women's mental health is shaped by biological, reproductive, and sociocultural factors that remain insufficiently integrated into psychiatric practice and training. This symposium will bring together complementary perspectives to improve understanding and care across the female life course.

Structured rationale:
Title: Advancing Women's Mental Health: Hormones, Perinatal Care, and Education

Female Hormones and Mental Health (Prof. Aninda Sidhana, India)

Drug and Alcohol Use in Pregnancy (Prof. Arpit Jashwantbhai Parmar, India)

How to improve education on women's mental health world wide (Prof. F Thibaut, France)

Post partum psychosis and immune dysfunction (Prof. Harish Thippeswamy, India)

Women's mental health is shaped by biological, reproductive, and sociocultural factors that remain insufficiently integrated into psychiatric practice and training. This symposium will bring together complementary perspectives to improve understanding and care across the female life course.

Prof. Aninda Sidhana (Sriganganagar, India) will focus on hormonal transitions and their impact on mood and behavior. Fluctuations across the menstrual cycle, premenstrual phase, and other reproductive stages can significantly influence psychiatric symptoms. A digital tool designed to help patients track mood symptoms in relation to menstrual cycles, supporting more precise assessment, shared decision-making, and personalized treatment strategies will be presented.

Prof. Arpit Jashwantbhai Parmar (New Delhi, India) will address perinatal mental health, a critical period associated with increased vulnerability to to mood, anxiety, and substance use disorders. He will review risk factors, early identification, and integrated care models that bridge psychiatry, obstetrics, and primary care. Particular attention will be paid to stigma, barriers to care, and the consequences of untreated illness for both mother and child.

Prof. Harish Thippeswamy (NIMHANS, India) will summarise the existing literature about postpartum psychosis presents a unique opportunity to unravel the links between the intense hormone changes and related genetics and immune dysfunction. Understanding the immune dysfunction could provide novel insights regarding the pathophysiology and potential newer treatment targets for postpartum psychosis.

Prof. Florence Thibaut (France) will examine education in women's mental health. Despite growing evidence of sex and gender differences in disease presentation, treatment response, and service access, these insights are not consistently translated into medical curricula or clinical training. Findings from a recent consensus initiative identifying 18 top-priority topics in women's health will be discussed, with implications for curriculum development and professional training.

Together, these sessions aim to strengthen gender-informed psychiatric practice, promote early intervention, and support systemic improvements in education and care delivery.

S-10
Advancing Women's Mental Health: Hormones, Perinatal Care, and Education

S-10-002
Drug and Alcohol Use in Pregnancy

Speaker: Arpit Parmar Jashwantbhai, India

Abstract

Abstract Title:
Drug and alcohol use during pregnancy:

Abstract:
Pregnancy complicated by substance use disorder (SUD) is among the most complex clinical scenarios in psychiatric practice. This symposium aims to explore the intersection of maternal neurobiology and fetal development, with attention to the acute and chronic effects of drug and alcohol use during pregnancy. The symposium also aims to highlight the profound stigma a woman with SUD faces during this vulnerable period and to focus on trauma-informed approaches to alleviate some of these problems. We will discuss specific biological risks, including the impact of epigenetics, altered neurodevelopment, and the role of psychiatric comorbidities. We will also examine the impact of SUD on fetal development, especially during the neonatal and childhood periods. Ultimately, the symposium aims to provide a roadmap for compassionate, evidence-based care for this group of patients.

S-10
Advancing Women's Mental Health: Hormones, Perinatal Care, and Education

S-10-003
Post partum psychosis and immune dysfunction

Speaker: Harish Thippeswamy, India

Abstract

Abstract Title:
Post partum Psychosis and Immune Dysfunction

Abstract:
Postpartum psychosis (PP) is a psychiatric emergency and is associated with high risks. The onset is within the initial weeks after delivery, and the aetiology remains enigmatic. In recent years, the role of immune dysfunction has gained traction. It is well-known that autoimmune conditions flare up during the postpartum period. Emerging research suggests a potential role for microglia in modulating neuronal plasticity during the postpartum period. The physiological adaptive changes in the immune system could result in the manifestation of postpartum psychosis among vulnerable women. Abnormalities in immune cells, such as monocytes and T cells, may alter the immune setpoint, triggering the manifestation of PP. The higher rates of thyroid abnormalities and reports of antineuronal antibodies lend credence to the role of immune dysfunction. The area of immune dysfunction warrants exploration, given the promising role of monoclonal antibodies in several neuropsychiatric conditions. Research could pave the way for newer therapeutics for the treatment of PP.

S-10
Advancing Women's Mental Health: Hormones, Perinatal Care, and Education

S-10-004
How to improve education on women's mental health world wide

Speaker: Florence Thibaut, France

Abstract

Abstract Title:
How to improve education on women's mental health worldwide

Abstract:
Women's mental health is shaped by biological, reproductive, and sociocultural factors that remain insufficiently integrated into psychiatric practice and training. This symposium will bring together complementary perspectives to improve understanding and care across the female life course.

Prof. Florence Thibaut (France) will examine education in women's mental health. Despite growing evidence of sex and gender differences in disease presentation, treatment response, and service access, these insights are not consistently translated into medical curricula or clinical training. Findings from a recent consensus initiative identifying 18 top-priority topics in women's health will be discussed, with implications for curriculum development and professional training.

Our session aim to strengthen gender-informed psychiatric practice, promote early intervention, and support systemic improvements in education and care delivery.

S-11
Gut-Brain Axis and Mental Health

Chair: Sailaxmi Gandhi, India

Co-Authors:

Nishanth K N

Topic:
10) Other

Abstract

Objectives:
1. To explain the role of the gut–brain axis in psychiatric illnesses.

2. To describe the influence of diet on gut microbiota and mental health.

3. To examine the impact of unhealthy diet, poor sleep, and sedentary lifestyle on mental health outcomes.

4. To highlight the importance of fibre, probiotics, physical activity, and regular routines in gut–brain communication.

5. To present findings from a systematic review on gut–brain axis–related interventions and mental health outcomes.

Structured rationale:
The gut–brain axis plays an essential role in maintaining physical and mental health through continuous bidirectional communication between the gastrointestinal system and the brain. This interaction is mediated through immune pathways, the vagus nerve, the enteric nervous system, and microbial metabolites such as short-chain fatty acids, branched-chain amino acids, and peptidoglycans. Recent research highlights the role of gut microbiota in influencing mood, cognition, and behaviour. Alterations in gut microbiota have been associated with psychiatric, neurodevelopmental, and neurodegenerative disorders, including schizophrenia, anxiety disorders, autism spectrum disorders, obesity, Parkinson's disease, and Alzheimer's disease.

Lifestyle factors strongly influence gut–brain communication. Diet, sleep, physical activity, and daily routines shape gut microbiota composition and metabolic functioning. Individuals with severe mental illness often follow unhealthy dietary patterns, have disturbed sleep–wake cycles, excessive screen time, and sedentary lifestyles. These factors may contribute to gut dysbiosis, inflammation, metabolic disturbances, and worsening psychiatric symptoms. Despite growing evidence, lifestyle-focused gut–brain approaches remain under-emphasized in routine mental health care.

This symposium provides an integrated overview of the gut–brain axis and its relevance to mental health. The first session focuses on clinical and research perspectives of the gut–brain axis in psychiatric illnesses. The second session discusses the role of diet and nutrition as key modulators of gut microbiota and mental health. The third session explores the impact of sleep, physical activity, and daily routines on strengthening gut–brain communication. The fourth session will present findings from a systematic review, where the speaker will synthesise current evidence on gut–brain axis–based interventions and their implications for mental health outcomes. Together, these sessions highlight evidence-based lifestyle approaches that can support mental well-being and improve quality of life.

S-11
Gut-Brain Axis and Mental Health

S-11-001
Gut–Brain Axis in psychiatric illnesses: Clinical Perspectives and research

Speaker: Nishanth K N, India

Abstract

Abstract Title:
Gut–Brain Axis in psychiatric illnesses: Clinical Perspectives and research

Abstract:
Importance of gut brain axis in maintaining homeostasis has long been studied and documented. In the recent years much evidence has evolved about the role of gut microbiota in gut brain axis regulation. This regulation happens through immune system, vagus and enteric nervous system and microbial metabolites such as short chain fatty acids, branched chain amino- acids and peptidoglycans. The research has further received traction due to the association of the microbiota gut brain ecosystem with physiological changes in psychiatric illnesses, neurodevelopmental disorders and neurodegenerative diseases. Much recent work has implicated the gut microbiota in many conditions including autism, anxiety, obesity, schizophrenia, Parkinson's disease, and Alzheimer's disease. Further translational research regarding the role of gut brain axis in psychiatric conditions and treatment implications would be interesting to see.

S-11
Gut-Brain Axis and Mental Health

S-11-002
Diet, Nutrition, and the Gut–Brain Axis

Speaker: Simrat Kaur, India, India

Abstract

Abstract Title:
Diet, Nutrition, and Gut Brain Axix: Intervention in mental health care.

Abstract:
Approximately 15–20% of individuals will encounter mental health illnesses, including depressive episodes or anxiety disorders, at some point in their lives. Thirty to forty percent of individuals with depression do not sufficiently react to pharmaceutical or psychosocial interventions. Stress and depression can modify taste thresholds, perceptions of sweet and fatty meals, and dietary selections. The diet primarily shapes gut flora, which then influences mental health via the bidirectional gut-brain axis.

Food choices directly influence mood, stress levels, and cognitive function via modulating neurotransmitter production, such as serotonin, and systemic inflammation. The brain and the gut maintain continuous communication through the vagus nerve, the immune system, and hormonal pathways. The gut microbiota functions as a biological factory that metabolises ingested food and converts it into essential compounds that affect the brain.

The gut microbiome can be both beneficial and detrimental; it is influenced by nutrition and has been demonstrated to impact mental health, including personality, mood, anxiety, and depression. Recent data shows that dysbiosis of gut microbiota correlates with illnesses like schizophrenia, anxiety, autism spectrum disorders, obesity, Parkinson's disease, and Alzheimer's disease.

Optimising diet for mental wellness involves actively nurturing the good bacteria while removing irritants.

The gut-brain axis is a bidirectional biochemical communication network linking the gastrointestinal tract and central nervous system. Imbalances in gut bacteria (dysbiosis) can drive inflammation, alter neurotransmitter production (like serotonin and dopamine), and trigger anxiety or depression. Targeted dietary and psychological interventions can restore this balance to improve mental health.

S-11
Gut-Brain Axis and Mental Health

S-11-003
Sleep, Physical Activity, and Daily Routines: Strategies to Strengthen the Gut–Brain Axis

Speaker: Sailaxmi Gandhi, India

Abstract

Abstract Title:
Sleep, Physical Activity, and Daily Routines: Nursing Strategies to Strengthen the Gut–Brain Axis in Mental Health

Abstract:
Background:

The gut–brain axis plays a significant role in mental health through bidirectional communication between the gastrointestinal system, immune pathways, and the central nervous system. Emerging evidence suggests that lifestyle factors such as sleep, physical activity, excessive screen exposure, and daily routines influence gut microbiota composition, circadian rhythm regulation, neuroinflammation, and emotional functioning. Individuals with severe mental illness commonly experience sleep disturbances, sedentary behaviour, irregular routines, and poor lifestyle habits, which may contribute to gut dysbiosis, metabolic disturbances, and worsening psychiatric symptoms. Despite increasing evidence supporting lifestyle-based interventions targeting the gut–brain axis, these approaches remain under-utilised in routine psychiatric nursing care.

Objectives:

1. To explain the relationship between sleep, circadian rhythm, physical activity, and the gut–brain axis.

2. To examine the impact of disrupted sleep and sedentary lifestyle on mental health outcomes.

3. To discuss evidence-based nursing strategies that promote sleep hygiene, physical activity, and daily routine regulation.

4. To highlight the role of caregivers and adherence monitoring in sustaining healthy lifestyle practices.

Presentation Overview:

This presentation focuses on practical nurse-led strategies to strengthen the gut–brain axis through lifestyle modification in mental health settings. The session will discuss the influence of disrupted sleep–wake cycles, excessive screen exposure, and physical inactivity on gut microbiota and psychiatric symptoms. Evidence-based approaches including sleep hygiene

education, structured daily routines, physical activity promotion, and lifestyle counselling will be emphasized.

Participants will also be introduced to caregiver-supported interventions and adherence monitoring strategies that promote sustained behavioural changes. The presentation highlights the expanding role of psychiatric nurses in integrating lifestyle-based gut–brain axis interventions into routine mental health care to improve psychiatric outcomes, physical health, and quality of life.

Conclusion:

Lifestyle-based interventions targeting the gut–brain axis may serve as feasible and holistic strategies in mental health care. Psychiatric nurses play an important role in promoting healthy routines, behavioural adherence, and caregiver involvement to support both physical and psychological well-being.

S-11
Gut-Brain Axis and Mental Health

S-11-004
The Gut–Brain Axis and Mental Health: A Systematic Review

Speaker: Maha S, India

Abstract

Abstract Title:
Gut Microbiota Alterations in Bipolar Disorder: A Systematic Review

Abstract:
Background: The gut–brain axis is a bidirectional communication network linking the gastrointestinal system and the central nervous system through neural, immune, endocrine, and metabolic pathways. Emerging evidence suggests that alterations in gut microbiota may contribute to the pathophysiology of Bipolar Disorder through mechanisms involving neuroinflammation, immune dysregulation, altered neurotransmitter activity, and metabolic disturbances. Individuals with bipolar disorder commonly experience sleep disturbances, dietary irregularities, a sedentary lifestyle, and psychotropic medication exposure, all of which may influence gut microbial composition and intestinal health.

Objectives: To systematically review current evidence on gut microbiota alterations in bipolar disorder and examine their association with mood symptoms, inflammation, metabolic abnormalities, and psychotropic medication use.

Methods: Human studies published between January 2005 and January 2026 were reviewed using Medline, PsycINFO, and EMBASE databases. Studies examining gut microbiota composition, microbial diversity, inflammatory markers, metabolic parameters, and gut-related interventions among individuals with bipolar disorder were included.

Results: Findings demonstrated significant alterations in gut microbial diversity and composition among individuals with bipolar disorder compared with healthy controls. Reduced microbial diversity, increased inflammatory activity, altered intestinal permeability, and metabolic abnormalities were frequently reported. Several studies also suggested associations between gut dysbiosis and the severity of mood symptoms, sleep disturbances, and psychotropic medication exposure. Emerging evidence indicates that microbiota-targeted interventions, including dietary modifications and probiotic supplementation, may have potential benefits as adjunctive approaches in bipolar disorder management.

Conclusion: Gut microbiota alterations appear to play an important role in the biological mechanisms underlying bipolar disorder. Understanding gut–brain axis interactions may contribute to the development of novel adjunctive interventions targeting inflammation, metabolism, and emotional regulation. Integrating gut-focused approaches into psychiatric care may support holistic management and improve long-term outcomes in bipolar disorder.

S-12

Trauma-Focused Intervention Across the Lifespan: Mechanisms, Modalities, and Clinical Applications

Chair: Susmita Halder, India

Co-Authors:

Supratik Kundu

Topic:
10) Other

Abstract

Objectives:
The symposium aims to understand the psychopathological manifestation of trauma across different age groups; its underlying neurobiological and psychological mechanisms focusing on tailored trauma focused psychotherapy and its outcome, effectiveness and clinical implications.

Structured rationale:
Psychological trauma is a pervasive risk factor for multiple psychiatric disorders, yet its clinical expression varies considerably across developmental stages. Current diagnostic and treatment approaches often apply uniform models of trauma, overlooking age-dependent neurodevelopmental processes, differences in cognitive–emotional organization, and variability in treatment response. This contributes to heterogeneity of presentation, partial treatment response and chronicity. A lifespan perspective is critical in linking developmental neurobiology with evolving psychological processes. Understanding how trauma mechanisms change from childhood to adulthood can inform selection and integration of tailored psychological and pharmacological treatments. This symposium therefore proposes a developmentally informed, mechanism-based framework for trauma care, aiming to improve precision in assessment and intervention planning across age groups.

S-12
Trauma-Focused Intervention Across the Lifespan: Mechanisms, Modalities, and Clinical Applications

S-12-001
Age-Specific Psychopathological Manifestations of Trauma

Speaker: Debaleena Ghosh, India

Abstract

Abstract Title:
Age-Specific Psychopathological Manifestations of Trauma

Abstract:
The symposium aims to understand the psychopathological manifestation of trauma across different age groups; its underlying neurobiological and psychological mechanisms focusing on tailored trauma focused psychotherapy and its outcome, effectiveness and clinical implications. Psychological trauma is a pervasive risk factor for multiple psychiatric disorders, yet its clinical expression varies considerably across developmental stages. Current diagnostic and treatment approaches often apply uniform models of trauma, overlooking age-dependent neurodevelopmental processes, differences in cognitive–emotional organization, and variability in treatment response. This contributes to heterogeneity of presentation, partial treatment response and chronicity. A lifespan perspective is critical in linking developmental neurobiology with evolving psychological processes. Understanding how trauma mechanisms change from childhood to adulthood can inform selection and integration of tailored psychological and pharmacological treatments.

This symposium therefore proposes a developmentally informed, mechanism-based framework for trauma care, aiming to improve precision in assessment and intervention planning across age groups.

S-12
Trauma-Focused Intervention Across the Lifespan: Mechanisms, Modalities, and Clinical Applications

S-12-003
Tailored Trauma-Focused Psychotherapies in Developmental Context

Speaker: Madhurima Dey Sarkar, India

Abstract

Abstract Title:
Tailored Trauma-Focused Psychotherapies in Developmental Context

Abstract:
Trauma manifests differently across developmental stages due to variations in neurobiological maturation, emotional regulation, cognitive processing, attachment patterns, and social context. Consequently, trauma-focused psychotherapies require developmental tailoring rather than a uniform treatment approach across age groups. This presentation aims to discuss how developmental context influences the assessment, formulation, and psychotherapeutic management of trauma-related psychopathology.

The presentation will explore age-sensitive trauma-focused interventions across childhood, adolescence, and adulthood, highlighting how therapeutic goals, emotional processing, therapeutic alliance, and symptom expression differ across the lifespan. Particular emphasis will be placed on integrating developmentally informed approaches within evidence-based trauma therapies, including trauma-focused cognitive behavioural approaches, acceptance-based interventions, attachment-oriented work, and emotionally focused therapeutic processes.

Clinical illustrations will be used to demonstrate how trauma may present through behavioural dysregulation, dissociation, anxiety, affective disturbances, interpersonal difficulties, and maladaptive coping across different developmental periods. The presentation will further discuss the importance of flexibility in intervention planning, consideration of relational and environmental factors, and the role of psychological resilience and meaning-making in recovery.

A developmental framework for trauma-focused psychotherapy may contribute toward more precise, individualized, and clinically effective interventions across diverse age groups.

S-13
Organ Transplantation and Psychiatry: Biological, Psychological, and Ethical Interfaces Across the Transplant Continuum

Chair: Shirshendu Sinha, USA

Co-Authors:

Debjani Bandyopadhyay

Topic:
10) Other

Abstract

Objectives:
This symposium aims to examine the critical role of psychiatry in organ transplantation across the pre-transplant, peri-transplant, and post-transplant phases. The session will explore psychiatric assessment frameworks for transplant eligibility, the management of pre-existing psychiatric disorders, and the identification of psychosocial risk factors that influence transplant outcomes. It will also review emerging evidence on neuropsychiatric complications associated with immunosuppressive therapies, adherence challenges, and long-term psychological adjustment following transplantation. Additionally, the symposium will address culturally informed ethical considerations in transplant psychiatry, including issues related to consent, donor–recipient dynamics, and disparities in access to transplantation services across diverse healthcare systems. Through international perspectives, the symposium seeks to highlight both shared challenges and region-specific solutions, fostering collaborative approaches to improving mental health outcomes among transplant recipients.

Structured rationale:
Advances in surgical techniques and immunosuppressive therapies have significantly improved survival rates following organ transplantation, transforming transplantation into a standard therapeutic modality for end-stage organ failure. However, psychiatric and psychosocial factors remain key determinants of transplant eligibility, treatment adherence, graft survival, and quality of life. Patients undergoing transplantation frequently experience complex emotional responses, including anxiety, depression, adjustment difficulties, and cognitive changes, which may affect medical outcomes if left unrecognized or untreated. Furthermore, transplant recipients face long-term challenges related to medication adherence, identity transformation, fear of graft rejection, and social reintegration. Despite growing recognition of transplant psychiatry as a subspecialty, variations exist globally in screening protocols, intervention strategies, and integration of psychiatric care within transplant teams. A multidisciplinary and culturally sensitive approach is therefore essential to optimize both medical and psychological outcomes. This symposium provides an opportunity to integrate international clinical experiences and research findings, advancing the role of biological psychiatry in transplant medicine and strengthening collaborative models of care.

S-13
Organ Transplantation and Psychiatry: Biological, Psychological, and Ethical Interfaces Across the Transplant Continuum

S-13-001
Pre-Transplant Psychiatric Assessment and Psychosocial Risk Evaluation: International Perspectives

Speaker: Shirshendu Sinha, USA

Abstract

Abstract Title:
Organ Transplantation and Psychiatry: Biological, Psychological, and Ethical Interfaces Across the Transplant Continuum

Abstract:
This symposium examines the pivotal role of psychiatry across the continuum of organ transplantation, including pre-, peri- and post-transplant phases. It highlights psychiatric assessment for transplant eligibility, management of pre-existing mental health conditions, and identification of psychosocial factors influencing outcomes. The session also reviews emerging evidence on neuropsychiatric complications of immunosuppressive therapies, adherence challenges, and long-term psychological adjustment.

With advances in transplant medicine improving survival, psychiatric and psychosocial variables remain critical determinants of outcomes, including adherence, graft survival, and quality of life. Transplant recipients frequently experience substance use disorders, anxiety, depression, cognitive changes, and complex adjustment processes that may impact recovery if unaddressed. Additionally, ethical and cultural considerations—such as informed consent, donor–recipient dynamics, and disparities in access—are increasingly recognized as integral to care.

Drawing on international perspectives, this symposium aims to identify shared challenges and region-specific solutions, emphasizing multidisciplinary and culturally sensitive approaches to optimize both medical and mental health outcomes in transplant populations.

S-14
The bio-clinical intersection of perinatal psychiatry: from placental programming to pharmacokinetic precision

Chair: Priyanka Banokar, India

Co-Authors:

Malay Dave

Topic:
5) Women's mental health

Abstract

Objectives:
Learning Objectives for the Symposium

1. Evaluate the "Placental-Brain Axis": Participants will be able to describe the role of the placenta as a dynamic mediator between maternal stress and fetal neurodevelopment, identifying key epigenetic and metabolic pathways that contribute to early-life programming of psychiatric illness.

2. Optimize Trimester-Specific Dosing: Participants will gain the clinical competency to adjust psychotropic regimens based on gestational pharmacokinetic shifts, specifically accounting for changes in V_d (Volume of Distribution) and CYP450 enzyme induction.

3. Execute Evidence-Based Risk-Benefit Analysis: Participants will be equipped to counsel patients using the latest global safety data, moving beyond "teratogenic potential" to include neonatal adaptation and long-term neurobehavioral outcomes in their clinical decision-making.

Structured rationale:
The structural rationale for this symposium is built upon a translational continuum, designed to move the clinician from the molecular foundations of fetal development to the practical complexities of pharmacological management.

By structuring the session in this specific sequence, we address the "why," the "how," and the "what" of modern perinatal psychiatry.

1. The Biological Foundation: "The Why"

The symposium begins with the Placental-Brain Axis to establish the long-term stakes of perinatal mental health. By framing the placenta as a "functional diary" of maternal stress, we move the conversation beyond immediate symptom management.

• Rationale: To provide a biological imperative for intervention. Understanding that maternal psychiatric illness can epigenetically program the fetal brain shifts the clinician's perspective from "treating a patient" to "protecting a developmental trajectory."

2. The Physiological Bridge: "The How"

Once the need for intervention is established, the second segment addresses the Pharmacokinetic (PK) hurdles unique to pregnancy. This serves as the technical bridge between biology and bedside.

• Rationale: To deconstruct the "one-size-fits-all" dosing myth. By detailing the induction of CYP450 enzymes and the expansion of the Volume of Distribution (V_d), we provide the physiological explanation for why standard doses often fail in the second and third trimesters, leading to avoidable relapses.

3. The Clinical Application: "The What"

The final segment concludes with Safety Profiles, applying the biological and physiological concepts to real-world decision-making.

• Rationale: To resolve the "prescriber's dilemma." After establishing the risks of untreated illness (Part 1) and the mechanics of dosing (Part 2), this section provides the evidence-based safety data (teratogenicity vs. neonatal adaptation) required to finalize a treatment plan.

The "Dyadic" Conclusion

The ultimate structural goal is to synthesize these three pillars into a dyadic care model. The symposium concludes that the health of the mother and the fetus are not competing interests, but a singular biological unit where optimized maternal psychopharmacology is the primary tool for neurodevelopmental prophylaxis.

S-15
From neurodevelopment to neurodiversity: biological mechanisms and emerging frontiers in autism

Chair: Tamoghna Bandyopadhyay, India

Co-Authors:

Aritra Chakraborty

Topic:
10) Other

Abstract

Objectives:
This symposium aims to examine autism through the evolving lens of biological psychiatry, tracing its journey from a classical neurodevelopmental disorder to a condition understood within the broader framework of neurodiversity. The session will integrate advances in genetics, synaptic biology, brain connectivity, immune mechanisms, and developmental neuroscience to highlight emerging biomarkers and translational implications. By bridging molecular research with lifespan perspectives and functional outcomes, the symposium seeks to contextualise autism within a changing world that increasingly values dimensional and individualised approaches. The objective is to foster biologically informed, ethically grounded, and clinically meaningful frameworks for understanding autism in contemporary psychiatric practice.

Structured rationale:
Autism has traditionally been conceptualised as a neurodevelopmental disorder rooted in early brain alterations. However, rapid advances in genetics, synaptic biology, connectomics, and neuroimmunology have transformed our understanding of its biological heterogeneity. Simultaneously, the emergence of the neurodiversity paradigm challenges deficit-based models and calls for dimensional, lifespan-oriented perspectives. In a changing world, biological psychiatry must integrate mechanistic discoveries with evolving social and ethical frameworks. This symposium is timely because it bridges molecular mechanisms, brain network science, and translational research with contemporary conceptual shifts, offering a comprehensive biological framework that informs diagnosis, intervention, and individualised care in autism.

S-15
From neurodevelopment to neurodiversity: biological mechanisms and emerging frontiers in autism

S-15-003
From Disorder to Neurodiversity: Lifespan Biology and Translational Implications

Speaker: Tamoghna Bandyopadhyay, India

Abstract

Abstract Title:
From neurodevelopment to neurodiversity: biological mechanisms and emerging frontiers in autism.

Abstract:
This symposium will explore contemporary biological perspectives on autism spectrum disorder (ASD), spanning neurodevelopmental mechanisms, genetic and epigenetic influences, neural circuitry, and biomarkers. Emphasis will be placed on advances in neuroimaging, translational neuroscience, and precision medicine that are reshaping our understanding of autism across the lifespan. The symposium will also examine the evolving paradigm of neurodiversity, highlighting how biological research can coexist with strengths-based and person-centred approaches. By integrating basic science, clinical findings, and emerging therapeutic innovations, the session aims to provide a comprehensive overview of current knowledge and future directions in autism research and care.

S-16
Development of the International Consortium for Preclinical Psychiatric Research

Chair: Rachel Hill, Australia

Co-Authors:

Jee Hyun Kim

Topic:
10) Other

Abstract

Objectives:
The objective of this symposium is to launch the official WFSBP CPPR task force, dedicated to the expansion of the Australian based Consortium for Preclinical Psychiatric Research (CPPR) through international nodes. Secondary objectives are to learn about current collaborative projects currently funded through the CPPR, and to explore the appetite for expansion of these collaborative endeavours across multiple nations, including India, Africa and the UK. The vision of this task force is the coalescing of preclinical psychiatric / mental health research into a collaborative global network.

Speakers will include the founder of the CPPR and President of Biological Psychiatry Australia, A/Prof. Rachel Hill. A/Prof. Hill will provide a detailed overview of the CPPR and the funded projects currently ongoing.

Dr. Reinmar Hager (The University of Manchester) will next present on the development of the UK node of the CPPR and the alignment of the CPPR goals with British Biological Psychiatry research groups.

Prof. Vidita Vaidya (Tata Institute of Fundamental Research) will next report on the diverse research programs focussed on preclinical and clinical research in mental health within India, and the capacity for collaborative gain through connecting India with the WFSBP CPPR task force.

Structured rationale:
Globally, mental disorders remain in the top 10 individual leading causes of burden in 2019, with no reduction since 1990. Yet the European Medicines Agency only approved one new treatment in the last three years for mental disorders compared to 68 for oncology. This catastrophic failure to progress new treatments for mental disorders has left many people with access only to palliative or symptomatic treatments which for many are poorly tolerated or ineffective. The absence of course-modifying treatments or diagnostic biomarkers for mental disorders constitutes a major unmet health need. A key challenge to progress in biological psychiatry includes access to appropriate samples from patients, as well as limited standardised empirical biological data related to robust characterisation of the disorder presentations. As

such, biological psychiatry attempts to create model types that are comparable to biological psychiatric conditions observed in post mortem human brain samples.

Within Australia, biological psychiatry is represented by world leading psychiatrists, neuroscientists and molecular biologists who have recently formed a Consortium for Preclinical Psychiatric Research. This consortium, founded and chaired by A/Prof. Rachel Hill (Monash University), currently consists of over 60 members from 14 different Universities / Institutes across Australia (https://www.monash.edu/medicine/scs/research/consortium-for-preclinical-psychiatric-research). The vision statement for the CPPR is 'To improve biological understanding of mental illness and fast-track the discovery of new treatments through collaboration'. The CPPR coalesces a large national tissue resource of post-mortem human and animal brain tissue and peripheral clinical samples with comprehensive phenotype/ diagnostic linked information, held in an open access repository - the CPPR – Knowledge databank (https://doi.org/10.6084/m9.figshare.25114685.v2).

An initial aim of CPPR is to provide the co-ordination and leadership for standardised creation of large-scale molecular data generated through omics technologies, across a range of currently disparate preclinical model types used to study causality of mental disorders. By harmonising the models through standardised data collection, we aim to better understand biological causality and develop robust, and empirically assessed models that can be deployed for development in novel drug treatment, diagnosis, treatment stratification and prognostics. The visionary nature of this consortium has already attracted considerable funding from Wellcome Trust and Bioplatforms Australia.

Our next goal is to expand the CPPR as a global consortium.

S-16
Development of the International Consortium for Preclinical Psychiatric Research

S-16-001
Introducing the Consortium for Preclinical Psychiatric Research

Speaker: Rachel Hill, Australia

Abstract

Abstract Title:
The Australian founding node of the Consortium for Preclinical Psychiatric Research

Abstract:
The Consortium for Preclinical Psychiatric Research (CPPR) was founded in 2024 by A/Prof. Rachel Hill (Monash University) and currently consists of over 60 members from 14 different Universities / Institutes across Australia. The vision statement for the CPPR is 'To improve biological understanding of mental illness and fast-track the discovery of new treatments through collaboration'. In this symposium A/Prof. Hill will present on two major projects initiated by the Australian CPPR and discuss the utility of expanding these projects as global collaboratives.

The first project is funded by Wellcome Trust and aims to develop an online resource, called the Animal Models in Neuropsychiatric Disorders (A MiND) Resource. This resource will evaluate and rank animal models and behavioural assays for psychiatric disorders, helping researchers select the most suitable model and assay for a given question, improving reproducibility, and reducing unnecessary animal use.

The second project developed from a large national tissue resource of post-mortem human and animal brain tissue and peripheral clinical samples with comprehensive phenotype/ diagnostic linked information, held in an open access repository - the CPPR – Knowledge databank. Funded by Bioplatforms Australia, the CPPR are using these banked tissue samples to create standardised large-scale molecular data generated through omics technologies, across a range of currently disparate preclinical model types used to study causality of mental disorders. By harmonising the models through standardised data collection, we aim to better understand biological causality and develop robust, and empirically assessed models that can be deployed for development in novel drug treatment, diagnosis, treatment stratification and prognostics.

A final objective of this symposium is to launch the official WFSBP CPPR task force, dedicated to the expansion of the Australian based Consortium for Preclinical Psychiatric Research (CPPR) through international nodes.

S-16
Development of the International Consortium for Preclinical Psychiatric Research

S-16-002
Establishing a UK node of the Consortium for Preclinical Psychiatric Research, alignment with UK priorities

Speaker: Reinmar Hager, United Kingdom

Abstract

Abstract Title:
The UK CPPR – a unique opportunity to transform preclinical research

Abstract:
Preclinical research in the UK has a long and highly successful history of making significant advances in fundamental research that contributes to improved treatment and patient benefits. Combining multi-omics approaches in both animal and cellular models with novel behavioural and molecular technologies have enabled the interrogation of how genetic and environmental factors interact in complex ways across development and can result in trajectories leading to increased disease risk. Yet, challenges remain, in particular around increasing translational relevance. The UK node of the Consortium for Preclinical Psychiatric Research aims to address key challenges by bringing together research groups working across disciplines on cellular, animal and postmortem samples, and to leverage the strength of the UK research ecosystem integrating research institutes, specialized repositories such as brain banks and university and hospital based research groups.

S-16
Development of the International Consortium for Preclinical Psychiatric Research

S-16-003
Showcasing the diverse research programs across preclinical and clinical mental health research in India

Speaker: Vidita Vaidya, India

Abstract

Abstract Title:
Serotonergic Psychedelics - from mitochondria to mood

Abstract:
In my talk, I will discuss the preclinical studies that indicate potent effects of serotonergic psychedelics on bioenergetics, neuronal plasticity and mood-related behaviors in rodent models. These studies uncover underlying mechanisms targeted by serotonergic psychedelics that are relevant to their potential therapeutic actions.

S-17

From the Field to the Synapse: A Psychoneuroimmunoendocrinological and Biopsychosocial Analysis of Farmer Suicides in India

Chair: Prashant Chaudhari, India

Co-Authors:

Krishnapriya Murlimanohar

Topic:
10) Other

Abstract

Objectives:
To critically examine farmer suicides in India through an integrated Psychoneuroimmunoendocrinological (PNIE) and Biopsychosocial framework, and to identify actionable biological and clinical targets that can be incorporated into psychiatric and primary care practice.

Structured rationale:
By the end of this symposium, participants will be able to:

Recognize the limitations of purely psychosocial and economic models in explaining variability in suicide risk among farmers exposed to similar stressors.

Understand the neurobiological impact of chronic low-dose pesticide exposure, including:

Acetylcholinesterase inhibition

Serotonergic and dopaminergic dysregulation

Microglial activation and inflammatory signaling

Appreciate the role of nutritional vulnerabilities, particularly Vitamin B12 deficiency and hyperhomocysteinemia, in modulating depressive and suicidal phenotypes in rural populations.

Apply the PNIE framework to conceptualize how environmental stressors are biologically transduced via:

HPA-axis dysregulation

Immune activation

Neurotransmitter alterations

Differentiate environmental depression from conventional major depressive disorder, with emphasis on clinical presentation in farming communities (somatic phenotype, irritability, impulsivity).

Identify practical screening and intervention strategies suitable for integration into:

Primary Health Centres (PHCs)

District mental health services

National Mental Health Programme (NMHP)

Evaluate the role and limitations of non-pharmacological interventions (e.g., meditation, counselling) when significant biological burden is present.

Formulate a biologically informed suicide-prevention approach that integrates toxin exposure history, nutritional assessment, pharmacotherapy, and psychosocial support.

Long-Term Goal

To stimulate research, policy adaptation, and clinical innovation that moves farmer suicide prevention beyond reactive crisis management toward biologically informed, preventive psychiatric care.

S-17
From the Field to the Synapse: A Psychoneuroimmunoendocrinological and Biopsychosocial Analysis of Farmer Suicides in India

S-17-001
Farmer Suicide in India: Clinical Realities Beyond the Psychosocial Narrative

Speaker: Faisal Mohammad, India

Abstract

Abstract Title:
From Synapse Outward — Organophosphate, HPA & Microbial Crosstalk in Environmental Depression

Abstract:
The farmer's brain is a chronically perturbed neuroimmunoendocrine organ. This talk dissects the mechanistic chain from chronic low-dose organophosphate (OP) exposure to depressive end-state. Acetylcholinesterase inhibition is the first hit, but OPs additionally produce 5-HT1A receptor down-regulation, mesolimbic dopaminergic disruption, microglial priming via TLR-4 / NLRP3 activation, and oxidative injury that lowers BDNF–TrkB signalling in hippocampus and prefrontal cortex. The pharmacokinetic profile of lipophilic OPs — long half-life in adipose tissue, daily reintroduction without PPE, no clean detoxification window — creates an effectively continuous exposure regime, fundamentally distinct from the discrete acute poisoning model that dominates toxicology textbooks. Layered upon this is rural nutritional context: 64–67% prevalence of B12 deficiency in Maharashtra rural surveys (Yajnik et al., Pune cohort), vegetarian baseline, alcohol-induced malabsorption, and elevated homocysteine producing endothelial and neuronal injury that synergises with OP neurotoxicity. The third axis is psychoneuroimmunology: chronic exposure plus chronic stress elevates IL-6 and TNF-α and activates indoleamine-2,3-dioxygenase (IDO), shunting tryptophan into the kynurenine arm, depleting serotonin and generating NMDA-agonist quinolinic acid — the molecular substrate of sickness behaviour that clinicians and families misread as laziness or moral weakness. Persistent hypercortisolaemia and glucocorticoid resistance explain the documented SSRI non-response in field studies. A unified circuit model — Fields → Immune → Synapse — frames depression as the predictable phenotype of an organism whose allostatic budget is exhausted by toxin + stress + nutrient deficit. The therapeutic implication: SSRIs alone are insufficient; biological correction (IM B12 1000 mcg weekly × 8 → monthly, folate, cortisol monitoring, removal from exposure where possible) must run in parallel. A PHC-deployable biomarker panel (homocysteine, CRP/IL-6, AChE proxy, AM cortisol) is previewed.

Key references: Voorhees JR et al. npj Aging 2018; Soreq H et al. Trends Mol Med 2020; Miller AH & Raison CL. Nat Rev Immunol 2016; Dantzer R et al. Nat Rev Neurosci 2008; Yajnik CS et al. J Assoc Physicians India 2006; Ecotoxicol Environ Saf 2024 OP–suicide cohort.

S-17
From the Field to the Synapse: A Psychoneuroimmunoendocrinological and Biopsychosocial Analysis of Farmer Suicides in India

S-17-002
Environmental Depression: Neurotoxic and Inflammatory Pathways in Farming Communities

Speaker: Anshu Varma, India

Abstract

Abstract Title:
From Biology Back to the Field — Biopsychosocial Integration & a Synapse-First Suicide-Prevention Architecture

Abstract:
Speaker: Dr Krishnapriya Murlimanohar

Knowing the biology is necessary but not sufficient; translating it into PHC-deployable suicide prevention is the decisive step. This talk synthesises the preceding clinical phenotype (Verma) and molecular mechanism (Faisal) into an operational framework — Screen → Correct → Support — engineered for India's primary care infrastructure. A four-tier screen is detailed: (i) brief somatic-mood items (PHQ-S, GAD-S adapted for somatic-dominant presentation); (ii) nutritional indices (B12, folate, homocysteine); (iii) toxicology surrogates (AChE proxy or validated symptom-cluster scoring where lab access is absent); (iv) HPA markers (AM cortisol when feasible). Correction includes IM B12 + folate as low-cost high-impact first-line nutritional rehabilitation, structured removal from active exposure where possible, and treatment of the glucocorticoid-resistant phenotype with anti-inflammatory adjuncts (NAC, omega-3, addressing inflammatory load) prior to or alongside SSRI initiation. Support extends beyond counselling-only models to community-level economic stabilisation, ASHA-worker–delivered psychoeducation that reframes somatic complaints as biological signals, and pesticide access reduction — among the most evidence-based suicide-prevention interventions globally (WHO LIVE LIFE 2021; Mann JJ JAMA 2005). Loan-waiver and meditation-only interventions are critically appraised: necessary but insufficient and prone to substitution error when biology is ignored. The talk culminates in a paradigm-shift statement — loan waivers stabilise land; psychotherapy stabilises the mind; PNIE stabilises the farmer. Confidence: high for biological components (SORT A); medium for integrated programme (SORT B; pilot data). A three-state implementation roadmap (Maharashtra → Karnataka → Telangana) tied to NCRB mortality endpoints is proposed.

Key references: Mann JJ et al. JAMA 2005; Patel V et al. Lancet 2017 (MANAS/PRIME); WHO LIVE LIFE Suicide Prevention 2021; Vijayakumar L. Indian J Psychiatry 2020; Köhler-Forsberg O et al. Acta Psychiatr Scand 2019.

S-17
From the Field to the Synapse: A Psychoneuroimmunoendocrinological and Biopsychosocial Analysis of Farmer Suicides in India

S-17-003
Bridging Psychiatry and Primary Care: A Biologically Informed Suicide Prevention Model

Speaker: Pankaj Patil, India

Abstract

Abstract Title:
Clinical Signals of Manufacturing Drift — Why Psychiatry Is the Sentinel

Abstract:
Part 1: Clinical Signals of Manufacturing Drift — Why Psychiatry Is the Sentinel

Indian pharmacovigilance has repeatedly been blind-sided by manufacturing failures detectable only through their behavioural phenotype; the Coldrif paediatric DEG cluster is the catastrophic-end illustration of a slow-burn systemic problem. This talk argues that CNS drugs are the most sensitive bioassay of manufacturing quality and presents anonymised case clusters that operationalise the claim. Case A — escitalopram brand switch in a stably remitted patient producing emotional blunting and partial efficacy loss despite identical labelled dose, traceable to altered dissolution and excipient-mediated changes in CNS serotonergic tone. Case B — valproate manufacturer switch in a 19 year old euthymic bipolar patient: breakthrough hypomania, tremor, GI intolerance with serum valproate “within range,” illustrating that serum concentration is a necessary but inadequate surrogate of brain exposure for narrow-therapeutic-index (NTI) CNS drugs. Case C — paroxetine switch precipitating SSRI discontinuation syndrome (“electric shocks,” dizziness, anxiety) misdiagnosed as relapse: the patient was relabelled rather than re-formulated. Case D — clozapine post-procurement change producing sedation and hypersalivation reframed by clinicians as “tolerance loss.” The common pathophysiology across signals is peak–trough variability, altered Cmax / Tmax, excipient-driven absorption variance, and polymorphic dissolution behaviour — none of which are captured by single-dose healthy-volunteer bioequivalence studies. The orphan-comparator problem is introduced (e.g., lemborexant has only one marketed brand in India: tolerability change cannot be scientifically attributed to formulation versus illness progression). The clinical take-home: psychiatric symptom drift after generic switch is data, not noise, and must be reported and triangulated.

Key references: Borg heini G. Clin Ther 2003; Margolese HC et al. CNS Drugs 2010; Berg MJ et al. Neurology 2008; FDA Dissolution & Excipient Impact Reviews; WHO Coldrif Investigation Report 2024.

S-18
Quantitative EEG in Precision Psychiatry: Current evidence, Translational potential and Clinical utility

Chair: Shubhmohan Singh, India

Co-Authors:

Nishant Goyal

Topic:
10) Other, 1) Psychoses

Abstract

Objectives:
To evaluate the neurophysiological basis, clinical utility, and computational integration of qEEG in precision psychiatry.

Structured rationale:
Psychiatry remains largely symptom-based despite growing evidence of circuit-level dysfunction underlying mental disorders. Quantitative EEG provides a scalable, non-invasive method to measure oscillatory and connectivity abnormalities that can inform treatment stratification and longitudinal monitoring. Integrating qEEG within computational and biomarker-driven frameworks may advance personalized, biologically grounded psychiatric care.

S-18
Quantitative EEG in Precision Psychiatry: Current evidence, Translational potential and Clinical utility

S-18-002
Disorder-Wise Evidence and Clinical Utility

Speaker: Harshit Harshit, India

Abstract

Abstract Title:
Disorder-Wise Evidence and Clinical Utility

Abstract:
The clinical utility of qEEG varies across psychiatric disorders. In major depressive disorder, frontal alpha asymmetry and elevated anterior cingulate theta are among the most consistently replicated findings. Although diagnostic specificity remains modest, meta-analytic data support the predictive value of qEEG for antidepressant and repetitive transcranial magnetic stimulation (rTMS) response, positioning qEEG as a tool for treatment stratification rather than diagnosis. In schizophrenia, increased delta/theta power, reduced gamma synchrony, and impaired long-range connectivity reflect disrupted excitatory–inhibitory balance and large-scale network dysfunction. BSNIP findings further support biologically distinct psychosis biotypes based on electrophysiological and cognitive markers, though broader validation is required before routine implementation. Delirium demonstrates robust generalized slowing with increased delta/theta and reduced alpha peak frequency, supporting diagnostic and monitoring utility. Similarly, in dementia and mild cognitive impairment, alpha slowing and theta augmentation correlate with molecular biomarkers. Evidence in anxiety and substance use disorders remains limited and preliminary.

S-18
Quantitative EEG in Precision Psychiatry: Current evidence, Translational potential and Clinical utility

S-18-003
Translational Integration and Precision Psychiatry

Speaker: Pankaj Kumar, India

Abstract

Abstract Title:
Translational Integration and Precision Psychiatry

Abstract:
Implementing qEEG in psychiatric care requires systematic strategies grounded in circuit-based models such as RDoC, which emphasise dimensional neural mechanisms over categorical diagnoses.8 Personalisation may be guided by well-validated biomarkers as discussed previously. As highlighted in the DSM-6 biomarker roadmap, candidate biomarkers must meet standards of reproducibility, validation, and clinical utility before integration into diagnostic frameworks.9 Longitudinal electrophysiological markers may aid treatment monitoring and relapse prediction. However, challenges—including normative database variability, protocol standardization, cost-effectiveness, and regulatory oversight—must be addressed to ensure responsible and scalable implementation.10

S-19

Recent advances in biological treatment of erectile dysfunction: a practical approach for the biological psychiatrist

Chair: Ambrish Singal, India

Co-Authors:

Shyama Singal

Topic:
10) Other

Abstract

Objectives:
This symposium aims to provide an advanced, clinically relevant understanding of erectile dysfunction (ED) from a biological psychiatry perspective, emphasizing the translation of neuroscience into practical treatment strategies.

The objectives are to:

• Reframe erectile dysfunction as a multidimensional neurobiological disorder rather than a purely vascular condition.

• Examine the central neurochemical pathways governing sexual function, including dopaminergic, oxytocinergic, melanocortin, and serotonergic systems.

• Review recent and emerging biological treatments with demonstrated or promising clinical applicability.

• Equip psychiatrists with mechanism-based approaches to assessment and treatment, particularly in patients with psychiatric comorbidities or medication-induced sexual dysfunction.

• Promote evidence-informed, multidisciplinary care models that enhance patient outcomes and quality of life.

Structured rationale:
Erectile dysfunction is increasingly recognized as a disorder arising from the integrated functioning of brain, hormones, vasculature, and psychological processes. Advances in neurobiology have reshaped our understanding of sexual arousal as a centrally regulated phenomenon involving a delicate balance between excitatory and inhibitory neurotransmitter systems. This paradigm shift holds particular relevance for biological psychiatrists, who routinely manage conditions—and prescribe treatments—that directly influence these pathways.

Despite its high prevalence, ED remains under-recognized in psychiatric settings. Patients often hesitate to report symptoms, while clinicians may lack familiarity with rapidly evolving biological therapies. Untreated sexual dysfunction can significantly impair self-esteem, intimate relationships, treatment adherence, and overall psychological wellbeing, thereby worsening the course of primary psychiatric illness.

Recent years have witnessed substantial progress in mechanism-driven treatments that extend beyond traditional peripheral agents. Centrally acting pharmacotherapies and melanocortin receptor agonists have expanded the therapeutic landscape by directly targeting neural circuits of desire and arousal. Regenerative strategies such as stem cell therapy and platelet-rich plasma are being actively investigated for their restorative potential, while low-intensity extracorporeal shockwave therapy has generated growing interest as a modality capable of improving penile vascular health. Additionally, advances in vascular interventions, including penile artery stenting, suggest a future in which selected patients may benefit from precision-based restoration of blood flow.

However, the expanding range of treatments has created a translational gap between scientific innovation and everyday psychiatric practice. Clinicians require structured guidance on patient selection, safety considerations, ethical boundaries, and real-world feasibility.

This symposium addresses that gap by integrating neurobiological insight with pragmatic clinical frameworks. Participants will gain actionable strategies for biologically informed assessment, prescribing, augmentation, and referral pathways, along with guidance for managing antidepressant-associated sexual dysfunction and complex presentations where psychological and organic factors intersect.

By positioning erectile dysfunction within the domain of biological psychiatry, this symposium encourages a shift from symptom-focused care toward mechanism-based intervention. Ultimately, improving sexual functioning is not solely about restoring performance—it is about strengthening intimacy, preserving relationships, enhancing treatment adherence, and promoting holistic mental health.

S-20
When Bioequivalence is not Biologic Equivalence: Manufacturing Variability and Neurobehavioral Outcomes in Psychiatry

Chair: Prashant Chaudhari, India

Co-Authors:

Anshu Varma

Topic:
10) Other

Abstract

Objectives:
By the end of this symposium, participants will be able to:

Explain the limitations of current bioequivalence standards for CNS drugs.

Recognize clinical patterns suggestive of formulation-related variability.

Understand excipient and dissolution impacts on neurobiological exposure.

Discuss the need for CNS-specific regulatory pharmacology frameworks.

Structured rationale:
Biological psychiatry assumes pharmacological consistency across approved formulations. However:

Bioequivalence (BE) studies are conducted in healthy volunteers.

CNS drugs have nonlinear brain-response relationships.

Manufacturing variability, excipient effects, and batch drift may alter neurobehavioral outcomes.

Psychiatric symptoms often precede laboratory-detectable pharmacokinetic shifts.

This symposium examines whether current BE frameworks are sufficient for CNS drugs and proposes a biologically informed pharmacovigilance model.

S-20
When Bioequivalence is not Biologic Equivalence: Manufacturing Variability and Neurobehavioral Outcomes in Psychiatry

S-20-001
Topic: Pharmacokinetics, Bioequivalence & Brain Exposure

Speaker: Krishnapriya Murlimanohar, India

Abstract

Abstract Title:
Regulatory Overhaul — Psychiatry at the Table and the CNS Bioequivalence & Manufacturing Consortium

Abstract:
The recurring Indian pharmaceutical poisoning narrative — from 1986 DEG through 2022 Gambia cough-syrup deaths to the 2024–25 Coldrif cluster — is not a story of unlucky outliers but of architectural failure: psychiatry has never been at the regulatory table, and the slow-burn neurobehavioral signal that would have detected drift years earlier has been treated as anecdote. This concluding talk synthesises Parts 1 and 2 into a regulatory blueprint. Three-phase rollout is proposed. Phase 1 (12 months): a CNS Pharmacovigilance Registry (CPR) coupled to the existing PvPI infrastructure, mandating structured reporting of psychiatric symptom drift on generic switch, with anonymised case-cluster review by a multidisciplinary panel (psychiatry, clinical pharmacology, manufacturing science, regulatory affairs). Phase 2 (24 months): pilot of CNS-Specific BE 2.0 on three index classes (mood stabilisers, antidepressants, antipsychotics), with a sponsored steady-state crossover platform jointly operated by ICMR and CDSCO. Phase 3 (36 months): incorporation of CNS-specific BE into the National List of Essential Medicines (NLEM) procurement criteria for public-sector psychiatric drugs, with manufacturer GMP audits triggered automatically by registry signals. Structural critiques are addressed head-on: cost (offset by reduced relapse, suicide, and hospitalisation), industry pushback (mitigated by phased implementation and transition windows), and the epistemic risk that psychiatric reports may be over-attributed to formulation (managed by formal triangulation protocols and pre-registered confounder analysis). The deeper argument is cultural: psychiatry must stop accepting “same molecule = same experience” as a regulatory premise. Psychiatric symptoms are not noise; they are the highest-resolution early data the regulator possesses — the patient’s brain is the most sensitive available biosensor. The talk closes with a formal call-to-action: WFSBP-aligned CNS Bioequivalence & Manufacturing Consortium with a registry-first deliverable inside 12 months.

Key references: WHO Coldrif Investigation Report 2024; Kesselheim AS et al. JAMA 2008; CDSCO Pharmacovigilance Programme of India guidance; IOM “Crossing the Quality Chasm” 2001; Vrijens B et al. Br J Clin Pharmacol 2012.

S-20
When Bioequivalence is not Biologic Equivalence: Manufacturing Variability and Neurobehavioral Outcomes in Psychiatry

S-20-002
Topic: Manufacturing Drift, Excipients & System-Level Gaps

Speaker: Faisal Mohammad, India

Abstract

Abstract Title:
Why Healthy-Volunteer Bioequivalence Fails the CNS Patient — Toward CNS-Specific BE 2.0

Abstract:
Bioequivalence (BE) regulation in India and globally rests on a 1980s framework: single-dose, fasted, healthy adult, 80–125% AUC / Cmax confidence-interval criteria. This framework was engineered for non-CNS, non-NTI drugs taken transiently; it was never designed to protect the chronically dosed CNS patient. This talk dissects the PK–PD mismatch quantitatively. First, brain exposure is not equivalent to plasma exposure — blood–brain barrier transport, P-glycoprotein efflux polymorphism, lipophilicity-dependent partitioning, and active-metabolite ratios all decouple plasma AUC from CSF / tissue exposure. Second, formulation changes alter dissolution profiles in ways the 80–125% window can hide — polymorph differences in carbamazepine, valproate, and lamotrigine have produced documented neurobehavioral failures despite "passing" BE. Third, healthy-volunteer PK ignores patient-specific variables: gut transit altered by anticholinergic load, hepatic CYP induction / inhibition, altered protein binding in inflammatory states common to psychiatric populations. Fourth, steady-state behaviour is not modelled — yet psychiatric drugs are essentially always at steady state in real-world use; single-dose BE is therefore the wrong experiment. A CNS-Specific BE 2.0 framework is proposed: (i) narrower BE margins (90.00–111.11%, equivalent to EMA NTI criteria; FDA RSABE alternative discussed); (ii) mandatory steady-state crossover studies in stable patient cohorts; (iii) neurobehavioral endpoints (cognition, sleep architecture, mood-state stability) alongside PK; (iv) excipient transparency and dissolution-profile (f2) equivalence; (v) post-marketing surveillance integration with psychiatric pharmacovigilance signals from Part 1. The proposal is benchmarked against EMA's NTI guidance and FDA's RSABE / Q1-Q2 sameness requirements to demonstrate that CNS-Specific BE 2.0 is regulatorily feasible, not utopian.

Key references: Davit BM et al. AAPS J 2009; EMA Guideline on Investigation of Bioequivalence 2010; FDA Guidance on Bioequivalence Studies with PK Endpoints for Drugs Submitted Under an ANDA 2021; Yu LX et al. Pharm Res 2014; Kesselheim AS et al. JAMA 2008; Schall R & Luus HG. Eur J Clin Pharmacol 1993.

S-21

Horses Are Common, but Zebras Do Exist: Uncommon Medical Conditions and Triggers Masquerading as or Exacerbating Primary Psychiatric Disorders Across the Lifespan

Chair: Shobit Garg, India

Co-Authors:

Mohammad Zia Ul Haq Katshu

Topic:
10) Other, 1) Psychoses

Abstract

Objectives:
1. Identify clinical red flags prompting search for underlying medical/non-psychiatric etiologies or triggers.

2. Demonstrate targeted diagnostic strategies and multidisciplinary approaches.

3. Highlight management implications, including reversibility when rare causes are addressed.

4. Promote systematic history-taking (including supplement use) and low threshold for “zebra hunting.

Structured rationale:
The aphorism “When you hear hoofbeats, think horses, not zebras” promotes diagnostic efficiency, but rare medical conditions, metabolic vulnerabilities, vascular pathologies, genetic disorders, paraneoplastic processes, and even exogenous triggers (e.g., supplements) can present with or exacerbate predominant psychiatric symptoms. Delayed recognition leads to refractory illness and avoidable morbidity.

This expanded symposium now includes six diverse cases across adolescence to late life, arranged by patient age, to illustrate evolving presentations and the need for vigilant medical evaluation in atypical, refractory, or triggered psychiatric syndromes.

S-22

Transcending the Transdiagnostic: Precision-Based Scalable AI Technologies to Bridge Mental Healthcare to "The World's Unreached"

Chair: Priyaranjan Avinash, India

Co-Authors:

Anshu Prasad

Topic:
10) Other

Abstract

Objectives:
1. Review Current Evidence Base in AI-based multimodal digital phenotyping for mental health, including validated methodologies, clinical outcomes, implementation challenges.

2. Demonstrate AI-Driven transformation of multimodal clinical data into actionable insights that enhance diagnostic accuracy and therapeutic outcomes.

3. Address regulatory and ethical considerations and challenges in integrating Al tools into real-world healthcare workflows.

4. Demonstrate through a pilot research study outcome, real-world AI implementation in clinical practice.

Structured rationale:
Mental health disorders account for over 10% of the global disease burden. The consequences are devastating, with over 720,000 people die by suicide each year, with 77% of these deaths occurring in low- and middle-income countries. Lower and middle income countries (LMICs) allocate less than 1% of their health budgets to mental healthcare, compared to 5-10% in high-income nations. The traditional care delivery models cannot scale to meet these demands. Current evolution of transdiagnostic interventions has reorganized diagnostic systems to capture a more holistic etio-phenomenological landscape of mental illnesses. Multimodal digital phenotyping offers unprecedented opportunities to capture mental health complexity in real-world settings without requiring specialized infrastructure. AI-driven technologies can democratize mental healthcare by operating asynchronously, crossing linguistic and cultural barriers, and reaching underserved populations through ubiquitous mobile platforms at a fraction of traditional costs. This symposium explores how we can transcend these limitations, leverage multimodal data for precision insights, and deploy scalable AI solutions to bridge the mental healthcare gap for the world's unreached populations

S-23
Stress, screens, and synapses: psycho-biological transitions from Gen Y to Gen Z

Chair: Tamoghna Bandyopadhyay, India

Co-Authors:

Nishant Saha

Topic:
10) Other

Abstract

Objectives:
This symposium aims to examine autism through the evolving lens of biological psychiatry, tracing its journey from a classical neurodevelopmental disorder to a condition understood within the broader framework of neurodiversity. The session will integrate advances in genetics, synaptic biology, brain connectivity, immune mechanisms, and developmental neuroscience to highlight emerging biomarkers and translational implications. By bridging molecular research with lifespan perspectives and functional outcomes, the symposium seeks to contextualise autism within a changing world that increasingly values dimensional and individualised approaches. The objective is to foster biologically informed, ethically grounded, and clinically meaningful frameworks for understanding autism in contemporary psychiatric practice.

Structured rationale:
Autism has traditionally been conceptualised as a neurodevelopmental disorder rooted in early brain alterations. However, rapid advances in genetics, synaptic biology, connectomics, and neuroimmunology have transformed our understanding of its biological heterogeneity. Simultaneously, the emergence of the neurodiversity paradigm challenges deficit-based models and calls for dimensional, lifespan-oriented perspectives. In a changing world, biological psychiatry must integrate mechanistic discoveries with evolving social and ethical frameworks. This symposium is timely because it bridges molecular mechanisms, brain network science, and translational research with contemporary conceptual shifts, offering a comprehensive biological framework that informs diagnosis, intervention, and individualised care in autism.

S-23
Stress, screens, and synapses: psycho-biological transitions from Gen Y to Gen Z

S-23-003
From Psychological Trends to Neurobiological Signatures: Clinical and Translational Implications

Speaker: Tamoghna Bandyopadhyay, India

Abstract

Abstract Title:
Stress, screens, and synapses: psycho-biological transitions from Gen Y to Gen Z.

Abstract:
This symposium will examine the psycho-biological impact of rapid technological and societal changes across Generations Y and Z. Drawing on evidence from neuroscience, psychiatry, and behavioural sciences, speakers will explore how digital immersion, social media exposure, altered sleep patterns, chronic stress, and changing social environments influence brain development, cognition, emotional regulation, and mental health. The session will discuss emerging findings on neuroplasticity, stress biology, reward pathways, and digital behaviour, while highlighting both vulnerabilities and adaptive strengths of younger generations. The symposium aims to bridge biological mechanisms with contemporary psychosocial realities, informing future research and clinical practice.

S-24
Circadian Control of Glial Metabolism: When the Brain's Clock Runs Out of Energy

Chair: Bhaskar Mukherjee, India

Co-Authors:

Divya Sharma

Topic:
10) Other

Abstract

Objectives:
This symposium addresses a critical paradigm shift in neuroscience: the realization that the brain's metabolic landscape is not a static background process, but a tightly orchestrated temporal dance. By the conclusion of this symposium, participants will be able to:

1. Deconstruct Glial-Specific Molecular Clocks

Identify the autonomous circadian oscillators within astrocytes and microglia, distinguishing their regulatory mechanisms from the neuronal SCN "master clock."

Analyze how Bmal1 deletion specifically in the glial lineage alters global brain metabolism and synaptic plasticity.

2. Map Temporal Energy Flux

Evaluate the rhythmic nature of glycogenolysis and the transport of monocarboxylates (lactate/pyruvate) between glia and neurons.

Quantify the impact of circadian phase on mitochondrial respiration rates and the production of reactive oxygen species (ROS) within the neurovascular unit.

3. Correlate Circadian Misalignment with Neuro-Pathology

Synthesize the link between "metabolic exhaustion" in glia and the accumulation of protein aggregates (Amyloid-\beta, Tau).

Describe the mechanism by which desynchronized microglia shift from an anti-inflammatory "surveillance" state to a chronic pro-inflammatory "neurotoxic" state due to ATP depletion.

4. Design Chronotherapeutic Strategies

Appraise the efficacy of metabolic "zeitgebers," such as time-restricted feeding and NAD+ boosters, in resynchronizing glial clocks.

Formulate experimental models that integrate "time-of-day" as a primary variable in neuropharmacological studies to optimize drug delivery and efficacy.

Structured rationale:
The metabolic demands of the human brain are disproportionate to its size, consuming approximately 20% of the body's total energy. To manage this burden, the brain relies on the Astrocyte-Neuron Lactate Shuttle (ANLS) and glymphatic clearance, both of which exhibit profound circadian periodicity.

However, modern society exists in a state of "circadian tyranny." Chronic light pollution, shift work, and sleep fragmentation decouple the master SCN clock from peripheral glial oscillators. When the glial clock "runs out of energy," we see more than just fatigue; we see the bioenergetic roots of neurodegeneration.

This symposium provides a structured deep-dive into how the molecular machinery of the circadian clock—specifically the Bmal1 and Rev-Erb\alpha pathways—regulates glial mitochondrial flux and substrate availability. By framing glial dysfunction as a chronometabolic failure, we move beyond describing pathology toward predicting and preventing it. We will argue that the "energy crisis" seen in Alzheimer's and Parkinson's is, at its core, a failure of temporal metabolic partitioning.

S-25

Biomarker-informed relapse prevention plan for alcohol use disorders: can this be the new pathway to recovery?

Chair: Raghav Shah, India

Co-Authors:

Manaswi Gautam

Topic:
6) Addiction

Abstract

Objectives:
The objective of our symposium is to discuss a proposed relapse prevention plan for substance use disorders which includes relevant biomarkers. The biomarkers can be related to direct use of alcohol or may assess the end organ damage due to it. We want to present the review of currently available evidence and then give our critical analysis along with the desirable structure of such a relapse prevention plan.

Structured rationale:
We hypothesise that a relapse prevention plan which incorporates the results of relevant biomarkers will be more efficacious than the one without it as it aligns better with the recovery motivatoin arising out of the physical damage due to alcohol.

S-26
Gut to Brain: The Microbiome-Gut-Brain Axis in Substance Use Disorders

Chair: Sukanto Sarkar, India

Co-Authors:

Kamini Verma

Topic:
6) Addiction, 10) Other

Abstract

Objectives:
This symposium aims to:

1. Discuss evidence connecting gut microbiome with substance use disorders (SUDs).

2. Explain pathways of microbiome-gut-brain communication relevant to addiction (immune, neural, endocrine, and metabolic).

3. Review substance specific evidence : alcohol, opioids, and stimulants.

4. Discuss translational implications and emerging microbiome-targeted interventions for SUDs.

Structured rationale:
Many pathways link gut dysbiosis to addiction phenotypes. Microbial-derived metabolites (e.g., short-chain fatty acids and tryptophan derivatives) modulate the neuroimmune signaling, epigenetic regulation, and neurotransmitter systems involved in reward and stress. Substance exposure can disrupt intestinal tight junctions, increasing gut permeability and systemic inflammation, with downstream effects on neuroinflammation and reward circuitry. Neural and endocrine signaling (including vagal pathways and enteroendocrine hormones) further integrate peripheral microbial signals with central motivational and stress systems.

S-26
Gut to Brain: The Microbiome-Gut-Brain Axis in Substance Use Disorders

S-26-003
Opioid dependence and withdrawal modulation by microbiome interventions

Speaker: Apurba Mahato, India

Abstract

Abstract Title:
From Gut to Brain: Gut–Brain Axis in Substance Use Disorders

Abstract:
Background:

Substance use disorders are chronic relapsing conditions similar to chronic physical condiyions like diabetes and hypertension. High relapse rates are seen despite the available pharmacological and psychosocial treatments. They are traditionally conceptualized as disorders of brain reward circuitry but growing evidence suggests that addiction is a systemic condition involving bidirectional communication between the gut microbiome and the central nervous system via the microbiome-gut-brain axis. Alterations in gut microbial composition, intestinal barrier integrity, immune signaling, and microbial metabolite production are increasingly implicated in vulnerability to addiction, drug reward, withdrawal severity, stress responsivity, and relapse risk.

Rationale and Mechanisms:

Many pathways link gut dysbiosis to addiction phenotypes. Microbial-derived metabolites (e.g., short-chain fatty acids and tryptophan derivatives) modulate the neuroimmune signaling, epigenetic regulation, and neurotransmitter systems involved in reward and stress. Substance exposure can disrupt intestinal tight junctions, increasing gut permeability and systemic inflammation, with downstream effects on neuroinflammation and reward circuitry. Neural and endocrine signaling (including vagal pathways and enteroendocrine hormones) further integrate peripheral microbial signals with central motivational and stress systems .

Evidence Across Substances:

Preclinical studies demonstrate causal links between microbiome manipulation and addiction related behaviors. In opioid dependence models, fecal microbiota transplantation and antibiotic mediated microbiome depletion attenuate naloxone-precipitated withdrawal, supporting a role for gut microbes in physical dependence and withdrawal severity

In stimulant models, germ-free and antibiotic-treated animals show altered cocaine-induced psychomotor activation and conditioned reward, with restoration following microbial conventionalization and metabolite supplementation, highlighting microbiome regulation of reward learning and relapse related phenotypes

Alcohol use disorder is consistently associated with dysbiosis, reduced short-chain fatty acid–producing taxa, increased intestinal permeability, and systemic inflammation; early translational studies suggest that microbiome-targeted approaches, including fecal microbiota transplantation and dietary modulation, may influence craving and consumption, though robust clinical trials are needed

Methods and Translational Considerations:

Experimental models (germ-free, antibiotic depletion, fecal microbiota transplantation, and metabolite rescue) have been instrumental in establishing mechanistic links, but methodological heterogeneity and off-target effects of antibiotics limit direct translation. Human studies remain largely associative, underscoring the need for standardized protocols, longitudinal cohorts, and multi-omics approaches to establish causality, identify biomarkers, and stratify treatment responders .

Symposium Structure:

Four complementary presentations will integrate: (1) mechanistic frameworks of microbiome-gut-brain communication in addiction; (2) alcohol-related dysbiosis and gut barrier dysfunction; (3) opioid dependence and withdrawal modulation by microbiome interventions; and (4) stimulant-related reward and relapse phenotypes shaped by microbial communities. Cross-cutting modifiers (stress, social context, diet, and host factors) and ethical/regulatory considerations for microbiome interventions will be discussed.

Conclusion:

Positioning addiction within a microbiome-gut-brain framework advances understanding of systemic drivers of craving, withdrawal, and relapse. Microbiome-informed biomarkers and adjunctive interventions (probiotics, prebiotics, dietary strategies, targeted metabolites, and fecal microbiota transplantation) represent promising, yet early-stage, translational avenues. Rigorous, standardized clinical trials are essential t

S-27
The Inflammatory Bridge: Linking Psychiatric Disorders with Systemic Pathophysiology

Chair: Nachiketa Desai, India

Co-Authors:

Malay Dave

Topic:
10) Other

Abstract

Objectives:
* Identify the molecular pathways (e.g. IDO/Kynurenine) linking peripheral inflammation to central(CNS) issues.

* Evaluate how barrier permeability (in the gut and blood brain barrier(BBB)) allows systemic inflammatory substances to enter CNS.

* Use inflammatory biomarker categorization (e.g., CRP levels) to inform personalized treatments for patients who do not respond to standard options.

* Interplay of other Illnesses(Non-Communicable Diseases(NCDs)) with Psychiatric Disorders.

Structured rationale:
Psychiatric disorders often occur alongside non-communicable diseases (NCDs), yet medical models usually treat them as separate issues. This symposium highlights the need for a combined pathophysiological model. We point out the shared molecular factors, particularly inflammatory signaling and oxidative stress. This supports the idea of "immunopsychiatry" as a way to improve treatment for patients who do not respond well to standard therapies.

S-29
Neurobiology and clinical evidence of relaxation techniques in psychiatry

Chair: Geethu Parvathy, India

Co-Authors:

Sneha Mary Minz

Topic:
10) Other

Abstract

Objectives:
To review the neurobiological mechanisms of relaxation techniques in Psychiatry, like mindfulness, progressive muscle relaxation, meditation, non-sleep deep rest, etc.

To critically appraise the current evidence regarding the use of such techniques in various psychiatric disorders.

To identify the indications, contraindications, adverse effects, strengths and limitations of relaxation-based interventions in psychotherapeutic treatments.

To identify the gaps in current research and future directions of relaxation-based interventions.

To promote evidence-based practices while recommending relaxation-based interventions to patient populations.

Structured rationale:
There has been a significant surge in research related to the application of relaxation-based interventions in various psychiatric disorders and their neurobiology recently (1). A variety of relaxation techniques were introduced and studied, including progressive muscle relaxation, mindfulness-based interventions, breathing exercises and guided meditation(2-5).

These techniques were studied for their efficacy, safety as well as the underlying neurobiological mechanism of action, mostly in anxiety and depressive disorders, and gained widespread popularity, not only in psychiatric practice, but also among the popular culture as methods to reduce stress and anxiety and improve overall mental wellbeing, that anyone can use without causing any adverse effects compared to pharmacotherapy(6,7).

The relaxation techniques are centered on focusing one's attention: on muscle relaxation in progressive muscle relaxation, or on the present moment, non-judgmentally, in mindfulness, or on breath movements in breathing techniques or on the instructor's commands in guided meditation(8).

Evidence has shown that regular practice of relaxation techniques can bring neurobiological changes, including structural changes such as an increase in cortical thickness in the prefrontal and anterior cingulate cortices, a reduction in size and reactivity of amygdala, as well as changes in neurotransmitter activity such as increased GABAergic, serotonergic activities, increased BDNF levels and reduction in cortisol levels(9). The relaxation techniques also bring about other physiological changes, such as a reduction in heart rate variability and a reduction in vagal tone(10).

Despite extensive evidence of its benefits, the adverse effects and potential risks of relaxation techniques are often overlooked(11). An adverse event or harmful event is any incident that may cause harm to the patient, and an adverse reaction refers to unexpected harm resulting from a justifiable action(12).

Research shows that about 8.3% of people experience various adverse events while practicing relaxation techniques, but the reported prevalence highly varies among observational and experimental studies(13). Psychological distress, like anxiety, depressive symptoms or dissociative symptoms are more common than somatic distress. The majority of the events occurred during or immediately after practicing meditation. Cognitive effects like false memories and involuntary movements were also reported(14). Most studies reveal that the adverse events associated with relaxation techniques are highly underreported, and the general population need to be educated about the potential risks, and informed consent of the participant must be taken before suggesting such methods for therapeutic purposes(15).

References

1. https://doi.org/10.1177/1745691617709589
2. https://doi.org/10.1016/B0-12-343010-0/00182-3
3. https://doi.org/10.2147/PRBM.S437277
4. https://psycnet.apa.org/record/2005-02461-003
5. https://doi.org/10.1016/j.xcrm.2022.100895
6. https://doi.org/10.1080/j.1440-1614.2006.01794.x
7. https://doi.org/10.1146/annurev-psych-042716-051139
8. https://doi.org/10.1007/978-1-4939-1951-2_26
9. https://doi.org/10.3390/biomedicines12112613
10. https://doi.org/10.1016/j.neubiorev.2022.104711
11. https://doi.org/10.1016/j.copsyc.2025.102197
12. https://www.who.int/publications/i/item/WHO-IER-PSP-2010.2
13. https://doi.org/10.1111/acps.13225
14. https://doi.org/10.1007/s12671-022-01915-6
15. https://doi.org/10.1007/s12144-021-01503-2

S-29
Neurobiology and clinical evidence of relaxation techniques in psychiatry

S-29-001
Relaxation techniques: Neurobiological mechanisms

Speaker: Sneha Mary Minz, India

Abstract

Abstract Title:
Relaxation techniques : Neurobiological mechanisms

Abstract:
Relaxation-based techniques include mindfulness meditation, progressive muscle relaxation, deep-breathing and guided imagery. These techniques are used as non-pharmacological interventions in psychiatric care for various psychiatric disorders. Relaxation involves immobility of body, control over focus of attention, low muscle tone, and cultivation of specific frame of mind. Wherein, progressive muscle relaxation relieves muscle tension, guided imagery requires visualization of a pleasant, restful scene and engagement of all senses and mindfulness involves intentional awareness in focusing on the present like other meditation practices. Majority studies suggest that various relaxation techniques reduce stress and improve overall mental well-being without any adverse effects as compared to pharmacotherapy.

Relaxation techniques consistently shift autonomic balance toward parasympathetic dominance, indexed by increased heart rate variability and vagal tone, with concurrent reduction in sympathetic markers. Physiologic changes during relaxation are the opposite of those induced by adrenergic stress response. Neuroimaging studies have shown that regular practice of relaxation techniques can bring structural changes in brain such as increase in cortical thickness in prefrontal and anterior cingulate cortices, reduction in the size and reactivity of amygdala, suggestive of improved top-down regulation of emotion. Resting state fMRI studies indicates normalization of default mode network and salience network connectivity. At the molecular level, changes in neurotransmitter activity such as increased GABAergic and serotonergic transmission, increased BDNF levels and reduced pro-inflammatory cytokines suggestive of neuroplastic and anti-inflammatory pathways. Neuroendocrine studies suggest attenuation of hypothalamic-pituitary-adrenal axis hyperactivity, reflected with lower cortisol and CRH levels. Neurobiological findings provide a mechanistic framework for use of relaxation techniques as biological-grounded adjuncts in management of various disorders. Trauma survivors may have high baseline sympathetic arousal reflecting amygdala hypersensitivity, where relaxation can be wrongly interpreted as loss of control by salience network. Functional circuits involved in relaxation may cause harm if the dose or technique or patient phenotype is not matched.

S-29
Neurobiology and clinical evidence of relaxation techniques in psychiatry

S-29-002
Physiological and Cognitive Effects of Relaxation Techniques.

Speaker: Harshini Shalin Thakker, India

Abstract

Abstract Title:
The Physiological And Cognitive Effects Of Relaxation Techniques

Abstract:
Relaxation techniques are therapeutic strategies developed to help an individual reduce tension and anxiety. Relaxation techniques have not only been an integral part of the non-pharmacological interventions of psychiatric disorders but have also been used as complementary therapies to treat patients experiencing various types of distress. Stress is defined as a state of worry or mental tension caused by a difficult situation. Our body responds to stress by activation various physiological and hormonal mechanisms which work as our fight and flight response. And it is these physiological and hormonal changes which present as the physical symptoms of stress namely sweating, dizziness, palpitations, restlessness, breathing difficulty etc and cognitive symptoms of stress namely reduced concentration, impaired working memory, poor judgement etc. Relaxation techniques aim to counter these physiological and hormonal changes and restore a state of homeostasis. Thus, the impact of relaxation therapies can be observed in both physiological and cognitive domains. Physiologically it is observed that relaxation techniques activate the parasympathetic drive to counter the sympathetic fight and flight response. This is observed with the reduced pulse rate, lower BP readings, reduced respiratory rate, reduced urinary frequency, reduced gut issues, normalised sugar levels in the blood etc. On the other hand, the cognitive domain witnesses improved working memory, better attention span and concentration along with better executive functioning. Overall the impact of relaxation therapy is to restore physical and mental homeostasis in order to improve the functionality of an individual.

S-29
Neurobiology and clinical evidence of relaxation techniques in psychiatry

S-29-003
Clinical Evidence and Therapeutic Applications of Relaxation techniques in Psychiatry.

Speaker: Geethu Parvathy, India

Abstract

Abstract Title:
Clinical Evidence and Therapeutic Applications of Relaxation Techniques in Psychiatry.

Abstract:
A wide variety of relaxation techniques are used, both in psychopharmacological practice and by the general population under the popular belief that these techniques are healthy and free of any adverse events. Numerous studies have already established the neurobiological mechanisms and effects of such methods. There is strong evidence which suggests a higher prevalence of psychological and cognitive adverse events associated with relaxation techniques, which may suggest that they may not be recommended for all. In this symposium, we aim to explore the evidence-based application of relaxation techniques in Psychiatry, including their indications, adverse events, contraindications, along with future directions.

S-30

Precision Psychiatry - Moving from categorical diagnostic systems to mechanism based diagnostic frameworks

Chair: Navoneela Bardhan, India

Co-Authors:

Rahul Mathur

Topic:
10) Other

Abstract

Objectives:
1. To examine the coherence of current categorical diagnostic systems drawing on insights from large-scale consortia such as the Psychiatric Genomics Consortium and highlighting the limitations of symptom-based frameworks such as the Diagnostic and Statistical Manual of Mental Disorders.

2. To evaluate emerging biological stratification approaches including neuroimaging markers, pharmacogenomic predictors, immunological and metabolic findings.

3. To assess the potential of multi-modal integration models combining genetic markers, neuroimaging findings, inflammatory and metabolic findings for treatment response prediction.

4. To critically appraise the challenges of translating these into real world clinical care.

Structured rationale:
The current diagnostic systems such as the Diagnostic and Statistical Manual of Mental Disorders (DSM 5) are categorical diagnostic system based on symptom clusters. It is widely heterogenous in terms of symptoms, biology as well as treatment response. For eg, two patients meeting criteria for major depression may share very few overlapping symptoms while two patients with major depression with same symptoms may not respond similarly to the same medication. This leads us to explore mechanism based stratification integrating genomics, neuroimaging, inflammatory and metabolic markers for more biologically informed diagnoses and enhanced treatment accuracy

S-31
The synaptic silicon: engineering biologically grounded AI

Chair: Divya Sharma, India

Co-Authors:

Nachiketa Desai

Topic:
10) Other

Abstract

Objectives:
Explain the shift from backpropagation to local learning rules (STDP) and structural priors in neural modeling.

Assess the application of heavy-tailed weight distributions to capture the metabolic and computational cost-effectiveness of the biological brain.

Determine the role of digital phenotyping and intracranial signaling in facilitating real-time, circuit-level interventions in neuropsychiatric disorders.

Analyze the "translucency problem" and the diagnostic difficulties of interpreting neuro-mimetic AI based on functional imaging logic.

Critique the ethical foundations of "Artificial Agency" and the role of biological homeostatic constraints in AI alignment.

Structured rationale:
This symposium is founded on a convergence thesis: that the next leap in AI for Biological Psychiatry will not be achieved by more data, but by more biological truth. This is intended to integrate structural connectomics, real-time feedback loop, and precision pharmacology into a single framework for neuro-psychiatric restoration. We hope to examine the technical reality of biologically grounded AI—systems that inherit the structural constraints of the connectome, the temporal dynamics of synapses, and the ethical burdens of agency. For the clinician, this offers a forum to translate bench-side discoveries into algorithmic architectures that will provide a roadmap for a leap in efficiency.

S-32
Artificial intelligence in psychiatric disorders : current status of research, ethics and technological aspects

Chair: Ashish Pakhre, India

Co-Authors:

Manaswi Gautam

Topic:
10) Other

Abstract

Objectives:
To understand the conceptual basis of artificial intelligence in psychiatric disorders

To understand the ethical, technical and legal issues related to artificial intelligence data in mental health

To describe the current status of research using artificial intelligence in mental disorders detection and management

Structured rationale:
AI can recognise mental disorders at an early stage when interventions may be given, personalised treatments based on the patient's specific characteristics. It is important to enhance the understanding of interactions of biological, psychological and social domains. Pathophysiology of psychiatric disorders is often heterogeneous, and recognition of biomarkers will allow a proper, objective, and clear identification of mental illnesses. AI can generate better screening tools, prepare risk models to figure out the predisposition of developing mental illnesses.

S-33
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

Chair: Ravjot Kaur, India

Co-Authors:

Shivam Tyagi

Topic:
10) Other

Abstract

Objectives:
To review the evolving understanding and diagnostic framework of Attention-Deficit/Hyperactivity Disorder (ADHD) in the context of its underlying neurobiology, including genetic factors, neural circuits and inflammatory pathways, as well as emerging biological markers and their implication for current and future treatment strategies.

Secondary Objectives

1. To review the evolution of the conceptualization and diagnostic criteria of ADHD, examining how advances in genetics, neurodevelopmental research, and longitudinal studies have informed recent revisions, including ICD-11, and reinforced its recognition as a biologically based, lifespan neurodevelopmental disorder.

2. To examine historical and contemporary neurobiological models of ADHD, including the catecholamine hypothesis, dysfunctional fronto-striatal circuitry, delayed cortical maturation, alterations in large-scale brain networks such as impaired task-positive and default mode network interactions, and subcortical-cortical dysconnectivity.

3. To discuss emerging neurobiological insights, including glutamatergic dysregulation, gene-related synaptic mechanisms associated with increased neural noise, gut–brain axis interactions, and atypical sensory processing within a neurodevelopmental framework.

4. To critically appraise biological markers and indicators of illness severity and prognosis, including neuroimaging, electrophysiological measures, inflammatory and haematological indices and their potential clinical relevance and limitations.

5. To review established and evolving treatment approaches in ADHD, including stimulant and non-stimulant medications and, to consider the role of recent neurobiological understanding in development of future therapeutic strategies.

Structured rationale:
Attention-Deficit/Hyperactivity Disorder (ADHD) remains one of the most prevalent

neurodevelopmental conditions encountered in clinical practice, with well-documented impact across childhood, adolescence, and adulthood. While the diagnosis continues to be based on observable behavioral symptoms, the scientific understanding of ADHD has expanded considerably over the past two decades. Accumulating evidence from genetics, developmental neuroscience, neuroimaging, and psychopharmacology has shifted the field toward viewing ADHD as a disorder of brain development with identifiable biological underpinnings.

Twin and molecular genetic studies have demonstrated substantial heritability, while neuroimaging research has consistently implicated frontostriatal circuits and distributed brain networks involved in attention, executive control, and reward processing. More recent work suggests that cortical maturation may be delayed rather than fundamentally abnormal, and that alterations in large-scale network dynamics, including interactions between task-positive and default mode systems, contribute to symptom expression. In parallel, interest has expanded beyond monoaminergic models to include glutamatergic mechanisms, inflammatory pathways, gut–brain interactions, and sensory processing differences.

Diagnostic frameworks have evolved in response to these findings, particularly in recognizing ADHD as a lifespan condition and situating it more clearly within neurodevelopmental disorders. At the same time, research into potential biological markers of illness severity, prognosis, and treatment response has increased, though their clinical utility remains under careful scrutiny.

Given the rapid pace of developments across these domains, there is a need to consolidate current knowledge and examine its relevance for clinical practice. A focused review of emerging neurobiological insights alongside treatment updates may help clarify where the evidence stands and where further work is required, particularly in translating mechanistic findings into meaningful improvements in patient care.

S-33
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

S-33-001
Introduction of ADHD, diagnostic revision pertaining to biological basis

Speaker: Nishchita Raj, India

Abstract

Abstract Title:
Attention-Deficit/Hyperactivity Disorder (ADHD): Diagnostic Evolution and Biological Underpinnings

Abstract:
Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental condition characterised by persistent patterns of inattention and/or hyperactivity-impulsivity that interfere with functioning or development. Affecting approximately 5-7% of children and 2-5% of adults globally, ADHD significantly impairs academic performance, occupational functioning, social relationships, and psychological well-being. Beyond behavioural manifestations, ADHD represents a fundamental disruption in executive function, impulse regulation, and sustained attention mechanisms.

Diagnostic Revisions

The ADHD diagnosis has evolved substantially. The DSM-III (1980) initially recognised "Attention Deficit Disorder," emphasising inattention. The DSM-IV (1994) introduced three presentations (predominantly inattentive, predominantly hyperactive-impulsive, and combined), refining behavioral phenotyping. Critically, DSM-5 (2013) represented a paradigm shift by acknowledging ADHD's persistence into adulthood, acknowledging symptom manifestation differences across developmental stages, and allowing ADHD diagnosis alongside autism spectrum disorder, reflecting emerging neuroscience demonstrating shared neurobiological substrates.

Contemporary revisions increasingly incorporate neurobiological validation—diagnostic frameworks now acknowledge that behavioural criteria reflect underlying neurodevelopmental differences rather than moral failings or poor parenting.

Biological Basis

Neurochemical Dysregulation: ADHD involves dysfunction in catecholaminergic systems. Dopaminergic hypofunction in the prefrontal cortex and striatum impairs executive control, working memory, and reward processing. Noradrenergic deficits compromise sustained attention and arousal regulation. These neurochemical imbalances constitute the neurobiological foundation for behavioural symptoms.

Genetic Predisposition: ADHD demonstrates high heritability (70-80%). Candidate genes include dopamine receptor and transporter genes (DRD4, DAT1), catecholamine-related genes (COMT), and genes affecting neurotransmitter synthesis. Polygenic inheritance and gene-environment interactions explain heterogeneity.

Structural and Functional Brain Differences: Neuroimaging reveals reduced prefrontal cortex and striatal volumes, altered anterior cingulate cortex function, and compromised default mode network connectivity. Diffusion tensor imaging demonstrates white matter microstructural abnormalities, particularly in tracts connecting prefrontal and striatal regions critical for inhibitory control.

Developmental Perspectives: Emerging evidence suggests ADHD reflects abnormal neurodevelopmental trajectories, with delayed maturation of prefrontal networks and dysregulated synaptic pruning during adolescence.

Conclusion

ADHD's diagnostic evolution reflects growing recognition of its neurobiological essence. Understanding catecholaminergic dysfunction, genetic architecture, and structural-functional brain abnormalities validates ADHD as a legitimate neurobiological disorder, guiding evidence-based treatment approaches and reducing stigma.

S-33
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

S-33-002
Updates in neurobiology of ADHD

Speaker: Shivam Tyagi, India

Abstract

Abstract Title:
Updates in neurobiology of ADHD

Abstract:
Attention-deficit/hyperactivity disorder (ADHD) has traditionally been conceptualized as a neurodevelopmental disorder arising from catecholaminergic dysfunction, particularly reduced dopamine and norepinephrine signaling within the prefrontal cortex (PFC), accompanied by abnormalities in frontostriatal circuitry. While these models remain influential, recent advances in neuroimaging, genetics, and systems neuroscience have led to a more integrated understanding of ADHD pathophysiology.

Contemporary evidence suggests that ADHD reflects a delay in neurodevelopmental maturation rather than an entirely aberrant developmental trajectory. Longitudinal neuroimaging studies have demonstrated a delay of approximately three years in cortical maturation, with peak cortical thickness in the PFC occurring around 10.4 years in individuals with ADHD compared with 7.5 years in typically developing children. Functional network analyses further indicate impaired suppression of the default mode network (DMN) during goal-directed tasks, resulting in inadequate dissociation between the DMN and task-positive network (TPN). This network-level dysfunction may contribute to attentional lapses and cognitive variability.

Emerging research has expanded the neurobiological framework beyond traditional catecholamine models. Alterations in the gut–brain axis suggest that gut microbiota may influence ADHD symptoms through immune, metabolic, and neurotransmitter-mediated pathways. Neurochemical studies have also implicated glutamatergic dysregulation, particularly elevated glutamate concentrations within the medial prefrontal cortex, indicating excitatory–inhibitory imbalance. Additionally, atypical subcortical-cortical connectivity involving the caudate nucleus and nucleus accumbens has been associated with deficits in executive function, reward processing, and motivational control.

Recent genetic and electrophysiological findings propose increased neural noise as another potential mechanism, with genes such as HOMER1 contributing to synaptic signaling abnormalities and reduced signal fidelity. Furthermore, atypical sensory processing, characterized by heightened sensory sensitivity and avoidance behaviors, is increasingly recognized, affecting nearly half of individuals with ADHD. Collectively, these findings support a multidimensional model in which delayed brain maturation, network dysregulation, neurotransmitter imbalance, altered connectivity, gut–brain interactions, and sensory processing abnormalities interact to produce the heterogeneous clinical manifestations of ADHD.

S-33
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

S-33-003
Biological markers and indicators of illness severity and prognosis

Speaker: Ravjot Kaur, India

Abstract

Abstract Title:
Biological Markers and Indicators of Illness Severity and Prognosis in ADHD

Abstract:
ADHD is increasingly understood as a biologically based neurodevelopmental disorder involving genetic, neurodevelopmental, and inflammatory mechanisms. Recent research has focused on biological markers that may help understand illness severity, prognosis, and treatment response.

This presentation reviews emerging evidence on neuroimaging findings, electrophysiological measures, inflammatory pathways, and haematological markers in ADHD. Alterations in brain network connectivity, cortical maturation, and inflammatory indices have shown possible associations with symptom severity and functional impairment.

The presentation will also discuss the clinical relevance and limitations of current biomarkers and their potential role in improving prognostic assessment and individualized treatment approaches in ADHD.

S-33
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

S-33-004
Updates in treatment strategies in ADHD

Speaker: Neal Kasbe, India

Abstract

Abstract Title:
Emerging advances in the neurobiology and treatmnet of attention-deficit/hyperactivity disorder

Abstract:
Treatment Updates in ADHD

1. Historical Foundations

Pharmacological treatment of ADHD began in 1937 when Bradley observed behavioral improvement in children receiving amphetamine. This discovery laid the foundation for stimulant-based therapy. Methylphenidate, introduced in 1956, was initially understood simply as a dopamine transporter (DAT) and norepinephrine transporter (NET) blocker. Early treatment approaches focused primarily on symptom reduction without knowledge of receptor-level or circuit-based neurobiology. However, several limitations of traditional stimulants drove newer research: nearly 30% of patients show partial or no response, many discontinue due to tolerability issues, and conventional agents inadequately address emotional dysregulation, glutamatergic abnormalities, and default mode network (DMN) dysfunction.

2. Stimulants — Mechanistic Advances

Modern understanding of methylphenidate extends beyond DAT/NET blockade. It improves prefrontal cortex (PFC) signal-to-noise ratio through D1 and α2A receptor stimulation. D1 activation enhances task-positive network engagement and working memory, while α2A stimulation suppresses DMN hyperactivity, explaining cognitive improvement beyond simple arousal. Dose-dependent effects are now biologically understood: low doses optimize PFC function, whereas higher doses increasingly activate striatal motor circuits. Stereoselective pharmacology has also become relevant, with d-methylphenidate demonstrating substantially greater potency than l-isomers.

Amphetamines additionally reverse DAT and NET transporters, producing catecholamine efflux, while VMAT2 displacement releases vesicular dopamine stores. Lisdexamfetamine, a prodrug cleaved gradually by red blood cell enzymes, provides smoother pharmacokinetics and lower abuse potential due to slower peak concentrations.

Formulation engineering represents another major advance. OROS-methylphenidate mimics twice-daily dosing through osmotic release, while transdermal methylphenidate bypasses first-pass metabolism and reduces appetite suppression. Serdexmethylphenidate and ion-exchange amphetamine preparations further improve duration and plasma stability.

3. Non-Stimulants and Emerging Therapies

Atomoxetine selectively inhibits NET, increasing norepinephrine and indirectly dopamine within the PFC without affecting striatal reward pathways. Its delayed therapeutic onset is now attributed to α2A receptor upregulation and synaptic remodeling. It is particularly useful in ADHD with anxiety, tic disorders, or sensory dysregulation.

Viloxazine extended release, approved in 2021, combines norepinephrine reuptake inhibition with serotonergic effects through 5-HT2B antagonism and 5-HT2C agonism. These mechanisms may improve emotional dysregulation and provide faster onset than atomoxetine.

Guanfacine XR and clonidine XR act as α2A adrenergic agonists that directly reduce neural noise and strengthen PFC connectivity. Guanfacine's α2A selectivity causes less sedation and improves top-down cortical regulation. Combination therapy with stimulants provides complementary catecholaminergic and receptor-level effects.

Investigational therapies include centanafadine, a triple DAT-NET-SERT inhibitor targeting emotional dysregulation; TAAR-1 agonists, which modulate dopamine and glutamate signaling; and metadoxine extended release, which restores GABA-glutamate balance and may preferentially benefit inattentive ADHD.

4. Pharmacogenomics and Precision Psychiatry

Precision prescribing is increasingly relevant in ADHD management. CYP2D6 polymorphisms strongly influence atomoxetine and viloxazine metabolism. DAT1 and SERT polymorphisms may predict stimulant or serotonergic treatment response, while COMT Val158Met variants influence baseline PFC dopamine tone and medication tolerability. These advances are gradually shifting ADHD treatment from empirical trial-and-error toward genotype-informed individualized care.

S-35

Mind, self & brain: from citta to cortex in contemporary biological psychiatry: integrating predictive brain models, network neuroscience, and classical indian psychological constructs.

Chair: Sunil Mittal, India

Topic:
10) Other

Abstract

Objectives:
This symposium aims to advance conceptual and mechanistic understanding of self-related phenomena in biological psychiatry by integrating contemporary network neuroscience and predictive processing models with structured phenomenological distinctions derived from classical Indian psychological frameworks.

The primary objectives are:

1. To examine how distributed neural systems—particularly Default Mode Network, salience, and executive control networks—contribute to construction and stabilization of the sense of self.

2. To evaluate whether phenomenological constructs such as mental fluctuations (vṛtti), ego-construction (ahamkara), latent conditioning (saṃskāra), and discriminative processing (buddhi) can be operationalised within contemporary computational and network-based models of brain function.

3. To clarify how disruptions in self-processing manifest transdiagnostically across depression, obsessive–compulsive disorder, psychosis, and dissociative conditions.

4. To assess neurobiological correlates of altered self-experience in meditation, pharmacologically induced states, and psychiatric disorders, with emphasis on network flexibility and predictive inference.

5. To generate testable hypotheses and conceptual tools that refine dimensional and biomarker-oriented approaches within biological psychiatry.

The symposium does not seek to resolve metaphysical questions regarding the ontological status of consciousness. Rather, it aims to determine whether refined phenomenological taxonomies of self-experience can constrain and sharpen mechanistic models of psychiatric illness.

Structured rationale:
Biological psychiatry has achieved major advances in elucidating molecular mechanisms, synaptic plasticity, and large-scale neural networks underlying psychiatric disorders. However, core clinical phenomena central to psychiatric practice—such as disturbed agency, pathological rumination, ego-dissolution, depersonalisation, and identity instability—remain

insufficiently specified at the mechanistic level. While neural circuits can be identified, the structure of subjective experience they subserve is often described in broad or heterogeneous terms.

Contemporary neuroscience increasingly conceptualizes the self as an emergent construct arising from distributed network interactions and hierarchical predictive processing. The Default Mode Network has been implicated in autobiographical and narrative self-processing; salience and executive systems contribute to switching, regulation, and precision weighting. Yet translating these findings into clinically meaningful models of self-disturbance remains an ongoing challenge.

Classical Indian psychological traditions developed systematic phenomenological distinctions between sensory processing, discriminative cognition, ego-construction, and latent conditioning. When treated descriptively rather than metaphysically, these frameworks function as structured taxonomies of mental processes that may refine how psychiatry defines and studies self-related dysfunction.

This symposium is based on the premise that high-resolution phenomenological distinctions may constrain neurobiological interpretation and improve conceptual precision. By mapping classical constructs onto contemporary network and computational models, we aim to explore convergences, clarify divergences, and identify areas where empirical testing is possible.

The rationale is therefore not philosophical validation but methodological integration. If self-disturbance is a transdiagnostic dimension cutting across mood, obsessive–compulsive, psychotic, and dissociative disorders, then refining its conceptual architecture is directly relevant to biomarker development, dimensional classification, and treatment innovation.

This integrative approach aligns with current trends in network neuroscience, computational psychiatry, and dimensional models of psychopathology. It seeks to expand the explanatory framework of biological psychiatry while remaining firmly grounded in mechanistic, testable, and clinically applicable science.

S-36
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

Chair: Nishchita Raj, India

Co-Authors:

Shivam Tyagi

Topic:
2) Schizophrenia

Abstract

Objectives:
By the end of this symposium, participants would be able to:

1. Learn about the mechanistic role of Damage-Associated Molecular Patterns (DAMPs) in the pathophysiology of schizophrenia. Attendees will learn how, in response to cellular stress, neurons and glia release damage-associated molecular patterns (such as HMGB1, S100 proteins, heat shock proteins, and nucleic acids), activate pattern recognition receptors (such as TLRs and RAGEs), and start cascade neuroinflammatory responses that lead to psychotic symptoms, cognitive dysfunction, and progressive neurodegeneration in individuals with schizophrenia.

2. Examine the clinical data that connects DAMP dysregulation to the symptoms and consequences of schizophrenia. Participants will critically evaluate empirical studies showing increased levels of DAMP in the cerebrospinal fluid and bloodstream in individuals with schizophrenia, as well as correlations with the severity of symptoms (cognitive, negative, and positive symptoms), first-episode psychosis, treatment resistance, and the course of the illness. They will comprehend how different neuroinflammatory endophenotypes may be found in biologically homogeneous patient subgroups by DAMP profiling.

3. Determine DAMPs as novel biomarkers for the diagnosis and prognosis of schizophrenia: Participants will learn how to assess DAMP measurements as possible diagnostic biomarkers to distinguish schizophrenia from other mental illnesses, forecast treatment response trajectories, identify patients who may experience cognitive decline, and track the course of the disease over time. This entails being aware of assay procedures, standardization issues, and clinical implementation tactics.

4. Turn DAMP biology into innovative therapeutics – Participants will learn about new approaches to treating DAMP-mediated neuroinflammation, such as: (a) anti-inflammatory drugs that block DAMP receptor signaling, (b) microglial modulators that lessen DAMP-induced neuroinflammatory reactions, (c) neuroprotective measures that lower DAMP release and cellular stress, and (d) precision medicine techniques that combine DAMP profiling with customized medication.

5. Use an integrative model of schizophrenia that takes immune dysfunction into account. Participants will combine their understanding of how genetic susceptibility, environmental stressors (such as substance abuse, childhood trauma, and prenatal infections), and dysregulated neurotransmitter systems combine with DAMP-mediated neuroinflammation to create the complex clinical phenotype of schizophrenia. Clinical conceptualization and treatment planning will be improved by this biopsychosocial understanding.

6. Future studies can be designed to fill in the gaps in the literature on DAMP-schizophrenia. Participants will determine important research questions that need to be looked into, such as longitudinal DAMP trajectories from prodromal to chronic illness phases, mechanistic connections between particular DAMPs and symptom dimensions, the relative effectiveness of DAMP-targeted versus conventional treatments, and the best biomarker panels for clinical use.

Secondary learning objectives

1. Recognize how DAMP-triggered innate responses affect T cell-mediated adaptive immunity and neuroinflammatory amplification, as well as the interplay between innate immunity and adaptive immunity.

2. Recognize limitations in current DAMP research.

3. Discuss ethical and practical considerations in implementing biomarker-driven precision psychiatry in resource limited setting.

Structured rationale:
Background

1% of people worldwide suffer from schizophrenia, yet existing dopamine-based therapies fail to appropriately address cognitive symptoms and treatment resistance (20–30% of patients). Recent developments in immunopsychiatry highlight the crucial role of neuroinflammation, and damage-associated molecular patterns (DAMPs) may be able to connect a variety of risk factors to the pathophysiology of mental illness.

Scientific foundation

Stressed cells emit natural "danger signals" called DAMPs (HMGB1, S100 proteins, HSPs, and ATP), which activate pattern recognition receptors and cause neuroinflammation. Research indicates that patients with schizophrenia have higher DAMP levels, which are associated with more severe symptoms and cognitive impairment. DAMP release is triggered by environmental risk factors such as stress, trauma, and infections, indicating a common route.

Clinical significance

Critical unmet requirements are addressed by DAMP research:

Biomarkers: Metrics that are objective for monitoring treatment, diagnosis, and prognosis.

Finding neuroinflammatory subtypes that need focused treatments is part of personalized medicine.

Innovative Treatments: Anti-inflammatory strategies to supplement conventional antipsychotics.

Early Intervention: Using neuroprotective techniques to stop the course of disease.

Gaps in Knowledge

Insufficient clinical translation frameworks, a lack of standardized measuring techniques, a lack of knowledge on how genetic differences affect DAMP signaling, and unknown mechanisms connecting certain DAMPs to symptoms are some of the current difficulties.

Goals of the Symposium

Understand the concept of DAMPs and how it is crucial to the psychopathology of Schizophrenia

Compile the most recent DAMP studies on schizophrenia.

Establish consensus on the measurement and interpretation of biomarkers

Explore therapeutic targets in DAMP pathway.

Discuss standardization and methodological issues.

Encourage interdisciplinary cooperation

Expected Impact

By utilizing precision medicine techniques, this symposium has the potential to transform the diagnosis and treatment of schizophrenia by expediting the translation of DAMP research into clinical practice. DAMP-based approaches could help patients who are resistant to treatment and alleviate cognitive symptoms that are currently unresponsive to current medications by combining biological and psychological viewpoints.

S-36
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

S-36-001
Brief introduction to Damage-Associated Molecular Patterns (DAMPs) and Schizophrenia

Speaker: Neal Kasbe, India

Abstract

Abstract Title:
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

Abstract:
DAMPs in the Psychopathology of Schizophrenia

Schizophrenia is increasingly conceptualized as a disorder involving chronic low-grade sterile neuroinflammation. Unlike classical inflammation caused by infection, sterile inflammation is triggered by endogenous molecules known as Damage-Associated Molecular Patterns (DAMPs). These molecules, including HMGB1, S100B, extracellular ATP, heat shock proteins, and oxidized mitochondrial DNA (ox-mtDNA), are released from stressed, injured, or dying cells and function as “danger signals.” Genetic vulnerability, oxidative stress, excitotoxicity, mitochondrial dysfunction, and aberrant synaptic pruning in schizophrenia create persistent cellular stress, leading to chronic DAMP release.

HMGB1 (High Mobility Group Box 1) is one of the most important DAMPs implicated in schizophrenia. Released during oxidative stress and NMDA receptor hypofunction, it activates Toll-like receptors (TLR2/TLR4) and RAGE receptors, triggering NF-κB-mediated inflammatory cascades with release of IL-6, TNF-α, and IL-1β. Elevated HMGB1 levels have been demonstrated in cerebrospinal fluid and serum of schizophrenia patients. HMGB1 also disrupts glutamatergic neurotransmission, thereby worsening NMDA receptor hypofunction, a central neurobiological abnormality in schizophrenia.

S100B, predominantly released from astrocytes, is among the most replicated inflammatory biomarkers in schizophrenia. At high concentrations, S100B activates RAGE receptors, causing neuroinflammation and blood-brain barrier disruption. BBB disruption facilitates entry of peripheral cytokines and immune cells into the CNS, amplifying neuroinflammatory responses. Extracellular ATP, released during synaptic stress and neuronal injury, activates purinergic P2X/P2Y receptors and stimulates the NLRP3 inflammasome, leading to caspase-1 activation and IL-1β/IL-18 release. This contributes to microglial priming and dysregulated dopamine-adenosine interactions within the striatum.

Heat shock proteins such as HSP70 and HSP90 are released during psychosocial and drug-induced stress and act as TLR4 ligands, linking stress exposure to innate immune activation. Oxidized mitochondrial DNA, an emerging DAMP in psychiatry, activates the cGAS-STING pathway and induces type-I interferon responses, contributing to synaptic dysfunction and cognitive impairment. DAMP-induced cytokines also dysregulate the hypothalamic-pituitary-

adrenal axis, resulting in cortisol abnormalities and further cellular stress, thereby reinforcing the stress–psychosis interaction proposed in the two-hit model of schizophrenia.

DAMPs initiate a self-perpetuating microglial activation loop. Activation of pattern recognition receptors converts microglia into a pro-inflammatory M1 phenotype, leading to release of reactive oxygen species, cytokines, and proteases that further damage neurons and generate additional DAMPs. This mechanism may explain progressive gray matter loss and cognitive decline. DAMP-mediated inflammation also activates the kynurenine pathway through IDO1 stimulation, shifting tryptophan metabolism toward kynurenic acid, an NMDA receptor antagonist, thereby worsening glutamatergic hypofunction and contributing to cognitive deficits.

Clinically, DAMP-related pathways map onto symptom dimensions of schizophrenia. Positive symptoms are linked to dopamine excess and NMDA dysfunction, negative symptoms to prefrontal hypodopaminergia and inflammatory motivational deficits, and cognitive impairment to IL-1β-mediated disruption of long-term potentiation and aberrant synaptic pruning. Human studies support this model, demonstrating elevated HMGB1, S100B, IL-6, and microglial activation markers in schizophrenia, along with genetic associations involving TLR4, RAGE, and NLRP3 pathways.

S-36
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

S-36-002
Recent neurobiological aspects of psychoses

Speaker: Shivam Tyagi, India

Abstract

Abstract Title:
Recent neurobiological aspects of schizophrenia

Abstract:
Schizophrenia is a complex neuropsychiatric disorder whose neurobiological understanding has evolved from a predominantly dopaminergic model to a multidimensional framework involving neurodevelopmental, synaptic, immune, and neural circuit abnormalities. Early theories were centered on the dopamine hypothesis, which proposed that excessive dopaminergic activity caused positive symptoms such as hallucinations and delusions. Structural neuroimaging studies demonstrating ventricular enlargement and cortical tissue loss, together with the two-hit model of prenatal vulnerability followed by adolescent stressors or substance exposure, further supported a neurodevelopmental basis for the disorder.

Current evidence suggests a revised dopamine hypothesis characterized by hyperdopaminergia in subcortical regions and hypodopaminergia in the prefrontal cortex, accounting for positive, negative, and cognitive symptoms. Additionally, glutamatergic and GABAergic dysfunction, particularly N-methyl-D-aspartate (NMDA) receptor hypofunction and impaired inhibitory interneuron activity, results in excitation–inhibition imbalance and disrupted cortical connectivity. Recent genetic discoveries have highlighted the role of complement component 4A (C4A)-mediated excessive synaptic pruning during adolescence, contributing to gray matter loss and altered neural network development. Large genomic studies have also identified several rare risk genes involved in synaptic organization and neurodevelopment.

Growing evidence implicates neuroinflammation in schizophrenia pathogenesis. Microglial activation and sterile inflammatory responses triggered by damage-associated molecular patterns (DAMPs), including HMGB1 and S100B, may contribute to synaptic loss and progressive brain volume reduction. Abnormal thalamocortical connectivity further suggests impaired thalamic gating of cognitive and sensory information. Emerging concepts include glymphatic system dysfunction, potentially linking sleep disturbances with impaired clearance of inflammatory mediators, and maladaptive neuroplasticity characterized by reduced neurotrophic signaling and aberrant synaptic remodeling. Therapeutic advances targeting muscarinic M1/M4 receptors and trace amine-associated receptor 1 (TAAR1) offer non-dopaminergic approaches to symptom management. Novel biomarkers such as artificial intelligence-assisted retinal imaging and digital therapeutics, including the CONVOKE program, may facilitate precision psychiatry. Collectively, schizophrenia is increasingly conceptualized as a disorder of abnormal neurodevelopment, synaptic dysregulation, immune activation, and disrupted neural circuitry.

S-36
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

S-36-003
Implication of DAMPs in psychopathology of Schizophrenia

Speaker: Nishchita Raj, India

Abstract

Abstract Title:
Implication of Damage-Associated Molecular Patterns in the Psychopathology of Schizophrenia

Abstract:
Background & Rationale

Schizophrenia remains a devastating neuropsychiatric disorder with an incomplete understanding of its etiopathogenesis. Emerging evidence implicates neuroinflammation as a critical pathogenic mechanism, with damage-associated molecular patterns (DAMPs) emerging as pivotal drivers. DAMPs are endogenous molecules released from damaged or stressed cells that activate pattern recognition receptors (PRRs), particularly Toll-like receptors (TLRs) and the NLRP3 inflammasome, triggering sterile inflammation independent of pathogenic infection.

Proposed Mechanisms

DAMPs implicated in schizophrenia include high-mobility group box 1 (HMGB1), heat shock proteins (HSP70 and HSP90), and extracellular ATP. These molecules activate microglial PRRs, inducing excessive production of pro-inflammatory cytokines (IL-1β, TNF-α, and IL-6) and chemokines. This neuroinflammatory cascade promotes (1) synaptic pruning via complement deposition and microglial engulfment of dendritic spines, disrupting glutamatergic and GABAergic neurotransmission; (2) blood-brain barrier compromise, exacerbating neuroinflammation; (3) oxidative stress and mitochondrial dysfunction in neurons, impairing energy metabolism critical for neurotransmission; and (4) impaired neuroplasticity and reduced neurotrophic factor (BDNF) expression, compromising cognitive function and emotional regulation. Peripheral DAMPs may breach a compromised BBB or activate systemic innate immunity, amplifying central nervous system inflammation through both direct and indirect mechanisms.

Post-mortem and neuroimaging studies demonstrate microglial activation and elevated pro-inflammatory markers in schizophrenia brains. Elevated circulating HMGB1 and HSP70 have been documented in schizophrenia patients, correlating with symptom severity. Animal models using DAMP-derived ligands (lipopolysaccharide and poly I:C) recapitulate schizophrenia-like behaviors, including cognitive impairment and social withdrawal, which are reversible by anti-inflammatory interventions.

Therapeutic Implications & Future Directions

Understanding DAMP-mediated neuroinflammation in schizophrenia opens unprecedented therapeutic avenues: (1) PRR antagonists and inflammasome inhibitors to reduce microglial activation; (2) anti-HMGB1 monoclonal antibodies to neutralise circulating DAMPs; (3) biomarker-driven patient stratification enabling precision medicine approaches; (4) combination pharmacotherapy integrating anti-inflammatory agents with antipsychotics to enhance treatment efficacy and reduce treatment resistance.

Future research should employ multimodal neuroimaging, cerebrospinal fluid biomarker analysis, and mechanistic cell biology studies to delineate DAMP-specific contributions to distinct symptom domains and identify DAMP signatures predicting treatment response.

Conclusion

DAMPs represent a mechanistic bridge between immune dysfunction and schizophrenia psychopathology, offering novel therapeutic targets for this chronic, debilitating disorder.

S-36
Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia: From Neuroinflammation to Novel Therapeutic Targets

S-36-004
Recent updates and treatment related aspects of DAMPs in Schizophrenia

Speaker: Ravjot Kaur, India

Abstract

Abstract Title:
Recent Updates and Treatment-Related Aspects of Damage-Associated Molecular Patterns (DAMPs) in Schizophrenia

Abstract:
Schizophrenia is increasingly being understood as a disorder involving immune dysregulation and neuroinflammation in addition to neurotransmitter abnormalities. Damage-Associated Molecular Patterns (DAMPs) such as HMGB1, S100 proteins, ATP, and heat shock proteins are released during cellular stress and may contribute to ongoing inflammatory changes in the brain. Recent studies have shown elevated DAMP levels in patients with schizophrenia, particularly in relation to symptom severity, cognitive impairment, and treatment resistance.

This presentation reviews current developments in DAMP-related research and their possible therapeutic relevance. Anti-inflammatory and neuroprotective approaches including minocycline, N-acetylcysteine, omega-3 fatty acids, and agents targeting microglial activation are being studied as adjunctive treatments alongside antipsychotics. Interest is also growing in therapies aimed at pathways involving HMGB1, Toll-like receptors, and RAGE signaling. The role of DAMPs as potential biomarkers for identifying inflammatory subtypes of schizophrenia and guiding individualized treatment approaches will also be discussed. Although further research is needed to improve standardization and clinical applicability, DAMP-focused interventions may offer new directions for addressing cognitive symptoms and treatment resistance in schizophrenia.

S-37
Optimizing rTMS Outcomes in Real-World Psychiatry: From Fundamentals to Personalization

Chair: Sourav Das, India

Co-Authors:

Nishant Goyal

Topic:
7) Mood disorders, 10) Other

Abstract

Objectives:
Repetitive Transcranial Magnetic Stimulation (rTMS) has emerged as an evidence-based neuromodulation technique for several psychiatric disorders. However, translating controlled trial efficacy into real-world clinical effectiveness remains a significant challenge, particularly in resource-constrained settings. This symposium aims to bridge this gap by integrating foundational knowledge with pragmatic clinical strategies to optimize treatment outcomes. The session will focus on protocol selection, dosing strategies, real-world adaptations, and emerging directions such as personalized and biomarker-guided approaches. Emphasis will be placed on scalability and applicability in routine psychiatric practice.

Structured rationale:
By integrating evidence-based fundamentals with real-world clinical insights and future directions, this symposium aims to enhance the effectiveness, accessibility, and scalability of rTMS in contemporary psychiatric practice.

We believe this symposium aligns closely with the conference theme, “Biological Psychiatry in a Changing World – Exploring New Frontiers,” and will be of significant interest to clinicians and researchers alike.

S-37
Optimizing rTMS Outcomes in Real-World Psychiatry: From Fundamentals to Personalization

S-37-002
Improving Clinical Outcomes in rTMS: Practical Strategies

Speaker: Sourav Das, India

Abstract

Abstract Title:
Improving Clinical Outcomes in rTMS: Practical strategies.

Abstract:
Although rTMS has established efficacy in depression and obsessive-compulsive disorder (OCD), many patients show incomplete or poor response. Inadequate matching between symptom domains, dysfunctional neural circuitry, and stimulation protocols may contribute significantly to treatment failure.

To discuss a symptom-domain-based approach for individualized rTMS protocol selection in depression and OCD using clinically relevant neurocircuitry models.

This presentation integrates current evidence with practical clinical experience to examine the relationship between psychiatric symptom clusters, neural networks, and rTMS targets. Practical aspects such as coil selection, targeting methods, motor threshold determination, and protocol reproducibility were also reviewed.

Distinct symptom clusters were associated with specific dysfunctional brain circuits. Depressed mood and anhedonia were linked to hypoactivity in the left dorsolateral prefrontal cortex (DLPFC), supporting high-frequency left DLPFC stimulation. Anxiety and rumination were associated with hyperactivity in right DLPFC circuits, favoring inhibitory low-frequency right-sided protocols. Psychomotor retardation and fatigue involved supplementary motor area and anterior cingulate dysfunction, while cognitive dysfunction benefited from bilateral DLPFC approaches. In OCD, obsessive intrusions were linked to orbitofrontal-striatal pathways, compulsive urges to pre-SMA dysfunction, and severe treatment-resistant OCD to broader cortico-striato-thalamo-cortical hyperactivity, supporting deep TMS approaches. The importance of accurate targeting, individualized dosing, and protocol fidelity was emphasized.

Many apparent rTMS failures may reflect inadequate personalization rather than true treatment resistance. Symptom-guided, neurocircuitry-informed protocol selection may improve clinical outcomes in depression and OCD.

S-37
Optimizing rTMS Outcomes in Real-World Psychiatry: From Fundamentals to Personalization

S-37-004
Future Directions in rTMS: Toward Personalization

Speaker: Nishant Goyal, India

Abstract

Abstract Title:
Future Directions in rTMS: Toward Personalization

Abstract:
Repetitive transcranial magnetic stimulation (rTMS) has emerged as an important non-invasive neuromodulation technique, with established efficacy in treatment-resistant depression and growing applications across psychiatry and neurology. The future of rTMS lies in moving beyond standardised protocols toward individualised, mechanism-informed interventions. Personalisation may involve tailoring stimulation parameters, cortical targets, session frequency, and treatment duration according to each patient's clinical profile, neurophysiology, symptom dimensions, and treatment response. Advances in neuroimaging, electroencephalography, neuronavigation, and computational modelling are increasingly enabling precise identification of dysfunctional brain networks and optimisation of stimulation sites. Biomarkers such as resting-state functional connectivity, cortical excitability, and electrophysiological signatures may help predict responders, guide dose adjustments, and monitor therapeutic progress. Emerging approaches, including accelerated rTMS, theta burst stimulation, closed-loop stimulation, and multimodal combinations with psychotherapy or pharmacotherapy, further expand the possibilities for individualized care. However, challenges remain, including variability in response, limited access to advanced targeting methods, cost considerations, and the need for robust clinical validation. Future research must focus on large-scale, biomarker-driven trials and real-world implementation frameworks. A personalized rTMS paradigm holds promise not only for improving efficacy and tolerability but also for transforming neuromodulation into a precision psychiatry tool aligned with the needs of individual patients.

S-38
Oligodendroglia as treatment target in schizophrenia

Chair: Peter Falkai, Germany

Co-Authors:

Johann Steiner

Topic:
2) Schizophrenia, 1) Psychoses

Abstract

Objectives:
Despite the critical role of residual symptoms in schizophrenia, only limited treatment options are available. Objectives: to disentangle oligodendrocyte and glia-related mechanisms (e.g. disturbed myelin plasticity and astroglial alterations) in schizophrenia and their relationship to cognitive deficits from a neuroimaging and post-mortem view. To identify the role of oligodendrocyte precursor cells in brain regions, especially the hippocampus, from schizophrenia patients. The modulatory role of cell type-specific schizophrenia polygenic risk scores (PRS) on hippocampal volume changes after aerobic exercise is addressed. Brain imaging and iPSC findings suggest that restoration of neural networks requires modulation of oligodendrocytes, especially a stimulation of oligodendrocyte precursor cell differentiation underlying remyelination in the brain. The overall aim of this symposium is to provide the background for targeted treatment of cognitive deficits in schizophrenia by providing a mechanistically informed treatment with drugs that restore myelination and OL maturation. Together these mechanisms are referred to as myelin plasticity, and comprehension of disturbed myelin plasticity as the substantial basis for dyscognition in schizophrenia will be discussed. Providing a causal treatment of cognitive deficits will be a major turning point in the treatment of not only schizophrenia but also bipolar disorder and major depression, where cognitive disturbances play a similar role. Clinical, neuroimaging, post-mortem and genetic data will be presented to characterize glial deficits and to describe novel treatment targets in schizophrenia. Overall, the symposium highlights OL and myelination deficits and their potential to be novel treatment targets of cognitive deficits in schizophrenia.

Structured rationale:
Johanna Seitz-Holland will present findings from two large multi-site studies integrating clinical, cognitive, neuroimaging, and blood based data. Her group applied advanced diffusion weighted magnetic resonance imaging to examine how cognitive deficits, white matter microstructure, and general health are related in individuals with schizophrenia and other psychotic disorders. The first study identified cognitive impairments across eight cognitive domains in 900 individuals with schizophrenia and demonstrated that these deficits were associated with alterations in white matter microstructure. The second study showed that a subset of individuals in the early course of psychosis already exhibit cognitive deficits and brain patterns resembling those observed in Alzheimer's disease. Together, these studies underscore the central role of cognitive impairment across the psychosis spectrum and highlight white matter

microstructure as a key factor linking psychosis to cognitive dysfunction. Johann Steiner summarizes converging glial cell findings on reduced oligodendrocyte (OL) numbers and BCAS1 immunoreactive regenerative OL populations in hippocampal subfields to explain myelin dysfunction in schizophrenia. OL loss arises via two complementary routes, one is the loss of mature oligodendrocytes and the other one failed maturation of OL precursor cells (OPCs) together with ultrastructural myelin abnormalities and downregulated myelin genes as primary glial pathologies rather than classical neurodegeneration. These reversible, targetable glial processes emphasize remyelination and novel glia-protective strategies. Oligodendrocyte-related risk variants are not among the top associations in genome-wide association studies (GWASs). However, in an enrichment analysis study of cell type-specific gene expression, oligodendrocytes and oligodendrocyte precursor cells (OPCs) showed enrichment in genes associated with schizophrenia. Sergi Papiol evaluated the modulatory role of cell type-specific schizophrenia polygenic risk scores (PRS) on volume changes after three months aerobic exercise in hippocampal subfields. The analyses showed that the PRS associated with OPCs significantly influenced the volume changes in the CA4/DG subfield in schizophrenia patients. A higher OPC-associated genetic risk burden was associated with a less pronounced volume increase during the exercise intervention. Peter Falkai reports on novel clinical treatment approaches targeting myelination and oligodendroglia differentiation in schizophrenia, including aerobic exercise, clemastine and quetiapine. Using an imaging transcriptomics approach, he shows that normalization of functional dysconnectivity after exercise is associated with increased expression of genes related to OLs. Quetiapine is known to enhance myelination in the frontal cortex and other brain areas, which translates into improved cognition. Compared with schizophrenia patients not treated with the drug, quetiapine-treated patients showed significant improvement in working memory and verbal learning and memory cognitive domains.

S-38
Oligodendroglia as treatment target in schizophrenia

S-38-003
Oligodendrocytes as part of schizophrenia risk

Speaker: Daniel Martins-De-Souza, Brazil

Abstract

Abstract Title:
Molecular targets associated to glial dysfunction in schizophrenia

Abstract:
Schizophrenia is conceptualized as a disorder of brain connectivity, in which oligodendrocytes emerge as central players. Here, I present an integrated view of biochemical mechanisms underlying glial dysfunction based on proteomics analyses of postmortem brain tissue, stem cell–derived models, and pharmacological approaches, including NMDA receptor antagonism (MK-801) and cannabinoid modulation.

Our findings converge on three interconnected processes. First, glutamatergic imbalance, particularly NMDA receptor hypofunction, disrupts oligodendrocyte maturation and protein homeostasis. Second, metabolic dysfunction, characterized by mitochondrial impairment, oxidative stress, and altered lipid metabolism, compromises the high energetic demands required for myelination. Third, dysregulation of RNA processing further impacts the expression of key myelin-related proteins.

Together, these alterations define a state of oligodendrocyte vulnerability that leads to impaired myelin maintenance and, consequently, large-scale connectivity deficits in the brain. Importantly, pharmacological interventions targeting these pathways modulate overlapping biochemical networks, suggesting that part of their effects may involve restoration of oligodendrocyte function.

This integrative framework supports a shift toward glia-centered models of psychiatric disorders and highlights oligodendrocytes as promising targets for future therapeutic strategies.

S-38
Oligodendroglia as treatment target in schizophrenia

S-38-004
Myelin-related drugs to treat cognitive deficits in schizophrenia

Speaker: Peter Falkai, Germany

Abstract

Abstract Title:
Myelin-related drugs to treat cognitive deficits in schizophrenia

Abstract:
Schizophrenia (SZ) is a severe mental disorder characterized by positive and negative symptoms, as well as cognitive dysfunction. Although positive symptoms can be treated well with antipsychotics, current pharmacological treatments have no significant beneficial effect on cognitive deficits. They occur as early as the prodromal phase of the illness and significantly determine social function. Consequently, only about 15% of patients with SZ reach recovery, i.e., a state where they have normal everyday functioning despite having residual symptoms such as cognitive dysfunction. Understanding the pathophysiology of cognitive dysfunction in SZ will lead to mechanistically informed treatments that solve the outlined unmet need. Using design-based stereology, our group identified a decreased number of oligodendrocytes (OLs) in the CA4 hippocampal subregion as the basis for disturbed myelination and impaired cognition, while others showed such a decrease in the prefrontal cortex. Animal studies demonstrated that clemastine enhances remyelination by increasing the differentiation of OL precursor cells (OPCs) and that aerobic exercise increases the rate of remyelination and proliferation of OPCs; this clinically meaningful effect of aerobic exercise is stronger in combination with clemastine. An imaging transcriptomics approach showed that in patients with SZ, normalization of functional dysconnectivity after three months aerobic exercise training is associated with increased expression of genes related to OLs and OPCs. Furthermore, aerobic exercise improved everyday functioning, measured by the Global Assessment of Functioning (GAF) scale, and dyscognition in SZ and increases hippocampal volume, especially in the hippocampal CA4 subregion. This regional volume change correlates negatively with global and cell-specific polygenic risk scores (PRSs), indicating that OPCs are involved in the genetic risk mechanisms and disturbed plasticity underlying SZ. In an ongoing, randomized, rater-blind, controlled prospective clinical trial in patients with SZ, effects of 90 days' aerobic exercise training plus clemastine with aerobic exercise training is compared to placebo and aerobic exercise. Primary outcomes are working memory and everyday functioning. As secondary outcomes, global cognition, other cognitive domains, and disease symptoms, resting-state magnetic resonance imaging, and diffusion tensor imaging to test connectivity and myelin integrity are assessed. In the multicenter PsyCourse study in 212 patients with SZ we found that quetiapine-treated patients showed significant improvement in prefrontal (working memory) and temporal (verbal learning and memory) cognitive domains compared with SZ patients not treated with the drug. These clinical trials might lead to a new, mechanistically informed pharmacological treatment for cognitive dysfunction in SZ and generate biomarkers to identify the patients who benefit most from this strategy. Our studies support an oligodendrocyte pathology in schizophrenia and suggest that reducing functional dysconnectivity reflect a potential therapeutic target to improve cognitive impairments.

S-39

HPA Axis Dysregulation in Psychiatric Disorders and the Neuroendocrine Modulatory Effects of Music.

Chair: Abhimanyu Sinha, India

Co-Authors:

Ashish Pakhre

Topic:
10) Other

Abstract

Objectives:
1.To review current evidence on HPA-axis dysregulation across major psychiatric disorders.

2 To examine molecular and neuroendocrine mechanisms linking chronic stress exposure to psychopathology.

3 To evaluate experimental and clinical evidence supporting music-induced modulation of HPA-axis activity.

4.To explore the translational implications of music-based interventions as adjunctive strategies targeting stress neurobiology in psychiatric care.

Structured rationale:
Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis is one of the most consistently replicated biological abnormalities in stress-related psychiatric disorders. Altered cortisol secretion, impaired glucocorticoid receptor sensitivity, disrupted circadian rhythmicity, and persistent stress activation contribute to structural and functional changes in the hippocampus, amygdala, and prefrontal cortex, reinforcing maladaptive emotional regulation. Major depressive disorder, anxiety disorders, post-traumatic stress disorder (PTSD), bipolar disorder, and schizophrenia have all been associated with disorder-specific patterns of HPA dysfunction[1].

Contemporary biological psychiatry increasingly conceptualizes psychiatric illness through the framework of stress neurobiology and impaired glucocorticoid feedback inhibition.

Emerging experimental and clinical evidence suggests that music a biologically salient and evolutionarily conserved stimulus can influence neuroendocrine stress regulation. Animal studies demonstrate that exposure to music attenuates stress-induced increases in corticosterone, suggesting a modulatory effect on HPA-axis activation[2].

Human investigations report reductions in salivary cortisol and improved autonomic balance following structured music listening, particularly when rhythmically organized or emotionally

meaningful[3]. Neurobiological models propose that music engages limbic–hypothalamic pathways central to stress processing, including the amygdala, hippocampus, and medial prefrontal cortex, providing mechanistic plausibility for endocrine modulation[3].

Despite increasing interest in non-pharmacological interventions, integration of music-based modulation within core stress neurobiology frameworks remains underexplored in biological psychiatry. Understanding whether music can meaningfully regulate HPA-axis activity has implications not only for symptom reduction but also for targeting underlying biological dysregulation across psychiatric disorders.

S-40
Advances in Neurosurgical Interventions for Treatment-Resistant OCD

Chair: Shyam Sundar Arumugham, India

Co-Authors:

Y C Janardhan Reddy

Topic:
10) Other

Abstract

Objectives:
Around 20% of patients with obsessive-compulsive disorder (OCD) do not respond to non-invasive treatments. Neurosurgical interventions, including deep brain stimulation (DBS) and ablative procedures, have emerged as effective treatment options for carefully selected patients with severe and treatment-refractory illness. Advances in neuroimaging, systems neuroscience, and computational approaches are transforming our understanding of the neural circuits underlying OCD and informing more precise neuromodulation strategies.

This symposium aims to provide an overview of contemporary developments in neurosurgical treatments for OCD, integrating perspectives from clinical psychiatry, neurosurgery, neuroscience, and patient experience.

The specific objectives of the symposium are:

1. To review clinical outcomes and current evidence for neurosurgical interventions in treatment-resistant OCD, including DBS and ablative procedures, and discuss their role within modern treatment algorithms.

2. To discuss practical challenges in implementing neurosurgical treatments, including patient selection, multidisciplinary evaluation, and ethical/legal considerations.

3. To examine the neural circuitry underlying OCD, focusing on the different cortico-striato-thalamo-cortical networks, their functions, and their relevance for neurosurgical targeting.

4. To discuss the structural connectivity of DBS targets, including the diffusion MRI tractography findings and network-level analyses of different DBS targets that aim to refine targeting and improve treatment outcomes.

5. To discuss predictors of treatment response, including clinical, electrophysiological, and neuroimaging markers that may enable more personalized neuromodulation strategies.

6. To incorporate a lived experience perspective, providing insight into patient expectations, recovery trajectories, and the psychosocial impact of neurosurgical treatments.

By integrating clinical, neurobiological, and experiential perspectives, this symposium aims to advance understanding of circuit-based interventions and identify future directions for improving outcomes for severe, treatment-resistant OCD.

Structured rationale:
Over the past two decades, neurosurgical treatments for OCD have evolved from permanent lesion-based approaches to neuromodulatory procedures such as deep brain stimulation (DBS). These procedures target different nodes within cortico-striato-thalamo-cortical (CSTC) circuits. Gamma ventral capsulotomy is the commonly employed lesion-based procedure. Common DBS targets include ventral capsule/ventral striatum and anteromedial subthalamic nucleus. Long-term follow-up studies have demonstrated that around 50 to 70% of patients with severe, chronic and treatment-refractory OCD show meaningful improvement. Nevertheless, considerable variability in treatment response remains. Further, different surgical techniques and neurosurgical targets have shown similar response rates. This symposium combines perspectives from clinical, ethical, neuroimaging, and neuroscience perspectives to discuss the advances in neurosurgical treatments for OCD.

Shyam Sundar Arumugham will discuss the outcomes of gamma ventral capsulotomy and anteromedial subthalamic nucleus DBS from India. He will also discuss the practical challenges involved in neurosurgical procedures, including legal hurdles, stigma, and multidisciplinary care. A lived experience perspective will be provided to highlight the expectations and psychosocial impact of surgery on patients and caregivers. The clinical, neuropsychological, and electroencephalographic predictors of treatment response will be discussed.

Himanshu Tyagi will review the biological rationale for DBS in OCD. Key clinical trial evidence and long-term follow-up studies from the UK and other multi-centre studies will be discussed,. Particular attention will be given to the heterogeneity of response, predictors of outcome, and the integration of DBS with behavioural therapies. The presentation will also examine emerging directions in the field, including connectomic targeting, adaptive stimulation approaches, and the role of multidisciplinary assessment in patient selection. Finally, the talk will consider the broader implications of DBS for understanding OCD pathophysiology.

Pablo Andrade will discuss the neural circuitry of OCD, focusing on the CSTC pathways and their functional relevance. The talk will discuss the findings from diffusion imaging-based patient-specific connectomic data, which highlights the shared and differential connectivity of different DBS targets for OCD, including VC/VS, amSTN, and medial forebrain bundle. This has implications for the emerging field of connectomic targeting.

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S-40
Advances in Neurosurgical Interventions for Treatment-Resistant OCD

S-40-001
Clinical outcome, practical challenges, and predictors of response to gamma ventral capsulotomy and subthalamic nucleus deep brain stimulation for OCD

Speaker: Shyam Sundar Arumugham, India

Abstract

Abstract Title:
Clinical outcomes and predictors of response to gamma knife capsulotomy and subthalamic nucleus DBS for OCD

Abstract:
This talk will discuss the outcomes of gamma ventral capsulotomy and anteromedial subthalamic nucleus DBS from India. He will also discuss the practical challenges involved in neurosurgical procedures, including legal hurdles, stigma, and the need for multidisciplinary care. A lived experience perspective will be provided to highlight the expectations and psychosocial impact of surgery on patients and caregivers. The clinical, neuropsychological, and electroencephalographic predictors of treatment response will be discussed.

S-41

Biological Psychiatry at the Crossroads: Multimodal Biomarkers and Convergence Hubs in the Architecture of Mental Disorders

Chair: Rezaul Hamid, India

Co-Authors:

Muhamad Aly Rifai

Topic:
10) Other

Abstract

Objectives:
This symposium examines a fundamental challenge being faced by contemporary biological psychiatry: the growing accumulation of biological signals without an integrative framework capable of organizing them into a coherent model of psychiatric pathophysiology.

The objectives of this session are:

1. To critically examine the current fragmentation of biomarker research in psychiatry, where neuroimaging, electrophysiological, biochemical, and genetic findings are often studied in isolation without a unifying biological framework.

2. To review major classes of biological markers currently investigated in psychiatry, including functional neuroimaging (fMRI), functional near-infrared spectroscopy (fNIRS), positron emission tomography (PET), electrophysiological markers such as EEG, P50 sensory gating, and P300 cognitive potentials, as well as cerebrospinal fluid (CSF) biochemical markers, inflammatory mediators, and neuroendocrine measures.

3. To explore the concept of multimodal biomarker integration, emphasizing how convergent biological signals may better capture network-level dysregulation underlying psychiatric disorders than single-marker approaches.

4. To introduce the concept of neural convergence hubs, particularly brainstem and midbrain integrative structures, as key organizational nodes linking cortical, limbic, and autonomic systems.

5. To discuss the periaqueductal gray (PAG) as a model convergence hub, integrating emotional processing, defensive behaviour, pain modulation, and autonomic regulation, and to examine its potential relevance in stress-related and affective disorders.

6. To evaluate emerging technologies that allow simultaneous or complementary assessment of brain function, including advanced neuroimaging, electrophysiological monitoring, and hemodynamic techniques.

7. To stimulate discussion on future directions for biomarker-driven biological psychiatry, including the development of integrated biomarker models capable of informing diagnosis, prognosis, and therapeutic innovation.

This symposium aims to move the conversation beyond isolated biological findings toward a systems-level understanding of psychiatric disorders grounded in multimodal biological evidence.

Structured rationale:
Biological psychiatry has developed a wide range of investigative tools capable of probing the biological substrates of mental disorders. Despite these advances, biomarker research in psychiatry remains fragmented across methodological domains. Single-modality biomarkers have repeatedly struggled to achieve diagnostic specificity, highlighting the need for integrative biological frameworks capable of explaining the complex and overlapping clinical presentations observed in psychiatric disorders.

Increasing evidence suggests that psychiatric conditions arise from disturbances across distributed neural systems linking cortical, limbic, and brainstem structures rather than dysfunction within isolated brain regions. Within these systems, certain anatomical regions may function as convergence hubs integrating emotional, autonomic, stress, and defensive responses.

The periaqueductal gray (PAG), a midbrain structure interconnected with the amygdala, hypothalamus, prefrontal cortex, and brainstem autonomic centres, represents a compelling example of such an integrative node. Its role in defensive behaviour, panic responses, stress modulation, and pain regulation highlights its potential relevance to several psychiatric conditions characterized by dysregulated emotional and physiological responses.

Advances in multimodal technologies—including neuroimaging, electrophysiological monitoring, and hemodynamic techniques such as functional near-infrared spectroscopy—now provide opportunities to examine these integrative systems through complementary biological signals.

This symposium proposes that progress in biological psychiatry will depend on integrating multimodal biomarkers within network-level models of brain function, enabling a deeper understanding of how biological systems converge to produce psychiatric symptoms.

Learning Objectives

At the end of this symposium, participants will be able to:

1. Explain the limitations of single-modality biomarker approaches in psychiatry and the need for integrative biological frameworks.

2. Identify major classes of biomarkers used in biological psychiatry, including neuroimaging, electrophysiological, biochemical, and autonomic measures.

3. Understand the concept of neural convergence hubs and discuss how structures such as the periaqueductal gray (PAG) integrate emotional, autonomic, and defensive processes.

4. Evaluate the potential of multimodal biomarker integration for advancing systems-level models of psychiatric disorders and guiding future translational research.

S-41
Biological Psychiatry at the Crossroads: Multimodal Biomarkers and Convergence Hubs in the Architecture of Mental Disorders

S-41-001
Multimodal Biomarkers and Network Convergence in Biological Psychiatry: Toward an Integrative Framework

Speaker: Rezaul Hamid, India

Abstract

Abstract Title:
Biological Psychiatry at the Crossroads: Multimodal Biomarkers and Convergence Hubs in the Architecture of Mental Disorders

Abstract:
Background: Biological psychiatry stands at a critical crossroads where advances in neuroimaging, electrophysiology, inflammatory biology, genetics, and computational neuroscience have generated vast amounts of biological data without a sufficiently integrated framework to explain psychiatric disorders. Single-modality biomarkers have repeatedly shown limited diagnostic specificity, highlighting the need for systems-level models capable of integrating distributed neural, autonomic, and behavioural processes.

Objectives: This symposium aims to: (1) examine the current fragmentation of biomarker research in psychiatry; (2) review major biological markers including neuroimaging, EEG, hemodynamic, biochemical, inflammatory, and neuroendocrine measures; (3) explore multimodal biomarker integration as a framework for understanding network-level dysregulation; (4) introduce neural convergence hubs as organizational nodes linking cortical, limbic, and autonomic systems; and (5) discuss translational implications for diagnosis, prognosis, and therapeutic innovation.

Methods: The symposium will feature expert presentations synthesizing evidence from multimodal neuroimaging, electrophysiology, functional near-infrared spectroscopy (fNIRS), and systems neuroscience. Particular emphasis will be placed on the concept of convergence hubs, especially the periaqueductal gray (PAG), as integrative structures involved in emotional regulation, defensive behaviour, stress modulation, pain processing, and autonomic control. Cross-disciplinary discussions will examine how complementary biological signals may enhance understanding of psychiatric pathophysiology beyond categorical diagnostic models.

Results (Expected): Emerging evidence suggests that multimodal biomarker integration may provide a more biologically coherent understanding of psychiatric disorders than isolated biomarkers alone. Convergent network models incorporating cortical, limbic, autonomic, and brainstem systems may improve translational relevance, facilitate early detection, refine phenotypic characterization, and support development of precision-based therapeutic approaches.

Conclusion: Future progress in biological psychiatry will likely depend on integrative frameworks capable of organizing diverse biological signals into network-based models of

mental disorders. By bridging neurobiology, technology, and clinical psychiatry, multimodal convergence approaches may help transform fragmented biomarker findings into clinically meaningful translational systems.

S-42
Psychedelic Assisted Therapy and it's Implications in Psychiatry

Chair: Diptanu Pandey, India

Co-Authors:

Suvayan Saha

Topic:
10) Other

Abstract

Objectives:
The objective of this review is to provide a comprehensive overview of psychedelic-assisted therapy (PAT), focusing on the classification of psychedelic substances, their neurobiological mechanisms of action, the therapeutic framework used in psychedelic-assisted psychotherapy, current clinical evidence across psychiatric disorders, and the evolving legal and regulatory landscape. Additionally, the review aims to examine safety considerations, potential risks, and emerging directions for research and clinical implementation.

Given the renewed scientific interest in psychedelic compounds over the past two decades, the present work seeks to synthesize contemporary findings from clinical trials, neuroimaging studies, and theoretical models to better understand the therapeutic potential of these substances. Particular emphasis is placed on the role of classic serotonergic psychedelics and non-classic agents in modulating neural networks implicated in psychopathology, as well as the integration of pharmacological effects with structured psychotherapeutic interventions.

Structured rationale:
Psychiatric disorders such as major depressive disorder, post-traumatic stress disorder, anxiety disorders, and substance use disorders remain leading causes of global disability. Despite advances in psychopharmacology and psychotherapy, a substantial proportion of patients experience treatment-resistant symptoms, highlighting the need for novel therapeutic approaches.

Psychedelic compounds were widely studied during the mid-20th century; however, research largely halted following regulatory restrictions in the late 1960s and early 1970s. In recent years, advances in neuroscience, improved research methodologies, and carefully controlled clinical protocols have led to a resurgence of interest in psychedelic research.

Emerging evidence suggests that psychedelics may produce rapid and sustained improvements in certain psychiatric conditions. These effects are hypothesized to arise from mechanisms including 5-HT2A receptor–mediated modulation of cortical networks, disruption of rigid patterns of brain activity such as those involving the Default Mode Network (DMN), and enhanced neuroplasticity. When combined with structured psychotherapeutic support, these

neurobiological effects may facilitate emotional processing, cognitive flexibility, and lasting behavioral change.

Given the growing body of clinical evidence and increasing policy discussions surrounding the medical use of psychedelics, a systematic synthesis of current knowledge is necessary. Understanding the mechanisms, therapeutic models, benefits, limitations, and ethical considerations of PAT is essential for guiding future research, informing clinical practice, and shaping responsible regulatory frameworks.

S-43
Biological Psychiatry Across Diagnoses: Moving Beyond Labels Without Losing the Clinic

Chair: Uttam Chandra Garg, India

Co-Authors:

Kashypi Garg

Topic:
10) Other

Abstract

Objectives:
Contemporary biological psychiatry has increasingly challenged traditional diagnostic boundaries, demonstrating that many biological processes relevant to mental illness, such as stress system dysregulation, altered neural circuitry, immune and metabolic changes, and impaired neuroplasticity cut across established diagnostic categories. While these transdiagnostic insights have advanced scientific understanding, their relevance to everyday clinical practice remains uncertain for many clinicians, who must continue to make decisions within diagnostic frameworks, balancing it with their clinical acumen and experience. This symposium aims to bridge this gap by examining how transdiagnostic biological findings can be meaningfully integrated into clinical psychiatry without undermining the pragmatic utility of diagnoses. Rather than advocating for the abandonment of diagnostic systems, the symposium adopts a balanced perspective that views diagnoses as clinically useful constructs, while acknowledging their biological limitations.

Structured rationale:
The first presentation will outline key transdiagnostic biological mechanisms observed across major psychiatric disorders, highlighting shared pathways related to stress, emotion regulation, cognition, and illness progression. Emphasis will be placed on human studies and clinically interpretable findings. The second presentation will critically examine the evidence supporting transdiagnostic biological models, discussing both their strengths and methodological challenges, including heterogeneity, replication issues, and limited clinical applicability. The final presentation will focus on clinical implications, exploring how transdiagnostic biological knowledge can inform assessment, prognosis, and treatment planning across diagnostic categories. Practical examples will illustrate how clinicians can incorporate biological insights—such as illness staging, neuroprogression, and systemic health factors—into routine practice without overreliance on unvalidated tests or premature interventions.

S-44

Biological Architecture of Psychiatric Disorders: Insights from Indian Cohorts Across Genes, Cells, and Networks

Chair: Biju Viswanath, India

Co-Authors:

Bharath Holla

Topic:
7) Mood disorders, 6) Addictions

Abstract

Objectives:
1. To examine genetic susceptibility and epigenetic regulation in alcohol use disorder and related liver disease through GWAS, population-specific allele variation, and brain DNA methylation analyses.

2. To characterize genetic risk in bipolar disorder through GWAS and trans-ancestry polygenic scores, and examine their clinical relevance across psychiatric disorders.

3. To characterize the cellular and functional features of iPSC-derived neurons and brain organoids generated from the CBM BD cohort.

4. To examine the spatiotemporal expression and interactomic patterns of genes linked to psychiatric and neurological syndromes, and drug targets.

Structured rationale:
Major psychiatric disorders are highly heritable and multifactorial. However, South Asian populations, particularly from India, remain underrepresented in psychiatric genomics, creating a critical gap in understanding disease biology. Recent efforts have focused on developing large cohorts and integrative multi-omics approaches to address this disparity.

This symposium highlights ongoing cohort studies, emerging genetic signals, and their global relevance. Covering trans-ancestry genome-wide association studies (GWASs), functional genomics, cellular models, and DNA methylation, it emphasizes how multi-scale approaches are advancing our understanding of psychiatric disorders. Dr. Biju Viswanath, chair, will provide an overview of key initiatives, including CBM and ABIGNET.

In the ensuing series of presentations, Dr. Bhagyalakshmi Shankarappa will present an overview of ongoing projects and cohorts investigating diverse psychiatric disorders, with a focus on integrating genetic and epigenetic approaches. It will highlight findings from GWASs of alcohol use disorder (AUD) and alcohol-related liver disease, emphasizing how population differences in allele frequency of alcohol-metabolizing genes (ADH and ALDH) contribute to susceptibility and clinical outcomes. Differences in DNA methylation between clinical subgroups will provide

insight into regulatory mechanisms underlying disease progression. Region-specific methylation patterns and epigenetic clock analyses from a pilot EPIC array study on postmortem brain samples will be presented. Together, these approaches highlight the role of DNA methylation in gene–environment interactions and the value of postmortem brain tissue in understanding neuropsychiatric disorders.

Dr. Jayant Mahadevan will be presenting outcomes of a GWAS of BD from India and a meta-analysis with the existing evidence. He will further be providing an update on the early findings from the Indian site as part of a large multi-country GWAS effort in bipolar disorder - ABIGNET. The talk will include the evidence of the impact of Trans-Ancestry Polygenic Scores (TA-PGS) on various clinical features of the BD cohort and in other ongoing studies in schizophrenia, and Obsessive Compulsive Disorder (OCD).

Dr. Kalyani Karunakaran will present the insights from functional analyses of genomic variants discovered in ongoing studies. These will include her examination of the spatiotemporal expression and interactomic patterns of genes linked to psychiatric and neurological syndromes with overlapping symptom clusters, ages-of-onset, and disease progress. These analyses suggest that many risk genes converge on similar biological systems. These approaches may also help understand mechanisms underlying complex drug effects (e.g., polarity switch observed with mood stabilisers and antidepressants).

Dr. Pradip Paul will present details of the CBM cellular bio-repository and highlight cellular and functional analyses of the CBM BD cohort, conducted using iPSC-derived neurons and brain organoids. Further, he will present key findings on the mechanism of action of lithium, using iPSCs derived from lithium responders and non-responders in BD.

Overall, we discuss the biology of major psychiatric illnesses using CBM and other ongoing studies in the Indian population. Correlating genetic findings with deep clinical assessments, genomic variations, and phenotypes may help us understand disease mechanisms in psychiatry. These findings have the potential to accelerate the discoveries in psychiatry genomics and molecular neuroscience.

S-44
Biological Architecture of Psychiatric Disorders: Insights from Indian Cohorts Across Genes, Cells, and Networks

S-44-001
Genetic and Epigenetic Architecture of Psychiatric Disorders: Bridging Population Gaps through Indian Cohorts and Brain Methylation Studies

Speaker: Bhagyalakshmi Shankarappa, India

Abstract

Abstract Title:
Exploring Genetic Susceptibility and Epigenetic Regulation in Alcohol Use Disorder

Abstract:
This presentation will provide an overview of ongoing genetic and epigenetic studies investigating alcohol use disorder (AUD) and alcohol-related liver disease (ALD), with a focus on understanding biological mechanisms underlying disease susceptibility and progression. Findings from genome-wide association studies (GWASs) of AUD and ALD will be highlighted, particularly the contribution of population-specific variation in alcohol-metabolizing genes such as ADH and ALDH to differences in alcohol sensitivity, dependence risk, liver injury, and clinical outcomes across populations.

The presentation will further explore the role of epigenetic regulation in disease progression through analysis of DNA methylation patterns across clinically distinct subgroups. Differential methylation at loci implicated in inflammation, lipid metabolism, and alcohol metabolism will be discussed in the context of gene–environment interactions and chronic alcohol exposure. In addition, preliminary findings from a pilot EPIC methylation array study conducted on postmortem brain tissue samples will be presented.

Together, these integrated genomic and epigenomic approaches provide insight into the molecular pathways contributing to AUD and ALD and demonstrate the importance of combining peripheral and brain-based epigenetic studies. The work also highlights the value of postmortem human brain tissue as a resource for advancing research into neuropsychiatric and substance use disorders.

S-44
Biological Architecture of Psychiatric Disorders: Insights from Indian Cohorts Across Genes, Cells, and Networks

S-44-002
Understanding the Contribution of Common and Rare Genetic Variations to Bipolar Disorder in India

Speaker: Jayant Mahadevan, India

Abstract

Abstract Title:
Understanding the Contribution of Common and Rare Genetic Variations to Bipolar Disorder in India

Abstract:
Background: Genetic factors contribute significantly to the risk of bipolar disorder (BD) with heritability estimates ranging between 60% to 90%. Recent studies have significantly improved our understanding of both common and rare variants associated with BD. These studies include individuals of European, African-American and East Asian ancestries. However, South Asian ancestry, which includes India, has the lowest representation of all ethnic groups. The paucity of data is concerning as it hinders the potential application of genetic findings to clinical practice. It also underscores the urgent need to conduct genome wide association studies (GWAS) of psychiatric phenotypes from India.

Methods: Patients with BD (n = 787) and unaffected controls (n = 660) genotyped using Illumina Global Screening Arrays were used for the first analysis. Standard pipelines were used for quality control (QC). Population structure was examined with principal component analysis (PCA) and imputation was done using TOPMed on the Michigan Imputation Server. GWAS was performed using Regenie, followed by meta-analysis. Post GWAS analysis was conducted using FUMA. Cross-ancestry PGS were constructed for BD with PGS-CS-auto and SBayesRC using publicly available GWAS summary statistics from European ancestry populations. Patients with BD (n=500) and unaffected controls (n=500) recruited as part of the Asian Bipolar Genetics Network (ABIGNET) and who underwent blended genome exome sequencing (BGE) will be used for the second analysis.

Results: Global PCA with 1000G data showed that the Indian samples positioned between European and East Asian clusters, with overlap on all the South Asian population labels, and also displayed intra-regional variability, extending along the PC1 axis but condensed along PC2. GWAS showed no significant genetic loci associated with BD in our sample. However, a high genetic correlation was noted with multi-ancestry GWAS of BD. PGS of BD derived from a multi-ancestry sample showed moderate-to-high predictive power for BD in our sample.

Discussion: This study demonstrates an overlapping common variant architecture of BD in India compared to the rest of the world. The low sample size for this analysis contributed to an absence of significant genome wide hits. However, the predictive power of PGS in our sample is promising. The ABIGNET study which has already collected 5000 cases of well characterized

BD-1 and 5000 controls (Target:10000 cases and 10000 controls) from across India is likely to offer further insights into both the common and rare genetic variant contributions to BD.

S-44
Biological Architecture of Psychiatric Disorders: Insights from Indian Cohorts Across Genes, Cells, and Networks

S-44-003
Cellular Models of Psychiatric Disorder: Insights from iPSC-Derived Neurons, Organoids, and Drug Response

Speaker: Pradip Paul, India

Abstract

Abstract Title:
Deciphering cellular mechanisms of psychiatric disorders using induced pluripotent stem cells

Abstract:
The idea that psychiatric diseases have their roots in fetal brain development has been around for decades, but not explored much in bipolar disorder (BD). Brain cells derived from human induced pluripotent stem cells (iPSC) offer an unprecedented opportunity to model specific aspects of BD pathophysiology. To investigate cellular aberrations in BD, we generated lymphoblastoid cell lines (LCLs) and iPSC derived neural cells, and performed cellular and functional assays. The cells were also exposed to lithium in vitro to investigate if disease-associated phenotypes are reversed. The findings revealed aberrations in cell proliferation, mitochondrial membrane potential; and the reversal with in-vitro lithium. Patient-derived LCLs and iPSC derived neural cells exhibited reduced viability, rescued in clinical lithium responders. Dysfunctions in cellular phenotypes central to neural precursor cells (NPCs) were also observed: in their capacity to proliferate and migrate. Much like in LCLs, in patient-derived NPCs, we observed a reversal in cellular phenotypic abnormalities with in vitro lithium exposure. The iPSC derived human cortical organoids also detected abnormalities for organisational, proliferation and migration defects in BD. Aforementioned studies demonstrate subtle changes in fetal brain development associated with BD. The relevance of cellular phenotypes examined here to clinical phenotypes is strengthened by rescue of observed defects with lithium exposure.

S-44
Biological Architecture of Psychiatric Disorders: Insights from Indian Cohorts Across Genes, Cells, and Networks

S-44-004
Understanding neuropsychiatric disease and drug biology using spatiotemporal transcriptomics and interactomics

Speaker: Kalyani B Karunakaran, India

Abstract

Abstract Title:
Neuropsychiatric disease and drug biology through the lens of spatiotemporal transcriptomics and interactomics

Abstract:
Background: Clinical syndromes often overlap across psychiatric and neurological classifications, which are difficult to capture using genetic correlations alone. To examine cross-disorder links, we analyzed spatiotemporal expression and interactome patterns of risk genes associated with five syndromes with overlapping symptoms and ages of onset: obsessive-compulsive disorder (OCD), Huntington's disease (HD), Parkinson's disease (PD), schizophrenia (SZ), and bipolar disorder (BD). We also hypothesized that the clinical effects of drugs are driven by connections between disease genes and drug targets in the interactome.

Methods: GWAS-derived genes were mapped onto BrainSpan RNA-sequencing data. Spatial and temporal clusters were identified using hierarchical and time-series approaches. Syndrome-specific protein-protein interaction networks (BioGRID/HPRD) were modularized using MCODE. Hypergeometric and Monte Carlo tests assessed gene and network enrichment.

Results: Disease genes alone showed no significant interconnectivity. However, inclusion of first-order interactors revealed extensive connectivity exceeding random expectation, which persisted after hub removal. OCD and PD networks showed prenatal expression signatures, while HD and BD co-emerged. Highly interconnected functional modules drove these cross-disorder patterns. Hence, shared expression signatures give rise to proteins occupying adjacent interactomic neighbourhoods and participating in similar cellular functions.

Drug analyses showed that first-generation antipsychotic targets inducing Parkinsonism directly interact with PD risk genes. We also examined mood polarity switching in drugs: category I (antidepressants inducing mania), category II (antimanic drugs inducing depression), and category III (used for mania/depression, with no such side effects). Category I and III targets mapped to a shared depressive disorder (DD) risk space (BD-DD-SZ, BD-DD), with category III robust to hub removal, while category II mapped to BD-SZ and SZ-specific spaces, disrupted after hub removal.

Conclusions: These findings support a network-based model in which genetic risk operates through distributed, coherent interactome modules across brain regions and development. In addition, clinical drug effects may arise from non-random interactions between disease genes and drug targets.

Clinical syndromes often overlap across psychiatric and neurological classifications, which are difficult to capture using genetic correlations alone. To examine cross-disorder links, we analyzed spatiotemporal expression and interactome patterns of risk genes associated with five syndromes with overlapping symptoms and ages of onset: obsessive-compulsive disorder (OCD), Huntington's disease (HD), Parkinson's disease (PD), schizophrenia (SZ), and bipolar disorder (BD). Disease genes alone showed no significant interconnectivity. However, inclusion of first-order interactors revealed extensive connectivity exceeding random expectation, which persisted after hub removal. OCD and PD networks showed prenatal expression signatures, while HD and BD co-emerged. Highly interconnected functional modules drove these cross-disorder patterns. Hence, shared expression signatures underlie proteins occupying adjacent interactomic neighbourhoods and participating in similar cellular functions. We then hypothesized that interactome connections between disease genes and drug targets likely drive the clinical effects of drugs. We found that the targets of first-generation antipsychotics inducing Parkinsonism directly interact with PD risk genes. We also examined drug-induced mood polarity switching: category I (antidepressants inducing mania), category II (antimanic drugs inducing depression), and category III (used for mania/depression without such side effects). Category I and III targets mapped to a shared depressive disorder (DD) risk space (BD-DD-SZ, BD-DD), while category II targets mapped to BD-SZ and SZ-specific spaces that were disrupted after hub removal. These findings support a network-based model in which genetic risk operates through distributed, coherent interactome modules across brain regions and development. In addition, clinical drug effects may arise from non-random interactions between disease genes and drug targets.

S-45
Circuit-Based Psychiatry: From Symptom Clusters to Network Modulation — The New Frontier in Biological Psychiatry

Chair: Rezaul Hamid, India

Co-Authors:

Muhamad Aly Rifai

Topic:
10) Other

Abstract

Objectives:
This symposium aims to formally introduce Circuit-Based Psychiatry as an essential paradigm shift in biological psychiatry. The objectives are:

1. To critically examine the structural limitations of symptom-cluster–based diagnostic systems in addressing neurobiological heterogeneity, cross-diagnostic overlap, and persistent treatment resistance.

2. To demonstrate that emerging evidence from functional connectivity research compels a transition from categorical description to network-informed psychiatric formulation.

3. To articulate a structured framework in which core neural systems — including the Default Mode Network, Salience Network, Central Executive Network, cortico-striato-thalamo-cortical circuits, and reward pathways — serve as organizing principles for understanding psychopathology.

4. To reframe neuromodulation (rTMS, TBS, VNS) as targeted network interventions rather than disorder-specific techniques.

5. To examine biomarker-guided approaches, including functional near-infrared spectroscopy (fNIRS), as scalable tools for circuit-informed clinical decision-making.

6. To present addiction as a translational model demonstrating how circuit dysfunction clarifies diagnosis, predicts treatment response, and guides neuromodulatory strategies.

7. To initiate a high-level discussion on the ethical, regulatory, and training implications of adopting a circuit-based framework in global psychiatric practice.

This symposium seeks to catalyse a disciplined yet decisive shift from descriptive psychiatry toward network-informed precision models.

Structured rationale:
Biological psychiatry has advanced in neuroscience, imaging, and neuromodulation, yet its diagnostic architecture remains largely anchored in symptom aggregation. While categorical systems improved reliability, they have not resolved the fundamental problem of biological non-specificity. Persistent heterogeneity within diagnoses, high rates of comorbidity, and inconsistent treatment response reflect a mismatch between descriptive classification and underlying neural organization.

Converging evidence from connectomics and functional neuroimaging demonstrates that psychiatric disorders are not discrete entities but patterns of dysregulation across distributed neural networks. Core systems — including the Default Mode Network, Salience Network, Central Executive Network, cortico-striato-thalamo-cortical loops, and mesocorticolimbic reward circuitry — cut across traditional diagnostic boundaries and offer a more biologically coherent framework for understanding psychopathology.

Simultaneously, the maturation of neuromodulation technologies (rTMS, TBS, VNS) and cortical monitoring tools such as fNIRS has created the capacity not only to observe but to selectively influence network dynamics. These developments render a purely symptom-based framework increasingly insufficient for guiding precision intervention.

Circuit-Based Psychiatry is proposed as the next evolutionary stage of biological psychiatry — integrating phenomenology with network science to create a structured model in which symptoms are mapped onto circuit dysfunction and treatment is conceptualized as targeted modulation.

Aligned with the congress theme “Biological Psychiatry in a Changing World – Exploring New Frontiers,” this symposium advances circuit-based formulation as a necessary progression rather than an optional innovation.

Circuit-based psychiatry is not an optional innovation; it is the logical next step in the maturation of biological psychiatry.

S-45
Circuit-Based Psychiatry: From Symptom Clusters to Network Modulation — The New Frontier in Biological Psychiatry

S-45-001
Circuit-Based Psychiatry: Reframing Diagnosis from Symptom Aggregation to Network Dysfunction

Speaker: Rezaul Hamid, India

Abstract

Abstract Title:
Circuit-Based Psychiatry: From Symptom Clusters to Network Modulation — The New Frontier in Biological Psychiatry

Abstract:
Background:

Despite major advances in neuroscience, neuroimaging, and neuromodulation, contemporary psychiatric classification remains predominantly symptom-based. While categorical diagnostic systems improved inter-rater reliability, they continue to face significant limitations including biological non-specificity, diagnostic heterogeneity, high comorbidity, and inconsistent treatment response. Emerging evidence from connectomics and functional neuroimaging suggests that psychiatric disorders may be better conceptualized as dysregulations within distributed neural circuits rather than isolated categorical entities.

Objectives:

This symposium aims to introduce and critically examine Circuit-Based Psychiatry as an evolving framework in biological psychiatry. The objectives are to:

1. Evaluate the limitations of symptom-cluster–based diagnostic systems.

2. Explore how functional connectivity research supports network-informed psychiatric formulation.

3. Examine the role of major neural systems—including the Default Mode Network, Salience Network, Central Executive Network, cortico-striato-thalamo-cortical circuits, and reward pathways—in psychopathology.

4. Reframe neuromodulation techniques such as rTMS, TBS, and VNS as targeted network interventions.

5. Discuss biomarker-guided approaches including functional near-infrared spectroscopy (fNIRS) for precision psychiatry.

6. Present addiction as a translational model of circuit dysfunction and network-based intervention.

7. Address the ethical, regulatory, and training implications of adopting a circuit-based framework in clinical practice.

Methods:

The symposium integrates contemporary evidence from functional neuroimaging, connectomics, translational neuroscience, and neuromodulation research. Expert presentations will synthesize conceptual models, emerging biomarkers, and real-world clinical applications of circuit-informed psychiatric care, including neuromodulatory interventions and multimodal treatment integration.

Results / Expected Outcomes:

The symposium is expected to demonstrate that many psychiatric syndromes reflect overlapping dysfunctions across core neural networks rather than discrete disease entities. It will highlight how circuit-informed approaches may improve diagnostic precision, guide biomarker-assisted treatment selection, and optimize neuromodulation strategies. The sessions will also illustrate how addiction psychiatry provides a practical translational model for targeted modulation of reward and executive control circuits.

Conclusions:

Circuit-Based Psychiatry represents a logical evolution of biological psychiatry, integrating phenomenology with network neuroscience to move toward precision-oriented psychiatric care. By shifting focus from descriptive symptom aggregation to neural circuit dysfunction, this framework has the potential to transform diagnosis, treatment planning, and neuromodulation strategies in modern psychiatry. The symposium aligns with the broader movement toward biologically informed, network-based models of mental illness in a rapidly evolving neuroscientific landscape.

S-46

Determination of Predominant Polarity and its importance in managing patients suffering from Bipolar disorder across lifespan

Chair: Ranjan Bhattacharyya, India

Co-Authors:

Naba Raj Koirala

Topic:
7) Mood disorders, 1) Psychoses

Abstract

Objectives:
In bipolar disorder, predominant polarity refers to the pattern in which a patient experiences more episodes of one pole of mood disturbance than the other over time.

Types of Predominant Polarity

Depressive predominant polarity

The majority of episodes are depressive

Most common pattern

Associated with:

Higher suicide risk

Greater functional impairment

Often seen in Bipolar II disorder

Manic (or hypomanic) predominant polarity

The majority of episodes are manic/hypomanic

More common in Bipolar I disorder

Associated with:

More hospitalizations

Increased risk-taking behavior

Structured rationale:
Determining predominant polarity in bipolar disorder is clinically very important because it directly influences management, prognosis, and long-term planning. Importance of Determining Predominant Polarity

1. Guides Treatment Selection

Depressive predominant polarity

Focus on preventing depressive episodes

Use: mood stabilizers (e.g., lithium, lamotrigine), cautious use of antidepressants

Manic predominant polarity

Focus on preventing mania

Use: antimanic agents (lithium, valproate, antipsychotics)

Helps in individualized pharmacotherapy

2. Predicts Course and Prognosis

Depressive polarity → more chronic course, residual symptoms, poorer functioning

Manic polarity → more episodic but severe relapses and hospitalizations

3. Suicide Risk Assessment

Higher risk in patients with depressive predominant polarity

Important for monitoring and preventive strategies

4. Helps in Maintenance Therapy Planning

Determines long-term prophylaxis strategy

The choice of drug depends on which pole needs stronger prevention

5. Reduces Risk of Treatment-Induced Switching

In depressive polarity, careless antidepressant use may trigger mania

Knowing polarity helps avoid mood switching

S-47
Non Suicidal Self Injury and Suicide: Understanding the biology and interventions

Chair: Hemendra Singh, India

Co-Authors:

Manaswi Gautam

Topic:
10) Other

Abstract

Objectives:
1. To review the current understanding of the psychological and biological underpinnings of self-injurious behavior

2. Evidence for utilization of biological therapies in treatment and prevention of self-injurious behavior

Structured rationale:
Suicide is the third leading cause of death worldwide and India ranks 49th in most suicides in the world. Unlike the global trends, India has shown an increase in suicide rates in the past 10 years from 11.2% in 2012 to 12.4% in 2022, a 10% increase. The 1-year prevalence of non-suicidal self-injury in India is 30-32%, almost double that of the western population. Initial theories of self-injurious behaviors focused on psychodynamic factors such as aggression turned inwards, identity formations, response to self-hatred or guilt, repressed impulses, primitive defence mechanisms, and cognitive factors such as frustration, helplessness, venting anger, establishing control or a cry for help. However, advances in neuroimaging, genetic testing, and biotechnology have highlighted the role of pre-frontal cortex functioning, serotonin, glutamate, BDNF, and risk genes and epigenetic factors on decision making, emotional regulation, impulsivity, and aggression – risk factors leading to in self-injurious behavior. This emphasises on the need for better understanding the biology of self-injurious behavior and to explore treatment strategies that are effective in its prevention. Few biological treatments with anti-suicidal action (Modified ECT and drugs such as Lithium, Clozapine, Ketamine) are available currently and their mechanisms of action are poorly understood. Understanding the neurobiological aspects of self-injurious behaviors can lead to development of better treatment strategies.

Dr Anitha Gautam will be highlighting on the hidden epidemiology of suicide. Majority of individuals visited general hospital setting before death by suicide. Dr Manaswi Gautam will present on the psychological theories of self-injurious behavior. As history of past suicide attempt is an important risk factor for future suicide, hence there is need understand the prevailing patterns and causes for self-injurious behavior. Dr. Hemendra Singh will discuss about the various aspects of Non-suicidal self-injury and suicidal behaviour and its prevention

in general hospital setting and Dr Swati C will be discussing about biological aspects of suicide and efficacy of biological interventions on suicide prevention.

Key words: Suicide attempts, Non-suicidal self-injury, Suicide prevention, neurobiology

S-47
Non Suicidal Self Injury and Suicide: Understanding the biology and interventions

S-47-003
Aspects of Non-suicidal self-injury and suicidal behaviour and its prevention in general hospital setting

Speaker: Hemendra Singh, India

Abstract

Abstract Title:
Intentional Self harm and Suicide Prevention in General Hospital Settings: An Indian Perspective

Abstract:
Self-harm is a major public health challenge in India, with emergency department and psychiatric care at the general hospitals setting is the first point of care for individuals at risk. However, the absence of standardized hospital based brief intervention and inadequate post-discharge follow-up plans pose significant barriers to for suicide prevention. This symposium will critically examine hospital-based self-harm prevention strategies focusing particularly insights from general hospital settings in Bangalore, India. The current session addresses three important aspects: (1) Suicide Risk Assessment- use of risk and protective factors and challenges in the general hospital settings for suicide risk assessment and management (2) Brief intervention module in General Hospitals – highlighting real world experiences of general hospital-based interventions for suicide prevention, challenges in implementing in the general hospital setting; and (3) Post-Discharge Care – the importance of assertive telephonic follow-up, and crisis response planning to reduce re-attempts. By integrating clinical experiences, research insights, and standard care policy perspectives, this session aims to provide a comprehensive framework for strengthening suicide prevention efforts in Indian general hospitals.

S-47
Non Suicidal Self Injury and Suicide: Understanding the biology and interventions

S-47-004
Suicide: biological aspects and interventions

Speaker: Swati Chandramouli, India

Abstract

Abstract Title:
Suicide: neurobiology and biological interventions

Abstract:
Suicide is a transdiagnostic phenomenon; often occurring in the absence of other formal psychiatric diagnosis. It has a complex biopsychosocial etiology and multidimensional clinical presentation. In this session, we will focus on the neurochemical (Serotonin, norepinephrine, dopamine), neuroendocrine (hypo-pituitary-adrenal axis) and neuroanatomic (ventromedial prefrontal cortex) endophenotypes and, genetic factors of suicide and its interaction with the clinical endophenotypes (aggression, impulsivity etc.) contributing to suicidal behavior. The session will conclude with the currently available biological interventions (Lithium, modified ECT etc.) and their efficacy.

S-48

Experimental ways to understand and individualise neuromodulation in mood disorders: Updates from electroconvulsive therapy, recurrent transcranial magnetic stimulation and (transcutaneous) vagal nerve stimulation (tVNS)

Chair: Philipp Sämann, Germany

Co-Authors:

Alexandros Balaskas

Topic:
7) Mood disorders, 10) Other

Abstract

Objectives:
This symposium will deal with neuromodulation in mood disorders that meanwhile offers a range of techniques to influence brain activity. We would like to address clinicians who are also interested in the neurobiology of neuromodulation. For each present method, we will briefly point out current clinical application recommendations, but then also turn to more specialised experimental studies on mechanistic aspects of these treatments. We cover both two established techniques – electroconvulsive therapy (ECT) and recurrent transcranial magnetic stimulation (rTMS) – but also two more recently promoted techniques that are currently entering the canon of evidence based treatments: transcutaneous vagal nerve stimulation (tVNS) and transcranial focussed ultrasound (tFUS) that rely both on very different stimulation principles. For ECT and rTMS, clinical trials support a general effect, yet with open question regarding placebo effects, non-response and clinical or biological prediction markers. For tVNS and tFUS the clinical trial literature is still sparse, not allowing for generally accepted guidelines and with new and growing indication fields such as pain, addiction or suicidality. All these techniques have benefitted strongly from additional experimental studies in animals and healthy participants that focus on how neuroimaging or peripheral readouts change during treatment. We will thus present and discuss a number of experimental approaches: First, animal studies play a role to understand effects of ECT at the cellular and circuit level, or to understand effects of tVNS on central and peripheral components of the autonomic nervous system. One specific objective here is to learn about imaging effects of ECT in animals and humans from longitudinal volumetric and diffusion imaging studies. Second, also in rTMS, neuroimaging studies that map stimulation effects in clinical populations have provide insights into the systemic circuit level. Here, more explorative network level results compete with very specific hypotheses on circuit changes. The objective here is to inform about E-field simulation as an approach to quantify and predict rTMS effects, particularly for DLPFC stimulation in depression. Neuroimaging has created an increasing body of knowledge on ways to taylor the treatment to the individual anatomy or even dysfunction. This may gain even more weight in anatomically highly precise techniques such as tFUS, as here the individual anatomical target calculations is particularly important. We will thus discuss that knowledge of circuit pathology in depression is currently being re-used for newer techniques such as tFUS. One example for this the longer history of subgenual anterior cingulate modulation in depression. Third, neuromodulation

experiments point out that there is a close entanglement of the central nervous system with the peripheral (autonomous) nervous system and other peripheral pathways: Two complementary examples for this will be discussed: rTMS seems to influence the immune system more than expected and could thus be helpful in mixed depression/fatigue syndromes. In reverse, stimulation of the auricular branch of the vagal nerve seems to have critical effects on the central arousal system, as for example estimated from pupillometry as elegant non-invasive biomarker technique.

Structured rationale:
The symposium is broad on one hand in terms of the modulation techniques covered, but we think that this design attracts clinicians and clinician scientists. All presenters are clinicians or translations researchers with a knowledge of both experimental medicine and clinical applications or practice. Instead of presenting merely clinical effects of neuromodulation trials, we would also like to present different experimental approaches that all allow to advance in the understanding of neuromodulation, and indirectly add to the understanding of mood disorders.

The objectives listed in the overview text are not entirely running in parallel with the four speakers, but will be covered as a whole: Talks will generally point out short summaries of the level of clinical effect evidence for the respective technique, especially for mood disorders. Talks will then turn to experimental approaches that help to understand mechanistic ways of action of neuromodulation and exemplify this by presenting (recent or latest) specialised studies.

Methodologically, these experimental approaches will refer to neuroimaging (in humans and animals) as one main modality that helped to detect structural and functional effects of ECT and rTMS, two strongly established techniques, but also vagal nerve stimulation. E-fields as a way to map the reach out of rTMS and their role for subgenual anterior cingulate targeting will be highlighted, also with an outlook towards tFUS studies. As second technique, peripheral readouts for rTMS such as blood immune markers will be covered. As third technique, functional measures of the autonomous nervous system such as pupillometry or heart rate variability and their role to track vagal nerve stimulation will be explained.

S-48
Experimental ways to understand and individualise neuromodulation in mood disorders: Updates from electroconvulsive therapy, recurrent transcranial magnetic stimulation and (transcutaneous) vagal nerve stimulation (tVNS)

S-48-001
Towards precision biomarkers of ECT response: extracellular vesicles, VR-based psychophysiology, and immune profiling in the DetECT study

Speaker: Maxine Latzel , Germany

Abstract

Abstract Title:
Towards precision biomarkers of ECT response: extracellular vesicles, VR-based psychophysiology, and immune profiling in the DetECT study

Abstract:
Electroconvulsive therapy (ECT) is the most effective treatment for severe and treatment-resistant depression (TRD), yet reliable biomarkers of treatment response remain lacking. The DetECT study, running since 2022, is a prospective observational cohort investigating the biological mechanisms underlying ECT response through longitudinal clinical phenotyping and multimodal biomarker profiling. In addition to genomic, transcriptomic, proteomic, metabolomic, and immunological analyses, the study integrates neuron-derived extracellular vesicles, multiplex immune profiling, and quantitative pupillometry to capture peripheral and neurobiological correlates of treatment response. Recruitment and longitudinal sample collection are ongoing, generating a comprehensive multimodal dataset. The integrated analyses aim to identify biomarkers predictive of ECT response, improve our understanding of its underlying mechanisms, and advance precision medicine approaches for patients with TRD.

S-48
Experimental ways to understand and individualise neuromodulation in mood disorders: Updates from electroconvulsive therapy, recurrent transcranial magnetic stimulation and (transcutaneous) vagal nerve stimulation (tVNS)

S-48-002
Immune system effects of a high intensity rTMS protocol in depression

Speaker: Alexandros Balaskas, Germany

Abstract

Abstract Title:
Immune system effects of a high intensity rTMS protocol in depression

Abstract:
Emerging evidence suggests that inflammatory and immune dysregulation contribute to the pathophysiology of depression, as exemplified by depressive syndromes associated with Post-COVID condition (PCC). This presentation will discuss current perspectives on neuroimmune mechanisms in depression and introduce an ongoing randomized pilot study conducted at the Max Planck Institute of Psychiatry, Munich, investigating the immunological and clinical effects of standard daily and accelerated intermittent theta-burst stimulation (iTBS), a patterned form of repetitive transcranial magnetic stimulation (rTMS), in patients with depression and comorbid PCC. Peripheral immune and neurotrophic biomarkers, including pro- and anti-inflammatory cytokines, are assessed alongside depressive symptom severity and psychometric measures of fatigue, sleep quality, anxiety, and functional impairment. The aim is to determine whether accelerated iTBS protocols differentially modulate neuroimmune processes and clinical symptom burden, thereby informing future biomarker-guided neuromodulation strategies.

S-48
Experimental ways to understand and individualise neuromodulation in mood disorders: Updates from electroconvulsive therapy, recurrent transcranial magnetic stimulation and (transcutaneous) vagal nerve stimulation (tVNS)

S-48-003
Subgenual ACC neuromodulation: What can we learn from rTMS for tFUS?

Speaker: Philipp Sämann, Germany

Abstract

Abstract Title:
Subgenual ACC neuromodulation: What can we learn from rTMS for tFUS?

Abstract:
In this talk the functional anatomy of the subgenual ACC in the context of in treatment resistent major depressive disorders (MDD) will be explained, particularly its relation to stress system regulation and inflammatory processes. Then, the development of neuromodulationof this brain area will be recapitulated, reaching from deep brain stimulation (DBS) over recurrent TMS (rTMS) to transcranial focussed ultrasound (tFUS) that allows for more precise targeting of this brain area. All three approaches and their putative functional mechanisms will be explained. The indirect modulation of the subgenual ACC using cortical DLPFC stimulation will be reported, as well as imaging techniques to map its efficacy. Eventually, the rationale and so far available evidence for an efficacy of subgenual ACC white matter (sgACC-wm) stimulation will be presented, including the technical requirements of such a tFUS study.

S-48
Experimental ways to understand and individualise neuromodulation in mood disorders: Updates from electroconvulsive therapy, recurrent transcranial magnetic stimulation and (transcutaneous) vagal nerve stimulation (tVNS)

S-48-004
Understanding transcutaneous vagus nerve stimulation in depression: a multimodal approach to clinical translation

Speaker: Florian Höhna, Germany

Abstract

Abstract Title:
Understanding transcutaneous vagus nerve stimulation in depression: a multimodal approach to clinical translation

Abstract:
Transcutaneous vagus nerve stimulation (tVNS) is a promising non-invasive neuromodulation approach for depression, with growing evidence suggesting modulation of brainstem nuclei and distributed cortical-limbic networks involved in mood regulation, autonomic control, and inflammation. However, its neurobiological mechanisms and the extent to which they overlap with those of invasive vagus nerve stimulation (iVNS) remain poorly understood. To address these questions, we established the AddVNS study, a prospective, randomized, double-blind, sham-controlled trial combining longitudinal clinical assessments with structural and functional magnetic resonance imaging, psychophysiology, and immune and multiomic profiling. We provide an overview of the current state of the field, introduce the study design, and present the first data from AddVNS study.

S-49
From Bench to Bedside: Practical Clinical Applications of Biomarkers in Psychiatric Disorders

Chair: Suvayan Saha, India

Co-Authors:

Abhimanyu Sinha

Topic:
10) Other

Abstract

Objectives:
1.To review current categories of biomarkers relevant to psychiatric disorders, including inflammatory, genetic, neuroimaging, and electrophysiological markers.

2.To examine the potential of biomarkers to improve diagnostic precision and identify biologically meaningful subtypes of psychiatric disorders.

3.To discuss the role of pharmacogenomic and neurobiological markers in predicting treatment response and guiding personalized psychiatric care.

4.To evaluate challenges and future directions in integrating biomarkers into routine psychiatric clinical practice.

Structured rationale:
Psychiatric diagnosis and treatment have traditionally relied on symptom-based classifications, which often fail to reflect the biological heterogeneity underlying mental disorders. Over the past two decades, advances in neuroscience, molecular biology, and neuroimaging have led to the identification of multiple candidate biomarkers associated with psychiatric conditions. These biological markers hold promise for improving diagnostic accuracy, predicting treatment response, and guiding personalized therapeutic strategies.

Biomarkers in psychiatry encompass a wide range of measurable biological indicators, including inflammatory markers, genetic and epigenetic signatures, neuroimaging findings, electrophysiological measures, and peripheral metabolic markers. Elevated inflammatory markers such as C-reactive protein and pro-inflammatory cytokines have been associated with subsets of patients with major depressive disorder and schizophrenia, supporting the concept of immune-related subtypes of psychiatric illness[1]. Pharmacogenetic markers affecting cytochrome P450 enzymes have also demonstrated clinical utility in guiding psychotropic medication selection and dosing, helping reduce adverse effects and improving treatment outcomes[2].

Neuroimaging biomarkers have further contributed to identifying structural and functional abnormalities in neural circuits involved in mood regulation, cognition, and reward processing. Functional MRI studies have demonstrated alterations in prefrontal–limbic connectivity across several psychiatric disorders, providing potential markers for disease stratification and treatment monitoring[3]. Additionally, electrophysiological indicators such as EEG patterns and event-related potentials have been explored as predictors of antidepressant treatment response and cognitive dysfunction.

Despite the rapid growth of biomarker research, translation into routine psychiatric practice remains limited. Challenges include variability in study findings, lack of standardized clinical protocols, limited accessibility of advanced investigations, and uncertainty regarding their clinical utility in diverse populations. However, recent developments in precision psychiatry, computational psychiatry, and digital health technologies have renewed interest in integrating biological markers into clinical decision-making frameworks.

This symposium aims to bridge the gap between biomarker research and real-world psychiatric practice by focusing on practical clinical applications of biomarkers across major psychiatric disorders.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-001
Lipids as a biological marker for predicting violence, clinical outcome and engagement in patients with psychosis and history of violence

Speaker: Piyal Sen, United Kingdom

Co-Authors:
Veena Kumari
Alexandra I Blackmore
Mehr-Un-Nisa Waheed
Rebecca Mottram
Fern Taylor

Topic:
1) Psychoses

Abstract

Objective:
The objectives of the present study were to examine: (a) link between TC and violence, c) link between TC and outcome, and c) link between TC and treatment engagement.

Methods:
Anonymized routine data from 230 patients with schizophrenia, personality disorder, or a dual diagnosis who had been in medium/low secure care was utilized. Risks of violence was measured from HCR-20, START, days in seclusion and days on high observation levels. Lipid profile at admission (TC, high-density lipoprotein, triglycerides), outcome and engagement data was collected (length of stay, security level, engagement in nursing and psychology).

Results / Discussion:
For mean TC, there were significant inverse correlations with the days in seclusion, (r=-0.132, p=0.045), days on 2:1 observations (r=-0.129, p=0.05) and days on high levels of observation due to violence, 1:1 or 2:1 (r=-0.180, p=0.012). There was a trend-level correlation between lower TC and a positive change in security level (rho=0.123, n=211), and no significant correlation with Length of Stay, Engagement with Nursing or Psychology.

Conclusion:
This study showed a low TC-violence link for the most seriously violent patients with schizophrenia, PD and DD for both men and women. No conclusive link was demonstrated with length of stay nor engagement. The findings make the case for exploration of low TC as a possible biological marker for prediction of violence risk but perhaps not of clinical outcomes or engagement with treatment.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-002
Psychosis in Parkinson Disease: A Retrospective Study on 24-Hour Continuous Subcutaneous Infusion of Foslevodopa/Foscarbidopa

Speaker: Kensuke Ikenaka, Japan

Topic:
1) Psychoses

Abstract

Objective:
Atypical psychosis, characterized by severe delusions, paranoia, and auditory or somatic hallucinations, is a notable complication of the 24-hour continuous subcutaneous infusion foslevodopa/foscarbidopa therapy in Parkinson's disease (PD). This study aims to identify clinical predictors of CSCI-induced atypical psychosis to understand its potential mechanisms and evaluate alternative predictive measures for its early detection and management.

Methods:
We analyzed clinical data including psychosis information, Parkinson's Disease Questionnaire (PDQ39) and the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease (QUIP) from PD patients treated with foslevodopa/foscarbidopa continuous subcutaneous infusion (n = 23) and database of an independent sample of PD patients (n = 94) at Osaka University Hospital to identify predictors of atypical psychosis occurring during therapy.

Results / Discussion:
Linear regression identified QUIP as the sole predictor of atypical psychosis onset (coefficient = 0.17, Bonferroni adjusted p = 0.049). PD database cohort showed that combined PDQ38_20,27 score was significantly and positively correlated with QUIP (r = 0.38, Bonferroni adjusted p = 0.000145). PDQ38_20,27 score independently predicted psychosis onset and stratified survival curves ($p < 0.0001$) also in the continuous infusion therapy sample.

Conclusion:
Atypical PD psychosis with visual hallucination likely involves mesolimbic circuits associated with impulsive-compulsive behaviors associated with dopamine dysregulation. While QUIP is rarely used in clinical practice, the widely used PDQ39_20 and PDQ39_27 combination score offers a practical tool to identify individual psychosis risk to support tailored strategies for PD patients receiving advanced dopaminergic therapies.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-003
Neuromodulation-Driven Improvement In Auditory Hallucinations In Schizophrenia: In The Lens Of Source Monitoring And Talk/Listen Task.

Speaker: Subham Samantaray, India

Co-Authors:

Muralidhran Kesavan

Topic:
1) Psychoses

Abstract

Objective:
Auditory hallucinations (AH) in schizophrenia are hypothesized to arise from disturbances in self-agency involving corollary discharge (CD) and source monitoring (SM). We examined CD deficits using a Talk–Listen event-related potential (ERP) paradigm, & assessed SM performance using a encoding–retrieval paradigm; investigated associations between CD and SM, at baseline & after continuous theta burst transcranial magnetic stimulation(cTBS-TMS).

Methods:
25 schizophrenia patients with medication-resistant AH and matched healthy controls were assessed. CD dysfunction was quantified using N1 amplitudes during Talk and Listen conditions and the Corollary Discharge Index (CDI = N1 Listen − N1 Talk). SM was evaluated with externalization bias (EB) to internal source items. Patients received cTBS-TMS for 15 days over left temporoparietal junction, followed by repeat clinical, CD, and SM assessments.

Results / Discussion:
CD measures showed no significant post-intervention change (CDI: 1.77±1.07 vs 1.99±1.68; p=0.579). N1 amplitudes remained stable. In contrast, SM improved significantly: EB decreased (0.80±0.06 to 0.74±0.07; $p<0.001$, d=0.75). The percentage reduction in EB correlated strongly with the reduction in AHRS score.

Conclusion:
CD deficits appear trait-like and resistant to neuromodulation, whereas SM impairments are state-dependent and responsive to intervention. Patients showed reduced AH severity following cTBS-TMS. This encoding–retrieval dissociation refines mechanistic models of AH and highlights retrieval-based processes as potential therapeutic targets.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-004
High prevalence of tau pathologies in late-onset psychosis revealed by pet imaging

Speaker: Manabu Kubota, Japan

Co-Authors:
Shin Kurose
Kenji Tagai
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Hiroyuki Uchida
Hidehiko Tahakashi
Makoto Higuchi
Keisuke Takahata

Topic:
1) Psychoses

Abstract

Objective:
Late-onset psychosis (LOP) exhibits distinct clinical features compared with younger-onset psychosis. Although postmortem and epidemiological studies have suggested an association between LOP and neurodegenerative processes, particularly tauopathies, in vivo evidence remains limited. We aimed to investigate the involvement of Alzheimer's disease (AD) and non-AD tauopathies in LOP in vivo.

Methods:
Thirty-seven patients with LOP and 47 age-matched controls underwent PET scans with 11C-PiB and florzolotau (18F) (18F-florzolotau) to assess group differences in amyloid beta (Aβ) and tau accumulation. Diagnostic effects on regional 18F-florzolotau standardized uptake value ratios (SUVRs) were assessed. Associations between regional SUVRs and cognitive and clinical features were examined separately in Aβ-positive and Aβ-negative subgroups.

Results / Discussion:
Patients showed significantly higher Aβ and tau positivity rates than controls. A significant diagnostic effect was found on regional SUVRs (P = .004), with post-hoc analyses showing increased tracer retention in the parietal cortex. This effect remained robust when the analysis was restricted to Aβ-negative participants (P = .002). Among Aβ-positive patients, higher parietal tau burden was associated with lower Frontal Assessment Battery scores.

Conclusion:
This in vivo PET study revealed a high prevalence of AD and non-AD tau pathologies in LOP, indicating that LOP is associated with heterogeneous tau-related neurodegenerative processes.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-005
Unraveling schizophrenia endophenotypes: synergistic impacts of urokinase system polymorphisms, perinatal environmental exposures, and immunological polygenic risk score

Speaker: Vera Guven, Russia

Co-Authors:
Margarita Alfimova
Vera Golimbet
Ekaterina Semina
Yulia Chaika

Topic:
2) Schizophrenia

Abstract

Objective:
Schizophrenia is a highly heritable, polygenic disorder shaped by interactions between genetic vulnerability and environmental risk factors. Immune dysregulation and chronic inflammation are central to its pathophysiology. The urokinase system links neurodevelopment, synaptic plasticity, and immune activation. This study examines associations between PLAUR, PLAU and SERPINE1 variants, symptom severity, environmental stressors, and immune PRS.

Methods:
The sample comprised 522 schizophrenia spectrum patients (ICD-10 F20.x/F25.x) of European ancestry. Psychopathology was evaluated with PANSS, including negative symptom subdomains (Marder's model). Early-life risk factors were obtained from structured record review. SNPs were bioinformatically prioritized (eQTL/sQTL, MAF>5%, CADD≥10). Associations and gene-environment and gene-PRS interactions were tested using ANCOVA with FDR correction.

Results / Discussion:
Clinical characteristics were comparable across groups. PLAUR rs4760 showed significant main effects on PANSS negative and general symptom domains, while SERPINE1 variants were associated with overall psychopathology. Additive models indicated effects of obstetric complications on PANSS outcomes for 7 PLAUR SNPs. Post-hoc analyses of trend-level interactions (likely due to limited sample size) supported gene-environment-immune effects.

Conclusion:
Genetic variation in the urokinase system contributes to schizophrenia symptoms in a context-dependent manner. Preterm birth × PLAU/PLAUR risk alleles were associated with higher

positive symptoms, while PLAUR variants × immune PRS influenced negative symptoms. SERPINE1 SNPs × IL-6 PRS affected diminished expression, and multiple interactions impacted general psychopathology: together highlighting the relevance of the integrative approach.

FC-01
Schizophrenia and psychosis: biomarkers, imaging and clinical phenotypes

FC-01-006
Effectiveness of long-acting injectable second-generation antipsychotics in suicide attempt prevention among people with severe schizophrenia

Speaker: Juan J. Fernández-Miranda, Spain

Co-Authors:

Silvia Díaz-Fernández

Topic:
2) Schizophrenia

Abstract

Objective:
Long-acting injectable (LAI) antipsychotics are an effective treatment strategy to improve adherence, and they indirectly may decrease suicide behavior. The purpose of this study was to know the adherence to treatment of people with severe schizophrenia, the suicide attempts among them and the antipsychotic (AP) treatment characteristics (first, FGA vs. second, SGA; oral vs. LAI) related to compliance and suicidal behaviour.

Methods:
An 8-year prospective study of patients with severe schizophrenia (CGI-S scale ≥ 5) undergoing community-based, case managed treatment (N=200) was carried out. Assessment included the CGI-S and the WHO-DAS at the beginning and after three, 12, 24, 36 and 96 months. APs prescribed, laboratory tests, weight, adverse effects reported, hospital admissions and reasons for treatment discharge, including deaths by suicide, were recorded.

Results / Discussion:
After eight years 42% of patients continued treatment (CGI-S=4.1(0.9); $p<0.01$) and 37% were medical discharged (CGI=3.4(1.5); $p<0.001$); WHO-DAS decreased ($p<0.005$) in both groups; 10% of patients discontinued treatment. Twelve patients died, four of them by suicide. 60% of patients were treated with SGA-LAIs, with high tolerability. There were higher compliance ($p<0.01$) and fewer suicide attempts ($p<0.001$) in patients on LAIs.

Conclusion:
Adherence to treatment of patients with severe schizophrenia treated with second generation long-acting antipsychotics was high and seemed to be useful to decrease suicide behaviour compared with those on oral APs.

FC-02
Treatment-resistant psychosis and antipsychotic optimisation

FC-02-001
ENIGMA-TRS phase 3 program: rationale and study design towards a new therapeutic paradigm in treatment-resistant schizophrenia (TRS)

Speaker: Ravi Anand, Switzerland

Co-Authors:
Alessio Turolla
Giovanni Chinellato
Francesca Sansi
Richard Hartman

Topic:
2) Schizophrenia

Abstract

Objective:
Increasing evidence indicates that TRS has a distinct pathophysiology, characterized by glutamatergic rather than dopaminergic dysfunction (Moghaddam and Javitt, 2012). Unlike available antipsychotics (AP), which act via DA/5-HT receptor blockade, evenamide restores hippocampal glutamatergic activity (Uliana et al., 2025). This abstract presents key design features and challenges of the ENIGMA-TRS phase 3 program.

Methods:
ENIGMA-TRS comprises two international trials evaluating efficacy and safety of evenamide add-on:

- Study 023: 1-year, randomized, double-blind, placebo-controlled trial, with the primary efficacy endpoint at 12 weeks and long-term endpoints. It will enroll ~600 patients in Europe, Asia, Latin and North America

- Study 022: 12-week, randomized, double-blind, placebo-controlled trial, enrolling ~400 patients in Asia, Latin and North America

Results / Discussion:
To minimize inter-site variability in this global program, structured rater training was implemented to standardize assessments. Identification and selection of patients with TRS is based on TRRIP criteria (Howes et al., 2017), confirmed by an Independent Eligibility Committee, and supported by verification of therapeutic plasma concentrations of background antipsychotics. Both Study 023 and 022 are currently in the recruiting phase.

Conclusion:
Despite the complex design of ENIGMA-TRS and variability across >90 centers in 20 countries, robust mitigation strategies have been adopted to ensure a reliable program. Results from ENIGMA-TRS trials will confirm the benefits of evenamide - a glutamate modulator added on to AP - demonstrated in prior studies (Study 008A, Anand et al., 2025; Study 014/015, Anand et al., 2024), supporting a novel therapeutic paradigm for people living with TRS.

FC-02
Treatment-resistant psychosis and antipsychotic optimisation

FC-02-003
Glutamate modulation by evenamide is associated with clinically meaningful benefits in patients with treatment-resistant schizophrenia and inadequate response to antipsychotics: new clinical findings from post-hoc analyses

Speaker: Ravi Anand, Switzerland

Co-Authors:
Alessio Turolla
Giovanni Chinellato
Francesca Sansi
Richard Hartman

Topic:
2) Schizophrenia

Abstract

Objective:
Available antipsychotics showed efficacy on positive symptoms but failed to provide meaningful benefits on negative/cognitive ones. As the latter are mainly driven by hippocampal abnormalities, drugs acting at this site could tackle them. Evenamide was shown to normalize hippocampal hyperglutamatergic activity, reduce hyperdopaminergic state and reverse schizophrenia-like cognitive and social deficits in the MAM model (Uliana et al 2025).

Methods:
Post-hoc analyses of 2 trials of evenamide as add-on were conducted to evaluate its effect on social functioning and life engagement using:

- A MMRM linear regression model for Study 008A: a 4-week, randomized, double-blind, placebo-controlled trial in patients with inadequately controlled schizophrenia

- An OC analysis for Study 014/015: a 1-year, randomized, open-label, rater-blinded study in patients with treatment resistant schizophrenia

Results / Discussion:
In Study 008A, a statistically significant (p=0.012) drug-placebo difference was observed in the PANSS subdomain of social functioning, while a borderline significance (p=0.055) was observed in the life engagement subdomain. In Study 014/015, an increasing improvement was noted in social functioning and life-engagement domains reaching -4.4 points and -4.1 points from baseline at 1 year respectively.

Conclusion:
These findings in addition to the previously demonstrated benefits of evenamide on the PANSS total score, CGI-S/C, and responder rates (Anand et al. 2024 and 2025), strongly support the clinical meaningfulness of benefits achieved through glutamate modulation of hippocampal hyperglutamatergic activity. Uniquely, this mechanism of action can address both positive and negative symptoms, and its effects seem to increase and strengthen over time.

FC-02
Treatment-resistant psychosis and antipsychotic optimisation

FC-02-004
Clozapine in Indian Patients: Lower Dose Thresholds and Precision Strategies for Safer Treatment

Speaker: Satish Suhas, India

Co-Authors:
Rajkumar Sanahan
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Ganesan Venkatasubramanian
Pratima Murthy

Topic:
2) Schizophrenia

Abstract

Objective:
To synthesize evidence from India demonstrating (i) distinct pharmacokinetic profiles influencing clozapine exposure, (ii) the real-world impact of therapeutic drug monitoring (TDM), and (iii) clinical and demographic predictors of response. The goal is to propose an empirically grounded, India-specific framework for precision clozapine dosing.

Methods:
Four complementary datasets published from our center were examined: An exploratory pharmacokinetic study of 142 patients assessing concentration-to-dose ratios and dose required to reach 350 ng/mL serum clozapine. A retrospective cohorts comparing pre- and post-TDM outcomes in 90 inpatients and registry-based review of 250 individuals assessing response trajectories and predictors using elastic-net modelling.

Results / Discussion:
Across all datasets, Indian patients consistently displayed higher concentration-to-dose ratios compared with Western reference populations, with median doses of 120–150 mg/day sufficient to achieve a therapeutic serum level of 350 ng/mL. Ethnicity-driven differences remained robust after controlling for smoking status, BMI, and pharmacokinetic confounders.

Conclusion:
Taken together, these findings provide the strongest India-specific evidence to date that standardized clozapine dosing algorithms overestimate the dose requirement for South Asian

patients. Integrating TDM with ethnicity-informed dosing can improve safety, optimize tolerability, and potentially enhance adherence. This body of work supports the development of a “precision psychopharmacology” dosing framework tailored to Indian populations.

FC-02
Treatment-resistant psychosis and antipsychotic optimisation

FC-02-005
Do cultural factors and language influence efficacy outcomes? Results from Study 008A in Latin America compared to other countries

Speaker: Daniel Mosca, Argentina

Co-Authors:
Georgina Viczena
Eugenio Velasco
Ravi Anand

Topic:
2) Schizophrenia

Abstract

Objective:
There has been concern that results from CNS trials evaluating new molecules are mainly influenced by culture, background factors, and the language in which the trial population was assessed (Laughren 2020; FDA Stat Review; Zielen et al 2024). The results from evenamide add-on study 008A (Anand et al 2025), performed in multiple countries in patients who are inadequate responders to SGA, provide an opportunity to assess the results by region.

Methods:
Study 008A was a phase 3, 4-week, randomized, double-blind, placebo-controlled trial evaluating the efficacy and safety of evenamide 30 mg bid add-on in patients with schizophrenia poorly responding to SGA (incl. clozapine) conducted in 11 countries (Asia, Latin America, Europe). Plasma levels assessed patients' adherence to the background AP. Differences of the Latin American population compared to other regions are described.

Results / Discussion:
Differences were noted between the placebo groups in Latam and other regions including older age and higher proportion of patients living with family. The baseline PANSS score did not differ between Latam and RoW; a statistically significant greater CGI-S was noted in Latam. Statistically significant improvements were noted on the PANSS Total (greater drug-placebo difference in Latam compared to RoW), Positive and General Psychopathology.

Conclusion:
These data suggest that the use of a common language (Latam=1, RoW=17), similar cultural background, and clinical practice logistics (higher recruitment density per site (Kantrowitz et al 2025)), contributed to achieving a higher drug-placebo difference compared to the overall Study 008A data. Additional results and explanations will be presented at the Congress.

FC-02
Treatment-resistant psychosis and antipsychotic optimisation

FC-02-006
Results of Study 008A with evenamide add-on to second-generation antipsychotics in India: influence of cultural, religious, and common background factors on efficacy

Speaker: Pradeep Thilakan, India

Co-Authors:
Vikhram Ramasubramanian
Radhika Reddy
Ravi Anand

Topic:
2) Schizophrenia

Abstract

Objective:
It is believed that AP trials performed in India provide a greater signal of efficacy likely due to background factors, religious biases, caregiver structure, and overrating bias (Kalra et al 2012). The evenamide add-on study 008A (Anand et al 2025), performed in many countries including India in patients who are inadequate responders to SGA, provide an opportunity to evaluate the influence of cultural background on results.

Methods:
Study 008A was a phase 3, 4-week, randomized, double-blind, placebo-controlled trial evaluating the efficacy and safety of evenamide 30 mg bid as add-on in patients with schizophrenia non responding to SGA (incl. clozapine) conducted in 11 countries (Asia, Latin America, Europe). Plasma levels assessed patients' adherence to the background AP. Differences in background characteristics and efficacy between India and other countries are presented.

Results / Discussion:
Indian patients were younger with 10% more males enrolled and a higher proportion living with family. The duration of illness was significantly shorter in India; accordingly, patients had fewer prior psychiatric hospitalizations. A significant higher proportion of patients was taking risperidone in India. Despite the lower baseline PANSS and CGI-S scores, a statistically significant benefit was noted on the PANSS Total, PANSS Positive, and CGI-S.

Conclusion:
These results point out that the effect of evenamide on the PANSS Total score is statistically significant in India despite the study being performed in 9 languages, and the magnitude of effect observed in India does not differ compared to other countries (-3.0 vs -2.7). More details and considerations will be presented at the Congress.

FC-03
Neuromodulation and brain stimulation across disorders

FC-03-001
Effect of Adjunctive High-Definition Transcranial Direct Current Stimulation in Treatment of OCD- Study Protocol of a Double-Blinded Randomized Sham-Controlled Trial

Speaker: Aditya Somani, India

Co-Authors:
Sri Jyothi Myneni
P. Lakshmi Nirisha

Topic:
10) Other

Abstract

Objective:
PRIMARY: To compare the effect of adjunctive High-Definition Transcranial Direct Current Stimulation (HD-tDCS) on left pre-supplementary motor area (pre-SMA) with sham HD-tDCS on symptoms of OCD, immediately and after 4 weeks of last session.

SECONDARY: To evaluate the impact of HD-tDCS on patients' depression, and anxiety symptoms immediately and after 4 weeks of sessions.

Methods:
This study shall include 50 consenting patients with OCD, aged 18–50 years who score 16 or above on the Yale Brown Obsessive Compulsive Scale (Y-BOCS). For HD-tDCS, the anode shall be placed at center, at left pre-SMA with four surrounding return cathodal electrodes. They shall continue to receive treatment-as-usual. They shall be assessed at baseline, post-intervention, and at four-weeks' follow-up.

Results / Discussion:
The trial has been started after approval by Ethics Committee and registration at Clinical Trials Registry of India. The findings among the two groups shall be compared using repeated measure analysis of variance and employ intention-to-treat method. Additionally, a per-protocol analysis shall also be carried out for those who complete the study protocol.

Conclusion:
HD-tDCS offers a targeted, non-invasive method for modulating neural circuits implicated in OCD, particularly the cortico-striato-thalamo-cortical (CSTC) loop. The results of this study could help explore utility of HD-tDCS in treatment of OCD.

FC-03
Neuromodulation and brain stimulation across disorders

FC-03-002
rTMS in Catatonia: A proof of concept study

Speaker: Anindya Das, India

Co-Authors:

Anand Kumar

Topic:
10) Other

Abstract

Objective:
Neurobiological studies (mechanistic theories) implicate the overstimulation of the supplementary motor cortex (SMA) in catatonia. We use this understanding to treat catatonia with inhibitory theta-burst rTMS stimulation to the SMA.

Methods:
In a series of consecutively enrolled cases of catatonia, medically stable, and suggesting nil neurological or systemic cause, naive to established treatment (lorazepam/ ECT), were administered inhibitory theta burst rTMS stimulation to the SMA. Participants were evaluated with Bush-Francis Catatonia Rating Scale (BFCRS) at baseline and then at repeated intervals for the next six hours. Per-post BFCRS Scores are compared for inference.

Results / Discussion:
A total of ten cases were screened into the study. Salient improvement was observed in some cases, while those who did not improve also had poor response to conventional treatment (lorazepam/ECT).

Conclusion:
The potential of rTMS for the treatment of catatonia seems possible.

FC-03
Neuromodulation and brain stimulation across disorders

FC-03-003
World federation of societies of biological psychiatry (wfsbp) guidelines on neuromodulation, neurofeedback and neurosurgical treatment in eating disorders: findings from preliminary analysis of the literature

Speaker: Davide Gravina, Italy

Co-Authors:
Hubertus Himmerich
Ulrike Schmidt
Claudia Carmassi
Nazife Gamze Usta Sağlam
Colleen Loo
Iain Campbell
Andres Lozano
Andreas Karwautz
Siegfried Kasper
Janet Treasure

Topic:
10) Other

Abstract

Objective:
Non-invasive brain stimulation (NIBS) and other brain-directed interventions have been proposed as adjuncts to standard treatments for eating disorders (EDs). Despite growing evidence, international guidelines with standardized protocols are lacking. This World Federation of Societies of Biological Psychiatry (WFSBP) project aims to develop the first guidelines on neuromodulation, neurofeedback, and neurosurgery treatments for EDs.

Methods:
A systematic search of PubMed, Web of Science, and APA PsycInfo is underway to identify clinical studies. Randomized controlled trials (RCTs) are prioritized to ensure the higher level of evidence. Study quality will be assessed using the Scottish Intercollegiate Guidelines Network (SIGN) tool. Levels of Evidence (LoE) and Grades of Recommendation (GoR) will be assigned according to the WFSBP grading system (Hasan et al., 2019).

Results / Discussion:
Preliminary analysis of the literature revealed promising evidence. tDCS and rTMS show encouraging effects on cravings and binge eating in bulimia nervosa and binge eating disorder, and modest benefits on body weight and comorbid psychopathology in anorexia nervosa, with a

good safety profile. Invasive interventions such as DBS show positive effects on body mass index in treatment-refractory anorexia nervosa but a higher risk of side effects.

Conclusion:
Although formal SIGN ratings, LoE, and GoR are pending, preliminary evidence supports nuanced, diagnosis-specific recommendations for these interventions in EDs.

FC-03
Neuromodulation and brain stimulation across disorders

FC-03-004
rTMS Targeting the Precuneus in Continuous Schizophrenia with Persistent Positive and Negative Symptoms: A Case Report

Speaker: Anaf Kololichalil, India

Co-Authors:
Shaurya Garg
Nand Kumar
Gagan Hans

Topic:
2) Schizophrenia

Abstract

Objective:
rTMS in schizophrenia has mainly targeted the dorsolateral prefrontal cortex and temporoparietal regions. Neuroimaging increasingly implicates the precuneus, a key default mode network hub, in abnormal self-referential processing, delusions, and network dysconnectivity. This report describes clinical effects of high-frequency precuneus rTMS in continuous schizophrenia with partial treatment response.

Methods:
A 34-year-old male with ICD-11 continuous schizophrenia and comorbid cannabis and nicotine dependence (8-year duration) presented with severe persecutory delusions, aggression, and negative symptoms. After partial response to optimized haloperidol and LAI initiation, precuneus-targeted rTMS was given (20 Hz; 1600 pulses/session; twice daily ×16 sessions). Outcomes were assessed using BPRS.

Results / Discussion:
Following 16 rTMS sessions, marked clinical improvement was observed. BPRS scores reduced from 40 to 22. Clinically, reductions were noted in persecutory ideation, hostility, and negative symptoms, with improved engagement and affective responsiveness. No significant adverse events were reported.

Conclusion:
rTMS may be a promising network-based approach in schizophrenia by modulating default mode network dysfunction. This case suggests benefits of targeting connectivity hubs rather than conventional cortical sites. Controlled studies are needed to confirm efficacy, optimize parameters, and clarify underlying network mechanisms.

FC-03
Neuromodulation and brain stimulation across disorders

FC-03-005
Neuromodulatory effects of adjuvant itbs on eeg mu wave suppression in response to observed biological motion in schizophrenia patients with deficits in empathy

Speaker: Supratik Kundu, India

Co-Authors:
Sanjay Munda
Nishant Goyal

Topic:
2) Schizophrenia

Abstract

Objective:
To study the neuromodulatory effects of iTBS on EEG mu suppression and Interpersonal Reactivity Index scores in response to observed biological motion in schizophrenia patients with deficits in empathy.

Methods:
Inpatients were randomly assigned to receive either active or sham iTBS (blinded) for twenty sessions over two weeks. Active iTBS consisted of bursts of 3 pulses at 50 Hz repeated at 5 Hz (80% of resting motor threshold), delivered in 2-second trains every 10 seconds (990 pulses/session). EEG mu-wave suppression (8–13 Hz), Interpersonal Reactivity Index (IRI) and TMSens_Q were assessed pre- and post-intervention.

Results / Discussion:
In the third-person video task, active iTBS significantly increased EEG mu-wave suppression for Clip 1 ($p = 0.002$), whereas the sham group showed no significant change ($p = 0.179$). Active iTBS also produced a significant increase on the IRI Fantasy subscale ($p = 0.044$).

Conclusion:
Active iTBS enhanced mu-wave suppression and increased Fantasy subscale scores, suggesting possible improvements in cognitive aspects of empathy. Although the vmPFC is typically implicated in emotional empathy, these effects may reflect its integrative role in linking emotional and cognitive components of empathic processing. These preliminary findings warrant replication in larger samples to confirm efficacy and clarify underlying mechanisms.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-001
Somatic complaints, neuropsychiatric manifestations and radiological correlates

Speaker: Shivani Mankar, India

Co-Authors:
Lehak Khanna
Sanjiv Kale

Topic:
10) Other

Abstract

Objective:
Non-specific symptoms frequently present in clinical practice and are often presumed to be functional. This study aimed to correlate the radiological findings with neuropsychiatric symptoms in patients having underlying organic pathology—specifically, neurovascular compression by vascular loops involving cranial nerves, thus highlighting the importance of an integrated neuropsychiatric diagnostic approach.

Methods:
This observational case series was conducted at a tertiary care hospital and included patients referred from Neurology after MRI brain for EEG and psychiatric evaluation, as well as presenting directly to Psychiatry OPD. Complaints included headache, giddiness, irritability, blurring of vision and other non-specific symptoms. MRI findings were reviewed for vascular loops in relation to cranial nerves, their location, and classification.

Results / Discussion:
MRI brain imaging revealed vascular loops : Type I, II or III abutting the seventh and/or eighth cranial nerves or extending into the internal auditory canal. These findings could provide a plausible biological basis for neuropsychiatric and vestibular symptoms. The symptoms earlier considered functional or primary psychiatric were reinterpreted for identifiable organic correlates, leading to changes in diagnosis and management.

Conclusion:
Neurovascular compression syndromes may present with prominent neuropsychiatric symptoms and can be overlooked if organic causes are not actively considered. This case series highlights the role of neuroimaging and consultation-liaison psychiatry in improving diagnostic accuracy, and enabling more rational treatment planning, reinforcing psychiatry within a neurobiological framework.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-002
When psychiatry masks neurology: case series of anti-nmdar encephalitis

Speaker: Arundhati Bhagabati, India

Co-Authors:
Sunil Mittal
Devanshika Misra

Topic:
10) Other

Abstract

Objective:
Anti NMDA Receptor encephalitis is an autoimmune encephalitis with rapid onset and neuropsychiatric symptoms, including behavioural disturbances, insomnia, seizures, facial dyskinesia and autonomic dysfunction.

This presentation will highlight a case series of 4 patients with varied clinical presentation with the aim of recognizing the need for early identification of NMDAr encephalitis which would prevent debilitating sequelae.

Methods:
Case study

Results / Discussion:
NMDA encephalitis can present to a psychiatric setup with symptoms simulating acute psychosis and mood disorders. early identification can help mitigating fatal consequences.

Conclusion:
Patients of acute psychosis in the young adulthood need to be evaluated for neurological signs so as not to overlook the possibility of an evolving encephalitis so that appropriate treatment can be initiated at the earliest.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-003
Structural MRI–Guided Neuronavigation Versus Scalp-Based Targeting for Left DLPFC rTMS in Treatment-Resistant Depression: A Systematic Review and Meta-Analysis

Speaker: Sonakshi Jyrwa, India

Co-Authors:

Varidh Katiyar

Topic:
10) Other

Abstract

Objective:
To compare the clinical efficacy of MRI-guided neuronavigation with conventional scalp-based targeting (5-cm rule, F3, Beam F3) for left DLPFC rTMS in adults with Treatment-resistant depression (TRD). Primary outcomes were changes in MADRS or HAMD scores. Secondary objectives included comparison of response and remission rates.

Methods:
A systematic review and meta-analysis was conducted following PRISMA guidelines. PROSPERO Registration: CRD420261308482. Databases were searched for studies comparing MRI-guided neuronavigation with conventional scalp-based targeting for left DLPFC rTMS in TRD. Randomized and comparative studies were included. Data were extracted independently, risk of bias was assessed, and pooled effect sizes were calculated using a random-effects model.

Results / Discussion:
Two randomized trials compared neuronavigation-guided rTMS with the conventional 5-cm rule. MADRS reduction could not be pooled due to incomplete variance. Response rates (RR 1.32, 95% CI 0.54–3.24; I^2=64%) and Remission rates (p = 0.56, I^2 = 59%) showed no significant difference. Variations in stimulation parameters and study design indicate clinical heterogeneity, affecting pooled estimates.

Conclusion:
The pooled randomised evidence did not demonstrate statistically significant difference between neuronavigation-guided targeting over the conventional 5-cm method for response or remission outcomes in TRD. However, interpretation is limited by small sample sizes, methodological variability and moderate heterogeneity. Larger, harmonised, head-to-head trials are needed to establish whether precision targeting yields meaningful clinical benefit.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-004
A study on the severity of delirium in an intensive care unit of a tertiary care hospital

Speaker: Navoneela Bardhan, India

Co-Authors:

Ashwin Mandan

Topic:
10) Other

Abstract

Objective:
1. To assess the severity of delirium in these patients.

2. To determine the possible factors associated with the development of delirium.

Methods:
Patients admitted in the ICU of a tertiary care hospital (above 18 years) were included in this cross sectional study. Sociodemographic details, data related to medical and surgical history, substance use pattern, any pre existing psychiatric comorbidities and their investigation findings were entered into a semi structured proforma. The Confusion Assessment Method (CAM) ICU-7 scale was administered to assess the severity of delirium.

Results / Discussion:
This is an ongoing study. Among the 18 patients with delirium in the ICU, 38.89% had severe delirium while 61.11% had mild to moderate delirium according to the CAM ICU-7 rating scale. 4 (22.22%) had an infective etiology, 4 (22.22%) had alcohol withdrawal and 3 (1/6th) patients had metabolic eitiology. 22.22% patients had deranged electrolytes, 27.78% had leucocytosis, 27.78% had deranged renal profile and 11.11% had imaging findings.

Conclusion:
This study highlights that more than one third of delirium in ICU is severe, with various underlying etiological factors. These patients also had various deranged biological parameters. The identification of delirium in ICU settings with a detailed evaluation of these associated features simultaneously could go hand in hand to not only detect delirium early but also in better management of the underlying factors.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-005
Persistent neuroinflammation in Long COVID patients demonstrated by MAO-B PET and blood biomarkers

Speaker: Keisuke Takahata, Japan

Co-Authors:
Sho Moriguchi
Shin Kurose
Chie Seki
Masanori Ichihashi
Hiroyuki Uchida
Takahiko Tokuda
Masaki Takao
Koichi Fukunaga
Masaru Mimura
Makoto Higuchi

Topic:
10) Other

Abstract

Objective:
Long COVID is associated with chronic neuropsychiatric symptoms, including cognitive impairment, fatigue and depression. Although persistent neuroinflammation has been proposed as a central mechanism, in vivo evidence in humans remains scarce. This study aimed to evaluate astrocyte-mediated neuroinflammation in Long COVID patients using monoamine oxidase-B (MAO-B) PET and to examine its relationship with peripheral inflammatory biomarkers.

Methods:
Twenty patients with Long COVID (mean age 43.0 years; mean interval since infection 403 days) and 17 healthy controls (mean age 37.0 years) underwent 11C-SL25.1188 PET with arterial blood sampling and MRI scanning. Regional MAO-B binding was quantified using a two-tissue compartment model to derive total distribution volume (VT). Plasma biomarkers, including TNF-α, IL-6, GFAP, NfL were measured using single molecule array (SIMOA) assays.

Results / Discussion:
Compared with healthy controls, Long COVID patients showed significantly increased 11C-SL25.1188 VT values in the cingulate cortex, striatum, thalamus, brainstem, hippocampus, amygdala, and white matter ($p<0.05$, corrected). Regional 11C-SL25.1188 VT demonstrated

positive correlations with plasma TNF-α levels, most prominently in the midbrain (r=0.83), white matter (r=0.74), hippocampus (r=0.70), and amygdala (r=0.69; all p<0.001).

Conclusion:
Our results provide robust in vivo evidence of persistent neuroinflammation in Long COVID. MAO-B PET imaging, in conjunction with peripheral inflammatory biomarkers, represents a promising translational approach for characterizing chronic brain inflammation and its contribution to long-lasting neuropsychiatric symptoms in Long COVID.

FC-04
Neuropsychiatry, delirium and neurology–psychiatry interfaces

FC-04-006
A composite biological score using CBC-derived inflammatory indices and serum vitamin D serves as a low-cost objective tool to support suicide risk assessment in first-episode drug-naive psychiatric patients

Speaker: Shaifali Mahar, India

Topic:
10) Other

Abstract

Objective:
To compare NLR, PLR, MLR, SII and SIRI between first-episode drug-naive psychiatric patients with a recent suicide attempt (Group A) and those without any history of suicide attempt (Group B), diagnosed as per ICD-10; to compare serum Vitamin D levels between the groups; and to develop a composite biological risk score for suicide attempt.

Methods:
An observational study at the Department of Psychiatry, SMS Medical College, Jaipur. After informed consent and eligibility screening, fasting blood samples are collected for CBC with differential and serum Vitamin D. NLR, PLR, MLR, SII and SIRI are derived from CBC. HAM-D-17 and C-SSRS are administered, and findings are statistically compared between the two groups.

Results / Discussion:
Elevation in NR, SII and SIRI is seen in Group A (suicide attempt) compared to Group B (no attempt, with lower serum Vitamin D levels in Group A. The composite biological score outperforms individual markers in predicting suicide attempt.

Conclusion:
A composite biological score using CBC-derived inflammatory indices and serum Vitamin D serves as a low-cost objective tool to support suicide risk assessment in first-episode drug-naive psychiatric patients.

FC-05
Neurodevelopment and neural markers

FC-05-001
Insular cortex serves as a critical hub for promoting and maintaining social fear generalization

Speaker: Qing Han, People's Republic of China

Co-Authors:
Kai Yuan
Xiaoxing Liu
Lin Lu

Topic:
10) Other

Abstract

Objective:
To survive, animals must efficiently identify and avoid potential threats while minimizing the costs associated with unnecessary avoidance. Adaptive fear generalization allows animals rely on prior experience to assess risks when encountering novel stimuli, while excessive fear generalization is associated with various psychiatric disorders. Here, we aim to explore how do animals calibrate their fear generalization to an adaptive level.

Methods:
We use social defeat and the three-chamber social paradigm. Mice that experience acute social defeat avoiding novel ICR are defined as adaptive fear generalization, and those avoiding novel C57 are defined as overgeneralization. Whole-brain immunolabeling and clearing techniques (iDISCO+), immunofluorescence, calcium imaging, electrophysiology, multiple viral strategies and other molecular biological methods are employed for mechanism research.

Results / Discussion:
Acute social defeat leads defeated mice to avoid novel ICR, while chronic social defeat causes defeated mice to avoid novel C57 and ICR more than an empty cage. One day after ASDS, interaction with C57 and ICR activates the insular cortex (Insctx), whereas CSDS suppresses the activity of these neurons. Lesion or inhibition of Insctx slow down adaptive fear generalization in mice, while inhibit Insctx during CSDS promotes overgeneralization.

Conclusion:
Insular cortex activation mediates adaptive fear generalization, while its inactivation promotes overgeneralization.

FC-05
Neurodevelopment and neural markers

FC-05-002
Neural Correlates of Social–Cognitive Change from a Brief Integrated Schema–Mindfulness Intervention in Adolescents with Internalizing Difficulties: An ERP Study

Speaker: Debaleena Ghosh, India

Co-Authors:
Masroor Jahan
Avinash Sharma

Topic:
10) Other

Abstract

Objective:
The study aimed to examine the associated changes in neural processing of socio emotional stimuli in adolescents exposed to a brief integrated schema–mindfulness intervention. Specifically, the study investigated modulation of early perceptual (N170) and later attentional–evaluative (P300) event-related potentials during an emotional face recognition task, alongside changes in social cognition and internalizing symptoms.

Methods:
Forty adolescents aged 12–17 years presenting with internalizing behavioural problems were allocated to an intervention group (n = 20) or waitlist-control group (n = 20). The intervention comprised 12 sessions. Participants completed a computerized emotional face recognition task during EEG recording at baseline and post-intervention. N170 and P300 amplitudes were analysed.

Results / Discussion:
The intervention group demonstrated significant reductions in anxiety and depressive symptoms, improvement in social cognition relative to the waitlist-control group. ERP analyses indicated modulation of emotional processing. Following intervention, adolescents in the intervention group showed enhanced N170 responses to emotional faces and increased P300 amplitudes, suggesting improved perceptual sensitivity and attentional engagement.

Conclusion:
This study examined neurophysiological modulation of socio-emotional processing following a brief integrated schema–mindfulness intervention in adolescents, indexed by N170 and P300 responses during an emotional face recognition task, alongside changes in social cognition and internalizing symptoms.

FC-05
Neurodevelopment and neural markers

FC-05-003
From neuron to mind

Speaker: Sakshi Doshi, India

Co-Authors:
Neha Garg
Soumya Kamath
Sanjiv Kale

Topic:
10) Other

Abstract

Objective:
To highlight the importance of Sr Na^+ and NH_3 as biochemical indicators for the early detection of neuropsychiatric manifestations in critically ill patients referred for consultation-liaison psychiatry.

Methods:
This observational study was conducted in a tertiary care hospital. Patients referred to consultation-liaison psychiatry were screened for Sr. Na+ and Sr. NH3. Informed consent was taken from the caregivers of patients who satisfied the inclusion criteria. Ethics committee approval taken. The behaviour change was observed and rated.

Results / Discussion:
66.2% of ICU patients with delirium had hyponatremia and 33.7% had hyperammonemia. Hyponatremia presented with nausea/vomiting, headache, irritability, disorientation and behavioural changes; drugs were the commonest cause. Mean Sr. Na^+ was 120 mEq/L and NH_3 58.2 µg/dL. Findings were more common in males and elderly. Mean MDAS score was 20, with average hospital stay of 8 days. Early detection reduced hospital stay in 62.9%.

Conclusion:
Deranged Sr.Na and NH_3 are simple, objective markers of brain dysfunction in critical patients. Combining with clinical assessment, would enable early recognition of neuropsychiatric manifestations, when history is limited. These abnormalities precede overt neurological symptoms. Incorporating Na and NH_3 assessment into consultation-liaison psychiatry facilitates early detection, timely intervention, improves outcomes, and reduces hospital stay.

FC-05
Neurodevelopment and neural markers

FC-05-004
Predicting methylphenidate response in pediatric ADHD using qEEG and deep learning

Speaker: Merlin Mathew, India

Co-Authors:
Bichitra N Patra
Rajesh Sagar
Rohit Verma
Rachna Bhargava
Simran Kaur

Topic:
10) Other

Abstract

Objective:
To develop and evaluate a convolutional neural network (CNN) model using raw resting-state EEG data to predict treatment response.

To assess the predictive performance of the CNN model using accuracy, sensitivity, specificity, F1-score, and area under the ROC curve (AUC).

Methods:
Forty children (8–16 years) with ADHD underwent resting-state 32-channel EEG before starting methylphenidate. Responders were defined by ≥25% reduction in Conners' Parent Rating Scale scores at 4 weeks. Raw EEG data was used to train a time-series CNN, with 80% for training and 20% for testing.

Results / Discussion:
The CNN successfully differentiated responders from non-responders with an overall classification accuracy of 77.719% (AUC- 0.8889). The F1- score was 0.7347 for the responders' group and 0.800 for non-responders.

Conclusion:
Application of deep learning models such as CNNs may advance the development of individualized, EEG guided treatment strategies, ultimately improving therapeutic outcomes. Larger multi-centre studies are needed to elucidate the neural mechanisms underlying ADHD and it's response to treatment.

FC-05
Neurodevelopment and neural markers

FC-05-005
Assessment Of Nlr, Plr, Mlr, And Mpv As Inflammatory Markers In Children With Attention-Deficit/Hyperactivity Disorder

Speaker: Avdhesh Upadhyay, India

Abstract

Objective:
To evaluate systemic inflammatory markers—neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and mean platelet volume (MPV)—in drug-naive children aged 6–16 years diagnosed with ADHD according to DSM-5 criteria, and to compare these markers with age-, sex-, and BMI-matched healthy controls.

Methods:
This observational case-control study included 82 drug-naive children aged 6–16 years diagnosed with ADHD and 70 age-, sex-, and BMI-matched healthy controls. Complete blood count parameters were obtained from blood samples, and NLR, PLR, MLR, and MPV were calculated and compared between groups using appropriate statistical analyses.

Results / Discussion:
Children with ADHD showed significantly higher NLR, PLR, MLR, MPV, and neutrophil counts, along with significantly lower lymphocyte counts, compared to healthy controls. However, no significant association was found between these inflammatory markers and ADHD subtypes or symptom severity.

Conclusion:
The findings support the role of low-grade systemic inflammation in the pathophysiology of childhood ADHD. NLR, PLR, MLR, and MPV may serve as simple, inexpensive, and readily available inflammatory markers, although further large-scale longitudinal studies are needed to confirm their clinical utility.

FC-06
Trauma, Depression, biological ageing and women's mental health

FC-06-001
Female-Specific Genotype Associations with Suicidal Behavior: Insights from a Sex-Stratified Genetic Analysis

Speaker: Ruchika Kaushik, India

Co-Authors:

Chittaranjan Behera

Topic:
5) Women's mental health

Abstract

Objective:
Sex differences in suicidal behavior are well established, yet most genetic studies rely on combined-sex analyses, potentially obscuring female-specific biological risk factors. Building upon our previously reported combined analysis, the present study aimed to investigate female-specific genotype associations with suicidal behavior and related clinical and behavioral traits, with a focus on inflammatory pathway genes.

Methods:
This study represents a sex-stratified secondary analysis conducted within a larger case–control cohort from the Indian population. Female suicide cases and female non suicidal controls were included. Genotyping was performed for 5 cytokine gene at 7 single nucleotide polymorphisms previously implicated in suicidal behavior. Standardized psychological autopsy methods were employed to reconstruct clinical, behavioral patterns, and life stressors.

Results / Discussion:
Variants within inflammatory pathway genes demonstrated differential risk patterns among females, particularly in relation to behavioral correlates of suicidality. IL6 (-174C/T) genotypes showed stronger associations with suicide-related traits in females, suggesting a unique biological vulnerability profile. These findings highlight that pooling males and females may mask meaningful genetic signals relevant to women.

Conclusion:
This female-oriented molecular genetic analysis highlights sex-dependent neuroinflammatory mechanisms underlying suicidal behavior. Identification of female-specific genetic signatures emphasizes the necessity of sex-stratified designs in psychiatric genomics and supports the development of sex-informed molecular biomarkers for suicide risk.

FC-06
Trauma, Depression, biological ageing and women's mental health

FC-06-002
Biological aging markers in South African women with HIV and variable exposure to childhood trauma

Speaker: Kabelo Maloka, South Africa

Co-Authors:
Georgina Spies
Stefan Du Plessis
Sian Megan Joanna Hemmings
Soraya Seedat
Jacqueline Samantha Womersley

Topic:
5) Women's mental health

Abstract

Objective:
Biological aging measures are influenced by HIV and childhood trauma (CT). The extent to which these measures reflect shared or distinct processes, especially in individuals exposed to both HIV and CT, remains unclear. This study investigated intercorrelations among biological aging markers and the HIV/CT effects on these relationships.

Methods:
We included 199 women with (n = 103) and without HIV (n = 96). Partial correlations adjusted for age examined pairwise associations among epigenetic age acceleration measures (Horvath, Hannum, PhenoAge, and intrinsic and extrinsic epigenetic age acceleration) and residual brain-predicted age difference (BPAD). Linear regression models tested HIV/CT moderation effects, with false discovery rate (FDR) correction.

Results / Discussion:
Epigenetic age acceleration measures were strongly intercorrelated ($p < 0.001$) but were not correlated with residual BPAD. Higher CT scores were observed in women with HIV compared to controls. Nominal HIV-CT moderation effects did not survive FDR correction.

Conclusion:
Epigenetic clocks showed substantial overlap, but residual BPAD appears independent. Although HIV and CT showed nominal moderation effects, these were not robust after correction. Findings suggest that different biological aging measures may capture different aspects of biological aging and highlight the importance of trauma-focused approaches in aging research among women with HIV.

FC-06
Trauma, Depression, biological ageing and women's mental health

FC-06-003
Genome-Wide DNA Methylation Analysis Reveals Epigenetic Signatures of Early Life Stress in Major Depressive Disorder Patients

Speaker: Yogesh Dwivedi, USA

Co-Authors:
Aleena Francis
Richard Shelton

Topic:
7) Mood disorders

Abstract

Objective:
Early life stress (ELS) is a well-established risk factor for developing depression later in life. Understanding the specific mechanisms by which ELS increases vulnerability to depression is a crucial area of research. This study examined genome-wide DNA methylation profiles in individuals with major depressive disorder (MDD), stratified by ELS exposure, and included trauma-matched control groups.

Methods:
Subjects comprised of MDD with ELS (n=30), MDD without ELS (n=30), and non-psychiatric controls with and without ELS (n=30). ELS was evaluated using the Childhood Trauma Questionnaire. Genome-wide methylation analysis was performed using reduced representation bisulfite sequencing. To explore the functional relevance of genes associated with DMRs, the GENE2FUNC module of the Functional Mapping and Annotation platform was used.

Results / Discussion:
We identified 128 DMRs in MDD versus controls, 29 in MDD+ELS versus control+ELS, and 13 in MDD without ELS versus controls without ELS. Notable genes with DMRs in MDD+ELS included LONRF1, MAPK8IP1, SELENOO, PDXP, and CNNM3. Functional enrichment revealed pathways involving MAPK, synaptic vesicles, and JAK-STAT signaling. Conversely, DMRs in MDD without ELS involved genes such as BCL7B, BLCAP, and NNAT.

Conclusion:
This study provides compelling evidence of distinct DNA methylation signatures associated with ELS-induced MDD and MDD without ELS exposure, highlighting the complex interplay between genetic and environmental factors in the etiology of this complex disorder.

FC-06
Trauma, Depression, biological ageing and women's mental health

FC-06-004
Association between biological aging measures and neurocognitive and depressive symptoms in women with HIV and childhood trauma

Speaker: Kabelo Maloka, South Africa

Co-Authors:
Georgina Spies
Stefan Du Plessis
Sian Megan Joanna Hemmings
Soraya Seedat
Jacqueline Samantha Womersley

Topic:
7) Mood disorders

Abstract

Objective:
Neurocognitive impairment and depression are common among women with HIV and exposure to childhood trauma (CT). Biological aging markers may index vulnerability, but their links to mental health are unclear. We examined cross sectional associations between biological aging measures and global cognitive deficit scores (GDS) and depressive symptom scores (CES-D), and tested moderation by HIV and CT.

Methods:
We included 199 women with (n = 103) and without HIV (n = 96). Linear regression models evaluated associations between residual brain-predicted age difference and epigenetic age accelerations (Horvath, Hannum, PhenoAge, and intrinsic and extrinsic epigenetic age acceleration) with GDS and CES D, adjusting for age and education. HIV and CT main and interaction moderation effects were tested with false discovery rate (FDR) correction.

Results / Discussion:
Biological aging markers were not significantly associated with GDS or CES D after FDR correction. Although CT exposure was higher in women with HIV than in controls, neither HIV nor CT showed significant moderation effects.

Conclusion:
Overall, biological aging measures were not robustly linked to neurocognitive or depressive symptom scores. Larger studies are needed to clarify whether HIV and CT shape aging-related vulnerability to mental health outcomes in women.

FC-06
Trauma, Depression, biological ageing and women's mental health

FC-06-005
Spatiotemporal and Cross-Species Multi-Omics Profiling Reveals Potential Biomarkers for Depression

Speaker: Xiaoping Wang, People's Republic of China

Co-Authors:

Mingxue Gao

Topic:
7) Mood disorders

Abstract

Objective:
Major depressive disorder is increasingly linked to gut-brain axis dysfunction, yet a comprehensive multi-omics characterization integrating microbial, metabolic, and inflammatory dimensions remains lacking.The objective of this study is to provide a comprehensive multi-omics characterization of major depressive disorder by integrating gut microbial profiling, metabolomic analysis of short-chain fatty acids, and inflammatory cytokine assessment.

Methods:
This study employed a combined cross-sectional and longitudinal design in both MDD patients and healthy controls, integrating gut microbiome profiling, short-chain fatty acid quantification, inflammatory cytokine assessment, and pathway analysis, with some patients receiving SSRI intervention. The clinical findings were subsequently validated in a chronic unpredictable mild stress rat model through SCFA supplementation.

Results / Discussion:
MDD patients showed reduced gut microbial diversity and function, elevated pro-inflammatory markers, and decreased IL-10 and SCFAs (acetate, propionate, butyrate). Antidepressant treatment partially restored IL-10 and butyrate. A validated biomarker panel accurately distinguished MDD from controls. Animal models confirmed conserved disruptions in microbiota, SCFAs, and inflammation, reversible by SCFA supplementation.

Conclusion:
This integrative study establishes a systemic multi-omics signature of depression, underscores the role of microbial-derived metabolites in driving neuroinflammation, and identifies targetable pathways for future microbiota-based interventions.

FC-07
Digital Psychiatry, Novel Interventions and Emerging Mental Health

FC-07-001
Fear of Missing Out Among Children, Adolescents, and Young Adults: A Cross-Sectional Study

Speaker: Mayank Gupta, USA

Co-Authors:
Shreya Jain
Ridhimaa Chaudhary
Priyal Khurana
Nihit Gupta

Topic:
6) Addiction, 10) Other

Abstract

Objective:
FoMO, the persistent apprehension that others are engaged in more rewarding experiences, has emerged as a clinically significant construct of the digital age. Amplified by social media's continuous visibility of peers' activities, FoMO is associated with anxiety, depression, disrupted sleep, and compulsive social media use. We aimed to validate the FoMOS in India and characterise its sociodemographic correlates

Methods:
We conducted a cross-sectional online survey of 824 individuals aged 10 years or older residing in India. Internal consistency was assessed with Cronbach's α and corrected item–total correlations. Exploratory factor analysis (EFA) using principal axis factoring and confirmatory factor analysis (CFA) using maximum likelihood estimation evaluated factor structure. Multivariable linear regression identified sociodemographic predictors of FoMO scores

Results / Discussion:
824 participants were enrolled (mean age 21.71 years [SD 8.68]; 558 [67.7%] female; 766 [93.0%] urban; 647 [78.5%] students). The FoMOS showed good internal consistency (α=0.826). EFA supported a unidimensional structure (eigenvalue 3.95; 39.5% variance explained). The revised CFA one-factor model achieved acceptable fit (RMSEA=0.08, SRMR=0.04, CFI=0.94, TLI=0.91; all items $p<0.0001$).

Conclusion:
The FoMOS is a reliable and unidimensional measure of FoMO in Indian adolescents and adults. Age is the primary sociodemographic correlate. These findings support integration of the FoMOS into adolescent mental health screening, digital well-being programmes, and national public health policy targeting India's growing burden of FoMO-related morbidity.

FC-07
Digital Psychiatry, Novel Interventions and Emerging Mental Health

FC-07-002
Digital inclusion as a social determinant of mental health: a conceptual framework for healthy aging in the digital era

Speaker: Farshiya Parveen, India

Topic:
10) Other

Abstract

Objective:
To explore the relationship between digital exclusion and mental health among older adults and to propose a comprehensive conceptual framework explaining how digital inequities influence psychological well-being in later life. The paper also aims to identify factors that mediate the association between digital engagement and mental health outcomes among older adults.

Methods:
This conceptual paper synthesizes literature from geriatrics, public health, digital inclusion, and mental health. Evidence on digital access, digital literacy, technology anxiety, social support, health literacy, and mental health outcomes in older adults was reviewed. Based on this synthesis, a theoretical framework was developed to explain the multidimensional nature of the digital divide, including access, skills, motivation, and engagement.

Results / Discussion:
The framework identifies the digital divide as a multidimensional construct comprising access, skills, motivation, and engagement. Digital exclusion was associated with reduced access to health information and services, greater loneliness, lower self-efficacy, and limited social participation. Digital inclusion was linked to better social connectedness, health literacy, healthcare access, and perceived social support.

Conclusion:
This paper presents a theoretical framework explaining how digital inclusion and exclusion influence mental health among older adults. The model highlights the roles of digital access, skills, motivation, and engagement in shaping psychological well-being. It provides a basis for future research and may inform policies and interventions aimed at reducing digital inequities, promoting healthy aging, and improving mental health outcomes.

FC-07
Digital Psychiatry, Novel Interventions and Emerging Mental Health

FC-07-003
Real-World Effectiveness of Symptom Provocation and Intermittent Theta Burst Stimulation (iTBS) Over the Dorsomedial Prefrontal Cortex/Anterior Cingulate Cortex in Treatment-Refractory Obsessive–Compulsive Disorder

Speaker: Shalini Naik, India

Co-Authors:
Shubh Mohan Singh
Abhishek Ghosh
Arshia Sood

Topic:
10) Other

Abstract

Objective:
Real-World Effectiveness of Symptom Provocation and Intermittent Theta Burst Stimulation (iTBS) Over the Dorsomedial Prefrontal Cortex/Anterior Cingulate Cortex (DMPFC/ACC) in Treatment-Refractory Obsessive–Compulsive Disorder(OCD)

Methods:
A retrospective review of consecutive patients with treatment-refractory OCD who received simultaneous symptom provocation and intermittent theta burst stimulation (iTBS) over DMPFC/ACC. Symptom severity was assessed using the Yale–Brown Obsessive Compulsive Scale (Y-BOCS) and Hamilton Depression Rating Scale (HDRS), with pre–post changes analysed using paired-sample t-tests.

Results / Discussion:
Sixteen patients (mean age 39.2 years; 62.5% female) with a mean YBOCS score decreased significantly from 27.27 ± 8.99 to 18.87 ± 10.23 after 25.6 ± 9.1 sessions ($t(14)=3.79$, $p=0.002$; Cohen's $d=0.98$). Treatment was well tolerated, with no serious adverse events reported. Comorbid depressive symptoms were observed in 25% (4/16), with mean HDRS scores decreased from 21.33 ± 14.43 to 11.33 ± 6.11, no statistically significant difference.

Conclusion:
Symptom provocation combined with dmPFC/ACC-targeted iTBS was feasible, safe, and associated with significant improvement in obsessive–compulsive symptoms in this treatment-refractory cohort. Improvements in comorbid depressive symptoms were also observed. These findings support the potential of dmPFC/ACC-directed neuromodulation as a real-world treatment option for refractory OCD and warrant confirmation in larger prospective studies.

FC-07
Digital Psychiatry, Novel Interventions and Emerging Mental Health

FC-07-005
A cross sectional study on relationship of degree of smartphone use with sleep quality and levels of anxiety, depression, and stress among undergraduate medical students

Speaker: Stuti Kothari, India

Co-Authors:

Alok Kumar Tyagi

Topic:
10) Other

Abstract

Objective:
1. To assess the degree of smartphone use among undergraduate medical students.

2. To evaluate sleep quality, anxiety, depression, and stress levels among undergraduate medical students.

3. To determine the relationship between the degree of smartphone use and sleep quality.

4. To determine the relationship between the degree of smartphone use and levels of anxiety, depression, and stress among undergraduate medical students.

Methods:
Undergraduates (18–30 years, regular smartphone users >6 months) providing consent are included. Those with severe psychiatric, neurological, chronic medical illnesses, or disabilities are excluded. Tools used: sociodemographic proforma, Smartphone Addiction Scale (SAS), Pittsburgh Sleep Quality Index (PSQI), and DASS-21. Analysis will use descriptive statistics, Chi-square test, and Spearman's correlation.

Results / Discussion:
Data collection and analysis are currently in progress.

Conclusion:
This study is expected to provide insights into the association between smartphone use, sleep quality, and psychological distress among undergraduate medical students. The findings may help in developing interventions aimed at promoting healthy smartphone use, improving sleep hygiene, and enhancing mental well-being among medical students.

FC-08
Dementia, neurodegeneration: mechanisms and interventions

FC-08-001
A Prospective Resting-State Functional MRI study of Acute Retarded Catatonia and its response to treatment

Speaker: Brunda Ballal S, India

Co-Authors:
Gauthami Nair
Himanshu Joshi
Guru S Gowda
Krishna Prasad M
John P John
Venkata Senthil Kumar Reddi

Topic:
10) Other

Abstract

Objective:
Catatonia is a neuropsychiatric disorder associated with psychiatric and medical conditions; yet its neurobiological mechanisms remain incompletely understood. A previous study reported whole-brain functional hyperconnectivity in acute retarded catatonia (Parekh et al., 2022). This study aimed to replicate this finding and determine whether the increased resting-state functional connectivity (rsFC) reverses following resolution of catatonia.

Methods:
In this prospective resting-state fMRI study, 10 patients with acute retarded catatonia and 10 age- and gender-matched healthy controls underwent structural and functional imaging. Patients were scanned during acute catatonia and after resolution of catatonia. Whole-brain ROI-to-ROI rsFC analysis was performed. rsFC differences within the lorazepam-responders (n=5) and ECT-responders (n=5), before and after resolution were also examined.

Results / Discussion:
At baseline, patients with acute retarded catatonia showed increased whole-brain ROI-to-ROI rsFC in comparison to healthy control subjects. This whole brain rsFC was found to be significantly lower following resolution of catatonia as indicated by the pre>post ROI-ROI functional connectivity analysis results ($p < 0.05$, FDR-corrected). A similar pattern was also observed among lorazepam responders subgroup.

Conclusion:
Generalized resting-state hyperconnectivity may represent a neurobiological state marker of acute catatonia. The GABA-ergic agent lorazepam, by restoring the excitation-inhibition

balance, reverses this aberrant functional hyperconnectivity and thereby reverses the psychomotor refractory state. These findings provide translational insights into catatonia pathophysiology and suggest potential imaging markers of differential treatment response.

FC-08
Dementia, neurodegeneration: mechanisms and interventions

FC-08-003
Differential effects of long-term electroacupuncture and repetitive transcranial magnetic stimulation in preventing the progression of Alzheimer's disease in AppNL-G-F mice

Speaker: Zhang-Jin Zhang, Hong Kong

Topic:
4) Dementia

Abstract

Objective:
Both electroacupuncture (EA) and repetitive transcranial magnetic stimulation (rTMS) possess the potential in combating the progression of Alzheimer's disease (AD). In this study, we compared the effects of the two regimens as early long-term intervention in AppNL−G−F mice, a new amyloid precursor protein (APP) knock-in model that recapitulates multiple AD-associated pathologies.

Methods:
The 2-month-old freely moving model mice randomly received EA or rTMS for 2-3 sessions a week for 6 months. Cognitive tests were conducted in Y maze and Barnes maze sequentially at the age of 4, 6, and 8 months. The cortex and hippocampus were dissected thereafter for neurohistological and molecular analysis.

Results / Discussion:
Both regimens prevented cognitive deterioration at 6 months old. EA maintained its prevention to 8 months. EA reduced Aβ burdens with particular dense-core plaques and mitigated synaptic loss. rTMS had similar effects on Aβ plaques, but not on dense-core plaques. Both regimens displayed greater suppression on microgliosis in the cortex than in the hippocampus, equivalently inhibited astrocytosis, and suppressed dendritic degeneration.

Conclusion:
EA produced more long-lasting and broad-acting effects than rTMS in alleviating memory impairment and pathological products of AD. EA could serve as an early long-term intervention and rTMS as adjuvant therapy in slowing the progression of AD.

FC-08
Dementia, neurodegeneration: mechanisms and interventions

FC-08-004
Non-invasive long-term treatment of Alzheimer's dementia by neuronavigated focused mechanical shockwaves - an effective and safe strategy

Speaker: Ulrich Sprick, Germany

Co-Authors:
Ali Riza Günes
Martin Beglau
Martin Köhne

Topic:
4) Dementia

Abstract

Objective:
Investigation in prior studies showed that noninvasive MRI-navigated transcranial pulse stimulation (TPS) lead to benefits of cognitive functions in Alzheimer's dementia.

Methods:
100 real world out-patients suffering from AD (light/moderate symptoms) received 6 MRI-guided TPS-stimulations in 2 weeks (6.000 pulses per session, 0.25 mJ/mm2, 4 Hz). bilaterally into the frontal, parietal and temporal cortex. Single booster sessions were administered every 4 to 6 weeks up to 3 years. Patients were interviewed for side effects before each of the sessions. The effectiveness of treatment was measured by the Stroop-test and BDI.

Results / Discussion:
Mean age was 73.5 years (SD 9.8). 58 % females. 82% received at least one booster session during the follow up treatment. 0% showed severe side effects. 75% reported no side effects at all. 25 % showed mild transient side effects (like local stitching). Stroop-data showed a significant long-term improvement of executive functions (especially interference condition). Depressive symptoms significantly decreased over time with BDI scores < 10.

Conclusion:
This study investigated the largest cohort of real world AD-patients treated with noninvasive neuronavigated TPS so far. TPS proofed to be a safe and effective noninvasive stimulation method for long-term treatments up to 3 years. The treatments were tolerated without any serious side effects. Improvement of executive functions and depressive symptoms lasted over long intervals. Thus TPS may be considered as a promising tool of NIBS in AD.

FC-08
Dementia, neurodegeneration: mechanisms and interventions

FC-08-005
Depressive symptoms modify the relationship between fatigue and cognitive performance in individuals with coronary artery disease

Speaker: Julius Burkauskas, Lithuania

Co-Authors:
Julija Gecaite-Stonciene
Justina Portacenko
Vesta Steibliene
Nijole Kazukauskiene

Topic:
7) Mood disorders

Abstract

Objective:
Nearly one-third of individuals with coronary artery disease (CAD) continue to experience severe fatigue even after completing cardiac rehabilitation. Fatigue is known to be associated with deterioration in mental health, including emotional and cognitive domains. We aimed to examine the associations between cognitive function and subjective fatigue levels in individuals with CAD, with and without depressive symptoms.

Methods:
This cross-sectional study included 1,034 participants with CAD (57 ± 9 years, 77% men) evaluated 1–2 weeks after acute coronary syndrome. Participants completed the Hospital Anxiety and Depression Scale (HADS) and Multidimensional Fatigue Inventory-20. The Mini-Mental State Examination assessed global cognition (MMSE), while the Digit Symbol Substitution Test (DSST) and Trail Making Test (TMT) assessed processing speed and executive function.

Results / Discussion:
Multiple regression showed that in non-depressed (HADS-D<8, n=905) individuals reduced motivation was associated with worse set-shifting (TMT-B, β=.108, p=.003). Mental fatigue was associated with worse global cognitive functioning (MMSE, β=−.125, p<.001), incidental learning (DSST recall, β=−.129, p<.001), and set-shifting (TMT-B, β=.100, p=.005). No associations between fatigue and cognitive function emerged in individuals with depressive risk.

Conclusion:
In individuals with CAD, subjective fatigue – particularly reduced motivation and mental fatigue – is independently associated with poorer cognitive performance primarily among those without

depressive symptoms, suggesting that depression status may modify the relationship between fatigue and cognitive functioning early after acute coronary syndrome.

FC-08
Dementia, neurodegeneration: mechanisms and interventions

FC-08-006
Acute Neurocognitive Effects of Ketamine in Treatment-Resistant Depression

Speaker: Akanksha Dahiya, India

Topic:
7) Mood disorders, 10) Other

Abstract

Objective:
Ketamine, an NMDA receptor antagonist, produces rapid antidepressant effects in treatment-resistant depression (TRD), but its acute cognitive effects—and whether baseline cognition predicts response—remained unclear. This study examined neurocognitive performance before and after ketamine infusion in TRD patients, exploring associations between cognitive function and subsequent antidepressant response.

Methods:
Patients with TRD received a single open-label subanesthetic ketamine infusion. Neurocognitive function was assessed at baseline and 24 hours to 48 hours apart post-infusion using standardized tests. Depressive symptoms were measured (with BDI) at baseline and follow-up to check forbantidepressant response.

Results / Discussion:
Ketamine infusion was associated with a transient impairment in memory recall at 24 hours compared to baseline, while other cognitive domains (attention, processing speed, executive function) showed no significant decline. Better baseline performance on certain cognitive tasks correlated with greater antidepressant response. Acute cognitive side effects were generally mild and did not correlate strongly with degree of clinical improvement.

Conclusion:
A single ketamine infusion caused selective, short-lived memory impairment at 24 hours without broader neurocognitive decline. Baseline cognitive function may serve as a predictor of antidepressant response, suggesting potential utility as a biomarker. Findings support ketamine's acute safety profile regarding cognition but highlight the need for further research on cognitive predictors and longer-term effects in TRD.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-001
Novel and promising treatments for anxiety disorders

Speaker: Borwin Bandelow, Germany

Topic:
10) Other

Abstract

Objective:
Current pharmacological treatments for anxiety disorders, including SSRIs, SNRIs, benzodiazepines, and others, have demonstrated efficacy but are limited by several important drawbacks. There is a need for novel anxiolytic agents that are faster-acting, more efficacious, better tolerated, and have no abuse potential. This review summarizes emerging pharmacological treatments for anxiety disorders.

Methods:
Information was obtained not only through electronic literature searches but also from clinical trial registries (e.g., ClinicalTrials.gov), company press releases, and artificial intelligence platforms (e.g., ChatGPT, Perplexity), as many of the studies cited in this article have not yet been published in peer-reviewed journals.

Results / Discussion:
Several compounds are in development, including drugs targeting the serotonin system (toludesvenlafaxine, gepirone, FKW00GA), the trace amine-associated receptor 1 (TAAR1), , the GABA binding site (ENX-102, darigabat, and buagafuran), the nicotinic receptor (soclenitant and VQW-765), the endocannabinoid system (cannabidiol and SYT-510), pherins (fasedienol) and psychedelics (MM120, PSX-001, MSP-1014). Some of these drugs have reached phase 3.

Conclusion:
The translation of these compounds into clinically approved treatments for anxiety disorders may take several years, potentially delaying the emergence of significant therapeutic breakthroughs.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-002
Differential Heart Rate Trajectories Following LSD and Psilocybin in Clinical Psychedelic-Assisted Psychotherapy

Speaker: Daniele Zullino, Switzerland

Co-Authors:
Tatiana Aboulafia Brakha
Louise Penzenstadler

Topic:
10) Other

Abstract

Objective:
Classic psychedelics induce mild autonomic activation, but their cardiovascular dynamics in clinical populations remain poorly characterized. This study aimed to compare heart rate trajectories following LSD and psilocybin in patients receiving psychedelic-assisted psychotherapy for depressive or anxiety disorders and to examine whether these effects are influenced by subjective anxiety.

Methods:
This single-center study analyzed routine clinical data from 30 patients (mean age 51.6 ± 12.2 years; 15 female) treated under compassionate use at Geneva University Hospital. Participants received LSD (100–200 µg; n = 13) or psilocybin (15–30 mg; n = 17). Heart rate and anxiety (VAS 0–100) were recorded at seven time points (30–300 min). Linear mixed models assessed heart rate trajectories including substance, age, and anxiety as covariates.

Results / Discussion:
No significant main effect of substance was observed ($p = 0.42$). A significant time × substance interaction emerged ($p = 0.01$), indicating distinct heart rate trajectories. LSD showed a delayed, sustained increase peaking at 3–4 h, while psilocybin declined earlier. This interaction remained significant after adjustment for age and anxiety ($p = 0.006$). No tachycardia or serious cardiovascular events occurred.

Conclusion:
LSD and psilocybin exhibit distinct temporal patterns of autonomic activation in clinical psychedelic-assisted psychotherapy. These differences likely reflect pharmacokinetic and receptor-level mechanisms and support the cardiovascular safety of supervised clinical use, while highlighting the need for substance-specific monitoring strategies.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-003
An immune–autonomic vulnerability signature for suicide attempts: meta-analysis of inflammatory markers and heart rate variability

Speaker: Brinda Shree, India

Co-Authors:
Subhasmita Das
Pankaj Verma

Topic:
7) Mood disorders

Abstract

Objective:
To systematically evaluate whether inflammatory markers (CRP, IL-6) and reduced vagally mediated heart rate variability together characterize an immune–autonomic vulnerability profile in adults with a history of suicide attempts, compared with psychiatric controls, to inform biologically grounded suicide risk stratification

Methods:
A PRISMA 2020–guided systematic review and random-effects meta-analysis will search PubMed, Embase, PsycINFO, and Web of Science for observational studies comparing suicide attempters with psychiatric controls. Outcomes include CRP, IL-6, and resting HRV indices (RMSSD, HF-HRV, SDNN). Hedges g and I^2 will be calculated.

Results / Discussion:
Study screening and data extraction are currently underway. We expect suicide attempts to be associated with elevated inflammatory markers (CRP, IL-6) and reduced vagally mediated HRV compared with psychiatric controls, supporting a dual-domain immune–autonomic vulnerability phenotype. Pooled effect sizes and heterogeneity estimates will be reported.

Conclusion:
An integrated immune–autonomic vulnerability signature may provide objective biological support for suicide risk stratification beyond symptom-based assessment. Combining inflammatory and autonomic markers with routine clinical evaluation could advance precision suicide prevention and guide future prospective validation in diverse psychiatric populations.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-004
New treatment for depression and anxiety disorders based on crataegus pinnatifada

Speaker: Racid Doron, Israel

Co-Authors:
Keren Nitzan
Mushki Paz
Motty Franko
Itamar Barouch
Shahaf Uzan
Sasha Ariev
Yair Ben-Chaim
Alon Shamir

Topic:
7) Mood disorders

Abstract

Objective:
Anxiety and depression are major burdens, and SSRIs, despite widespread use, have limited efficacy and side effects such as sexual dysfunction and weight gain. This study evaluates the herbal compound Crataegus pinnatifida (Shan-Zha) as an alternative therapy, comparing its behavioral efficacy and side-effect profile with escitalopram.

Methods:
Mice underwent four weeks of unpredictable chronic mild stress to induce anxiety- and depression-like behaviors, then received one of four treatments: a novel herbal therapy, Shan-Zha, escitalopram, or saline. Behavioral outcomes, sexual behavior, and weight were assessed, alongside serotonin transporter levels in the prefrontal cortex and hippocampus to evaluate efficacy and side effects.

Results / Discussion:
The novel herbal treatment showed strong anxiolytic and antidepressant-like effects without sexual dysfunction or weight gain, unlike escitalopram. Shan-Zha was identified as the main active component driving these benefits, suggesting it may represent a promising complementary or potential alternative approach to SSRIs, with comparable efficacy and a more favorable side-effect profile.

Conclusion:
These differences in side effect profiles may be attributed to distinct biological mechanisms.

Ongoing studies explore these mechanisms to better understand the therapeutic potential of this novel treatmen

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-005
High-Dose Vitamin D3 and Resveratrol as an Augmentation Strategy for Persistent Depressive Symptoms in Medicated Patients with Major Depressive Disorder and Comorbid Hypothyroidism

Speaker: Julia Fedotova, Russia

Topic:
7) Mood disorders

Abstract

Objective:
To investigate the efficacy, safety, and potential anti-inflammatory mechanisms of combining high-dose vitamin D3 and resveratrol as an adjunctive therapy for persistent depressive symptoms in patients with MDD and hypothyroidism already receiving stable antidepressant and levothyroxine treatment.

Methods:
Patients were receivied with therapeutic doses of either venlafaxine (75–225 mg/day) or escitalopram (10–20 mg/day) for at least 3 months prior to augmentation. Participants were randomized 1:1 to receive either adjunctive vitamin D3 (5000 IU/day) plus trans-resveratrol (500 mg/day) or placebo. The primary outcome was the change in score HDRS from baseline to week 12. Secondary outcomes included changes in serum 25(OH)D and thyroid hormones.

Results / Discussion:
The combination therapy produced a marked and statistically significant improvement. This group showed a 47% reduction in HDRS scores ($p < 0.001$), compared to the placebo group. The response rate was 66.7% in the active group vs. 13.3% in placebo.

The additional treatment increased serum 25(OH)D to optimal levels (48.6 ± 8.7 ng/mL; $p < 0.001$) and significantly reduced pro-inflammatory cytokines IL-6 (-34%) and TNF-α (-28%) (both $p < 0.01$).

Conclusion:
The addition of high-dose vitamin D3 and trans-resveratrol to antidepressant (venlafaxine or escitalopram) and levothyroxine therapy for 12 weeks is a highly effective, safe, augmentation strategy for patients with MDD and comorbid hypothyroidism suffering from persistent depressive symptoms.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-006
Modeling Epistasis in Alcohol Use Disorder: Evidence from Candidate Gene Variants

Speaker: Bhagyalakshmi Shankarappa, India

Co-Authors:
Jayant Mahadevan
Pratima Murthy
Biju Viswanath
Harshad Devarbhavi
Sanjeev Jain
Meera Purushottam

Topic:
6) Addiction

Abstract

Objective:
Alcohol Use Disorder (AUD) and its complications, including alcohol-related cirrhosis, are influenced by both genetic and metabolic factors. This study aimed to investigate allele frequency distribution of selected polymorphisms in genes involved in alcohol metabolism (ADH2, ADH3, ALDH2) and one-carbon metabolism (MTHFR, MTR), and to evaluate their individual and interactive contributions to clinical manifestations, particularly cirrhosis.

Methods:
Men (N=243) with Alcohol Use Disorder (AUD) were classified as cirrhosis (AUD-C+ve) or non-cirrhosis (AUD-C−ve) based on ICD-10 criteria, recruited from SJMCH. Cirrhosis was assessed using Fibroscan/ultrasonography (LSM <14 kPa for controls). SNPs in ADH2, ADH3, ALDH2, MTHFR, and MTR were genotyped, and gene–gene interactions were analyzed using MDR.

Results / Discussion:
Allele frequencies varied between groups. MDR identified significant 3-locus models for alcohol metabolism (rs1789920, rs2066701, rs2238151) and one-carbon metabolism (rs1801131, rs1801133, rs1805087) (accuracy=0.59; CVC=10; p=0.008). ADH2–ADH3 and MTHFR–MTR interactions were associated with ~1.9-fold increased cirrhosis risk, with interactions contributing more than individual SNPs.

Conclusion:
Our findings highlight the importance of gene–gene interactions in modulating susceptibility to alcohol-related cirrhosis. Both alcohol metabolism and one-carbon metabolism pathways exhibit synergistic effects, emphasizing the need to consider epistatic interactions in genetic studies of AUD and its clinical outcomes.

FC-09
Mood, anxiety and suicide: mechanisms and novel interventions

FC-09-007
Inherited Genetic Variants in Autism Reveal Shared Neurobiological Mechanisms Linking Core Behaviours to Psychiatric Comorbidities

Speaker: Ashitha Siddappa Niranjana Murthy, India

Co-Authors:
Snijesh V P
Meghana R

Topic:
10) Other

Abstract

Objective:
Neurodevelopmental disorders are part of a spectrum of psychiatric conditions, yet the role of inherited variants in clinical manifestations remains unclear, especially in underrepresented populations. Using Autism Spectrum Disorder as a model, this study investigates convergent genetic pathways linking core behaviours, comorbidity, and clinically relevant mechanisms within a precision psychiatry framework.

Methods:
Whole exome sequencing data from 50 parent–child trios were analysed to identify rare, deleterious inherited variants. A novel gene prioritisation framework integrating evolutionary constraint metrics was applied. Functional enrichment, developmental brain expression (BrainSpan), and protein–protein interaction analyses were used to map genetic findings to behavioural domains and comorbidities.

Results / Discussion:
We prioritised 751 inherited genes, incl 2 high-risk ASD genes/subject. We report (i) a strong enrichment of sensory-processing genes (n=116)-overlapped with 62% of RRB genes-suggesting a mechanistic sensory–behavioural axis; (ii) 71.4% overlap of social-interaction genes with seizure-genes, indicating shared neurobiological substrates; and (iii) distinct prenatal (neurodevelopmental) vs postnatal (synaptic signalling) gene expression clusters.

Conclusion:
This study provides evidence for a inherited risk architecture in ASD, bridge core ASD symptoms with neurophysiological mechanisms and comorbidities- highly relevant to biological psychiatry. Importantly, this work underscores the need to incorporate population diversity and inherited variation into psychiatric genomics to advance biomarker discovery and precision medicine approaches in ASD.

FC-10
Prescribing, pharmacogenomics, and real-world psychopharmacology

FC-10-001
Screening of Psychopharmacological Agents for cellular health in iPSC-derived neural cells

Speaker: Karishma Ravikumar, India

Co-Authors:
Manasa Deepika
Mythri Sv
Amirtha Varshini
Kalyani Bindu
Jayant Mahadevan
Reeteka Sud
Pradip Paul
Biju Viswanath

Topic:
10) Other

Abstract

Objective:
To screen major psychiatric drugs, including lithium, clozapine, DOI, and ketamine, in induced pluripotent stem cells (iPSC) derived neural cells for cellular health.

Methods:
Analysis of cellular ATP and ROS levels using luminescence and fluorescent-based assays, respectively. Cell viability using flow cytometry with propidium iodide. Assessment of mitochondrial dynamics (analysis of membrane potential using JC-1 dye, imaging with MitoSox superoxide indicator dye for mitochondrial oxidative stress, and Mitotracker Deep Red dye for verifying mitochondrial integrity).

Results / Discussion:
Preliminary study conducted with neural stem cells upon 12-hour exposure to lithium, valproate, and clozapine showed the following observations: Cellular ROS (reactive oxygen species) remained the same, and ATP levels seemed to decrease. MtROS decreased with Valproate. Further, the above-mentioned drugs will be evaluated for their effect in additional model systems like neurons and neurosphere allowing longer exposure periods.

Conclusion:
Assessing cellular health upon exposure to these drugs would help us understand the underlying mechanisms of their action, and hence, this study could highlight potential areas for further exploration.

FC-10
Prescribing, pharmacogenomics, and real-world psychopharmacology

FC-10-002
Dynamic Alterations in TSHZ3 Expression Levels and Nuclear Localization During SH-SY5Y Neuronal Differentiation

Speaker: Ekaterina Marilovtseva, Russia

Co-Authors:
Veronika Prosvetova
Artemiy Kurishev
Vera Golimbet
Ekaterina Semina
Yulia Chaika

Topic:
2) Schizophrenia

Abstract

Objective:
Transcription factor TSHZ3 is implicated in neocortex development, its heterozygous deletion resulting in ASD-like behavior. SH-SY5Y cell line is one of the most convenient models for studying neuronal differentiation and function. Our previous research showed that heterozygous deletion of TSHZ3 enhancer affected SH-SY5Y differentiation. The aim of this work was to analyze TSHZ3 expression rate and localization during SH-SY5Y.

Methods:
SH-SY5Y were treated with 10 μM all-trans retinoic acid for 8 days (the first stage of differentiation). This was followed by 8 day-long culturing on ECM in the presence of 50 ng/mL BDNF (the second stage of differentiation). Undifferentiated and differentiated SH-SY5Y were analyzed with qPCR, immunoblotting, and immunofluorescence.

Results / Discussion:
The levels of TSHZ3 altered significantly during SH-SY5Y differentiation, reaching their peak at the first and slightly decreasing at the second stage. The ratio of TSHZ3 phosphorylated to nonphosphorylated forms increased during differentiation. TSHZ3 was mostly concentrated on the nuclear periphery in undifferentiated cells, evenly distributed within the nucleus at the first and partly detected back on the nuclear periphery at the second stage.

Conclusion:
TSHZ3 expression, nuclear localization, and, apparently, phosphorylation rate change significantly during SH-SY5Y differentiation, implicating the involvement of the protein in this process.

FC-10
Prescribing, pharmacogenomics, and real-world psychopharmacology

FC-10-003
Gene expression connectivity within postsynaptic density networks: a comparison of clozapine with a d2 receptor antagonist and a d2 receptor partial agonist and its translational relevance for treatment-resistant schizophrenia

Speaker: Licia Vellucci, Italy

Co-Authors:
Anita Nasti
Annarita Barone
Daniele Formisano
Carmine Tomasetti
Felice Iasevoli
Giuseppe De Simone
Andrea De Bartolomeis

Topic:
2) Schizophrenia

Abstract

Objective:
Antipsychotic medications are known to influence brain connectivity. The present study aimed to investigate the organization of brain functional connectivity in Sprague–Dawley rats after acute and chronic exposure to three antipsychotics with distinct pharmacodynamic profiles at the dopamine D2 receptor (D2R): haloperidol (0.8 mg/kg), clozapine (15 mg/kg), and aripiprazole (12 mg/kg).

Methods:
Functional connectivity was assessed through topographical molecular imaging of the activity-dependent immediate early gene Homer1a mRNA. Expression levels were quantified across 31 predefined brain regions of interest and visualized using heatmaps coupled with hierarchical clustering. Network organization was further characterized using graph-theoretical approaches, including analysis of nodal parameters and global network properties.

Results / Discussion:
Control animals exhibited a relatively unspecialized network architecture. Chronic haloperidol impaired network integration compared with aripiprazole. Acute aripiprazole induced a modular configuration involving prefrontal and sensorimotor regions. Clozapine produced a marked clustering of cortical regions already after acute administration. This effect was reflected by significant differences in Frobenius norm relative to haloperidol.

Conclusion:
Overall, these results indicate that antipsychotic drugs exert distinct and time-dependent effects on brain network topology. The observed patterns suggest the presence of drug-specific connectivity profiles at the level of synaptic gene expression, offering potential translational insights into the mechanisms that may underlie differences in clinical efficacy among antipsychotic treatments.

FC-10
Prescribing, pharmacogenomics, and real-world psychopharmacology

FC-10-004
CYP2D6 Allele and Metabolizer Phenotype Distribution in a Central Indian Depression Cohort: Interim Pharmacogenetic Findings

Speaker: Shubham Atal, India

Co-Authors:
Sadasivam Balakrishnan
Debasis Biswas
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Abhijit Rozatkar
Tamonud Modak
Umay Kulsum
Apoorva Nema
Steven Hollon
Vikram Patel

Topic:
7) Mood disorders

Abstract

Objective:
To determine the frequency of clinically relevant CYP2D6 gene polymorphisms, copy number variations (CNVs), and predicted metabolizer phenotypes in adults with major depressive disorder (MDD) enrolled in a primary care randomized controlled trial in Central India, to assess potential implication on antidepressant drug prescribing.

Methods:
This pharmacogenetic analysis is nested within a primary care RCT (OptimizeD) in Bhopal. Adults with newly diagnosed moderate–severe MDD who consented to genomic testing were included. Key CYP2D6 variants (*2, *3, *4, *5, *6, *10, *41) and CNVs were genotyped using TaqMan assays by qPCR. Diplotypes were translated into predicted metabolizer phenotypes (PM, IM, NM, UM) using CPIC activity score methodology.

Results / Discussion:
Among 197 genotyped participants, the most frequent alleles were *2A (54.7%) and *2 (37.8%). Reduced-function alleles included *10 (17.0%) and *41 (9.6%), while no-function variants were *4 (8.6%), *5 deletion (3.3%), *3 (2.8%), and *6 (0.6%), with 12.2% participants showing gene multiplications. Phenotype analysis (n=197) showed NM 61.9%, IM 24.9%, PM 3.1%, and UM 10.1%, indicating that 38% were predicted non-normal metabolizers.

Conclusion:
These findings have direct implications for antidepressant prescribing. CYP2D6 PMs may experience higher exposure and adverse effects with paroxetine, fluvoxamine, venlafaxine, TCAs, and vortioxetine, while UMs risk subtherapeutic response. The substantial IM proportion may have altered dose requirements. Further findings and treatment outcome associations will help inform pharmacogenetics based prescribing.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-001
Multisensory Integration Deficits in remitted Bipolar I Disorder: A Study of SVV, DVA, and Structural MRI.

Speaker: Rahina Abubacker, India

Co-Authors:
Pradeep Yuvaraj
Aravind Kumar
Aravinda Hr
Binukumar Bhaskarapillai
Muralidharan Kesavan

Topic:
7) Mood disorders, 1) Psychoses

Abstract

Objective:
To evaluate Subjective Visual Verticality and Dynamic Visual Acuity in remitted BD-I and examine their relationship with brain morphology and clinical variables.

Methods:
Fifty-three BD-I participants aged 18-50 years in remission and age-gender matched Healthy subjects underwent SVV (static, dynamic clockwise, dynamic anticlockwise, head tilt conditions) & DVA (static and dynamic visual acuity) tests. A representative subset (n=15 per group) underwent 3T MRI which were processed using CAT12/SPM with atlas-based ROI extraction.

Results / Discussion:
BD-I showed poorer DVA and greater SVV deviation in dynamic and head-tilt conditions, with no static difference. MRI showed cortical thinning in insular, temporal, parietal, and frontal regions, with increased thickness in visual areas. Vestibular measures correlated with limbic, cerebellar, and cortical regions. Clinical variables-manic episodes, total illness burden, remission duration, and age of onset, were associated with DVA and SVV.

Conclusion:
BD-I shows disruption of higher-order visual–vestibular integration rather than isolated peripheral dysfunction. Structural alterations correlate with SVV and DVA deficits, reflecting impaired sensory integration, motion perception, and spatial processing. Associations with clinical variables suggest disease-related modulation. SVV and DVA with MRI may serve as composite biomarkers.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-003
Integrating Frontal Lobe Assessment and Quantitative EEG as a biomarker in ADHD

Speaker: Mini Sharma, India

Co-Authors:
Deepak Kumar
Suman Kushwaha
Manoj Kumar

Topic:
10) Other

Abstract

Objective:
To study the correlation between the clinical profile of children with ADHD with frontal lobe functions and EEG changes in children of ADHD.

Methods:
This cross- sectional study included children aged 5-12 years diagnosed with ADHD using DSM- criteria at a tertiary care centre over 9 months. ADHD severity was assessed using Conners Severity rating scale (parent short version). Frontal lobe functions were evaluated using Frontal Assessment battery, EXIT-25 Battery and NIMHANS Battery. qEEG recording (21- channel) were analyzed using Fast Fourier Transformation with NeuroWorks software.

Results / Discussion:
Of 35 Participants, 33 met inclusion criteria with moderate to severe ADHD. Approximately 60% demonstrated frontal lobe dysfunction. Significant correlation were observed between ADHD severity and executive dysfunction on EXIT-25 & NIMHANS battery (p-value=0.02, 0.04 respectively). In age group 10-12 years a significant association was noted with NIMHANS battery (p-value=0.05). Around 75% participants exhibited elevated theta: beta ration qEEG.

Conclusion:
Frontal lobe dysfunction and qEEG abnormalities, particularly elevated theta–beta ratio, are prevalent in children with ADHD and correlate with clinical severity. The integration of neuropsychological assessment and qEEG may enhance objective characterization of ADHD and support its role as a potential biomarker framework.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-004
Adverse Chronobiological Phenotype is Associated with Smaller Gray Matter Volumes in Mood Disorders

Speaker: Benicio Frey, Canada

Co-Authors:
Andre Tonon
Katharine Dunlop
Pierre Blier
Raymond Lam
Roumen Milev
Daniel Mueller
Lena Quilty
Joshua Rosenblat
Valerie Taylor
Rudolf Uher

Topic:
7) Mood disorders, 8) Sleep disorders

Abstract

Objective:
Abnormal circadian and sleep-wake cycle markers, namely greater composite phase deviation (CPD) and reduced relative amplitude (RA), have recently been identified as predictors of depressive episode relapse (Tonon, Frey et al JAMA Psychiatry 2026). In the present analysis, we investigated clinical and brain structural correlates of this chronobiological phenotype in an independent sample of individuals with mood disorders.

Methods:
232 individuals with mood disorders were studied. CPD and RA were objectively measured using actigraphy. Participants were categorized into four groups: Low RA + High CPD (n=54), Low RA only (n=58), High CPD only (n=56), and High RA + Low CPD (n=64). Depression severity (MADRS), anxiety (GAD-7), insomnia (ISI), functioning (FAST) and quality of life (WHO-QOL) were compared across groups. 128 participants underwent T1-weighted MRI scans.

Results / Discussion:
Low RA + High CPD reported greater depressive symptom (padj=0.003), insomnia severity (padj<0.001), worse functioning (padj=0.02) and worse quality of life (padj<0.001). RA- and/or CPD-adverse phenotype displayed smaller gray matter volumes in the left pericalcarine and posterior cingulate cortices, and the right frontal pole, medial orbitofrontal, paracentral, pars opercularis, pericalcarine, and supramarginal cortices (p<0.01; FDR corrected).

Conclusion:
Individuals with mood disorders and objectively-measured alterations in circadian and sleep-wake cycle rhythms represent a distinct phenotype characterized by greater clinical severity and widespread cerebral gray matter volume reduction.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-005
Multiscale molecular and functional annotation of morphometric similarity network alterations in panic disorder

Speaker: Yubo Zhang, People's Republic of China

Co-Authors:
Linlin You
Guangwei Sun
Yucheng Yuan
Yue Zhou
Shangyang Li
Wenbin Guo
Chenguang Jiang
Wenhao Jiang
Yingying Yin
Yonggui Yuan

Topic:
7) Mood disorders

Abstract

Objective:
To characterize cortical morphometric similarity network (MSN) alterations in panic disorder (PD) and link their spatial pattern to clinical change, transcriptomic, cellular, neurochemical, and functional annotations.

Methods:
Individual MSNs were constructed from seven multimodal morphometric and microstructural features across 308 cortical parcels in 107 patients with PD and 237 healthy controls. Case-control differences, clinical correlations, AHBA-based PLS transcriptomics, enrichment, PET receptor mapping, and Neurosynth decoding were performed.

Results / Discussion:
PD showed MSN alterations in 34 parcels, with reductions in visual and superior parietal dorsal-attention regions and increases in default-mode, limbic, and frontoparietal territories. Higher right superior parietal MSN predicted greater 2-week HAMA improvement. PLS identified 3,257 robust genes, and the map aligned with CB1, μ-opioid, D2, and 5-HT4 receptor distributions.

Conclusion:
PD involves spatially organized cortical MSN reconfiguration extending beyond fronto-limbic models to visual and dorsal-attention systems. MSN features may capture clinically relevant

network phenotypes and provide molecular and neuromodulatory context for early symptom-change trajectories.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-006
Plasma metabolomic profiling in schizophrenia: associations with symptom severity and antipsychotic treatment

Speaker: Polina Brit, Russia

Co-Authors:
Evgeny Ermakov
Nikita Basov

Topic:
2) Schizophrenia, 8) Sleep disorders

Abstract

Objective:
Schizophrenia is clinically heterogeneous, and reliable peripheral biomarkers remain limited. Plasma metabolomics may capture systemic alterations related to inflammation, oxidative stress, neurotransmitter precursor metabolism, membrane remodeling and treatment effects. This study aimed to identify plasma metabolic changes in schizophrenia and assess their links with clinical features and antipsychotic exposure.

Methods:
The study included 41 patients with schizophrenia and 40 healthy controls. Patients were recruited during inpatient exacerbation. Plasma samples were analyzed by targeted HPLC-MS/MS. After preprocessing and normalization, profiles were compared between groups and assessed in relation to PANSS scores, chlorpromazine-equivalent dose and treatment response. Multivariate analysis, effect sizes and Bayes factors were used.

Results / Discussion:
Patients with schizophrenia showed increased xanthine and decreased metabolites related to membrane lipids, tryptophan and indole pathways. Tryptophan reduction was consistent across several MRM transitions, and pathway analysis supported glycine, serine and threonine metabolism. Xanthine was higher with greater symptom severity, while higher antipsychotic exposure was linked to lower energy-related metabolites.

Conclusion:
Plasma metabolomic profiling revealed systemic alterations in schizophrenia involving purine metabolism, membrane lipid remodeling, tryptophan-related pathways and energy metabolism. Links with PANSS scores and antipsychotic exposure suggest that metabolomic signatures may reflect both disease-related biology and treatment effects, supporting further studies of biological phenotyping in schizophrenia.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-007
A Cross-Sectional Comparative Study To Evaluate Thyroid Function Tests, Inflammatory Markers And Autonomic Function In Patients With Unipolar Depression And Bipolar Depression At Sms Medical College, Jaipur

Speaker: Azra Ansari, India

Topic:
7) Mood disorders

Abstract

Objective:
To evaluate and compare thyroid function tests (TSH, T3, T4, fT3, fT4, Anti-TPO), inflammatory markers (CRP, ESR, NLR, PLR), and autonomic function assessed by heart rate variability in patients with Unipolar Depression and Bipolar Depression, and to examine their association with depression severity.

Methods:
This observational cross-sectional study was conducted in the Department of Psychiatry, SMS Medical College, Jaipur. Sixty patients (30 with Unipolar Depression and 30 with Bipolar Depression), aged 18–45 years and diagnosed per DSM-5-TR criteria, were enrolled. Thyroid function tests, inflammatory markers, and heart rate variability were assessed and analyzed using SPSS version 26.0.

Results / Discussion:
Patients with both Unipolar Depression and Bipolar Depression did exhibit abnormalities in thyroid function, elevated inflammatory markers, and reduced heart rate variability. More pronounced alterations are anticipated in the Bipolar Depression group, with significant associations between these biological parameters and depression severity scores.

Conclusion:
Combined assessment of thyroid function, inflammatory markers, and autonomic function may help identify biological differences between Unipolar and Bipolar Depression. These markers could serve as useful adjuncts for early diagnosis, improved differentiation, and personalized treatment strategies in patients with mood disorders.

FC-11
Biomarkers and Brain Mechanisms Across Psychiatric Disorders

FC-11-008
To compare peripheral inflammatory biomarkers—Neutrophil-to-Lymphocyte R. (NLR), Monocyte-to-Lymphocyte R. (MLR), Platelet-to-Lymphocyte R. (PLR), and Systemic Immune-Infl. (SII) Index—between patients in active manic phases of Bip. Dis. & cli. remission

Speaker: Virendra Chaturvedi, India

Co-Authors:

Alok Kumar Tyagi

Topic:
7) Mood disorders

Abstract

Objective:
Bipolar I disorder (ICD-11: 6A61) links to low-grade inflammation. Cytokine testing is costly; Complete Blood Count (CBC) ratios are affordable. Cross-sectional data cannot distinguish static traits from episode fluctuations. This longitudinal, pre-post study tracks patients from mania to remission as their own controls to validate these biomarkers as objective clinical state-switch indicators.

Methods:
This longitudinal study at SMS Medical College, Jaipur, recruits 67 hospitalized Bipolar I (ICD-11: 6A61) patients. CBC differentials are taken at two points: active mania (YMRS >12) and 4-week sustained remission (YMRS <11). Automated cell counts will calculate NLR, MLR, PLR, and SII indexes. Standard statistical protocols will analyze the data.

Results / Discussion:
Results will be produced on completion of the study.

Conclusion:
Conclusion from Results would be drawn on completion of the study.

FC-12
Comorbidity, public health and behavioural disorders

FC-12-001
Prevalence of eating disorders in the community and treatment

seeking population: a cross-sectional study

Speaker: Ankita Gupta, India

Co-Authors:

Pragya Lodha

Topic:
10) Other

Abstract

Objective:
Eating disorders (ED) are marked by irregular eating habits and distress about body weight or shape. Often seen as a problem of the young, they also affect adults and older adults. They remain underdiscussed due to stigma, low awareness, poor history taking, and negative emotions linked to eating. This study aimed to estimate the prevalence of eating disorders in the community and among treatment-seeking patients at a tertiary general hospital.

Methods:
A survey-based cross-sectional study was carried out to find the prevalence of eating disorders in community population and treatment seeking patients visiting a tertiary general hospital. A total sample of 254 individuals above the age of 18 years were screened using the Eating Disorders Examination-Questionnaire (EDE-Q 6.0).

Results / Discussion:
On the EDE-Q scale, more participants in the community sample, 37 (28%) showed scores in the moderate range and 31 (23.5%) showed scores in the severe range compared to 20 (16.4%) and 13 (10.7%) in the patient sample. Eating concern, shape concern, restraint on eating and weight concern was also greater in the community sample.

Conclusion:
The study helps to understand this concerning prevalence of the eating disorders which warrants greater awareness and early intervention in order to reduce the community burden of eating disorders.

FC-12
Comorbidity, public health and behavioural disorders

FC-12-002
Pattern and prevalence of psychiatric morbidity in patients receiving anti tubercular medications– An observational study

Speaker: Syed Mehvish, India

Topic:
10) Other

Abstract

Objective:
To estimate the prevalence and pattern of psychiatric morbidity among patients receiving ATT, to determine the incidence of new-onset psychiatric disorders during treatment, and to compare these findings with the general population.

Methods:
This 12-month prospective observational study (Dec 2024–2025) included 350 consenting adults receiving ATT at Government Medical College, Srinagar, and affiliated hospitals. Baseline assessment was done at first OPD visit, with telephonic follow-ups at 4, 8, and 12 months. Psychiatric morbidity was evaluated using DSM-5 criteria and validated scales (HDRS, HARS, PANSS, Y-BOCS, YMRS).

Results / Discussion:
At baseline, 41.4% had psychiatric morbidity, most commonly depression (22.3%) and anxiety (13.1%). During follow-up, 38% of previously unaffected participants developed new-onset psychiatric illness. Morbidity was higher than in the general population ($p < 0.001$). Second-line and isoniazid regimens were linked to greater symptom severity, and poor ATT compliance correlated with psychiatric morbidity ($p < 0.001$).

Conclusion:
Psychiatric morbidity is highly prevalent among patients receiving ATT and frequently emerges during the course of treatment. Depression and anxiety constitute the predominant disorders and are significantly associated with poor treatment adherence. Routine mental health screening and integrated psychiatric care should be incorporated into TB treatment programs to improve outcomes.

FC-12
Comorbidity, public health and behavioural disorders

FC-12-003
ADHD Is Associated With Persistent Substance-Related Problems Under Regulated Cannabis Access: Longitudinal Evidence From the Cannabinothèque Pilot Project

Speaker: Louise Penzenstadler, Switzerland

Co-Authors:
Océane Humel
Nathan Cina
Maelle Dreifuss
Tatiana Aboulafia Brakha
Daniele Zullino

Topic:
6) Addiction

Abstract

Objective:
Attention-deficit/hyperactivity disorder (ADHD) is associated with altered reward processing and increased vulnerability to substance-related problems, particularly alcohol. It remains unclear whether this vulnerability persists under strictly regulated, non-commercial access conditions. This study examined longitudinal changes in problematic substance use in the Swiss Cannabinothèque pilot project and assessed ADHD as a vulnerability marker.

Methods:
The Cannabinothèque is a Swiss pilot program providing regulated, non-commercial access to standardized cannabis with prevention and counseling. Participants were followed longitudinally with repeated CUDIT assessments at five time points. ADHD was included as a between-subject factor in repeated-measures ANOVA models. Main effects of time, ADHD, and their interaction were tested, and alcohol-related problems were explored descriptively.

Results / Discussion:
A significant main effect of time on CUDIT scores was observed ($F = 4.90$, $p = 0.0006$). A strong main effect of ADHD was found ($F = 27.13$, $p < 0.000001$), with higher CUDIT scores in participants with ADHD across all time points. The time × ADHD interaction was not significant ($F = 0.42$, $p = 0.79$), indicating parallel trajectories. Descriptive analyses suggested more frequent alcohol-related problems in participants with ADHD.

Conclusion:
ADHD appears to function as a stable vulnerability marker for persistent substance-related problems, even under conditions of strict regulation and reduced environmental risk. These

findings highlight the importance of integrating neurodevelopmental vulnerability into the design of regulated access models and associated clinical interventions.

FC-12
Comorbidity, public health and behavioural disorders

FC-12-004
A study assessing depression, anxiety and quality of life (QOL) in patients of chronic low back pain (CLBP).

Speaker: Vasuda Gupta, India

Co-Authors:
Fiona Mahapatro
Sanjiv Kale

Topic:
7) Mood disorders

Abstract

Objective:
CLBP-pain >12 wks, is a global health issue, a leading cause of disability.Prevalence in India-48-66%. Pain is not purely physical; it is shaped by psychological, emotional, social factors. Depression is ass. with CLBP, worsening pain perception, impairing coping abilities, hindering treatment through negative thought patterns.CLBP management- biopsychosocial approach, combining medical, psychological interventions- cognitive-behavioral therapy.

Methods:
This cross-sectional study included 150 CLBP patients from a tertiary care orthopaedics OPD. Patients with pre-existing psychiatric or major medical illnesses were excluded. After ethical approval and consent, participants were assessed using the Hamilton Depression Rating Scale (HAM-D), Hamilton Anxiety Rating Scale (HAM-A), Numerical Rating Scale (NRS) for pain, and WHOQOL-BREF for quality of life. The study was conducted from 2022 to 2025.

Results / Discussion:
150 participants, 65.4%-depressive, 55.3%-anxiety, 64.7%-both. A significant correlation was observed between depression and anxiety severity. Longer duration of psychiatric symptoms was associated with higher anxiety, severe depression, and lower QOL. Pain severity showed a moderate correlation with anxiety, depression. Mental health symptoms reduced QOL, especially when long-standing. Pain duration alone showed no similar effect.

Conclusion:
Our study found that depression, anxiety, and low back pain negatively affect quality of life, with greater depression and anxiety linked to more severe pain and overall poorer well-being.

Key words:Chronic low back pain, Depression, Anxiety, quality of life.

WS-04
The Quest for Truth: The GALENOS Approach to Triangulating Evidence

Organisers: Soraya Seedat, South Africa
Gin S. Malhi, Australia

Abstract

Background:

More evidence on the efficacy, safety, and mechanism of actions of treatments for psychiatric disorders does not necessarily produce greater certainty. Evidence across human and preclinical research is fragmented, findings often discordant, and different sources of evidence carry different biases. Conventional systematic reviews typically synthesise evidence within particular study designs and rapidly become outdated. The Global Alliance for Living Evidence on aNxiety, depressiOn and pSychosis (GALENOS), supported by Wellcome, is a global ecosystem established to address these challenges through living evidence synthesis, bringing together human and non-human evidence, methodological expertise, and lived experience to inform research prioritisation in anxiety, depression and psychosis. GALENOS has developed the GALENOS Approach to Triangulating Evidence (GATE), a structured framework for integrating multiple evidence streams in a transparent, systematic and reproducible manner.

Aims:

This interactive session will introduce the principles underlying GALENOS living systematic reviews (LSRs) and demonstrate how triangulation can be used to interrogate convergence, complementarity and discordance across different sources of evidence. Particular attention will be given to the challenges of translating between animal and human research, and the integration of expertise derived from lived experience.

Format and content:

Using practical examples from completed and ongoing GALENOS LSRs, the presenters will take participants through the GATE process which bring together clinicians, human and animal researchers, epidemiologists, methodologists and people with lived experience, enabling evidence to be considered through multiple epistemological and methodological lenses. Real-world examples will illustrate for example, GALENOS triangulation of pro-dopaminergic interventions for anhedonia found evidence compatible with a role for dopamine while also revealing that the available evidence could not establish a greater effect of pro-dopaminergic than non-dopaminergic drugs—leading to specific priorities for future human and animal research.

Conclusion:

Scientific truth rarely resides in a single study, methodology or evidence stream. By combining living evidence synthesis with multidisciplinary deliberation and co-production, GALENOS seeks not simply to determine what the literature says, but to establish how confidently we can believe it, why uncertainty remains, and what research should come next. This approach has

the potential to reduce research waste, identify promising signals earlier, and accelerate the translation of discovery science into meaningful advances in psychiatry and mental health

WS-05
How to Fail at Lectures, Grants and Papers (or Not)

Organiser: Michael Berk, Australia

Co-Authors:
Jee Hyun Kim
Dan Siskind

Abstract

- How to give a terrible lecture
- How to ensure your grant application fails
- How to guarantee your paper is rejected

The aim of this workshop is in a tongue in cheek manner two present pitfalls end traps in the three critical academic elements; lectures grant applications and papers. Three eminent academics will present on each of these topics. The first lecture will cover the topic of how to give a terrible lecture, the second will focus on tips and tricks to ensure your grant application fails, and the final lecture will focus on techniques to ensure that your academic manuscript is rejected. The expectation that this will both be humorous and informative.

The learning objective is to increase skills in lecturing grant writing and paper generation.

WS-06
Clinical EEG in Psychiatry

Chair: Oliver Pogarell, Germany

Co-Authors:

Sebastian Olbrich

Abstract

Workshop theme:
Clinical electroencephalography (EEG) covers both standard (sEEG) and quantitative EEG (qEEG) and serves as an approach to analyze alterations of brain function in neuropsychiatric disorders. Though underutilized, EEG remains an important method for the assessment of organic factors influencing psychiatric presentations and monitoring the effects of treatment.

Through this interactive workshop clinicians will achieve an understanding of clinical areas where EEG may provide valuable differential diagnostic and/or predictive information.

Following a brief summary of historical developments, the psychiatrist will learn the basics of a normal EEG exam and understand both the limitations of EEG assessments and the general classes of medical and organic variables that are reflected in abnormal EEG patterns. Specific clinical indicators ("red flags") for sEEG studies will be emphasized, as well as strengths and limitations of the use of qEEG in specific clinical constellations.

Educational objectives:
1) Recognize the clinical EEG as auxiliary diagnostic tool in psychiatry.

2) Decide whether a clinical EEG is indicated for a particular patient.

3) Identify abnormalities and clinical consequences.

4) Understand how qEEG can be implemented in specific clinical constellations and

5) Assess the value of qEEG in therapeutic decisions in psychiatric disorders

WS-07
Drug-Assisted Treatment as Environmental Neurobehavioral Modulation

Chair: Daniele Zullino, Switzerland

Co-Authors:

Louise Penzenstadler

Abstract

Workshop theme:
This workshop explores drug-assisted treatment as a form of environmental neurobehavioral modulation rather than solely a pharmacological intervention. It focuses on how institutionalized, predictable access to psychoactive substances alters stress regulation, reward processing, and learning under uncertainty—core mechanisms implicated in addiction. Using heroin-assisted treatment as an established model, and cocaine-assisted treatment as a prospective extension, the workshop examines how replacing illicit market volatility with institutional predictability stabilizes behavior without coercion. Participants will engage with neurobiological, behavioral, and policy perspectives to understand how environmental design can modulate addiction-related brain processes and improve clinical and social outcomes.

Educational objectives:
After participating in this workshop, attendees will be able to:

1. Explain how environmental volatility and uncertainty influence neurobiological processes involved in addiction, including stress reactivity, reward processing, and learning under risk.

2. Describe drug-assisted treatment as a form of environmental and behavioral intervention that modulates neurobehavioral systems beyond its pharmacological effects.

3. Identify key behavioral and neurobiological mechanisms (e.g., stress reduction, predictability, habit stabilization) through which institutionalized access alters addiction-related behavior.

4. Apply principles of environmental stabilization and choice architecture to clinical, public health, or policy scenarios involving substance use disorders.

5. Critically evaluate emerging interventions, including cocaine-assisted treatment, using a neurobehavioral and policy-informed framework rather than a purely pharmacological comparison.

WS-09

Neuromodulation in Psychiatry - Transcranial Magnetic Stimulation (TMS) and Transcranial Direct Current Stimulation (tDCS) - A Hands-On Training Workshop

Chair: Muralidharan Kesavan, India

Co-Authors:
Preethi Veerappa Reddy
Rashmi Arasappa

Abstract

Workshop theme:
Neuromodulation in psychiatry has always been an important modality of treatment, right from the days of Electroconvulsive therapy (ECT). The newer techniques, like rTMS and tDCS, have come to the limelight in recent times as they are non-invasive, have minimal side effects, and can also be used to identify certain biomarkers for various psychiatric disorders.

Our team has expertise in different neuromodulatory treatments, and our theme is mainly to help the participants understand the basics of how these techniques work and know the efficacy of rTMS and tDCS in various psychiatric disorders. We would also focus briefly on the utility of these methods as research tools to identify biomarkers.

We want the participants of the workshop, mainly targeting young psychiatrists, to have hands-on experience of using the rTMS and tDCS machines, and learn the basic approved protocols for treating different psychiatric disorders, including Schizophrenia, Depression, OCD, Addiction, etc.

Educational objectives:
The educational objectives of the workshop:

1. Understanding the basic functioning and neurobiology behind the use of rTMS and tDCS in psychiatry

2. Understanding the available evidence and approved protocols for their use in psychiatry, and also hands-on training with the devices

3. Understanding the different types of biomarker research that can be done in psychiatry using TMS and tDCS

WS-10
Optimizing Clozapine Treatment in Schizophrenia: From Evidence to Everyday Practice

Chair: Satish Suhas, India

Abstract

Workshop theme:
Drawing on a body of clinical research and real-world evidence generated from high-volume schizophrenia services in low- and middle-income settings, this workshop aims to translate contemporary evidence on clozapine into practical, implementable strategies for everyday clinical care. Building on published work examining clozapine dosing patterns, therapeutic drug monitoring, metabolic variability, and safety outcomes in Asian populations, the workshop will critically examine why clozapine remains underutilized or suboptimally used despite its unmatched efficacy in treatment-resistant schizophrenia. Participants will be guided through biologically informed approaches to initiation, dose optimization, and monitoring, with particular attention to interindividual variability, adverse-effect burden, and long-term treatment sustainability. The workshop emphasizes pragmatic decision-making rooted in evidence but responsive to real-world constraints, including resource limitations, patient heterogeneity, and system-level barriers. Through clinical vignettes and data-informed discussions, the session will demonstrate how findings from translational and observational research can directly inform safer, more effective clozapine prescribing in routine practice.

Educational objectives:
By the end of this workshop, participants will be able to:

-Apply current evidence and guideline recommendations to optimize the initiation and dosing of clozapine in treatment-resistant schizophrenia, with attention to interindividual and population-level pharmacokinetic variability.

-Interpret and use clozapine therapeutic drug monitoring data in routine clinical practice to guide dose adjustments, assess adherence, and improve clinical outcomes while minimizing toxicity.

-Identify, anticipate, and manage common and serious adverse effects of clozapine using proactive, biologically informed strategies, reducing premature discontinuation and improving long-term treatment retention.

-Integrate real-world clinical data and case-based decision-making into everyday clozapine prescribing, enabling safer and more effective use across diverse healthcare settings and resource contexts.

WS-11

Targeting circuits, transforming outcomes: rewiring brain circuits through multimodal neuromodulation in mental health

Chair: Sunil Mittal, India

Co-Authors:
Sukanksha Sahni
Arundhati Bhagabati

Abstract

Workshop theme:
Advances in neuroscience have shifted psychiatric treatment from a purely symptom based model towards a circuit based understanding of mental disorders. neuromodulation techniques allow clinicians to directly influence dysfunctional neural networks implicated in depression, obsessive-compulsive disorder, psychotic disorders, and other complex psychiatric conditions.

this workshop presents a comprehensive framework for multimodal neuromodulation, integrating transcranial direct current stimulation (tdcs), electroconvulsive therapy (ect), deep transcranial magnetic stimulation (dtms), repetitive transcranial magnetic stimulation (rtms) within a structured treatment model alongside pharmacotherapy and psychotherapy (cognitive behavioural therapy).

Educational objectives:
Designed for psychiatrists, clinical psychologists, and mental health professionals, this workshop aims to provide a clinically applicable roadmap for integrating neuromodulation into evidence-based, multidisciplinary psychiatric practice. The workshop will highlight:

a. Mechanisms of action and clinical indications for ect, tdcs, rtms and dtms

b. Comparative advantages and patient selection criteria

c. Safety, ethical considerations, and practical implementation

d. Optimizing pharmacological strategies to enhance neuroplasticity

e. Timing and structuring psychotherapy (CBT) combined with neuromodulation techniques, specifically dtms.

f. Case based individual multimodal treatment planning and discussion through real life clinical cases that have shown significant improvement.

WS-12
Neurobiological Mechanisms and Clinical Translation of Vagus and Peripheral Cranial Nerve Stimulation

Chair: Rezaul Hamid, India

Co-Authors:
Sidharth Sood
Muhamad Aly Rifai

Abstract

Workshop theme:
Biological psychiatry is progressively shifting from neurotransmitter-centric explanations of mental illness toward circuit-based models that emphasize dysfunction within distributed cortico–limbic–autonomic networks. Major psychiatric disorders—including treatment-resistant depression, trauma-related syndromes, and attentional disorders—are increasingly conceptualized as disturbances in integrated neural systems regulating emotion, cognition, stress responsiveness, and physiological homeostasis.

Peripheral cranial nerve stimulation, particularly vagus nerve stimulation (VNS), trigeminal nerve stimulation (TNS), and cranial electrostimulation (CES), represents a translational bridge between circuit neuroscience and therapeutic intervention. Vagal and trigeminal afferent pathways project to the nucleus tractus solitarius and subsequently modulate key neuromodulatory nuclei such as the locus coeruleus and dorsal raphe, influencing noradrenergic and serotonergic tone, neuroplasticity, autonomic balance, and inflammatory signalling. These mechanisms provide a biologically coherent rationale for peripheral neuromodulation in selected psychiatric conditions.

The workshop examines the neurobiological foundations of peripheral nerve stimulation and critically reviews the clinical evidence supporting both invasive and non-invasive approaches. Data from long-term studies in treatment-resistant depression and randomized sham-controlled trials in ADHD will be discussed in relation to effect size, durability of response, safety, and regulatory considerations. Distinctions between implantable and external stimulation modalities will be clarified within a systems-based framework.

Beyond mechanism and trial evidence, the session incorporates emerging real-world clinical observations from a structured interdisciplinary neuromodulation laboratory operating in a rural, resource-constrained setting in India. Prospective data capture of VNS, TNS, and CES within routine psychiatric care pathways provides implementation-level insights into feasibility, safety monitoring, adherence patterns, and cost-sensitive integration strategies outside high-income academic centers. This experience highlights practical considerations for scalable neuromodulation models across diverse healthcare environments.

Thematic focus will include patient selection principles, integration with pharmacotherapy and psychotherapy, safety monitoring, and interdisciplinary workflow models. A live, device-neutral

demonstration will illustrate procedural fundamentals and operational considerations within structured clinical systems.

By synthesizing circuit neuroscience, evidence appraisal, and real-world implementation experience, this workshop situates peripheral cranial nerve stimulation within the evolving landscape of circuit-based psychiatry and global mental health innovation.

Educational objectives:
At the conclusion of this workshop, participants will be able to:

1. Explain the circuit-based model of psychiatric disorders, describing how dysfunction within cortico–limbic–autonomic networks contributes to mood, trauma-related, and attentional syndromes.

2. Describe the neuroanatomical and physiological mechanisms underlying Vagus, trigeminal, and cranial electrostimulation approaches, including projections to the nucleus tractus solitarius, locus coeruleus, dorsal raphe nuclei, and prefrontal–limbic circuitry.

3. Critically appraise the clinical evidence supporting invasive and non-invasive peripheral cranial nerve stimulation, including interpretation of study design, effect sizes, durability of response, safety data, and regulatory considerations.

4. Identify appropriate patient selection criteria and contraindications, including predictors of response, risk factors, and integration with pharmacological and psychotherapeutic treatment.

5. Apply structured clinical decision pathways to determine when peripheral neuromodulation may be biologically rational and clinically appropriate in cases of treatment resistance or autonomic dysregulation.

6. Demonstrate foundational practical competencies through live, device-neutral demonstration, including principles of electrode placement, parameter initiation, titration logic, safety monitoring, documentation standards, and patient counselling.

7. Evaluate real-world implementation data, including feasibility, adherence, safety monitoring, and cost-sensitive integration strategies from resource-constrained clinical settings, and assess scalability across diverse healthcare environments.

8. Engage in supervised hands-on exposure to diverse neuromodulation devices, gaining familiarity with device-specific operational principles, practical setup variations, and parameter adjustment strategies, while recognizing key regulatory frameworks, ethical considerations, documentation standards, and responsible clinical governance in the use of peripheral nerve stimulation technologies.

The workshop is designed to move beyond theoretical discussion and provide participants with a structured, evidence-informed, hands on and operationally feasible approach to incorporating peripheral cranial nerve stimulation into contemporary psychiatric practice.

WS-13
Hyperbaric oxygen therapy for post-traumatic stress disorder: from brain wounds to neuroplastic recovery

Chair: Keren Doenyas-Barak, Israel

Abstract

Workshop theme:
The workshop will explore hyperbaric oxygen therapy as a biologically targeted intervention for PTSD, addressing unmet clinical needs in patients with chronic and treatment-resistant symptoms. By combining neurobiological mechanisms, clinical trial data, cognitive outcomes, and early treatment-response markers, the workshop aims to bridge neuroscience and clinical psychiatry, offering a practical framework for understanding PTSD as a disorder of impaired brain recovery and neuroplasticity.

Educational objectives:
At the conclusion of this workshop, participants will be able to:

Describe key neurobiological alterations associated with PTSD and discuss their relevance to symptom chronicity and treatment resistance.

Explain the biological mechanisms underlying hyperbaric oxygen therapy (HBOT), with particular emphasis on the hyperoxic-hypoxic paradox and its effects on neuroplasticity, angiogenesis, inflammation, and mitochondrial metabolism.

Review and interpret current clinical evidence supporting the use of HBOT in PTSD, including findings from military and civilian populations with treatment-resistant symptoms.

Identify early indicators of treatment response to HBOT and discuss their potential role in patient stratification, treatment monitoring, and personalized therapeutic planning.

Evaluate the effects of HBOT on cognitive domains commonly impaired in PTSD, and relate cognitive changes to clinical and neurobiological outcomes.

Discuss the phenomenon of memory surfacing observed during HBOT, including proposed mechanisms, clinical implications, and considerations for therapeutic management.

Integrate mechanistic, clinical, and cognitive perspectives to develop a translational framework for understanding biologically targeted interventions in PTSD and trauma-related brain disorders.

WS-14
Neuroscience for Psychiatrists: Foundations and Clinical Applications

Organiser: Rajiv Tandon, USA

Co-Authors:

Matcheri Keshavan

Abstract

Advances in neuroscience over the past two decades have transformed our understanding of brain function and the neurobiology of psychiatric disorders. However, many early-career psychiatrists receive limited training in translating contemporary neuroscience concepts into clinical practice. This 3-hour pre-conference workshop is designed to provide a concise, clinically relevant overview of the neuroscience foundations of psychiatry.

The session will introduce key principles of brain organization, neural circuits, neurotransmission, and neuroplasticity, and discuss how alterations in these systems contribute to major psychiatric disorders including schizophrenia, mood disorders, anxiety disorders, and substance use disorders. Emphasis will be placed on linking modern neuroscience concepts such as circuit-based models of psychopathology and multi-neurotransmitter interactions, to clinical phenomena and treatment mechanisms.

Through focused lectures and illustrative clinical examples, the workshop aims to enhance neuroscience literacy among psychiatrists and mental health trainees and also to promote integration of contemporary neurobiological insights into diagnostic formulation and therapeutic decision-making

Learning Objectives

By the end of this workshop, participants will be able to:

1. Describe key principles of brain organization, neural circuits, neurotransmission, and neuroplasticity relevant to psychiatric practice.

2. Explain how disturbances in neural circuitry and neurochemical systems contribute to major psychiatric disorders such as schizophrenia, mood disorders, and anxiety disorders.

3. Apply contemporary neuroscience concepts to clinical formulation and to understanding mechanisms of action of psychiatric treatments.

Intended audience:

This workshop is intended for psychiatrists, psychiatry and other mental health trainees and medical students.

WS-15
Intergenerational Transmission of Psychiatric Disorders: Neurobiological Pathways from Pregnancy to Childhood

Chair: Rashmi Shukla, India

Co-Authors:

Bandna Gupta

Abstract

Workshop theme:
Psychiatric disorders often cluster within families due to a complex interplay of genetic predispositions, epigenetic factors, neuroendocrine mechanisms, and early environmental influences. Recent findings indicate that the transmission of risk can start as early as the intrauterine period. Maternal stress, depression, and anxiety experienced during pregnancy can affect fetal brain development through various biological mechanisms, including programming of hypothalamic–pituitary–adrenal (HPA) axis, inflammatory pathways, placental functions, and epigenetic changes(e.g. DNA methylation). These neurobiological changes can influence how offspring respond to stress, regulate their emotions, and develop cognitively, ultimately increasing their risk for neurodevelopmental and mood disorders. This workshop brings together findings from longitudinal birth studies, neuroimaging research, and molecular investigations to connect perinatal psychiatry with child mental health, focusing on prevention strategies that can benefit future generations.

Educational objectives:
By the end of this workshop, participants will be equipped to:

1. Describe essential neurobiological mechanisms related to the intergenerational transmission of psychiatric disorders, focusing on HPA axis dysregulation and inflammatory pathways. 2. Explain how epigenetic modifications connect prenatal stress to changes in offspring stress responses.

3. Analyze findings from neuroimaging and longitudinal cohort studies that investigate the link between prenatal maternal mental health and child outcomes.

4. Recognize potential opportunities for early screening and preventive interventions within perinatal and early childhood mental health services.

WS-16
Cognitive Training Based Psychological Intervention for Adult ADHD: Skills for daily living

Chair: Susmita Halder, India

Abstract

Workshop theme:
This workshop focuses on cognitive training–based psychological intervention for Adult ADHD, with an emphasis on addressing core cognitive and executive function deficits that contribute to functional impairment in adulthood. The theme integrates current theoretical models of Adult ADHD with evidence-based cognitive training approaches targeting attention regulation, working memory, inhibitory control, planning, and self-monitoring. Through a clinically oriented framework, the workshop highlights how structured cognitive training can be integrated with psychological principles to promote meaningful functional change in daily life, work, and interpersonal domains. Special attention is given to translating cognitive gains into real-world outcomes, individualizing interventions, and incorporating these approaches within comprehensive treatment plans for adults with ADHD.

The theme underscores a practical, evidence-informed, and lifespan-sensitive approach, making it relevant for clinicians seeking effective, skill-based interventions beyond symptom management.

Educational objectives:
Understanding the cognitive and executive function deficits underlying Adult ADHD and their impact on everyday functioning.

Highlighting the theoretical and evidence base for cognitive training–based psychological interventions in Adult ADHD.

Identify key components and techniques of integrated cognitive training programs applicable in clinical practice.

Apply cognitive training strategies in integration with other psychological techniques to enhance functional outcomes in adults with ADHD across real-world settings.

WS-17
Neurocognition In Bipolar Disorders - Assessment and Intervention

Chair: Muralidharan Kesavan, India

Co-Authors:
Preethi Veerappa Reddy
Rashmi Arasappa

Abstract

Workshop theme:
Over the last couple of decades, Neurocognition has emerged as an endophenotype and marker of neuroprogression in Bipolar Disorder (BD). It has been replicated in several studies that there are significant neurocognitive deficits even in the euthymic phase, and this leads to impaired functionality and thus poor quality of life.

With this background, the main theme of our workshop is for mental health professionals to be aware of the various cognitive domains that are affected in BD, and be able to screen for these deficits in their patients using simple bedside tests. Since pharmacological agents have not shown much efficacy in treating cognitive deficits, we also aim to introduce the various neuropsychological interventions in a case-based learning format so that mental health professionals can identify and intervene at early stages to improve cognitive deficits.

Educational objectives:
The educational objectives of the workshop:

1. To understand the various neurocognitive domains that are affected in Bipolar Disorder

2. To learn the screening tests for assessing neurocognition and understand how to interpret them

3. Case-based discussions of the neuropsychological interventions for neurocognitive deficits in Bipolar Disorder

WS-18

The non-motor brain in Parkinson's disease: Pharmacological strategies for the behavioral and cognitive challenges in PD

Chair: Debanjan Banerjee, India

Abstract

Workshop theme:
This interactive workshop will focus on the often-overlooked non-motor dimensions of Parkinson's disease, with special emphasis on cognitive impairment and behavioral manifestations such as depression, anxiety, apathy, psychosis, impulse control disorders, and sleep-related disturbances. Through a biologically informed and clinically pragmatic lens, the session will explore evidence-based pharmacological strategies targeting dopaminergic, serotonergic, cholinergic, and glutamatergic systems. Emphasis will be placed on rational drug selection, sequencing, dose optimization, and risk mitigation in older adults, integrating real-world case vignettes and recent advances to enhance personalized, brain-based care in Parkinson's disease.

Educational objectives:
1. To enhance recognition and neurobiological understanding of cognitive and behavioral manifestations in Parkinson's disease, beyond motor symptoms.

2. To provide practical, evidence-based pharmacological strategies for managing depression, psychosis, apathy, impulse control disorders, anxiety, and cognitive impairment in PD.

3. To guide rational medication selection, sequencing, and dose optimization, balancing efficacy with safety in older adults and comorbid populations.

4. To translate current evidence into day-to-day clinical decision-making using case-based discussions and common therapeutic dilemmas in PD.

WS-19

The Future Brain Physician: Integrating Neurodevelopment, Immunity, and Metabolism in Psychiatry

Chair: Prashant Chaudhari, India

Co-Authors:
Anshu Varma
Krishnapriya Murlimanohar

Abstract

Workshop theme:
This workshop proposes a systems-biology reorientation of psychiatric practice through the lens of Psycho-Neuro-Immuno-Endocrinology (PNIE). While contemporary psychiatry has benefited enormously from standardized diagnostic frameworks such as the DSM, clinical complexity increasingly reveals the limitations of purely categorical approaches. High genetic overlap across major disorders, frequent mixed phenotypes, fluctuating longitudinal trajectories, and variable treatment responses suggest that symptom-based classification alone does not fully capture underlying biology.

The workshop introduces a PNIE-informed model that conceptualizes psychiatric illness as dynamic dysregulation across interacting neural, immune, endocrine, autonomic, and metabolic systems. Rather than replacing existing diagnostic systems, this framework deepens biological formulation by focusing on mechanisms such as excitatory–inhibitory balance, neurodevelopmental architecture, glial maintenance and energy metabolism, stress-axis reactivity, circadian regulation, and inflammatory signaling.

Through clinical case discussions and translational neuroscience updates, participants will examine how a systems approach can clarify common dilemmas: antidepressant-induced activation, autonomic dysregulation in anxiety disorders, delirium as inflammatory–circadian collapse, psychosis as signal-to-noise failure, clozapine-related hematological interpretations, and the biological considerations of psychotropics in pregnancy.

The workshop also highlights emerging data in systems genomics, neuroinflammation, circadian biology, and receptor kinetics, demonstrating how these domains converge in real-world psychiatric care. Emphasis will be placed on integrating descriptive diagnosis with mechanistic reasoning, allowing clinicians to move from “What disorder is this?” to “Which biological systems are dysregulated in this patient?”

By situating psychiatry firmly within brain systems medicine, this workshop aims to bridge molecular research and bedside decision-making, fostering biologically coherent, personalized, and safer psychiatric care.

Educational objectives:
By the end of this workshop, participants will be able to:

Differentiate descriptive diagnosis from biological formulation, and articulate how DSM-based classification can be complemented by a systems neurobiology approach.

Explain the core components of the PNIE framework, including interactions among neural circuitry, immune signaling, endocrine stress systems, autonomic tone, metabolic regulation, and circadian rhythms.

Interpret common psychiatric presentations through systems biology, including:

Anxiety as autonomic hyperactivation

Depression as metabolic and inflammatory conservation states

Psychosis as excitatory–inhibitory integration imbalance

Delirium as inflammatory–circadian dysregulation

Apply PNIE-informed reasoning to clinical dilemmas, such as:

Antidepressant activation versus bipolar conversion

Autonomic contributors to treatment resistance

Biological interpretation of hematological changes during clozapine therapy

Risk–benefit considerations of psychotropics during pregnancy

Incorporate emerging biological evidence into clinical practice, including insights from systems genomics, receptor pharmacodynamics, neuroimmune research, and circadian neuroscience.

Develop a longitudinal systems perspective, recognizing that psychiatric illness reflects dynamic biological trajectories rather than static categorical states.

Participants will leave with practical tools to integrate mechanistic reasoning into routine psychiatric care while maintaining alignment with established diagnostic frameworks and evidence-based practice.

WS-20
Spirituality, Psychiatry, and Epigenetics: Translating Gene–Environment Science into Clinical Practice

Chair: Shraddha Jadhav, India

Abstract

Workshop theme:
This interactive workshop explores how spirituality-informed clinical practices can influence gene–environment interactions through epigenetic mechanisms relevant to psychiatric disorders. While genetics establishes vulnerability, epigenetics provides a dynamic interface between stress, trauma, resilience, and recovery. Emerging evidence suggests that structured contemplative practices—such as meditation, breath regulation, compassion training, and meaning-centered psychotherapy—may modulate inflammatory pathways, stress-response systems (HPA axis), autonomic regulation, and gene expression markers.

Participants will engage in structured skill-building modules to understand how spirituality-informed interventions can be integrated into psychiatric formulation, assessment, and treatment planning within an evidence-based framework. Case discussions and guided exercises will demonstrate how clinicians can incorporate spiritual history-taking, stress-biology mapping, and resilience-focused interventions into routine psychiatric care without compromising scientific rigor.

Educational objectives:
By the end of the workshop, participants will be able to:

1. Explain the role of epigenetic mechanisms (DNA methylation, histone modification) in psychiatric disorders.

2. Describe how stress and trauma influence gene expression and psychiatric vulnerability.

3. Evaluate current evidence linking contemplative practices with biological and epigenetic modulation.

4. Apply a structured spirituality-informed clinical model to psychiatric case formulation.

5. Develop ethically sound, culturally sensitive approaches to integrating spirituality into practice.

Interactive Format

- Case-based discussions
- Small group formulation exercises
- Guided experiential demonstration (breath-based autonomic regulation)
- Clinical framework worksheets for implementation

WS-21
The plasticity of healing: modifying the traumatized child's brain biology via trauma informed therapies

Chair: Sadiya Mohammad, India

Co-Authors:

Nahid Dave

Abstract

Workshop theme:
This workshop centers on understanding childhood trauma through a neurobiological, developmental, and relational lens, and explores the integrative use of Arts-Based Therapy (ABT), somatic work, Non-Directive Play Therapy (NDPT), and Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) in supporting recovery.

Childhood trauma including experiences of abuse, neglect, loss, chronic instability, or exposure to violence can significantly affect emotional regulation, behavior, learning, attachment, and physiological stress responses. Rather than viewing behavioral difficulties as oppositional or maladaptive, a trauma-informed perspective recognizes them as adaptive survival responses shaped by the child's nervous system. Effective intervention therefore prioritizes safety, co-regulation, and empowerment before engaging in structured cognitive processing.

The workshop will examine how Arts-Based Therapy, somatic interventions, and Non-Directive Play Therapy support trauma recovery in developmentally responsive ways alongside the structured framework of TF-CBT. Many children do not possess the verbal capacity to articulate traumatic experiences directly. Trauma is often encoded in sensory, somatic, and implicit memory systems; therefore, expressive modalities such as drawing, music, rhythm, storytelling, movement, somatic, sensorial and symbolic play allow children to externalize internal experiences, regulate physiological activation, process overwhelming affect, and regain a sense of control and agency.

TF-CBT will be discussed as an evidence-based, structured approach addressing psychoeducation, emotional regulation, trauma narrative development, and cognitive restructuring. In parallel, Non-Directive Play Therapy provides a relationally safe space in which the child leads the therapeutic process, facilitating mastery, emotional expression, and corrective relational experiences. Somatic work supports nervous system regulation by enhancing body awareness and completing stress responses, while Arts-Based Therapy offers symbolic and creative pathways for meaning-making.

The workshop emphasizes an integrative, trauma-informed practice in which these modalities are used thoughtfully and ethically based on the child's developmental stage, readiness, and presenting needs, rather than as a single prescriptive model

Educational objectives:
-Develop a neurobiological understanding of trauma and its impact on the developing brain and body.

-Identify trauma responses as they manifest in behavior and somatic presentation.

-Apply core principles of trauma-informed practice (safety, trust, collaboration, choice, empowerment).

-Demonstrate practical applications of Arts-Based Therapy (ABT), Somatic Work, Non-Directive Play Therapy (NDPT), and Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) as distinct yet complementary trauma interventions.

-Analyze case illustrations from school and community mental health settings.

-Understand the neurobiological changes induced by art-based expressive therapy.

WS-22
Indian Classical Music Therapy for Borderline Personality Disorder

Chair: Avinash Desousa, India

Co-Authors:

Seetharaman Iyer

Abstract

Workshop theme:
Borderline Personality Disorder (BPD) is characterized by pervasive instability in affect, interpersonal relationships, self-image, and marked impulsivity. Impulsivity—manifesting as self-harm, substance misuse, aggression, and suicidal behaviours—remains a major driver of morbidity and emergency service utilization. While evidence-based psychotherapies such as Dialectical Behaviour Therapy (DBT) and Mentalization-Based Therapy (MBT) have demonstrated efficacy, challenges related to accessibility, cultural acceptability, and persistent residual symptoms highlight the need for adjunctive, low-cost, culturally rooted interventions—particularly in low- and middle-income settings.

This workshop presents an innovative, research-informed model examining Indian Classical Music (ICM), specifically Carnatic music, as an adjunctive therapeutic intervention for BPD. Drawing upon neuroscientific evidence that music modulates autonomic arousal, cortical oscillations, and emotional salience networks, the workshop explores how structured rāga-based exposure may influence impulsivity, mood dysregulation, anxiety, and suicidality. Emerging literature suggests that Indian Classical Music, deeply embedded in affective evocation and regulation traditions, may offer a biologically plausible and culturally congruent therapeutic modality.

The workshop will outline the design of a controlled interventional study comparing pharmacological treatment alone with pharmacological treatment plus a structured 15-day ICM intervention. Standardized psychometric tools—including the Barratt Impulsiveness Scale (BIS-11), Montgomery–Åsberg Depression Rating Scale (MADRS), Hamilton Anxiety Rating Scale (HAM-A), and Suicide Behaviours Questionnaire–Revised (SBQ-R)—were used to evaluate changes across core symptom domains.

Educational objectives:
1. To understand the neurobiological and psychological rationale for using Indian Classical Music in emotion regulation and impulsivity modulation.

2. To examine the design and implementation of a structured ICM-based adjunctive intervention for BPD.

3. To analyze clinical outcome measures assessing impulsivity, mood symptoms, anxiety, and suicidality.

4. To explore feasibility, cultural acceptability, and scalability of rāga-based therapeutic modules in psychiatric practice.

WS-23
Pharmacological Optimization in Schizophrenia: From Neurobiological Hypotheses to Clinical Polypharmacy

Chair: Kristian Sæthre, Norway

Abstract

Workshop theme:
Optimizing schizophrenia treatment through a deeper understanding of biological mechanisms and the clinical application of evidence-based drug combinations.

Educational objectives:
To begin, we will examine some central hypotheses of schizophrenia, including the dopaminergic, glutamatergic, and serotonergic hypotheses. We will then explore the proposed mechanisms of action for key antipsychotic medications. A particular focus will be placed on clozapine.

Leveraging our theoretical understanding of these core hypotheses and the pharmacological effects of different agents, we will discuss synergistic medication combinations. Subsequently, we will review key clinical evidence regarding antipsychotic polypharmacy, emphasizing how specific drug combinations can improve treatment outcomes and symptom management. We will specifically examine the use of clozapine and amisulpride within the context of combined antipsychotic therapy.

Finally, we invite participants to share clinical insights on optimizing therapy. Our goal is to foster a productive dialogue on pharmacological optimization in schizophrenia and facilitate an exchange of expertise among all attendees.

WS-24
Advancing neuromodulation in psychiatry: emerging non-invasive brain stimulation (nibs) and precision techniques for depression

Chair: Rohit Verma, India

Co-Authors:

Shraddha Verma

Abstract

Workshop theme:
Non-invasive brain stimulation (NIBS) is an emerging treatment modality that enables the modulation of neural circuits underlying psychiatric and neurocognitive disorders. There is growing clinical utility of techniques such as repetitive transcranial magnetic stimulation (rTMS), theta burst stimulation (TBS), and transcranial direct current stimulation (tDCS), particularly in treatment-resistant depression (TRD) and other difficult-to-treat conditions. Moreover, advances in neuroimaging and electrophysiology are enabling increasingly precise targeting of brain networks. These advances are moving the field toward personalised treatment.

This workshop aims to provide a clinically oriented overview of both the established and emerging NIBS techniques. The focus will be on the mechanisms of action, clinical efficacy, and treatment applications in depression and other disorders. In addition, participants will learn about accelerated stimulation protocols, neuro-navigation, and biomarker-informed treatment strategies. The workshop will provide in-depth presentations, clinical case discussions, and interactive engagement with participants. The workshop aims to bridge advancing techniques with real-world clinical practice, highlighting the role of neuromodulation in modern psychiatric treatment.

Educational objectives:
1. Understand the neurobiological mechanisms of action of rTMS, TBS, and tDCS is modulating neural circuits and inducing neuroplasticity.

2. Discuss the current evidence base for NIBS interventions in depression and other disorders.

3. Highlight the advances in precision neuromodulation including neuro-navigation, neuroimaging-guided targeting, and accelerated stimulation protocols such as SAINT protocol.

4. Apply best practices for clinical application including patient selection, safety considerations, and integration with pharmacotherapy and psychotherapy.

5. Recognise emerging techniques and future directions in circuit-based psychiatry.

6. In depth understanding of NIBS with interactive session using Q&A

WS-25
WFSBP Young Investigators Workshop 2026

WS-25-001
Designing Translational Studies in Psychiatry

Organiser: Andreas Reif, Germany

Abstract

Despite a plethora of studies in fundamental neuroscience, advances in the treatment of mental disorders are only sparse: at best, we saw 5-10 new treatments in psychiatry in the last two decades which falls short of the progress we see in other disciplines. The reasons for that are manifold, include the complexity of the brain and its disorders, but also and most importantly an insufficient back- and forward translation. First, the use of "an animal model for [add your mental disorder of choice]" is by far too simplistic and does not account for the heterogeneity of mental disorders, as well as human-specific neurobiological mechanisms; second, psychiatry itself falls short to put forward a mechanism-based framework for mental disorders but rather relies on atheoretical diagnostic categories; to name but a few fundamental issues. Translational research in psychiatry will only succeed if specific mechanisms in animal and cell models are targeted and influenced and then are taken forward in the human model in a mechanism, but not diagnosis based framework; in reverse, precision psychiatry relies on specific mechanisms, that will be implied in only a subgroup of patients, but not on inference-based differences of means. in this workshop, these more fundamental and conceptional issues will be elaborated; their implications for translational studies will be discussed; and finally, we will suggest promising ways forward to overcome the "translational valley of death".

WS-25
WFSBP Young Investigators Workshop 2026

WS-25-002
Mastering the Art of High Impact Publishing

Organiser: Suhas Satish, Germany

Abstract

In an era of "publish or perish," the difference between a desk rejection and an acceptance in a top-tier journal often lies in the invisible nuances of manuscript strategy rather than just the raw data. This interactive workshop is designed for early-career researchers and clinicians who aim to elevate their academic footprint. Moving beyond basic writing tips, the session delves into the "editorial mind" i.e., understanding what high-impact biological psychiatry journals seek in a submission. Participants will explore the anatomy of a compelling manuscript, learn to pre-emptively address common reasons for rejection, and master the art of the rebuttal. Through practical exercises and real-world examples, this workshop provides a roadmap for navigating the complex journey from a finished draft to a published paper in a premier global journal.

Learning Objectives

By the end of this workshop, participants will be able to:

1. Strategize Submission Choice: Identify the hallmarks of "high-quality" journals and align their research narrative with specific editorial scopes.

2. Optimize Manuscript Structure: Implement advanced structural techniques to enhance the clarity, impact, and "readability" of their scientific arguments.

3. Navigate the Editorial Gauntlet: Understand the internal workflow of high-impact journals to better manage peer-review feedback and editorial expectations.

4. Master the Rebuttal: Construct professional, evidence-based responses to reviewers that increase the likelihood of final acceptance.

WS-26
Motivational Interviewing in Addiction Treatment: A Hands-on Skills Workshop

Chair: Nandhini Ponnuswamy Bojappen, India

Co-Authors:

Babli Kumari

Abstract

Workshop theme:
Substance use disorders are frequently characterized by ambivalence toward change, fluctuating motivation, and repeated cycles of relapse and recovery. Traditional directive approaches often encounter resistance when individuals are uncertain about modifying their substance use behaviors. Motivational Interviewing (MI), a collaborative, person-centered counseling approach, has emerged as an evidence-based method to enhance intrinsic motivation for change while respecting patient autonomy.

This workshop aims to introduce participants to the principles and practical application of Motivational Interviewing in the assessment and treatment of addiction. Grounded in the core MI spirit of partnership, acceptance, compassion, and evocation, the session will demonstrate how clinicians can effectively engage individuals with substance use disorders, explore ambivalence, and facilitate meaningful behavioral change.

The workshop will provide a concise overview of the theoretical foundations of MI, including the stages of change model and the role of change talk in promoting recovery. Participants will learn key MI skills such as open-ended questioning, affirmations, reflective listening, and summarizing (OARS), as well as strategies to recognize and strengthen change talk while responding constructively to sustain talk and resistance.

Through interactive demonstrations, case discussions, and structured role-play exercises, participants will gain hands-on experience applying MI techniques to common clinical scenarios encountered in addiction treatment settings. The session will also highlight the integration of MI with pharmacological and psychosocial interventions in addiction psychiatry.

By the end of the workshop, participants will develop practical communication skills that can enhance therapeutic engagement, strengthen treatment adherence, and support sustained recovery in individuals with substance use disorders.

Educational objectives:
Describe the core principles and spirit of Motivational Interviewing (MI) and its role in enhancing motivation for change in individuals with substance use disorders.

Identify and apply key MI communication skills—including open-ended questions, affirmations, reflective listening, and summarizing (OARS)—to facilitate patient-centered conversations about substance use.

Recognize and respond effectively to ambivalence, sustain talk, and resistance, while eliciting and strengthening change talk during clinical interactions.

Integrate Motivational Interviewing techniques into routine addiction treatment settings, including during assessment, brief interventions, and ongoing management alongside pharmacological and psychosocial treatments.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-001
Beyond primary psychosis: recurrent psychotic episodes secondary to acute intermittent porphyria

Presenter: Surabhi Gupta, India

Co-Authors:
Sandeep Grover
Ishita Upadhyay

Topic:
1) Psychoses, 10) Other

Abstract

Objective:
Acute Intermittent Porphyria is a rare disorder of heme biosynthesis characterized by neurovisceral and psychiatric manifestations. Psychiatric symptoms may mimic primary psychotic disorders, resulting in delayed diagnosis and inappropriate treatment. We report a case of recurrent psychosis associated with genetically confirmed Acute Intermittent Porphyria, highlighting the importance of evaluating organic causes in atypical psychosis.

Methods:
A 21-year-old female presented with recurrent psychotic episodes characterized by delusions, hallucinations, irritability, and disorganized behaviour, with complete inter-episodic recovery. She had three similar episodes over five years, each lasting 1–1.5 months and associated with abdominal pain and vomiting suggestive of porphyria. Clinical, biochemical, genetic, and psychopharmacological evaluations were reviewed.

Results / Discussion:
The latest episode was preceded by abdominal pain, vomiting, and constipation. Investigations revealed hyponatremia, elevated liver enzymes, raised urinary porphobilinogen, and a pathogenic hydroxymethylbilane synthase gene mutation confirming Acute Intermittent Porphyria. The patient improved with carbohydrate loading and non-porphyriogenic psychotropics, with Brief Psychiatric Rating Scale scores reducing from 54 to 23.

Conclusion:
This case highlights the importance of considering Acute Intermittent Porphyria in recurrent atypical psychosis with gastrointestinal symptoms and complete inter-episodic recovery. Evaluating organic causes of psychosis is essential to avoid diagnostic delays and inappropriate treatment. Early recognition and multidisciplinary management can improve outcomes.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-002
Neurobiology Beyond Neurotransmitters: Structural Brain Pathology as a Substrate for Treatment-Resistant Psychosis — Two Rare Case Reports

Presenter: Sumeet Kaur Madaan, India

Co-Authors:
Ranjit Singh
Manveer Kaur
Suresh Chand Gocher
Chanendra Sablania

Topic:
1) Psychoses, 10) Other

Abstract

Objective:
Psychosis is overwhelmingly understood through the lens of neurotransmitter dysregulation — yet this framework fails a clinically significant subset of patients. We aimed to examine structural brain pathology as an independent, non-neurotransmitter substrate for psychosis, using two rare cases to interrogate the limits of receptor-targeted pharmacotherapy and argue for a broader neurobiological model of treatment-resistant psychotic illness.

Methods:
Two cases from a tertiary psychiatry centre with treatment-resistant psychosis underwent psychiatric assessment, cognitive testing, neurological examination, EEG, and NCCT brain. Case 1: a 42-year-old female with adult-onset psychosis and congenital hemiparesis. Case 2: a 25-year-old male with childhood-onset refractory epilepsy and florid psychosis. Diagnosis followed established clinical criteria.

Results / Discussion:
Case 1: NCCT revealed open-lip schizencephaly. Olanzapine 20 mg, Risperidone 8mg, Trifluoperazine 20mg, and Valproate failed — paranoia and disorganisation persisted. Case 2: NCCT showed unihemispheric atrophy — diagnosed with Rasmussen's encephalitis in liason with neurology department. Levetiracetam 2g and Valproate 2g failed seizures; antipsychotics barely touched psychosis. Treatment pivoted to immunomodulatory steroids.

Conclusion:
Psychosis is not always a disease of synapses. Structural brain pathology — neurodevelopmental and neuroinflammatory — can independently drive treatment-resistant psychosis beyond pharmacological reach. Pharmacological failure here was mechanistic, not a

matter of dose. Neuroimaging is not optional in refractory psychosis — it is the question we forgot to ask. These cases demand aetiological reclassification over antipsychotic escalation.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-003
Psychosis at the crossroads of neurology and psychiatry: a case of seizure disorder with behavioural disturbance

Presenter: Sanjeev Sanjeev, India

Co-Authors:
Tanu Kundal
Ibadat Anmol Singh

Topic:
1) Psychoses, 10) Other

Abstract

Objective:
To present a rare and diagnostically challenging case of seizure disorder with psychosis in a young female presenting with acute behavioural disturbances, psychotic symptoms, seizure episodes, and MRI brain abnormalities, highlighting the importance of detailed neuropsychiatric evaluation and multidisciplinary management in differentiating organic psychosis from primary psychiatric disorders.

Methods:
Detailed psychiatric and neurological evaluations were performed in a 19-year-old female presenting with psychotic symptoms and seizure episodes. Mental status examination, MRI brain, and clinical rating scales (BPRS and YMRS) were used for assessment. Clinical findings and treatment response were analyzed through a multidisciplinary neuropsychiatric approach.

Results / Discussion:
The patient presented with acute psychotic symptoms and seizure episodes with postictal confusion. MRI brain showed bilateral fronto-parietal T2/FLAIR hyperintensities. Based on clinical and neuropsychiatric evaluation, a diagnosis of seizure disorder with psychosis was made. The patient improved with antipsychotic and antiepileptic treatment.

Conclusion:
This case emphasizes the importance of considering seizure-related psychosis in young patients presenting with acute psychotic symptoms and behavioural disturbances. Early neuropsychiatric evaluation and multidisciplinary management are essential for accurate diagnosis and improved clinical outcomes.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-005
The Nutritional Fingerprint of First Episode Psychosis: Vitamin B12, Vitamin D and Symptom Burden

Presenter: Sumeet Kaur Madaan, India

Co-Authors:
Ranjit Singh
Manveer Kaur
Suresh Chand Gocher

Topic:
1) Psychoses

Abstract

Objective:
Vitamin B12 — essential for dopamine synthesis, one-carbon methylation, and myelin integrity, is critically implicated in psychosis neurobiology. Evidence that B12 acts as a graded severity determinant in first episode psychosis (FEP) remains lacking. This study assessed: (1) B12 and Vitamin D deficiency prevalence in FEP versus controls; (2) the inverse association between serum B12 and BPRS severity; and (3) the combined clinical impact.

Methods:
Case-control study; tertiary psychiatric Centre, North India (N=120: 60 FEP patients [DSM-5] and 60 matched controls). Serum B12 and Vitamin D measured by chemiluminescent immunoassay (deficiency: B12<200 pg/mL; Vitamin D<20 ng/mL). Severity by BPRS. Analyses: chi-square, Fisher's exact, Spearman/Pearson correlations, ANOVA (SPSS v26). Ethics approved; informed consent obtained

Results / Discussion:
B12 deficiency more prevalent in FEP (48.3%) vs. controls (26.7%; OR=2.57, p=0.023). Serum B12 inversely correlated with BPRS severity (Spearman ρ=−0.300, p=0.020; Pearson r=−0.281, p=0.030; ANOVA p=0.047). Vitamin D deficiency linked to longer illness duration (28.2 vs. 15.1 months; p=0.005). Combined deficiencies worsened BPRS burden (F=3.50, p=0.037; d=0.75). Vitamin D alone did not differentiate groups (p=1.000).

Conclusion:
B12 deficiency is more prevalent in FEP and inversely correlated with severity, supporting a biologically graded nutritional vulnerability model. Vitamin D links to prolonged illness duration, not case status. Combined deficiencies worsen symptom burden. Routine B12 screening at FEP is recommended. Supplementation trials are warranted.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-006
Methylphenidate induced psychosis in adolescent with attention deficit hyperactivity disorder

Presenter: Niharika Bawankar, India

Co-Authors:
Paramjeet Singh
Vipul Sharma

Topic:
1) Psychoses, 10) Other

Abstract

Objective:
To report a rare case of methylphenidate-associated psychosis in an adolescent girl with ADHD and to highlight the importance of monitoring for psychotic symptoms during stimulant treatment.

Methods:
A case study of a 13-year-old female diagnosed with severe ADHD (Combined Presentation) according to DSM-5, who had been receiving sustained-release methylphenidate for approximately three years. Clinical history, mental status examination, physical examination, laboratory investigations, treatment modifications, and follow-up outcomes were documented and analysed.

Results / Discussion:
A 13-year-old girl with ADHD developed progressive auditory hallucinations after approximately three years of treatment with methylphenidate SR 20 mg/day. Clinical evaluation and investigations revealed no alternative medical or psychiatric cause. Reducing methylphenidate to 10 mg/day did not resolve symptoms. Following initiation of risperidone 2 mg/day and clonidine 0.05 mg/day, hallucinations improved significantly within four weeks.

Conclusion:
Methylphenidate-induced psychosis is a rare but important adverse effect in children and adolescents with ADHD. This case highlights the emergence of auditory hallucinations during long-term methylphenidate treatment and their subsequent improvement following pharmacological intervention. Clinicians should closely monitor for psychotic symptoms during stimulant therapy to ensure timely identification and appropriate management.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-007
Atypical psychiatric presentations of Neurocysticercosis:A case series with review of literature highlighting Mania and schizophrenia like psychosis

Presenter: Rashmi Shukla, India

Co-Authors:

Bandna Gupta

Topic:
1) Psychoses

Abstract

Objective:
Neurocysticercosis (NCC) is a prevalent neuroparasitic infection in endemic regions, primarily associated with seizures, but increasingly recognized as a cause of severe psychiatric syndromes. Biological mechanisms linking NCC to psychopathology remain underexplored. This case series aims to characterize atypical psychiatric phenotypes of NCC and to examine the neurobiological pathways underlying psychiatric disorders.

Methods:
Case one involved a 20-year-old male with acute-onset, treatment-resistant manic symptoms, later found to have an inflammatory NCC lesion in the right temporal lobe. Case two involved an 18-year-old female with insidious schizophrenia-like psychosis and seizure recurrence, associated with a reactive NCC lesion in the left occipital lobe. Multimodal assessment included standardized psychiatric rating scales, neuroimaging, neurological evaluation.

Results / Discussion:
Both cases demonstrated limited response to conventional psychotropic treatment alone. Neuroimaging identified NCC lesions with significant perilesional edema, leading to reclassification as psychiatric disorders due to a medical condition. Targeted biological interventions showed improvement in psychiatric symptoms.

Conclusion:
These findings support a neuroinflammatory and network-disconnection model of NCC-related psychopathology. NCC represents a biologically mediated, potentially reversible cause of severe psychiatric illness. Early neuroimaging, inflammatory modulation, and integrative neuropsychiatric management are critical for accurate diagnosis and optimal outcomes in atypical psychiatric presentations.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-008
Isolated Delusion of Time in Post-Traumatic Psychosis

Presenter: Madihah Faisal, India

Co-Authors:
Lahari R
Virupaksha Harave

Topic:
1) Psychoses

Abstract

Objective:
Traumatic brain injury is associated with neuropsychiatric sequelae including psychotic disorders. Temporal disorientation is typically attributed to delirium or neurocognitive disorders, whereas focal disturbances of a single aspect of cognition such as delusion of time are rarely described. We report a case of persistent delusion of time following TBI with structural and electrophysiological correlates.

Methods:
A 57-year-old woman with progressive social withdrawal sustained a head injury with retrograde amnesia and behavioural change. She attended work on Sunday believing it to be Saturday and remained at home Monday believing it to be Sunday. The belief was unshakeable with absent insight. MRI demonstrated left frontotemporal gliotic encephalomalacic changes and EEG showed left frontotemporal spike-wave discharges.

Results / Discussion:
MMSE showed intact cognition except for the temporal delusion with a score of 29/30. No hallucinations, mood symptoms, or fluctuating consciousness were present. Treatment with risperidone led to gradual resolution of symptoms and functional recovery over 6 months. The patient remained stable on treatment.

Conclusion:
Temporal disorientation does not necessarily indicate delirium or dementia. Selective disturbances of temporal belief can emerge from focal frontotemporal injury. Such presentations broaden the phenomenology of post-traumatic psychosis and emphasise modular cognitive networks underlying time perception. Early recognition and treatment may prevent prolonged occupational disability.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-009
A prospective study of determinants of short-term treatment response in early onset psychosis.

Presenter: Madhushree Baruah, India

Co-Authors:
Eesha Sharma
Urvakhsh M Mehta
K. John Vijay Sagar
Binu Vs

Topic:
1) Psychoses

Abstract

Objective:
To prospectively examine the demographic, developmental, and clinical predictors of short-term (6-12 weeks) response to antipsychotic treatment.

Methods:
Cases were subtyped using latent class analysis into four groups based on duration and severity. Treatment response was assessed at 6–12 weeks using change in BPRS-C scores and responder status. Associations were examined using bivariate analysis, linear regression, group comparisons, and binomial logistic regression to identify independent predictors.

Results / Discussion:
Greater BPRS-C change was associated with higher baseline severity ($r = 0.583$, $p < .001$) and shorter illness duration ($r = -0.265$, $p = .007$). Linear regression explained 42.7% variance. Responders differed in severity class, illness duration, and education. Duration of illness independently predicted response ($p = .04$, OR = 1.05).

Conclusion:
Shorter duration of illness and greater symptom severity are significant predictors of short-term treatment response. Duration of illness is potentially modifiable, and early identification and timely intervention may improve outcomes in early-onset psychosis.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-010
The ants never left- a 40 year untreated psychosis with delusional parasitosis

Presenter: Kamna Dadheech, India

Co-Authors:

Arun Bhat

Topic:
1) Psychoses

Abstract

Objective:
This poster highlights how long term untreated psychosis remains a challenge in society leading to secondary medical complications. It demonstrates how prolonged disengagement from mental health services can contribute to delusions becoming consolidated over time, poor quality of life and psychosocial decline. The objective is to underscore the importance of early intervention, continuity of care and extensive medical psychiatric approach.

Methods:
A 66-year-old man with 40 years of untreated psychosis due to poor insight presented with severe pruritus and infected eczema from scratching caused by delusional parasitosis. Illness began with suspiciousness and hallucinations, later progressing to somatic delusions, skin picking, and decline in functioning with negative symptoms. Combined psychiatric and dermatologic treatment reduced psychosis and scratching behaviour.

Results / Discussion:
This case illustrates the consequences of prolonged untreated psychosis, resulting in systematisation of delusional parasitosis, prominent negative symptoms, and dermatological complications. Years of untreated illness resulted in entrenched infestation beliefs, emphasizing the need for early, sustained and collaborative medical psychiatric care.

Conclusion:
Prolonged untreated psychosis may lead to deep seated medical complications and delusional parasitosis. Early diagnosis, sustained treatment, family involvement, and holistic approach is essential to prevent disability.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-011
Cumulative incidence of schizophrenia-spectrum disorders in children and adolescents with neurodevelopmental disorders

Presenter: Taeyoung Lee, Republic of Korea

Topic:
1) Psychoses

Abstract

Objective:
Adolescent-onset schizophrenia is rare but clinically severe. While neurodevelopmental disorders (NDDs) are recognized antecedents, the absolute risk of transition to psychosis in pediatric NDD clinic populations has not been clearly quantified relative to other psychiatric conditions.

Methods:
We conducted a retrospective cohort study using electronic medical records from Pusan National University Children's Hospital (2008-2023). We compared the cumulative incidence of schizophrenia-spectrum disorders between a cohort of youth diagnosed with NDDs (N = 558) and an internal clinical control group of youth treated for non-neurodevelopmental psychiatric disorders (N = 824) during the same period.

Results / Discussion:
The 10-year cumulative incidence of schizophrenia-spectrum disorders was significantly higher in the NDD group (2.3 %, 13/558) compared to the internal clinical control group (0.1 %, 1/824) (Log-rank test, $p < 0.01$). Sensitivity analysis in the ADHD subgroup found no association between stimulant medication use and psychosis onset.

Conclusion:
Youth with NDDs in a tertiary care setting face a distinct and significantly higher risk of developing schizophrenia-spectrum disorders compared to psychiatric controls. These findings underscore the importance of routine monitoring for emerging psychotic symptoms in this high-risk population.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-012
Long acting injectable antipsychotics predict treatment response to electroconvulsive therapy in psychosis

Presenter: Kyuyoung Lee, Republic of Korea

Co-Authors:

Heewon Jeong

Topic:
1) Psychoses

Abstract

Objective:
The objective of this study is to evaluate clinical outcomes for LAI use group and non-use group with psychosis patients treated with ECT. The study aimed to investigate predictors of treatment response.

Methods:
The study conducted a retrospective chart review of patients with psychosis who received ECT at Nowon Eulji Medical center, Eulji university, South Korea. The clinical outcomes were defined as decrease in the Clinical Global Impression - Severity scale scores. $\chi 2$ test was used to compare treatment response to ECT with LAI and without LAI. Multivariate logistic regression was employed to identify risk factors.

Results / Discussion:
In total, 64 subjects underwent ECT during the study period and 48.5% of patients were treated concurrently with both ECT and LAI. There was no difference in CGI-S scores between the two groups at baseline. Factors associated with a better response to ECT were LAI (P=0.04) and history of ECT (P=0.02), and factors not associated with treatment response included age, diagnosis, use of Clozapine, and history of hospitalization (P>0.05).

Conclusion:
In this study, LAI and history of ECT are associated with treatment response among patients treated by ECT. The LAI reliably predicts the treatment response in patients who are psychosis with ECT.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-013
Pharmacogenetic Testing of DRD3 and ABCB1 Polymorphisms in Clozapine-Induced Obsessive–Compulsive Symptoms

Presenter: Arshia Sood, India

Co-Authors:
Sandeep Grover
Naveen M
Amol Patil

Topic:
2) Schizophrenia, 10) Other

Abstract

Objective:
To examine the association of clozapine-induced obsessive-compulsive symptoms (OCS) or obsessive-compulsive disorder (OCD) with drug transport mechanisms via ABCB1 (rs1045462) and dopaminergic pathways via DRD3 (rs6280) single nucleotide polymorphisms (SNPs)

Methods:
A total of 200 treatment-resistant schizophrenia patients receiving clozapine underwent genetic subtyping for ABCB1 and DRD3 variants as part of a pharmacogenetic cohort. Clinical records of these patients were reviewed for new-onset OCS after clozapine initiation. Patients with pre-existing OCS were excluded. Data from 168 patients were analyzed, of whom 12 developed clozapine-associated OCS/OCD.

Results / Discussion:
New onset OCS/OCD was seen in 7.1% of patients. There was no significant association between OCS/OCD and either DRD3 ($\chi^2 = 2.299$, $p = 0.317$) or ABCB1 ($\chi^2 = 2.334$, $p = 0.311$) SNPs. Clinically, OCS/OCD appeared at a mean clozapine dose of ~267 mg/day and after a prolonged duration of treatment (~89 months). Improvement was observed with the addition of a selective serotonin reuptake inhibitor (SSRI) or clozapine dose adjustment.

Conclusion:
This study does not support a major role for DRD3 or ABCB1 SNPs in clozapine-induced OCS. Clinical findings of delayed onset, dose-dependence, and SSRI responsiveness suggest that clozapine-induced OCS is more likely driven by pharmacodynamic & pharmacokinetic mechanisms (e.g., serotonergic effects) than by genetic vulnerability alone. These findings warrant replication in larger samples to better elucidate the underlying mechanisms.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-014
Psychosis Beyond Intoxication: A Case of Evolving Treatment-Resistant Schizophrenia

Presenter: Sanjeev Sanjeev, India

Co-Authors:
Tanu Kundal
Ibadat Anmol Singh

Topic:
2) Schizophrenia, 1) Psychoses

Abstract

Objective:
To highlight the diagnostic challenge in differentiating cannabis-induced psychosis from paranoid schizophrenia and to demonstrate the progression of persistent psychotic symptoms into treatment-resistant schizophrenia requiring Clozapine therapy.

Methods:
This case report was based on detailed clinical evaluation, longitudinal assessment of psychotic symptoms and substance use, serial mental status examinations, collateral history from caregivers, and review of previous treatment records and investigations. Diagnostic formulation was made according to ICD clinical criteria with assessment for treatment-resistant schizophrenia.

Results / Discussion:
Longitudinal assessment revealed persistent psychotic symptoms, progressive socio-occupational dysfunction, and poor treatment response despite reduction in cannabis use and adequate antipsychotic trials, favouring a diagnosis of treatment-resistant paranoid schizophrenia over cannabis-induced psychotic disorder, leading to consideration of Clozapine therapy.

Conclusion:
This case highlights the diagnostic challenge in differentiating cannabis-induced psychosis from paranoid schizophrenia and emphasizes the importance of longitudinal assessment in identifying treatment-resistant schizophrenia requiring Clozapine therapy.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-015
Home-Based Bilateral Prefrontal tDCS Combined With Cognitive Emotional Training for Negative Symptoms of Schizophrenia: A Pilot Study

Presenter: Anindo Mitra, India

Topic:
2) Schizophrenia, 1) Psychoses

Abstract

Objective:
Meta-analytic evidence supports tDCS benefit for negative symptoms under specific stimulation conditions. This study evaluated home-based remotely supervised tDCS using a bilateral prefrontal F3-F4 montage, combined with Cognitive Emotional Training (CET), in patients with schizophrenia.

Methods:
Seven patients with schizophrenia underwent 20 sessions of home-based bilateral prefrontal tDCS (2 mA, 20 min; F3 anode, F4 cathode), paired with CET, a structured gamified paradigm targeting emotional processing, working memory, attention, and processing speed. Outcomes included PANSS positive and negative subscale scores, frontal EEG biomarkers, and CET task-performance indices.

Results / Discussion:
PANSS-negative scores improved from 29.1 ± 3.2 at baseline to 19.4 ± 2.8 post-intervention, reflecting a shift from moderate to mild severity. CET performance improved across working memory accuracy (+18%), sustained attention (+21%), and processing speed (-14% response latency). EEG showed improved Frontal Alpha Asymmetry and increased theta-beta variability, consistent with enhanced prefrontal engagement.

Conclusion:
Home-based bilateral prefrontal tDCS combined with CET was feasible and clinically promising for schizophrenia negative symptoms. The combination of bilateral prefrontal stimulation, concurrent CET, and home-based delivery has not been previously reported. Findings are preliminary given the small sample (n=7) and uncontrolled design.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-016
Listening to the sound of silence: mismatch negativity as a translational biomarker of cognitive dysfunction in schizophrenia

Presenter: Jyoti Jyoti, India

Co-Authors:
Shaily Mina
Kshitij Nair
Pankaj Verma

Topic:
2) Schizophrenia

Abstract

Objective:
Cognitive impairment is a core determinant of functional disability in schizophrenia. Despite advances in neurophysiological research, mismatch negativity (MMN) remains underexplored as a clinical biomarker. The limited integration of MMN cognitive measures represents a critical gap in translational psychiatry. This study aims to evaluate MMN as a biomarker of cognitive dysfunction in schizophrenia and its relationship with cognitive domains.

Methods:
In this case–control study, 60 participants were recruited: 40 with schizophrenia (20 remitted and 20 non-remitted) and 20 age- and gender-matched healthy controls. Symptom severity was assessed using PANSS. Neurocognitive functioning was evaluated using ACE-III, and social cognition using the Hinting Task (HT) and Emotion-Laden Sentences Toolbox for Emotion Recognition (ELSTER). MMN was recorded using an auditory oddball paradigm via EEG.

Results / Discussion:
Schizophrenia patients are expected to demonstrate reduced MMN amplitude and prolonged latency relative to controls. These deficits are anticipated to correlate with impairments in neurocognitive and social cognitive domains, as well as greater clinical severity. The study is ongoing, with a small number of participants remaining to complete the final dataset prior to poster presentation.

Conclusion:
MMN is expected to emerge as a sensitive, non-invasive biomarker of early information processing deficits in schizophrenia, with potential translational utility in enhancing objective assessment and informing prognostic and therapeutic strategies across cognitive domains.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-018
Effect of a Nurse-Led Dyadic Intervention on Treatment Adherence Among Inpatients Initiated on Clozapine Therapy: A Pilot Study

Presenter: Simrat Kaur, India

Co-Authors:
Sailaxmi Gandhi
Chethan B
Krishna Prasad M

Topic:
2) Schizophrenia

Abstract

Objective:
The primary objective was to evaluate the effect of a nurse-led dyadic intervention on medication adherence among patients undergoing clozapine therapy. Secondary objectives included assessing its impact on patients' attitude toward medication, anthropometric measures, haematological and biochemical parameters, adherence to white blood cell monitoring, severity of illness, quality of life, socio-occupational functioning, and family burden.

Methods:
A pilot study using a quantitative, posttest-only control group design was conducted in the psychiatry wards of a tertiary mental health care institute in Bengaluru. Eight patient–caregiver dyads of newly initiated clozapine patients were recruited using consecutive sampling and randomly assigned to an experimental group (n = 4) or control group (n = 4) through block randomisation where experimental group receives nursed led dyadic intervention.

Results / Discussion:
Medication adherence was higher in the experimental group at both follow-ups (MARS score at 3 months: 7 ± 2.9 vs 3 ± 2.4; at 6 months: 8.5 ± 1.7 vs 3 ± 0.8). These findings demonstrate the feasibility and acceptability of a nurse-led dyadic intervention during clozapine initiation.

Conclusion:
These findings demonstrate the feasibility and acceptability of a nurse-led dyadic intervention during clozapine initiation and indicate potential benefits for adherence, clinical outcomes, and caregiver burden, supporting the need for a larger randomized controlled trial.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-019
Catatonia in pregnancy with response only to MECT and limitations of it in 3rd trimester

Presenter: Sachin Pursnani, India

Co-Authors:
Panna Sharma
Rohit Verma

Topic:
2) Schizophrenia

Abstract

Objective:
In this report, we will review relevant literature on catatonia in pregnancy, with focus on treatment with ECT.

Methods:
A young pregnant woman with catatonic schizophrenia presented at 24 weeks with catatonia and psychosis, complicated by gestational diabetes and cholestasis. Lorazepam produced minimal response, necessitating mECT, which resolved catatonia after 11 sessions. mECT was paused late in pregnancy, restarted postpartum, improving psychosis, with maintenance mECT planned.

Results / Discussion:
A pregnant woman with catatonic schizophrenia presented at 24 weeks with catatonia and psychosis, complicated by gestational diabetes and cholestasis. Lorazepam showed minimal response, requiring mECT, which resolved catatonia. Psychosis persisted; mECT was paused late in pregnancy, restarted postpartum, leading to improvement and planned maintenance mECT.

Conclusion:
Though the literature on these topics is limited and typically presented in case reports format, results favour the use of ECT for pregnant catatonic patients. This case provides us with insight on treating a pregnant catatonic patient with multiple medical comorbidities with mECT which proves to be a safe and effective treatment option in such patients.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-020
The broken fabric of reality: a case report on truman show delusion

Presenter: Anjali Yadav, India

Co-Authors:
Sachin Pursnani
Ankesh Singh
Sumegha Mittal
Rohit Verma
Pratap Sharan

Topic:
2) Schizophrenia

Abstract

Objective:
In this report, we will review literature on truman show delusion

Methods:
23-year-old male law student presented with increased cannabis use, paranoia, disturbed sleep and delusional beliefs that people around him were acting, and that a fake reality was staged by a secret society to punish him for past sins. He described family members' emotions and activities as an act, stopped eating believing food and water needs were fake rules. Symptoms resolved within 6 weeks of antipsychotic treatment.

Results / Discussion:
Literature on Truman Show delusion is limited, reported since 2008. It is commonly viewed as a combination of paranoia, grandiosity and ideas of reference rather than a distinct disorder. Some consider it a delusional misidentification syndrome or an early stage of delusion formation marked by feelings of unreality, altered perception, depersonalization and derealization.

Conclusion:
The observations in this case suggest that a subset of schizophrenia patients may experience a Truman show delusion which is associated with themes of grandiosity, paranoia. This case highlights the bizarre nature of such delusion which is associated with delusion of persecution and response of this delusion to antipsychotic treatment.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-021
Homocysteine metabolism and Schizophrenia

Presenter: Aarti Midha, India

Topic:
2) Schizophrenia

Abstract

Objective:
Dysregulation of the homocysteine pathway has been implicated in many psychiatric conditions, including schizophrenia. Emerging evidence also highlights the relevance of co-occurring metabolic dysfunctions—such as insulin resistance, hypothyroidism and gut–brain axis disruptions—in contributing to medication-resistant presentations. This presentation discusses case studies of patients with schizophrenia who exhibited elevated Hcy levels (>10 µmol/L) alongside additional metabolic abnormalities.

Methods:
A case-study design was used, involving medication-resistant schizophrenic cases who remained symptomatic despite adequate antipsychotic therapy. Comprehensive clinical nutrigenomic testing (MTHFR and GSTM1 polymorphisms) and metabolic markers (insulin resistance/thyroid) were assessed, and individualised interventions were initiated, including methylfolate, methylcobalamin, pyridoxal-5-phosphate, N-acetylcysteine, and gut-targeted therapy. Patients were followed for 12–24 months.

Results / Discussion:
Medication-resistant schizophrenia cases demonstrated marked symptom improvement once homocysteine and other metabolic markers normalised, in addition to standard treatment.

Conclusion:
Homocysteine metabolism relates to gut health and Hormones. Correcting metabolic dysfunction may offer a valuable adjunctive strategy for medication-resistant schizophrenia and warrants further research.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-023
Prevalence of metabolic syndrome in patients of schizophrenia receiving clozapine

Presenter: Shweta Chanalia, India

Co-Authors:

Sandeep Grover

Topic:
2) Schizophrenia

Abstract

Objective:
To evaluate the prevalence of metabolic syndrome in patients with schizophrenia receiving clozapine.

Methods:
260 patients diagnosed with schizophrenia receiving clozapine were evaluated for metabolic syndrome. Metabolic syndrome was diagnosed based on the consensus criteria for Asian population.

Results / Discussion:
The mean age of the participants was 38.42 years (SD: 11.86) with, slight male preponderance (50.8%). The mean duration of illness was 15.68 (SD 8.68) years. Abdominal obesity was present in 80.4%, elevated triglycerides in 54.6%, low HDL in 53%, elevated blood pressure in 14% of participants. Overall, metabolic syndrome was present in 125 patients (48.1%), with a higher prevalence among females 66 (52.8%) compared to males (47.2%).

Conclusion:
Nearly half of patients with schizophrenia receiving clozapine have metabolic syndrome, with a higher prevalence among females. These findings highlight the need for regular metabolic monitoring, early lifestyle and pharmacological interventions in this high-risk group.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-024
Microarray analysis of postmortem brain tissues in methamphetamine dependent, methamphetamine induced psychosis and schizophrenia

Presenter: Arvin Haghighatfard, Iran

Co-Authors:

Bahar Ghezelayagh

Topic:
2) Schizophrenia

Abstract

Objective:
Over 40% of methamphetamine users develop psychosis, yet the molecular and neural mechanisms underlying methamphetamine-induced versus primary psychosis remain unclear. This study aims to identify molecular pathways involved in methamphetamine-induced psychosis using whole-genome expression profiling, to improve understanding of its pathophysiology and inform the etiology of substance-induced and primary psychotic disorders.

Methods:
Genome-wide expression profiling was conducted by using the Affymetrix U133 plus 2.0 Array for individuals with chronic methamphetamine use disorder without psychoses, chronic methamphetamine use disorder with methamphetamine induced psychosis, schizophrenic patients, and non-psychiatric participants. Each group was included Prefrontal cortex tissue of 15 postmortem subjects.

Results / Discussion:
Alteration of immune system and mitochondria pathways detected in methamphetamine-induced psychosis and schizophrenia. Serotonergic synapse and metabolic pathways changed only in methamphetamine-induced psychosis.

Conclusion:
Results detected several potential biomarkers for methamphetamine-induced psychosis and schizophrenia as well as an extension of our knowledge about the complex molecular pathways which cause a complicated mental situation called psychosis.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-025
Doses, plasma levels, and response of SGA long-acting injectable in patients with severe schizophrenia

Presenter: Juan J. Fernández-Miranda, Spain

Co-Authors:
Silvia Díaz-Fernández
Javier Cepeda-Piorno

Topic:
2) Schizophrenia

Abstract

Objective:
There is a need when optimizing antipsychotic treatment to know the plasma levels (PLs) achieved with the different doses and their relationship with effectiveness and toxicity, especially in patients with poor clinical progress. This research studies the dose-plasma levels (PLs) relationship of second-generation antipsychotics, together with the treatment outcomes achieved, in seriously ill patients with schizophrenia.

Methods:
One-year prospective study with patients (N=68) with severe schizophrenia treated with paliperidone three-month (PP3M) or aripiprazole one-month (ARIM). Subjects were divided into standard-dose or high-dose groups. PLs were divided into "standard PL" and "high PL" (above therapeutic reference range) groups. Doses/PLs relationship, severity, hospitalizations, tolerability, compliance, and their relationship with doses and PLs, were evaluated.

Results / Discussion:
There was no linear relationship between ARIM or PP3M doses and PLs achieved. In half of the subjects, standard doses reached PLs above the TRR. The improvements in clinical outcomes were related to high PLs, without worse treatment tolerability or adherence. All subjects remained in the study, regardless of dose or PL. Clinical severity and hospitalizations decreased significantly more in those patients with high PLs.

Conclusion:
Considering the non-linear dose-PL relationship of ARIM and PP3M in people with severe schizophrenia, PLs above the TRR are linked to better treatment outcomes without worse tolerability. The need in a notable number of cases for high doses to reach those effective PLs is highlighted.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-026
Reduced Basal Ganglia Perivascular Space in Treatment-Resistant Schizophrenia

Presenter: Taeyoung Lee, Republic of Korea

Co-Authors:
Jiseon Jang
Jun Soo Kwon

Topic:
2) Schizophrenia

Abstract

Objective:
Glymphatic system dysfunction is increasingly implicated in the pathophysiology of schizophrenia. However, it remains unknown whether treatment-resistant schizophrenia (TRS)—increasingly recognized as a neurobiologically distinct subtype—exhibits specific alterations in perivascular spaces (PVS), the structural conduits for glymphatic waste clearance.

Methods:
We quantified PVS volumes in the basal ganglia (BG) and white matter (WM) using 3T structural MRI in a matched cohort of healthy controls, patients with first-episode psychosis, and patients with TRS. A normative modeling framework trained on the HC group was applied to quantify individualized neurodevelopmental deviations in BG-PVS volume across the lifespan.

Results / Discussion:
TRS patients demonstrated a significantly reduced BG-PVS volume compared to both the HC and FEP groups. This reduction remained robust after controlling for age, total BG volume, and cumulative antipsychotic exposure, and was driven by group-level differences rather than within-group chronicity.Normative trajectory modeling revealed that TRS patients exhibited the earliest and most pronounced deviation from healthy BG-PVS developmental patterns.

Conclusion:
The selective reduction of basal ganglia PVS volume in TRS points to an intrinsic, neurodevelopmentally acquired impairment in glymphatic clearance rather than a secondary consequence of chronic illness or prolonged medication use. These region-specific structural alterations highlight a distinct neuroglial pathology in TRS and offer a potential neuroimaging biomarker for earlier identification of treatment-refractory trajectories.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-027
CYP450 Polymorphism Profile in Treatment-Resistant Schizophrenia

Presenter: Johny Kamal, India

Co-Authors:

Prerna Kukreti

Topic:
2) Schizophrenia

Abstract

Objective:
This study aimed to examine the allele frequency and metabolisation phenotype based on the genetic variation harboured by studied psychiatric patients in order to establish a base for the pharmacogenomics in Indian psychiatric clinical setting.

Methods:
A pharmacogenomic panel was applied to studied cohort that included CYP2D6 for aripiprazole ; CYP1A1 for olanzapine; ABCB1 for clozapine; RGS4 for risperidone ; and SLC22A1 for anticholinergic sensitivity. Predicted response was stratified as per CPIC nomenclature.

Results / Discussion:
Predicted response to olanzapine and Risperidone was seen to be low in most of the cases raising concerns about its empiric use . Clozapine response , mediated by ABCB1 encoded P-glycoprotein was observed to be compromised in a significant proportion of patients . In contrast , molecules metabolised through CYP2D6 demonstrated normal response in the large majority of patients, offering a genomically informed basis for preferential prescribing.

Conclusion:
These findings demonstrates genetic variability among the studied cases with high frequency of variations that may cause likelihood of adverse drug reactions or toxicity, establishing pharmacogenomic screening as an evidence-based clinical necessity rather than an adjunctive tool in contemporary psychiatric practice.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-029
Smoking through a tracheostome: nicotine dependence, psychosis and oro-sensory compulsive behaviour following caustic ingestion

Presenter: Arshiya Fathima M S, India

Co-Authors:

Minhajzafar Nasirabadi

Topic:
6) Addiction, 1) Psychoses

Abstract

Objective:
Permanent tracheostomy following deliberate caustic ingestion is a rare surgical outcome whose psychiatric consequences remain undescribed. We present a case where the tracheostome itself became the route of extreme nicotine delivery, producing treatment-refractory psychosis, severe malnutrition, and a compulsive oral behaviour outside existing nosology.

Methods:
Psychiatric and medical history was obtained through clinical interview and family collateral. Informed consent was obtained from the patient's guardian.

Results / Discussion:
A 28 year old male following tracheostomy, escalated to taking 125–150 beedis daily administered through the stoma. Psychotic features emerged in close temporal proximity and showed minimal response to antipsychotics. A behaviour not meeting criteria for bulimia nervosa, rumination disorder, or pica emerged-oral holding and spitting of food for thermal-sensory gratification, with vomit re-ingestion under deprivation.

Conclusion:
This case highlights stoma-mediated nicotine exposure as a potential psychosis-precipitating mechanism, CYP1A2 induction as a cause of apparent treatment resistance, and oropharyngeal injury as a substrate for novel compulsive sensory-seeking behaviour. It underscores the need for integrated medical psychiatric management in complex self-harm presentations.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-031
Early metabolic trajectory and reorganization of the metabolic network during the first weeks of clozapine treatment in schizophrenia

Presenter: Sergey Mosolov, Russia

Co-Authors:
Yuri Bellevich
Dmitriy Sosin
Sergey Mosolov

Abstract

Objective:
Clozapine is the gold standard for treatment-resistant schizophrenia (TRS) but carries a high cardiometabolic burden. We aimed to characterise the dynamics of anthropometric, bioimpedance and laboratory parameters during the first 4-6 weeks of clozapine monotherapy and to use correlation network analysis to assess the structure of early somato-metabolic risk and its reorganisation.

Methods:
Prospective observational single-centre study (n=101; schizophrenia, ICD-10 F20; clozapine monotherapy), assessed at baseline and after 4-6 weeks of treatment. Anthropometry, body composition (bioimpedance, Seca 515) and fasting laboratory parameters were measured. The Wilcoxon signed-rank test was applied; network analysis was performed in JASP 0.18.3.0 on rank-based correlations (edges at $p<0.05$) with bootstrap stability assessment.

Results / Discussion:
Over 4-6 weeks, body weight (73.78→75.75 kg; $p=0.016$), waist circumference (96.4→97.8 cm; $p<0.001$), fat mass (19.64→20.6 kg; $p<0.001$) and visceral fat (2.0→2.2 L; $p=0.021$) increased significantly. Total cholesterol (4.86→5.36 mmol/L; $p=0.026$) and LDL (2.78→3.06 mmol/L; $p<0.001$) rose, as did HbA1c ($p=0.025$). A network showed an adipose core; by visit 2 glucose moved to a glycaemic-inflammatory cluster, and ESR–triglycerides coupling doubled.

Conclusion:
Within the first weeks of clozapine monotherapy, interconnected axes of early metabolic risk emerge: adipose, atherogenic-inflammatory and glycaemic-compositional, reflecting a coordinated, system-level reorganisation of somato-metabolic structure rather than isolated parameter shifts.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-032
Assessment And Comparison Of Systemic Inflammatory Biomarkers And Their Changes Following Electroconvulsive Therapy In Patients Of Schizophrenia At Sms Medical College, Jaipur

Presenter: Arunima Parashar, India

Abstract

Objective:
To assess systemic inflammatory biomarkers (NLR, PLR, and MLR) in patients with schizophrenia undergoing ECT, compare their levels before and after six ECT sessions, evaluate changes in relation to clinical response, and examine whether baseline lymphocyte counts can predict treatment outcomes.

Methods:
This observational study included ICD-10 diagnosed schizophrenia patients undergoing ECT. Fasting blood samples were collected before the first ECT session and one day after the sixth session. NLR, PLR, and MLR were derived from complete blood counts. Clinical symptoms were assessed using PANSS and cognitive function using MMSE. Pre- and post-ECT parameters were statistically analyzed. (399 characters)

Results / Discussion:
Patients with schizophrenia showed elevated NLR, PLR, and MLR at baseline. After six ECT sessions, significant reductions in NLR and MLR were observed, accompanied by substantial improvement in PANSS scores. These findings indicate a reduction in systemic inflammation alongside clinical improvement following ECT.

Conclusion:
The findings support the role of systemic inflammation in schizophrenia and suggest that ECT may exert part of its therapeutic effect through modulation of inflammatory processes. Reduction in inflammatory biomarkers alongside symptom improvement highlights their potential utility as markers of treatment response in schizophrenia.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-033
Assessment Of Cognitive Outcomes In Patient Of Schizophrenia Under Going Ect Using Moca At Sms Medical College Jaipur

Presenter: Ishan Kumar Goyal, India

Abstract

Objective:
1. To assess MoCA scores before and after ECT (6th session) in hospitalized schizophrenia/schizoaffective disorder patients with predominantly positive psychotic symptoms (DSM-5-TR).

2. To classify patients into MoCA improvement (≥+2), deterioration (≤−2), or no change (±1) groups.

3. To compare pre- and post-ECT MoCA and BPRS scores.

4. To identify sociodemographic and clinical factors associated with cognitive change following ECT.

Methods:
This observational study at SMS Medical College, Jaipur enrolled schizophrenia (DSM-5-TR) patients referred for ECT. MoCA (Hindi) and BPRS were assessed pre-ECT and post-6th session. Bitemporal ECT with seizure threshold-based dosing was used. Patients were classified as MoCA improvement(≥+2), deterioration(≤−2), or no change(±1). Paired t-tests and descriptive statistics were applied.

Results / Discussion:
Pre- and post-ECT MoCA, BPRS, and GAF scores showed statistically significant improvement ($p<0.05$). Majority of patients demonstrated cognitive improvement post-ECT.

Conclusion:
Most patients with schizophrenia undergoing ECT showed preserved or improved cognition. Low baseline MoCA should not contraindicate ECT. This study provides foundational Indian data on cognitive outcomes following ECT in schizophrenia.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-034
Digging deep to uncover rare CYP genotype - Solanidine analysis during TDM revealed rare CYP2D6 poor metabolizer genotype: a case report

Presenter: Sverre Larne, Norway

Abstract

Objective:
BACKGROUND: CYP2D6 catalyzes the metabolism of around 25% of all clinically used drugs

• In Caucasians, 5-10% are homozygous carriers of variant alleles encoding no CYP2D6 enzyme activity and classified as poor metabolizers (PMs). These patients are at increased risk of adverse effects.
• Challenges
➢ selection of appropriate clinical CYP2D6 variant alleles

➢ costs required to identify CYP2D6 PMs

Methods:
We have included measurements of the potato-derived compound solanidine (Figure 1) and its CYP2D6-dependent metabolite 4-hydroxysolanidine in our LC-MS method. This allows routinely phenotyping of CYP2D6 as part of our therapeutic drug monitoring practice:

Results / Discussion:
In October 2024, a metabolic ratio of -7.86 was calculated in a therapeutic monitoring sample from a patient who had been genotyped as CYP2D6*1/*1 and interpreted as a normal metabolizer in 2020. The patient had a history of side effects during use of the CYP2D6 substrates perphenazine and aripiprazole at standard doses. Upon reanalysis with an extended genotyping panel, a rare CYP2D6

genotype, CYP2D6*7/*7, encoding PM status, was revealed.

Conclusion:
This report shows the clinical value and feasibility of routine analysis of solanidine and 4-hydroxysolanidine to identify patients predicted to be CYP2D6 PMs, including patients with rare variants.

• Solanidine measurements can be used as a complementary tool to genotyping
• Phenotyping can substantially reduce the number-needed-togenotype for identification of CYP2D6 PMs in clinical practice.

P-A
Postersession A: Psychosis & Schizophrenia

P-A-035
Interim data from ENIGMA – TRS 1 study of evenamide as add on therapy to antipsychotic Standard of Care in patients with treatment resistant schizophrenia (TRS). Difficulties in performing TRS study with TRRIP criteria: the CRO experience

Presenter: Bartosz Janikowski, Czech Republic

Abstract

Background:

Newron is performing phase 3 placebo-controlled studies in patients with TRRIP confirmed TRS criteria with evenamide (Howes et al., 2017). CRX has been contracted to perform the study in the field. Unique difficulties have been experienced in the performance of these studies.

Treatment resistant schizophrenia (TRS) remains an unmet medical need considering insufficient treatment modalities available to such patients either from efficacy or safety perspective as well as varied compliance to currently available medications.

Methods:

The study protocol limits the primary AP to 10 different SGAs that may be supplemented by other SGA or FGA. The requirement for the adequacy of treatment is firstly demonstration of plasma levels of the therapeutic range, duration of treatment of 6-week, documentation of two previous failures of therapeutic doses and duration, need for qualify raters as well as training them to criteria. Unique requirements include writing a patient narrative by the CRO Medical Monitors (MM) and review by the principal investigator (PI) prior to narrative being reviewed by an IEAC reviewer.

One aspect of the ENIGMA – TRS 1 study assesses plasma concentrations of standard of care as selected antipsychotics: Aripiprazole, Brexpiprazole, Cariprazine, Clozapine, Lurasidone, Olanzapine, Paliperidone, Quetiapine, Risperidone and Ziprasidone for subjects treated with those antipsychotics with daily therapeutic doses meeting local product labels requirements and correlated those with minimal duration of 6 weeks treatment with any of those medications.

Results:

As the study is performed in 20 countries worldwide major issues reside in getting approval by all regulatory approvals, languages use for the rating instruments as well as for the training of the raters.

Based on hundreds of patients entering screening, a large proportion failed the screening phase because of lack of adequate concentration of the primary AP. Difficulties have been noticed in ensuring adequate documentation of previous AP failures.

Initial results from the randomized subjects to date within 18 countries globally reveal number of patients that present with either lower than expected therapeutic plasma concentrations for received antipsychotic(s) despite administered daily recommended therapeutic doses or concentrations above upper expected concentration thresholds with no significant signs of toxicity.

Discussion:

Disparity between therapeutic doses of selected antipsychotics and plasma concentrations inform so efficacy and clinical presentation of treatment resistant subjects is not fully aligned, with potential lack of compliance being confounding factor especially given so plasma concentrations are not assessed as standard worldwide as well as so lack of efficacy might be related to suboptimal plasma concentrations despite recommended therapeutic doses prescribed.

P-B
Postersession B: Mood & Affective Disorders

P-B-001
When the Gut, Brain, and Heart Lose Synchrony: Autonomic Insights Into PSVT and Panic Symptoms

Presenter: Jigisha Das, India

Topic:
10) Other

Abstract

Objective:
The gut–brain–heart axis is a neuroautonomic network integrating gastrointestinal, brain, and cardiovascular function. Dysregulation of this axis has been linked to arrhythmogenesis, autonomic imbalance, and anxiety disorders. Altered vagal signaling, sympathetic overactivity, and gut-derived inflammatory mediators may disrupt CV electrophysiology and emotional regulation, contributing to palpitations, panic symptoms, and autonomic hyperarousal.

Methods:
A 28 year-old married female with no psychiatric history presented with recurrent palpitations, chest discomfort, dyspnea, tremulousness, and panic-like episodes for 5 years. ECG confirmed PSVT, and radiofrequency ablation reduced arrhythmia symptoms. Persistent bloating, constipation, dyspepsia, and delayed gastric transit continued despite treatment. Psychiatric evaluation revealed panic disorder, suggesting gut-brain-heart axis dysregulation.

Results / Discussion:
GI symptom flares were temporally associated with worsening palpitations, autonomic hyperarousal, and panic episodes, suggesting neuroautonomic dysregulation. Following RFA, tachyarrhythmia improved, but panic symptoms persisted. Sertraline 50 mg/day and clonazepam 0.5 mg/day produced significant clinical improvement. Gut dysbiosis, inflammatory cytokines, altered vagal tone, and sympathetic overactivity may contribute to symptom persistence.

Conclusion:
This case highlights the intricate interplay between cardiac arrhythmia, gastrointestinal dysmotility, and panic symptoms within the gut–brain–heart axis. Recognition of these bidirectional neuro autonomic interactions may improve diagnostic accuracy and support integrated multidisciplinary care involving cardiology, gastroenterology, and psychiatry, ultimately enhancing patient outcomes and quality of life.

P-B
Postersession B: Mood & Affective Disorders

P-B-002
Levosulpiride-Induced Akathisia Presenting With Suicidality in Depression: A Diagnostic Challenge

Presenter: Akanksha Shukla, India

Co-Authors:
Parvaiz Alam
Dinesh Kataria
Bhavuk Garg
Mini Sharma

Topic:
10) Other, 7) Mood disorders

Abstract

Objective:
Akathisia is a distressing movement disorder that may be mistaken for anxiety, agitation, or worsening mood symptoms. Levosulpiride, a commonly used gastrointestinal prokinetic with dopamine D2 antagonist action, can rarely cause extrapyramidal adverse effects, including akathisia. This case highlights the diagnostic difficulty of identifying akathisia in depression with suicidality.

Methods:
A single case was assessed using clinical history, medication timeline, mental status and neurological examination. Barnes Akathisia Rating Scale, Hamilton Depression Rating Scale and Naranjo Adverse Drug Reaction probability assessment were applied. Routine investigations were done, followed by dechallenge and rechallenge with the suspected drug.

Results / Discussion:
A 55-year-old woman had depressive symptoms for 2 months. During this period, she went to a general practitioner for abdominal pain and was prescribed levosulpiride 75 mg. Two days later, she developed severe restlessness and suicidal ideas. BARS score was 4. The symptoms resolved after levosulpiride was stopped. On rechallenge, akathisia recurred. Her Naranjo score was 7, suggesting a probable adverse drug reaction.

Conclusion:
This case shows the need to ask about recent gastrointestinal medications in patients with depression who develop sudden restlessness or agitation. Levosulpiride-induced akathisia can be very distressing and may be associated with suicidal ideas. Early recognition and stopping the drug can lead to quick improvement.

P-B
Postersession B: Mood & Affective Disorders

P-B-003
Adverse childhood experiences and resilience in patients diagnosed with - depression and their correlation with sociodemographic profile of these patients

Presenter: Onkar Deokate, India

Co-Authors:

Veena Gholap

Topic:
10) Other

Abstract

Objective:
To Study The Prevalence of Adverse Childhood Experiences(ACE) in Patients suffering with Depression.

To study the Resilience in these patients.

To study the correlation of ACE, resilience and Sociodemographic profile of these patient.

Methods:
Cross-sectional study in a Mumbai teaching hospital Psychiatry OPD using convenience sampling (n=70). Adults (>18) with DSM-V depression consenting will be included; those with suicidal ideation or acute illness excluded. Data: demographics and clinical details. Tools: ACE Questionnaire and 14-item Resilience Scale. Analysis via Excel 2019 using descriptive statistics, Chi-square, and t-test ($p<0.05$).

Results / Discussion:
Results will be discussed in view of available literature

Conclusion:
Conclusion will be discussed in view of results

P-B
Postersession B: Mood & Affective Disorders

P-B-004
Structural psychoradiomic features of clinical symptom improvement following mindfulness-based cognitive therapy in patients with panic disorder: A randomized controlled trial

Presenter: Hyun-Ju Kim, Republic of Korea

Co-Authors:

Ki-Sung Park
Sang-Hyuk Lee

Topic:
3) Postpartum depression

Abstract

Objective:
This study aimed to identify structural MRI-based radiomic features associated with clinical improvement following Mindfulness-Based Cognitive Therapy using Neuroscience (NMBCT) in patients with panic disorder (PD).

Methods:
PD patients were randomized to NMBCT+TAU (n=23) or TAU (n=20). T1 MRI and clinical assessments were acquired at baseline and 8 weeks. Radiomic features were extracted from fear-related regions using PyRadiomics. Features were selected using Welch's t-test, and a random forest classifier with leave-one-out cross-validation was applied. Feature importance and clinical associations were examined using SHAP and Spearman correlation.

Results / Discussion:
Out of 2,140 extracted features, 213 showed significant group differences and were included in the model. SHAP analysis revealed that reduced texture heterogeneity in the right anterior insula and altered frontal radiomic patterns were strongly associated with NMBCT response. Several radiomic features showed significant correlations with improvements in PDSS, ASI-R, and sleep quality.

Conclusion:
Psychoradiomic features, particularly within the insula, were associated with clinical symptom improvement following NMBCT in patients with PD. These findings support psychoradiomics as complementary neurobiological information for symptom improvement and advancing precision psychiatry in PD.

P-B
Postersession B: Mood & Affective Disorders

P-B-005
Chronic Sympathomimetic stimulation to Catecholaminergic Dysregulation: A neurobiological Case analysis of Mephentermine-Induced Mania

Presenter: Jigisha Das, India

Co-Authors:

R Anitha

Topic:
6) Addiction

Abstract

Objective:
Mephentermine is an indirect-acting sympathomimetic related to amphetamines that increases synaptic norepinephrine and dopamine. Often misused for gym performance, chronic exposure may cause neuro adaptive changes in mesolimbic and fronto-limbic circuits. Sustained catecholaminergic stimulation can trigger affective switching, behavioral dyscontrol, and manic syndromes, though such reports remain limited.

Methods:
Mr. Y, a 23 year-old unmarried male with no past or family psychiatric history, presented with irritability, aggression, suspiciousness, decreased sleep, increased energy, boastful speech, and self-harm behavior. He used daily IM mephentermine (3–4/day) for 4 years. BPRS: 46; YMRS: 37. Diagnosed with substance-induced bipolar disorder, manic type (DSM-5).

Results / Discussion:
Temporal correlation between high-dose Mephentermine use and mania supported a substance-induced etiology. Dopaminergic overactivity in mesolimbic circuits and noradrenergic dysregulation in prefrontal regions likely drove impulsivity and aggression. After stopping mephentermine and starting valproate 1500 mg, risperidone 2 mg, and lorazepam 2 mg, marked improvement occurred within weeks.

Conclusion:
This case underscores the neurobiological impact of chronic sympathomimetic misuse in precipitating manic syndromes. Early identification of pharmacologically driven affective states and integrated mood stabilization with de-addiction strategies are crucial for favorable outcomes.

P-B
Postersession B: Mood & Affective Disorders

P-B-006
Exploring Oculomotor Dysfunction in Remitted Bipolar I Disorder: Insights from VNG, MRI and Clinical Correlates

Presenter: Pradeep Yuvaraj, India

Co-Authors:
Rahina Abubacker
Aravind Kumar
Aravinda Hr
Binukumar Bhaskarapillai
Muralidharan Kesavan

Topic:
7) Mood disorders

Abstract

Objective:
To explore oculomotor function in remitted BD-I compared with healthy subjects using VNG, and to examine its relationships with MRI measures and clinical variables.

Methods:
Fifty-three participants with BD-I in remission and age-gender matched healthy subjects underwent VNG assessment, including spontaneous, saccade, smooth pursuit, gaze, optokinetic, and caloric tests. A representative subset (n=15 per group) underwent 3T MRI. Structural MRI data were processed using CAT12/SPM with atlas-based ROI extraction.

Results / Discussion:
BD-I showed abnormalities in smooth pursuit (reduced gain), saccades (reduced precision and velocity), and optokinetic responses (reduced gain, altered slow-phase velocity). Gaze, spontaneous nystagmus, and calorics were normal, indicating preserved peripheral function. VNG parameters correlated with cortical, limbic, and cerebellar regions. Illness duration and age of onset showed associations with oculomotor measures.

Conclusion:
BD-I shows central oculomotor dysfunction, with impaired smooth pursuit, saccades, and optokinetic responses, reflecting fronto-parietal, temporo-insular, and visual network abnormalities. Preserved peripheral and brainstem pathways suggest higher-order dysfunction. Associations with MRI and clinical variables indicate illness-related changes, supporting VNG as an objective biomarker of multisensory and cognitive dysfunction in BD-I.

P-B
Postersession B: Mood & Affective Disorders

P-B-007
Transcranial magnetic stimulation for treatment-resistant depression in affective disorders: randomized, single-blind, comparative study of personalized neuronavigated and standard protocols

Presenter: Sergey Mosolov, Russia

Co-Authors:
Nikita Maslenikov
Bogdan Antonovich
Larisa Mayorova
Eduard Tsukarzi

Topic:
7) Mood disorders, 10) Other

Abstract

Objective:
Repetitive transcranial magnetic stimulation (rTMS) is a safe, non-invasive neuromodulation technique widely used for the treatment of depression, but the therapeutic response to the standard method is limited and highly variable. The aim of the study is to compare the response rate of personalized cortical targeting based on resting-state fMRI network connectivity data with that of standard rTMS in patients with treatment-resistant depression.

Methods:
38 patients with recurrent (n=25) and bipolar depression (n=13) (ICD-10) resistant to antidepressant treatment were randomized in two groups: personalized neuronavigated rTMS where the target was individually localized after fMRI resting-state analysis as a part of the CEN (n=18) and standard rTMS of DLPFC (n=20). Both groups received 20 Hz rTMS (15 sessions) and stable medication. The criterion of response was ≥50% HDRS-17 score reduction.

Results / Discussion:
HDRS-17 score reduction after 3 weeks was statistically significant in both groups: 42.3% ($p = 0.00023$) in the neuronavigated rTMS group and 33.8% ($p = 0.0004154$) in the standard rTMS group. There were no significant differences between the two groups in the response rate (55.6% vs. 35.0%, $p = 0.32755$) and the remission rate (27.8% vs. 20.0%, $p = 0.72379$). rTMS was well tolerated in both groups, and no participants discontinued the study.

Conclusion:
Our preliminary results showed that the efficacy of personalized neuronavigated rTMS is not significantly better than that of standard rTMS, although this may be due to the small sample size and lack of statistical power. The increased activity of the anterior nodes of the default

mode network (DMN) could be a potential predictor of response to neuronavigated rTMS. Further large-scale controlled studies are needed to confirm these findings.

P-B
Postersession B: Mood & Affective Disorders

P-B-008
Delayed-onset olanzapine-associated dizziness in bipolar disorder: a case of diagnostic overshadowing.

Presenter: Linkan Verma, India

Topic:
7) Mood disorders, 8) Sleep disorders

Abstract

Objective:
Adverse effects of antipsychotic medications are commonly recognized early in treatment; however, delayed-onset somatic symptoms may complicate attribution, particularly in patients with dissociative or functional symptoms. We report a case of possible delayed-onset olanzapine-associated dizziness in bipolar disorder in which diagnostic overshadowing contributed to delayed recognition of a potential medication-related adverse effect

Methods:
A 19-year-old female with bipolar affective disorder, stable for 8 months on divalproex sodium (500 mg/day) and olanzapine (10 mg/day), developed intermittent spinning dizziness after a 5-week gastrointestinal illness. She had prior depressive symptoms and dissociative episodes, with later diagnostic revision to bipolar disorder following a manic episode. Orthostatic BP, laboratory, neurological, ENT, and NCCT head evaluations were normal.

Results / Discussion:
Dizziness persisted despite recovery from gastrointestinal symptoms and showed only partial improvement with vestibular treatment. Accidental omission of olanzapine for 3 days was followed by marked symptom improvement, with no recurrence after discontinuation, while mood stability was maintained on divalproex monotherapy. Naranjo assessment score was 3, suggesting a possible medication-related adverse effect.

Conclusion:
This case highlights the risk of diagnostic overshadowing in psychiatric practice and emphasizes that psychotropic adverse effects may emerge or become clinically apparent after prolonged treatment stability, particularly following intercurrent physiological stressors. Clinicians should remain open to medication-related contributions when evaluating new somatic symptoms in psychiatric patients.

P-B
Postersession B: Mood & Affective Disorders

P-B-010
Home-Based Bifrontal tDCS Combined With Cognitive Emotional Training in Post-Manic Depression: A Detailed Case Report With Neurophysiological and Cognitive Outcomes

Presenter: Anindo Mitra, India

Topic:
7) Mood disorders, 10) Other

Abstract

Objective:
Post-manic depression is a pharmacologically difficult phase of bipolar disorder. Antidepressants carry a documented switch risk in this period, and non-pharmacological alternatives with mechanistic data are lacking. We report a case with clinical, neurophysiological, and cognitive outcome data following home-based bifrontal tDCS combined with Cognitive Emotional Training (CET) as an antidepressant-sparing approach.

Methods:
A 35-year-old male with DSM-5 Bipolar I Disorder presented in a depressive phase following a confirmed manic episode. YMRS was 2 and HAM-D was 18 with BDI of 17 at tDCS initiation, confirming post-manic depression without active mania. No antidepressant was prescribed. The patient received 20 sessions of home-based bifrontal tDCS with phone-based and outpatient supervision, combined with structured CET.

Results / Discussion:
Post-treatment assessment showed clinically meaningful depressive improvement with YMRS remaining at 2 and no affective switch. EEG showed marked normalization of frontal asymmetry across all frequency bands. Elevated gamma variability stabilized, indicating reduced cortical hyperarousal. CET showed improvements in attentional control, working memory span, emotional response time, and processing speed with preserved accuracy.

Conclusion:
Home-based bifrontal tDCS produced measurable prefrontal network normalization and cognitive-affective improvement in post-manic depression without affective switching. These findings support further investigation of tDCS as an antidepressant-sparing neuromodulation strategy in this phase of bipolar disorder.

P-B
Postersession B: Mood & Affective Disorders

P-B-012
Nitrous oxide for the treatment of depression: a systematic review and meta-analysis

Presenter: Kiranpreet Gill, United Kingdom

Co-Authors:
Angharad De Cates
Chantelle Wiseman
Susannah Murphy
Ella Williams
Catherine Harmer
Isabel Morales-Muñoz
Steven Marwaha

Topic:
7) Mood disorders

Abstract

Objective:
To evaluate the efficacy, safety and clinical relevance of nitrous oxide (N2O) for depressive disorders by synthesising evidence from clinical trials and protocols, and conducting meta-analysis where possible, alongside evidence mapping to identify research gaps and guide future study design.

Methods:
Systematic review and meta-analysis of clinical trials and protocol papers on N2O for depression, following PRISMA guidelines. Data on depressive symptoms and adverse events (AEs) were synthesised using fixed or random-effects models. Evidence mapping summarised study characteristics across completed and ongoing trials.

Results / Discussion:
Seven trials (n=247) and four protocols were included. N2O (25-50%) produced rapid antidepressant effects, with meta-analysis showing significant symptom reduction (MD -2·74, 95% CI: -4·72 to -0·76; p=0·007) and 24 hours (MD -3·32, 95% CI: -5·09 to -1·55; p<0·0001), but not 1 week. AEs were mild and dose-dependent. Repeated dosing showed more sustained effects. Evidence mapping revealed early-phase, short-term focus.

Conclusion:
N2O demonstrates rapid, reproducible antidepressant effects in early-phase trials. Its future clinical value depends on whether these effects can be sustained over time through optimised

dosing and extended/repeated use. Improved trial design, outcome standardisation, and population diversity is required to clarify its full potential for the treatment of depression.

P-B
Postersession B: Mood & Affective Disorders

P-B-013
Mindfulness-based cognitive therapy using neuroscience modulates triple network functional connectivity and clinical symptom improvement in patients with panic disorder: A randomized controlled study

Presenter: Hyun-Ju Kim, Republic of Korea

Co-Authors:
Shinhye Kim
Sang-Hyuk Lee

Topic:
7) Mood disorders

Abstract

Objective:
Panic disorder (PD) involves maladaptive cognitive–affective processes linked to dysregulation of the default mode, salience, and executive control networks. This study examined whether mindfulness-based cognitive therapy using neuroscience (NMBCT) modulates triple network functional connectivity (FNC) and whether such changes are associated with clinical symptom improvement.

Methods:
Forty-three patients with PD were randomized to NMBCT plus treatment-as-usual (TAU; n=23) or TAU alone (n=20), with clinical assessments and T1-weighted and resting-state fMRI acquired at baseline and week 8. Triple network components were identified using group independent component analysis, and group-by-time effects on FNC were examined using linear mixed-effects models controlling for age, sex, head motion, and antidepressant equivalent dose.

Results / Discussion:
Compared with the TAU-only group, the NMBCT+TAU group showed greater reductions in APPQ and HAM-D scores and significant group-by-time interactions in FNC involving the anterior salience network, precuneus, and dorsal and ventral default mode networks. Preserved DMN-related connectivity was associated with greater reductions in depressive symptoms and increased reflective rumination.

Conclusion:
NMBCT induces functional reorganization within the triple network in PD, characterized by stabilization of DMN and salience network connectivity over time. These network-level changes were associated with improvement in depressive symptoms and adaptive modulation of rumination, suggesting enhanced regulation of self-referential and affective processing.

P-B
Postersession B: Mood & Affective Disorders

P-B-014
Effects on prefrontal cortical oxygenation during low-frequency repetitive transcranial magnetic stimulation for Adolescent Treatment Resistant Depression

Presenter: Robert Bodén, Sweden

Co-Authors:
Jonas Persson
Jonas Jester-Broms
Helena Strömbergsson

Topic:
7) Mood disorders

Abstract

Objective:
Low frequency repetitive transcranial magnetic stimulation (LF-rTMS) has been associated with a reduction of depressive symptoms. Functional near-infrared spectroscopy (fNIRS) detect changes in the oxygenation of hemoglobin (HbO) which has been correlated to neural activity. We investigate the effects of LF-rTMS on changes in frontal HbO in adolescents with treatment resistant depression and if these changes correlate with clinical outcome.

Methods:
Twenty adolescents, 13-18 years old with TRD were included and received LF-rTMS over F4 for 20-30 week-daily sessions. Frontal HbO was measured through fNIRS every 10th session, and change in oxygenation and sample entropy, (SampEnt) pre-post LF-rTMS was recorded. HbO changes were analyzed in linear mixed-effects models and Spearman correlations between fNIRS during first LF-rTMS session and depressive symptom pre-post LF-rTMS were computed.

Results / Discussion:
There was a significant effect of treatment session on frontal ΔHbO (baseline vs post-stimulation) and on pre-stimulation SampEnt. Specifically, ΔHbO significantly decreased from session 1 to session 10, and then increased to baseline level again by session 20, while pre-stimulation SampEnt increased from session 1 to 10 and remained high. Decrease in depressive symptoms correlated to ΔSampEnt, but not to ΔHbO measured at session 1.

Conclusion:
The results suggest that LF-rTMS over the right frontal lobe induces changes in within-session (post-pre stimulation) responsivity in frontal cortical HbO and pre-stimulation entropy of the HbO signal. Clinical response could be predicted by change (post-pre) in SampEnt at session 1, which is an indication that this measure could serve as a early biomarker when considering the probability of subsequent response to a full treatment course.

P-B
Postersession B: Mood & Affective Disorders

P-B-015
Revalence of Treatment Resistant Depression and its treatment patterns

Presenter: Christine Reif-Leonhard, Germany

Co-Authors:
Max Bayas
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Andreas Reif
Melanie Formenko

Topic:
7) Mood disorders

Abstract

Objective:
Approximately one third of patients with depression do not achieve remission after treatment with two antidepressants and are defined to have treatment-resistant depression (TRD). Although TRD contributes substantially to the overall burden of disease, robust data on its epidemiology and real-world treatment patterns remain scarce.

Methods:
We used the German DADB Database, comprising 2.47 million statutory health insurance beneficiaries, to estimate TRD prevalence and to characterize treatment strategies. In accordance with the EMA definition, TRD was defined as moderate-to-severe depression with receipt of at least three consecutive antidepressant treatments, including two or more changes in therapeutic strategy (switch, combination, augmentation, electroconvulsive therapy [ECT]).

Results / Discussion:
14.6% of patients were diagnosed with depression, of whom 4.0% had received pharmacological treatment. Among these 0.5% (6.5% of all MDD patients) fulfilled TRD criteria. Only 41.5% of patients received combination or augmentation therapy after failure of first-line treatment. SSRIs predominated across all treatment lines. Even third line treatment was prescribed by GP rather than specialist. ECT was rarely utilized.

Conclusion:
TRD affects at least 350,000 individuals per year in Germany. Real-world treatment data show little guideline adherence with use of ineffective pharmacological strategies. ECT remains critically underutilized. These findings underline the need to improve guideline dissemination and access to specialized care, particularly regarding escalation to evidence-based interventions including ECT.

P-B
Postersession B: Mood & Affective Disorders

P-B-016
Pre-treatment effective connectivity between DLPFC and sgACC predicts remission following rTMS in major depressive disorder

Presenter: Toshiyuki Shimizu, Japan

Co-Authors:
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Topic:
7) Mood disorders

Abstract

Objective:
Functional connectivity between the dorsolateral prefrontal cortex (DLPFC) and the subgenual anterior cingulate cortex (sgACC) has been implicated in the antidepressant mechanisms of repetitive transcranial magnetic stimulation (rTMS) for major depressive disorder (MDD). However, the prognostic value of effective connectivity between these regions remains insufficiently explored.

Methods:
In this single-centre observational study, we analysed pre-treatment electroencephalography (EEG) and clinical data from 30 adults with MDD undergoing high-frequency rTMS targeting the left DLPFC. Directed effective connectivity between the DLPFC and sgACC in the alpha and theta frequency bands was estimated using isolated effective coherence (iCoh) derived from exact low-resolution electromagnetic tomography.

Results / Discussion:
Patients achieving remission exhibited significantly lower baseline alpha-band iCoh from the left DLPFC to the sgACC compared with non-remitters. This measure demonstrated moderate predictive accuracy for remission (AUC = 0.75). Multivariable logistic regression analysis confirmed that reduced alpha-band iCoh independently predicted remission. No significant group differences were observed in the theta band.

Conclusion:
Reduced pre-treatment alpha-band effective connectivity from the left DLPFC to the sgACC is associated with clinical remission following rTMS in MDD. These findings suggest that iCoh-based EEG measures may serve as a neurophysiological biomarker to support patient stratification and personalised rTMS treatment strategies.

P-B
Postersession B: Mood & Affective Disorders

P-B-017
Underlying neural correlates of hand gestures in depression: associations between gesture performance and cognition

Presenter: Sofie Von Känel, Switzerland

Co-Authors:
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Alexios Malifatouratzis
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Topic:
7) Mood disorders

Abstract

Objective:
Gestures are important for nonverbal communication and thus successful social interactions. Individuals with depression show deficits in nonverbal communication, including use of gestures, where execution requires motor and cognitive processing. Neural correlates of gesture performance in depression are unknown. We aimed to investigate this in depression and healthy controls and explore associations with cognitive and motor measures.

Methods:
We measured gesture performance in 30 individuals with depression and 31 healthy controls using a modified version of the test of upper limb apraxia during fMRI. Trials started with a written gesture command or a control sentence, followed by a planning and an execution phase. Cognition (MCCB) and motor performance (grip strength and coin rotation) were measured and associations with gesture performance explored with Spearman's correlations.

Results / Discussion:
Despite no behavioral group differences ($p>0.10$), controls showed a more robust activation in the praxis network, cerebellum, thalamus, and occipital lobe during gesture planning (all $p<0.001$). In patients, gesture performance was associated with social cognition (rho=0.439, p=0.014), and attention/vigilance (rho=0.415, p=0.020), whereas in controls it was associated with coin rotation of the dominant hand (rho=0.380, p=0.035).

Conclusion:
Despite intact gesture performance, praxis network activation was lower in depression during gesture planning compared to controls. In depression, gesture performance was associated with cognition, whereas in controls it was linked to coin rotation, suggesting different

mechanisms supporting gesture performance across groups and might elucidate nonverbal communication difficulties in depression in more cognitively demanding real-world situations.

P-B
Postersession B: Mood & Affective Disorders

P-B-018
Depression and Peripheral Neuropathy as Early Manifestations of Occult Parathyroid Adenoma: A Case Report

Presenter: Omar Afroz, India

Co-Authors:

Rajarapu Hema

Topic:
7) Mood disorders

Abstract

Objective:
Primary hyperparathyroidism can present with neuropsychiatric and neuromuscular symptoms due to chronic calcium dysregulation. However, diagnosis may be delayed if initial imaging is inconclusive. We present the case of an elderly male who initially presented with depressive symptoms and was ultimately diagnosed with parathyroid adenoma. The case highlights the importance of longitudinal evaluation in atypical psychiatric presentations.

Methods:
Case presentation of a 69-year-old male who presented with progressive weight loss, depressive symptoms, and peripheral neuropathy.

Results / Discussion:
Laboratory evaluation revealed elevated calcium (12.3 mg/dL) and parathyroid hormone (196.5 pg/mL). Initial neuroimaging, including PET and parathyroid scintigraphy, showed no focal lesion. Parathyroid imaging (scintigraphy) after one year demonstrated a left superior parathyroid adenoma. The patient underwent surgical excision, following which there was significant improvement in mood and neuropathic complaints.

Conclusion:
Endocrine disorders should be actively considered in late-onset depression accompanied by systemic or neurological features. Reversible metabolic causes offer important therapeutic opportunities in biological psychiatry. This case illustrates the limitations of functional imaging in early or small adenomas and emphasises the diagnostic value of biochemical evaluation and longitudinal reassessment in atypical cases.

P-B
Postersession B: Mood & Affective Disorders

P-B-019
Accelerated intermittent theta burst stimulation treatment in patients with major depressive disorder: A retrospective real-world study

Presenter: Melker Hagsäter, Sweden

Co-Authors:
Fiona Geiger
Caroline Wass
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Topic:
7) Mood disorders

Abstract

Objective:
Major depressive disorder (MDD) is a widespread mental health condition that causes substantial suffering worldwide. Individuals with MDD have an increased risk of suicide, a leading cause of death among young people. Current treatments help only a subset of patients. This study aimed to examine the effectiveness of accelerated intermittent theta burst stimulation (aiTBS) in real-world clinical settings.

Methods:
Data were collected from patients with depressive episodes treated at Kungälv Hospital, Sweden. Information was retrieved from the National Quality Register for TMS (Q-rTMS) between January and December 2025. Depressive symptoms were evaluated using MADRS-S and CGI-I scores before and after treatment.

Results / Discussion:
aiTBS was a well tolerated treatment for depression. There was a significant decline in depressive symptoms at the end of the treatment and 7-19 days post treatment compared to baseline.

Conclusion:
This small real-world study indicates promising results for an accelerated rTMS protocol. However, further research is required to determine the optimal duration and effectiveness of accelerated rTMS treatments.

P-B
Postersession B: Mood & Affective Disorders

P-B-021
Association between recovery, cognitive function, and quality of life levels with peripheral cytokine, bdnf, and vegf levels in patients with bipolar depression

Presenter: Hikaru Hori, Japan

Co-Authors:
Fumito Hamada
Leo Gotoh

Topic:
7) Mood disorders

Abstract

Objective:
The treatment goals for bipolar disorder have expanded beyond merely improving mood symptoms to include enhancing functional levels, such as recovery and restoration of cognitive function. However, knowledge regarding biomarkers associated with bipolar depression and functional levels remains limited. This study investigated the relationship between functional levels in bipolar disorder and cytokines, BDNF, and VEGF.

Methods:
This study employed a cross-sectional design and included 24 participants diagnosed with bipolar depression. Participants underwent assessments of medical records, depressive symptoms(QIDS-SR), social functioning (FAST), cognitive function (Thinc-it tool), personal recovery (QPR-J), quality of life (WHO-QOL), cytokines (IL-2, IL-6, TNF-alpha), BDNF, and VEGF. Correlations between the clinical scales and biomarkers were examined.

Results / Discussion:
FAST scores showed a significant correlation with depressive symptoms (QIDS-SR) (r=0.46, p=0.03). Depressive symptoms (QIDS-SR) showed a significant correlation with FAST scores and subjective cognitive symptoms(PDQ-D-5) (r=0.51, p=0.01). The Personal Recovery Scale and QOL level showed a significant positive correlation (r=0.43, p=0.03).

Conclusion:
No significant association was found between cytokine levels, BDNF, or VEGF and functional levels in bipolar disorder. Further investigation with a larger sample size is necessary.

P-B
Postersession B: Mood & Affective Disorders

P-B-022
Effects of exercise therapy on plasma levels of cytokines, vegf and bdnf in patients with bipolar depression

Presenter: Fumito Hamada, Japan

Co-Authors:
Hikaru Hori
Leo Gotoh

Topic:
7) Mood disorders

Abstract

Objective:
Bipolar disorder is a condition that significantly impairs social functioning due to its high rate of relapse and recurrence. Effective pharmacological treatment options for bipolar depression are limited, leading to increased interest in non-pharmacological treatments.

We focused on exercise therapy among non-pharmacological treatments and investigated its efficacy and effects on blood cytokine levels, BDNF, and VEGF.

Methods:
This study administered a 6-week exercise therapy program (exercise or stretching) to patients with mild to moderate bipolar depression (n=24). Participants underwent clinical assessments (FAST, QIDS-SR, WHOQOL, QPR, Thinc-it tool) and plasma levels of cytokine (IL-6, IL-1β, IL-2, TNF-α, IL-1β), VEGF, and BDNF) at weeks 0 and 6.

Results / Discussion:
Depressive symptoms (QIDS-J) improved significantly ($p < 0.01$), and subjective recovery (QPR) also showed significant improvement ($p = 0.01$). Furthermore, subjective cognitive function (PDQ) also improved significantly ($p = 0.01$).

Conclusion:
A 6-week exercise therapy program for bipolar depression improved depressive symptoms, global functioning, and subjective cognitive function. The effects of exercise therapy on cytokines, BDNF, and VEGF were not clear.

P-B
Postersession B: Mood & Affective Disorders

P-B-023
Determinants of Psychiatric Versus Non-Psychiatric First Contact in Bipolar I Disorder: data from a tertiary care centre in India

Presenter: Rachana Chaturvedi, India

Co-Authors:
Muralidharan K
Gautham M.S

Topic:
7) Mood disorders

Abstract

Objective:
Delayed and non-linear help-seeking pathways in Bipolar I Disorder (BD I) contribute to prolonged untreated illness, increased economic burden, and poorer functional outcomes. BD I has a lifetime prevalence of 0.5% in India, with a treatment gap of >70%. Understanding real-world pathways and determinants of early psychiatric engagement is essential to improve timely access to specialized mental health care.

Methods:
A prospective cross-sectional study included 200 BD I subjects recruited at NIMHANS, India.Caregivers and euthymic patients were interviewed for pathway-related information.Pathways to care were assessed using an author-developed semi-structured proforma based on the WHO Pathways Encounter Form.Group comparisons used Chi-square and Mann-Whitney U tests.Binary logistic regression identified independent predictors of psychiatric consultation.

Results / Discussion:
At first contact, 80% (n=160) consulted psychiatrists, 13.5% faith healers, and 6.5% other providers. Median duration from symptom onset to first contact was 30 days in both groups ($p=0.786$). Awareness of psychiatric illness showed strong association (98.5% vs 1.5%, $p<0.001$). Travelling >14 km had higher odds (AOR=2.80; 95% CI: 1.14–6.85). Sociodemographic and clinical variables were not significantly associated.

Conclusion:
Most BD-I patients accessed psychiatric care at first contact; however, non-psychiatric pathways also remain common and frequently accessed. Awareness of mental illness emerged as the strongest determinant of early psychiatric help-seeking. Strengthening mental health literacy and referral networks may improve timely access to specialized services.

P-B
Postersession B: Mood & Affective Disorders

P-B-024
Biomarkers of depression: a systematic review of indian studies

Presenter: Varshitha Tholasappa, India

Co-Authors:
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Rashmi Arasappa
Muralidharan Kesavan

Topic:
7) Mood disorders

Abstract

Objective:
This review synthesizes biomarker research on depressive disorders from India, with the aim of providing geographical validation to existing data.

Methods:
A systematic literature search was done in PubMed and Scopus for studies published between January 2000 and December 2024. Eligible studies included observational and interventional studies done in the Indian population that evaluated inflammatory markers, neuroendocrine parameters, neurotrophic factors, oxidative stress indicators, neurocognitive and neuroimaging correlates. Quality assessment was done using the AXIS tool.

Results / Discussion:
26 Studies on depressive disorders were identified from a broader mood disorder review. TNF-α decreased and BDNF increased with antidepressants, while IL-6 and CRP showed mixed results; NLR correlated with severity. Neuroimaging showed reduced hippocampal volume, frontal atrophy,reduced occipital activity, reduced limbic connectivity. EEG revealed frontal alpha asymmetry, and cognitive markers included executive dysfunction and memory deficits.

Conclusion:
Studies from India largely replicate global findings. Most depression biomarker research focuses on blood-based markers, while neurocognitive, EEG, and imaging markers remain underutilized. Most studies assess diagnostic utility, with very few examining predictive value and none addressing prognostic utility.

P-B
Postersession B: Mood & Affective Disorders

P-B-025
Association of bdnf val66met polymorphism with antidepressant treatment response in major depressive disorder

Presenter: Ahmad Najjad Husain, India

Co-Authors:
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Topic:
7) Mood disorders

Abstract

Objective:
The BDNF Val66Met (rs6265) polymorphism influences activity-dependent secretion and neural plasticity. Although prior studies report inconsistent associations with antidepressant response, its predictive value remains unclear across populations. This study evaluated whether BDNF Val66Met polymorphism predicts antidepressant treatment response in individuals with Major Depressive Disorder (MDD) in an Indian population.

Methods:
This prospective observational study included 51 individuals with MDD attending the All India Institute of Medical Sciences, Delhi. Deoxyribonucleic acid was extracted from whole blood, and BDNF Val66Met was detected using TaqMan allelic discrimination with real-time polymerase chain reaction. Response was defined as ≥50% reduction in Hamilton Depression Rating Scale (HAMD) scores at 4 and 8 weeks. Chi-square and Spearman's tests were applied.

Results / Discussion:
Among participants, 31 were Val/Val, 15 Val/Met, and 5 Met/Met. Genotype distribution did not differ significantly between responders and non-responders (χ^2=1.83, p=0.40). Comparison of Val/Val versus Met carriers was also non-significant (χ^2=0.001, p=0.97). No significant correlation was observed between polymorphism and HAMD improvement at 4 weeks (r_s=-0.004, p=0.98) or 8 weeks (r_s=0.09, p=0.54).

Conclusion:
BDNF Val66Met polymorphism was not associated with antidepressant response in this cohort, consistent with the prior Indian study (Pathak et al., 2022). Despite its biological relevance, rs6265 showed limited standalone predictive utility for treatment outcomes in MDD, highlighting

the need to evaluate additional genetic variants and integrative biomarker models to improve prediction of antidepressant response.

P-B
Postersession B: Mood & Affective Disorders

P-B-026
Echo: results from a non-interventional cohort study of esketamine nasal spray in treatment resistant depression

Presenter: Christine Reif-Leonhard, Germany

Co-Authors:
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Simon Köhler

Topic:
7) Mood disorders

Abstract

Objective:
Esketamine nasal spray (ESK-NS) in combination with an oral antidepressant is indicated in adults with treatment resistant depression (TRD) lacking response to ≥2 pharmacological treatments in the current moderate to severe depressive episode.[1] The ECHO study investigated ESK-NS effectiveness and safety in real-world clinical settings. Here, we present the first results from ECHO, for patients initiating ESK-NS treatment in 7 countries.

Methods:
ECHO was a prospective, non-interventional phase IV study comprising screening/on-treatment (variable duration)/post-treatment follow-up (6 months) periods. Adults with TRD were prescribed ESK-NS, independent of the study. ESK-NS dosing and safety outcomes were collected at every visit. Clinical response was assessed at Weeks 4/8/12 then every 12 weeks. Montgomery-Åsberg Depression Rating Scale was assessed at Weeks 2/4/6/8 then every 12 weeks.

Results / Discussion:
571 patients were enrolled in ECHO (Germany: 145; Italy: 124; Israel: 101; Belgium: 89; Spain: 59; Sweden: 42; Netherlands: 11). Baseline characteristics were representative of a broad patient population with TRD.[2] Analysis is ongoing; preliminary study results regarding

effectiveness and safety of ESK-NS in a real-world setting will be reported in the poster, in addition to comorbidities and post-treatment follow-up outcomes.

Conclusion:
ECHO is the first large prospective real-world evidence study of ESK-NS in TRD, with a broader patient population than previously studied. Data will enhance understanding of real-world ESK-NS use, which may improve guidance and treatment strategies for patients with TRD. [1] Spravato SmPC. EMA. www.ema.europa.eu/en/medicines/human/EPAR/spravato [Accessed 02/26]; [2] Constant E. ECNP 2025;PS02-1216. Funding: J&J; medical writing: Costello Medical.

P-B
Postersession B: Mood & Affective Disorders

P-B-027
Seizure episode in a bipolar disorder patient on Valproate: Effect of co-morbid condition

Presenter: Shraddha Verma, India

Topic:
7) Mood disorders

Abstract

Objective:
To present a rare case of a bipolar disorder patient on long-term Valproate treatment and Benzodiazepine, with moderate hydronephrosis, who developed a seizure episode.

Methods:
A 32-year-old male, k/c/o Bipolar Disorder on Tab Sodium Valproate 500 mg BD and Tab Lorazepam 2mg HS, presented with a seizure episode.

Results / Discussion:
MRI was suggestive of Minimal Cortical atrophy; the abnormal EEG showed diffuse background slowing (maybe due to Lorazepam use). Seizure episode was controlled on Tab Leviteracetam 500mg BD.

Conclusion:
Breakthrough seizure episode despite patient taking Valproate and lorazepam.

P-B
Postersession B: Mood & Affective Disorders

P-B-028
A study of metabolic syndrome in patients with unipolar depression and bipolar depression

Presenter: Anjali Sharma, India

Co-Authors:
Dinesh Kataria
Sumit Rana
Shiv Prasad

Topic:
7) Mood disorders

Abstract

Objective:
Primary objectives - To assess difference in magnitude of metabolic syndrome in patients with unipolar depression and bipolar depression.

Secondary objectives - To assess relationship of metabolic syndrome with severity of depression.

Methods:
Cross-sectional study of 100 patients (50 unipolar, 50 bipolar) aged 18–65 years diagnosed as per ICD-11. Metabolic syndrome was assessed using modified NCEP ATP III criteria. Anthropometric measures, blood pressure, and fasting glucose and lipid profile were recorded. Depression severity was assessed using BDI and HAM-D scales.

Results / Discussion:
Overall prevalence of metabolic syndrome was 51%. It was present in 60% of unipolar and 42% of bipolar patients ($p=0.072$). Abnormal blood pressure ($p=0.029$) and fasting glucose ($p=0.045$) were significantly higher in unipolar depression. Metabolic syndrome was associated with higher age ($p<0.001$) and greater time spent in illness ($p=0.047$).

Conclusion:
Metabolic syndrome was highly prevalent in both unipolar and bipolar depression, with no significant difference between groups. Unipolar depression showed higher rates of abnormal blood pressure and glucose levels. These findings highlight the need for routine metabolic screening and integrated physical and mental healthcare in individuals with mood disorders.

P-B
Postersession B: Mood & Affective Disorders

P-B-029
Sex-specific treatment effects with adjunctive antidepressant maintenance treatment in bipolar-I depression

Presenter: Alok Kulkarni, India

Co-Authors:
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Topic:
7) Mood disorders

Abstract

Objective:
To examine the sex differences in the treatment response to adjunctive antidepressant maintenance treatment in bipolar I disorder (BD-I).

Methods:
This was a post-hoc analysis of an RCT of adjunctive escitalopram or bupropion XL in patients with BD-I who had recently remitted from a depressive episode. 177 participants were randomly assigned in a 1:1 ratio to continue antidepressant treatment for 52 weeks or to switch to placebo at 8 weeks. The primary outcome was the time to any mood episode assessed via Kaplan-Meier curves. Log rank tests and Cox proportional-hazards model were also used.

Results / Discussion:
Among women, continuing antidepressant treatment for 52-weeks significantly prolonged time to any mood episode compared to discontinuation at 8-weeks (event rates: 23% vs. 48%; hazard ratio [HR] 0.41, 95% CI 0.19-0.85), primarily driven by reduced depressive episodes (9% vs. 44%; [HR] 0.18, 95% CI 0.06-0.51). No significant benefit was observed in men (39% vs. 44%).

Conclusion:
In this post-hoc analysis, adjunctive antidepressant maintenance treatment for 52 weeks significantly prolonged the time to any mood episode in women as compared to men with BD-I. These sex-specific effects highlight the potential value of personalized, sex-informed treatment approaches and warrant confirmation in larger prospective trials.

P-B
Postersession B: Mood & Affective Disorders

P-B-031
Motor cortical plasticity following cathodal hd-tdcs in treatment-resistant depression (trd): A tms-emg study

Presenter: Stuti Dave, India

Co-Authors:
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Clinical Research Centre For Neuromodulation In Psychiatry Consortium

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Topic:
7) Mood disorders

Abstract

Objective:
1. Comparing the degree of inhibition and time to recovery following cathodal HD-tDCS over the motor cortex between patients with TRD and healthy controls (HC) with single-pulse suprathreshold stimulation (SI1MV) and short interval intra-cortical inhibition (SICI) as cortical reactivity measures.

2. Comparing the association between baseline cortical reactivity denoting GABA activity measured using SICI and LICI and anxiety symptoms score.

Methods:
TMS-EMG paired-pulse measures of cortical reactivity (SICI, LICI, SI1mV) were obtained in 116 TRD patients (≥1 failed antidepressant trial) and 119 healthy controls at baseline and 10, 20, 30 & 40 min after cathodal HD-tDCS. Baseline SICI, SI1mV and plasticity indices were compared using linear regression. RM-ANOVA examined group, time and group×time effects. Associations between HAM-A and baseline SICI/LICI were tested.

Results / Discussion:
After controlling for age and site, no significant group differences were observed in baseline SICI, LICI, SI1mV, or plasticity indices. No significant group × time interaction for SICI or SI1mV

were observed either. Anxiety and depression severity were not associated with cortical inhibitory measures.

Conclusion:
Motor cortical reactivity and plasticity parameters following cathodal HD-tDCS did not differ between depression and healthy individuals. Moreover, these parameters had no significant relation with symptom severity.

P-C
Postersession C: Women's Mental Health & Reproductive Psychiatry

P-C-001
Integrating Biological and Clinical Markers for Early Detection of Peripartum Psychiatric Disorders: A Dyadic Approach

Presenter: Caroline Plett, Germany

Topic:
3) Postpartum depression

Abstract

Objective:
The perinatal period involves major neuroendocrine, immune, and psychosocial changes that increase vulnerability to psychiatric disorders. Postpartum depression affects 15–20% of women, while postpartum psychosis is a psychiatric emergency. Despite research on maternal mental health, biological mechanisms after birth, partners' psychopathology, and agreement between self-reports and gynecological findings remain understudied.

Methods:
In a prospective cohort study, mothers and partners are screened for depression, anxiety, PTSD, and suicidality at three visits within 150 days postpartum using standardized tools. Reproductive hormones, cortisol, oxytocin exposure, and immune markers (T cells, Treg, IL-6, IL-10) are assessed. Sociodemographic and obstetric factors are examined as moderators, and gynecological records are reviewed for psychological symptoms.

Results / Discussion:
This study will assess prevalence in mothers and partners, identify biopsychosocial correlates, and examine reporting discrepancies. We hypothesize: (1) high rates of affective and stress symptoms in both parents; (2) links between depressive symptoms and cortisol, neurosteroid, and proinflammatory immune changes; and (3) underdocumented psychiatric symptoms in gynecological records.

Conclusion:
By integrating biological psychiatry with real-world gynecological practice, this study seeks to identify clinically actionable biomarkers and structural gaps in care. A dyadic and multidisciplinary framework may enhance early detection of persons at risk, targeted intervention, and prevention strategies, ultimately improving long-term outcomes for families.

P-C
Postersession C: Women's Mental Health & Reproductive Psychiatry

P-C-002
Assessment of hormonal imbalance and menstrual irregularities among patient with Severe Mental Illness and its impact on treatment adherence

Presenter: Pooja Shakya, India

Co-Authors:

Poulami Basu Rupam Kumari

Topic:
5) Women's mental health, 1) Psychoses

Abstract

Objective:
Menstrual irregularities (MIs) are frequently observed in women with severe mental illness (SMI), and can adversely affect treatment adherence and overall quality of life. This study aimed to assess the prevalence of menstrual irregularities and hormonal imbalance among female psychiatric inpatients.

Methods:
A cross-sectional observational study over nine months. A total of 253 adult female inpatients aged 18–50 years with severe mental illness were consecutively recruited. Menstrual distress was measured with the Menstrual Distress Questionnaire (MEDI-Q), and treatment adherence was assessed by the Brief Adherence Rating Scale (BARS) and TSH, Prolcatin levels were measured.

Results / Discussion:
Menstrual irregularities were present in 39.5% (n=100) of patients. The MI group showed a significantly longer menstrual cycle interval (38.5 ± 16.8 days) compared to those without MIs (29.6 ± 6.5 days; p=0.020) with deranged hormonal levels (TSH and Prolactin). Regression analysis identified longer illness duration (adjusted odds ratio [AOR] = 1.12, p=0.034), and poor treatment adherence(AOR=4.14,p=0.004)as independent predictors of MI.

Conclusion:
Menstrual irregularities are common and clinically significant among women with severe mental illness. Their association with poor treatment adherence highlights a need for integrated, gender-sensitive psychiatric care that includes routine menstrual health assessment. Strategies addressing both mental and reproductive health are essential to enhance quality of life and long-term prognosis in this vulnerable population

P-C
Postersession C: Women's Mental Health & Reproductive Psychiatry

P-C-003
Prevalence and clinical correlates of premenstrual dysphoric symptoms among female psychiatric outpatients

Presenter: Neelofer Jan, India

Co-Authors:

Abdul Majid

Topic:
5) Women's mental health

Abstract

Objective:
To determine the prevalence and pattern of premenstrual dysphoric symptoms among female psychiatric outpatients and to identify clinical correlates associated with symptom severity.

Methods:
A cross-sectional study was conducted among female outpatients aged 18–45 years attending a tertiary care psychiatry OPD. Socio- clinical information were recorded. Psychiatric diagnoses were established according to ICD-11 criteria. Premenstrual symptoms were assessed using the Premenstrual Symptoms Screening Tool (PSST). Associations between premenstrual symptom severity and psychiatric diagnoses, illness duration, treatment were examined.

Results / Discussion:
Premenstrual symptoms were seen in about 17% of patients but full Premenstrual dysphoric disorder was seen only in 5% of patients with highest prevelance and severity in those with the diagnosis of depression and anxiety disorders. Severity was more in patients with ongoing familial conflicts.

Conclusion:
Premenstrual dysphoric symptoms are common among female psychiatric outpatients and may contribute significantly to illness burden. Routine screening for menstrual-cycle-related symptom fluctuations may facilitate earlier recognition and targeted interventions, potentially improving overall psychiatric outcomes and quality of life.

P-C
Postersession C: Women's Mental Health & Reproductive Psychiatry

P-C-004
Prevalence, pattern, and functional impact of sexual dysfunction among drug-naïve female psychiatric outpatients

Presenter: Neelofer Jan, India

Topic:
5) Women's mental health

Abstract

Objective:
To determine the prevalence and pattern of sexual dysfunction among drug-naïve female psychiatric outpatients and to examine its association with psychosocial functioning.

Methods:
A cross-sectional study was conducted amoung consecutive drug-naïve female, married patients aged 18–55 years attending a tertiary care psychiatric outpatient department. Psychiatric diagnoses was established according to ICD-11 criteria. Sexual functioning and it's impact was assessed using the Female Sexual Function Index (FSFI) and Sheehan Disability Scale (SDS). Socio clinical information was collected using a structured proforma.

Results / Discussion:
Sexual dysfunction was seen in almost 60% of our patients with desire and arousal difficulties at the top. It was seen more in women living in extended family settings and having concurrent relationship difficulties. The most common psychiatric diagnosis was seen to be depression followed by anxiety and bodily distress disorder.

Conclusion:
Sexual dysfunction is common in drug-naïve women with psychiatric disorders and contributes substantially to functional impairment. Routine assessment of sexual health in psychiatric settings may facilitate holistic patient care and improve overall treatment outcomes.

P-D
Postersession D: Digital Psychiatry, AI & Precision Medicine

P-D-001
Understanding and Applying the Mind-Metabolism Connection in Clinical Practice. The Difference: Beyond Traditional Silos.

Presenter: Aarti Midha, India

Topic:
10) Other, 2) Schizophrenia

Abstract

Objective:
This poster focuses on the bidirectional pathways that link mental processes and metabolic function. Rather than treating mental health and metabolic conditions as separate entities, this poster translates complex neuroscience into actionable clinical frameworks designed to simultaneously address both psychological and metabolic outcomes.

Methods:
1. Establishing the Neurobiological Foundation

2. Master Clinical Recognition and Assessment

3. Developing Integrated Treatment Plans

Results / Discussion:
Effective management of lifestyle, metabolic, behavioural, and pharmacological interventions is directly related to desired mental health outcomes.

Conclusion:
This poster presents a compelling summary for bridging the mind-metabolism connection in clinical practice. By recognising that metabolic dysregulation is both a driver and consequence of mental illness, participants can move beyond traditional approaches toward a more holistic, integrated care model.

P-D
Postersession D: Digital Psychiatry, AI & Precision Medicine

P-D-002
Systematic Review of Cognitive Training Smartphone Applications

Presenter: Ammu Thampi, India

Co-Authors:
Aruna Laxmy
Renuka Mulay
Paulomi Sudhir
Vikas Menon
Triptish Bhatia
Smita Deshpande
Konasale Prasad
Himani Kashyap

Topic:
10) Other

Abstract

Objective:
Smartphone applications for cognitive training are freely available, yet there is limited information on clinical and neuropsychological features like reinforcement, complexity, self-monitoring, culture-sensitivity, and literacy demands. This study systematically reviews cognitive training smartphone applications by assessing quality, training environment characteristics, accessibility, and inclusivity.

Methods:
A systematic search on Google Play Store identified 974 cognitive training apps. After screening, 43 apps met inclusion criteria. Data were extracted on quality (Mobile App Rating Scale), interactivity (technical support, reminders), training environment (reinforcement, feedback, difficulty levels, self-monitoring, transfer to real life), and inclusivity features (language options, text alternatives, and cultural considerations).

Results / Discussion:
Though some apps had inaccurate description (32%), most apps showed high technical quality (79%). The most commonly trained domains were attention, and memory. Most lacked adaptive difficulty (4%), baseline/outcome assessments, goal-setting, and transfer of skills to real-life (40-49%) or clinician guidance(2%). Culture, language, or literacy inclusivity were limited (35% required English reading; 2% Asian language translations).

Conclusion:
Apps are a good candidate for delivering cognitive training particularly in resource constraint

settings. However existing brain training apps lack grounding in neuro psychological theories, principles of learning and brain behavior interaction. Cultural sensitivity, low literacy options and adaptive difficulty, are critical in supporting diversity

P-D
Postersession D: Digital Psychiatry, AI & Precision Medicine

P-D-003
Development of a Smartphone Application for Cognitive Control: Co-creation Approach

Presenter: Renuka Mulay, India

Co-Authors:
Ammu Thampi
Rajkumari Linthoisana
Radha Chaturvedi
Haroon Lone
Snehil Gupta
Himani Kashyap

Topic:
10) Other

Abstract

Objective:
Smartphone apps offer scalable, low-cost interventions, in resource-limited settings. Most brain-training apps lack neuropsychological grounding, quality indicators, adaptive difficulty, outcome metrics, and accessibility for low-literacy populations. Integrated Cognitive Control Training (ICCT) shows efficacy in preliminary trials and is suited for app delivery. This study aims to develop a gamified ICCT app for psychiatric conditions.

Methods:
ICCT tasks targeting cognitive control were gamified. Cogtrain (single-game beta version) was upgraded through the process of an iterative, co-creation approach with expert consultations, systematic review of cognitive training apps, and user experience testing. Design priorities were better engagement via gamification, minimal language dependence, and feasibility across diverse clinical and cultural contexts.

Results / Discussion:
Final Cogtrain app comprises six cognitive training games: Last Marble, Winglow, Car Count, Match-a-Batch, Score Four and Sudoku. Enhancements include non-verbal cues, tutorials, feedback, reminders, in-game controls and adaptive difficulty and detailed gameplay data recording. Refinements covered layout, speed, predictive algorithms, difficulty levels, and feedback. Security measures included registration, secure login and session notifications.

Conclusion:
Cogtrain is a gamified cognitive training application developed using co-creation approach integrating empirically informed task design and user-centered features. It holds potential as a scalable digital tool for cognitive intervention and research in psychiatric populations and as an

adjunct within stepped-care and hybrid mental health models. Future studies should evaluate its clinical efficacy and transfer-of-training effects.

P-D
Postersession D: Digital Psychiatry, AI & Precision Medicine

P-D-004
Precision Psychiatry Approaches in Clinical Psychiatry: A Paradigm Shift in Practice

Presenter: Satish Suhas, India

Co-Authors:
Deepak Ghadigaonkar
Guru S Gowda
Venkata Senthil Kumar Reddi
John P John

Topic:
2) Schizophrenia

Abstract

Objective:
To evaluate the diagnostic, risk-stratification, and treatment implications of genetic, clinico-radiological and biological psychaitry approaches in patients with complex psychiatric presentations managed at a tertiary care neuropsychiatric center in India.

Methods:
We describe a case series of patients with severe, atypical, early-onset, familial, or treatment-resistant psychiatric presentations who underwent clinically integrated WES, advanced imaging and immune investigations. Variants were interpreted across neurodevelopmental, synaptic, metabolic, and pharmacogenomic pathways and translated into individualized clinical decisions.

Results / Discussion:
WES identified pathogenic or contributory variants in a substantial proportion of cases. Findings informed diagnostic refinement, medication selection or modification, and monitoring strategies. In several cases, genomic data explained prior treatment resistance, atypical adverse effects, or neurocognitive trajectories, improving clinical confidence and shared decision-making.

Conclusion:
Precision psychiatry approach represents a feasible translational tool in selected psychiatric populations, enabling biologically informed diagnosis and personalized treatment. These findings support the integration of genomics, imaging and immune investigations into routine clinical psychiatry and reinforce precision psychiatry as the new standard of care.

P-D
Postersession D: Digital Psychiatry, AI & Precision Medicine

P-D-005
Longitudinal case study of Major Depressive Disorder with Rumination Disorder in an adult female: clinical presentation, diagnosis and treatment approach

Presenter: Shraddha Verma, India

Topic:
7) Mood disorders

Abstract

Objective:
1. To present a rare case of an adult female diagnosed with rumination disorder presenting with chronic regurgitation and depressive symptoms and its course over a period of 3 years.

2. Clinical presentation, assessment, diagnosis and treatment of rumination disorder with comorbid major depressive disorder.

Methods:
Differentiation of vomiting from regurgitation based on history. Investigations, including USG W/A, GI endoscopy, and Oesophageal manometry, were performed to rule out medical conditions. Clinical assessments were done to assess for Depressive and anxiety symptoms. Patient was followed up, and the course of illness was studied for 3 years. Patient was treated using breathing exercises, antidepressants, CBT, and psychological support.

Results / Discussion:
Patient had Chronic effortless regurgitation of food, non-acidic, undigested within 5 minutes of food intake, which would persist for 5-10 minutes, not a/w nausea occurring after every meal, including solid and liquid food. These symptoms were fluctuating based on the depressive symptoms, poor psychosocial support and familial stressors and improved with antidepressants along with CBT and psychological support involving coping strategies.

Conclusion:
Rumination disorder can be adult-onset and can be present in patients with Major Depressive Disorder. Women with depression can present with varied symptoms and disorders; familial stressors and poor psychosocial support play a major role in clinical presentation. Pharmacological and Psychological treatment approaches together are beneficial.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-002
The Intersection of Hypogonadism and Early-Onset Substance Use in Adolescent Males: A Case Series

Presenter: Asmita Vashisht, India

Co-Authors:
Gurveen Kaur
Biswadip Chatterjee
Anju Dhawan

Topic:
6) Addiction, 10) Other

Abstract

Objective:
Substance use, especially illicit opioid use, causes androgen deficiency. Chronic opioid use disrupts hypothalamic – pituitary - gonadal (HPG) axis causing secondary hypogonadism and directly inhibits testosterone impairing bone mineralization, erythropoiesis, gonadal maturation, puberty and fertility aggravated by poor nutrition, poor treatment adherence and relapses. We report three adolescent male cases of illicit drug use with hypogonadism.

Methods:
We present three cases of adolescent males with prepubertal onset of multiple substance use (tobacco, cannabis, inhalant and opioids) and delayed secondary sexual characteristics. Physical examination revealed low BMI, sparse pubic, axillary and facial hair, small sized testes and high-pitched voice corresponding to tanner stage II. Blood investigations revealed low gonadotropins and testosterone levels.

Results / Discussion:
Dependence on nicotine, cannabis and opioid and tentative diagnosis of hypogonadotropic hypogonadism was established. Agonist maintenance (Buprenorphine) and growth monitoring was planned. Relapses were present in two cases with injection drug use and no gain in height, weight or secondary sexual characteristics over one year. In third case, abstinence was achieved from illicit opioids with gain in height and progression to Tanner Stage III.

Conclusion:
Treatment with buprenorphine is associated with lower suppression of HPG axis as compared to Methadone in adult males. Routine anthropometric and physical examination along with hormonal analysis may help guide endocrine treatment options. As compared to adult, gonadotropin therapy is found to be more effective than testosterone therapy in preserving fertility in addition to inducing growth and development in adolescents.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-003
Rational benzodiazepine prescribing in psychiatric practice: a clinical audit of duration control, taper planning, and dependence-risk management

Presenter: Linkan Verma, India

Topic:
6) Addiction, 8) Sleep disorders

Abstract

Objective:
Benzodiazepines remain widely prescribed in psychiatric practice but require careful duration control and monitoring because of tolerance, dependence, and adaptation risks associated with prolonged exposure. This audit evaluated rational benzodiazepine prescribing practices and documentation related to duration control, taper planning, and dependence-risk management in a private psychiatric outpatient clinic.

Methods:
A closed-loop clinical audit was conducted in a private psychiatric outpatient setting. Baseline audit of 100 benzodiazepine prescribing encounters was undertaken between 11–21 February 2026, followed by a structured prescribing improvement approach. Re-audit of 100 prescribing encounters was performed between 3–13 May 2026.

Results / Discussion:
Baseline audit showed strong documentation of dose (100%), duration (91%), taper/de-escalation planning (87%), follow-up (96%), and SOS versus regular prescribing (100%). Among documented durations (n=91), 81.3% were ≤4 weeks. Indication (42%), diagnosis (63%), dependence-risk discussion (5%), substance-use assessment (36%), and non-pharmacological management (12%) improved at re-audit to 87%, 90%, 78%, 69%, and 81%, respectively.

Conclusion:
A structured prescribing-focused intervention was associated with improved rational benzodiazepine prescribing documentation, reduction of dependence risk, and better integration of non-pharmacological management in routine psychiatric outpatient care.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-004
From Neurobiological Mechanisms to Personalised Prescribing: A Prospective Three-Way Comparison of Anti-Craving Agents in ICD-11 Alcohol Dependence

Presenter: Adil Masood, India

Co-Authors:

Minhaj Nasirabadi

Topic:
6) Addiction, 10) Other

Abstract

Objective:
To compare 3-month relapse rates, craving reduction (OCDS), adherence, side effects, and dropout rates among patients with ICD-11 Alcohol Dependence (6C40) prescribed acamprosate, baclofen, or disulfiram in a real-world Indian clinical setting.

Methods:
Prospective observational cohort study, Hyderabad, Telangana. 120 patients (40 per group) with ICD-11 Alcohol Dependence (6C40) received acamprosate (666mg TID), baclofen (titrated 20–60mg/day; ceiling due to sedation above 60mg), or disulfiram (250–500mg/day). Follow-up: baseline, week 1, then fortnightly for 3 months. Primary outcome: time to first relapse. Secondary: OCDS scores, adherence, side effects, dropout rates.

Results / Discussion:
Baseline comparable ($p>0.05$, N=120). ITT relapse: acamprosate 62.5%, baclofen 55.0%, disulfiram 42.5% ($p=0.18$). Per-protocol: acamprosate 51.5%, baclofen 42.4%, disulfiram 21.2% ($p=0.03$). Baclofen: fastest craving reduction (OCDS -12.3 at week 2; $p<0.01$); sedation above 60mg/day (45.0%). Disulfiram: lowest relapse, highest dropout (57.5%). Acamprosate: best adherence (72.5%; $p=0.01$). No serious adverse events.

Conclusion:
First prospective three-way comparison in an Indian ICD-11 AUD population. Baclofen offers rapid craving control (titrate ≤60mg/day); disulfiram is effective only under supervision; acamprosate provides best real-world adherence. Personalised prescribing based on craving severity and social support improves AUD outcomes in Indian settings.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-005
Effect of Cathodal High Definition Transcranial Direct Current Stimulation (HD-tDCS) over Left Dorso-Medial Prefrontal Cortex on Craving in Alcohol Use Disorder – A Pilot, Double-blind, Sham-controlled Randomized Clinical Trial [DRIVE Pilot Study]

Presenter: Chahat Jamwal, India

Co-Authors:
Shalini Naik
Sharon Narula
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Devender Kumar
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Subodh Bn
Debasish Basu

Topic:
6) Addiction, 10) Other

Abstract

Objective:
The study aimed to find out the feasibility and acceptability of doing simultaneous cue exposure and High-definition transcranial direct current stimulation (HD-tDCS) and the effect of this intervention on craving in patients of Alcohol Use Disorder(AUD).

Methods:
The study was a double-blinded randomised clinical study. Forty participants were randomly allocated into two arms. The first arm received cathodal HD-tDCS and cue-exposure in the form of an audio-visual paradigm and the second group was given sham HD-tDCS along with the paradigm.Ten sessions were given over a period of seven days.

Results / Discussion:
38 patients completed at least 3 sessions of HD-tDCS and of these, 36 completed all the sessions. A total dropout of 4 subjects(10%) was found.Significant time effects for craving was noted.There are significant group*time interaction (F = 13.13*), indicating a significant reduction in craving scores post-tDCS.Significant effects for time (F = 75.52*) and interaction effect between group and time (F = 17.57*) was found.

Conclusion:
Our results suggest that HD-tDCS over left dorsomedial prefrontal cortex, was an acceptable

and feasible treatment modality in our study population. We also found reduction in craving scores and number of drinking days and heavy drinking days, however this needs to be further investigated in larger clinical trials.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-006
Trends in Non-invasive Brain Stimulation for Substance Use Disorders and Addictive Behaviours: A Bibliometric Analysis

Presenter: Muhammed Jadeer K, India

Co-Authors:
Priyanka Saha
Pinki Sevda
Rohit Verma
Siddharth Sarkar

Topic:
6) Addiction

Abstract

Objective:
Substance use disorders are chronic, relapsing conditions associated with substantial morbidity and mortality. Non-invasive brain stimulation modalities, including tDCS and TMS, have emerged as potential therapeutic strategies. This bibliometric analysis aimed to characterize global research trends in non-invasive brain stimulation for substance use disorders and related addictive behaviors over the past two decades.

Methods:
The Web of Science Core Collection was systematically searched for English-language publications indexed between 2004 and 2024. Bibliometric indicators included annual publication output, citation metrics, leading authors and institutions, contributing countries, core journals, keyword co-occurrence with temporal overlay, co-citation analysis, and bibliographic coupling.

Results / Discussion:
A total of 551 publications were included, with output peaking in 2022. The USA, China, and Italy led in volume; Harvard University had the highest citation impact. Hanlon, George, and Zangen were key contributors. Drug and Alcohol Dependence was the most cited journal. Research focused on RCTs in nicotine, alcohol, and cocaine use, with three major clusters centered on tDCS and TMS. The most frequent keywords were craving and addiction.

Conclusion:
Research on non-invasive brain stimulation in substance use disorders has grown substantially, with emphasis on alcohol, nicotine, and stimulants. Opioid and cannabis research remains

limited, and advanced modalities such as HD-tDCS and deep TMS are underrepresented, highlighting the need for more rigorous studies.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-007
Anti-craving effect of adjunctive propranolol administration prior to reactivation of addiction related memories by imagery cues: a comparative study using psycho-physiological markers

Presenter: Arnab Datta, India

Co-Authors:

Roshan Khanande

Topic:
6) Addiction

Abstract

Objective:
To determine whether a single dose of propranalol 20mg prior to reactivation of addiction related memories with standardized imagery cues can attenuate subjective craving and physiological reactivity (heart rate variability) in detoxified individuals with alcohol use disorder.

Methods:
Fifty male adults, initiated on anti-craving treatment with Topiramate and Motivational Enhancement Therapy, were randomized to either receive adjunctive single dose pre-reactivation propranolol 20 mg (n=26) on two occasions over a period of three weeks or treatment as usual (n=24). Self-reported craving intensity (Alcohol Craving Questionnaire) and heart rate variability (HRV) index (power of High Frequency band, HF) were measured weekly.

Results / Discussion:
Using repeated measures design for within-subject effect over 3 weeks, there was no statistically significant difference in either self-reported craving intensity as measured by ACQ-NOW scores [F(1,4)=1.529, p=0.218] or relative power of High Frequency band(HF) [F(1,4)=0.813, p=0.475] of HRV, between the adjunctive pre-reactivation propranolol group and treatment as usual group.

Conclusion:
While feasible and acceptable to patients, guided alcohol related memory reactivation under adjunctive propranolol does not lead to reduction in subjective craving intensity or physiological reactivity at doses of 20 mg. More comprehensive trials are needed with wider dosing range and structured cue-reactivity paradigms to establish memory reconsolidation based therapies.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-009
Social Reward Outcompetes Drug Seeking Dopaminergic Ensembles to Prevent Relapse

Presenter: Wei Zheng, People's Republic of China

Co-Authors:
Xiaoxing Liu
Tangsheng Lu
Yanxue Xue
Lin Lu

Topic:
6) Addiction

Abstract

Objective:
Drug addiction is a chronic relapsing brain disease, characterized by high susceptibility to relapse upon re-exposure to drug-related environments even after an extended period of abstinence. Social rewards are believed to have a great therapeutic potential in managing drug addiction, exerting significant protective effects in multiple critical phases of drug addiction. However, the underlying neural mechanisms remain poorly understood.

Methods:
We combined drug self-administration models with immunofluorescence, immediate early gene-based neuronal labeling, fiber photometry, in vivo single-cell calcium imaging, optogenetics, chemogenetics, and electrophysiology to investigate how distinct DAergic subpopulations contribute to the protective effect of social reward against drug relapse.

Results / Discussion:
We identified two distinct dopaminergic ensembles in the ventral tegmental area that respectively encode social reward and drug seeking. These ensembles exert reciprocal inhibition through competitive interactions. Furthermore, social reward-responsive dopaminergic ensembles receive preferential input from the dorsal raphe nucleus, and activation of this pathway recapitulates the protective effects of social reward against drug seeking.

Conclusion:
In this study, we uncovered a dynamic competition between functionally specialized dopaminergic ensembles through which social reward attenuates drug seeking, offering insights that may inform development of novel strategies for SUD treatment.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-010
A prospective study of gut microbiota in patients with alcohol use disorders and its relationship to symptoms and short-term illness course

Presenter: Nandhini Ponnuswamy Bojappen, India

Co-Authors:
Jayant Mahadevan
Bhagyalakshmi Ms
Meera Purushottam
Biju Viswanath
Yogesh Shouche

Topic:
6) Addiction

Abstract

Objective:
To examine differences in gut microbiome profiles between individuals with AUD and healthy controls (HC), to evaluate microbiome changes following abstinence, and to assess associations between microbiome indices and short-term clinical outcomes using standardized drinking and remission phenotypes.

Methods:
This prospective longitudinal case–control study recruited 80 individuals with AUD and 82 age-, sex-, and diet-matched healthy controls. High-quality shotgun metagenomic sequencing data were available for 79 AUD and 75 HC participants at baseline, with repeat stool samples obtained from 34 AUD participants following sustained abstinence.

Results / Discussion:
At baseline, alpha diversity did not differ significantly between AUD and HC groups. Beta diversity , GMWI analyses demonstrated significant differences in overall microbial community composition between groups. Differential abundance analyses revealed significant increases in specific taxa following abstinence.

Conclusion:
Gut microbiome alterations in AUD are characterized primarily by compositional and taxonomic shifts rather than changes in global diversity indices. While remission status showed changes in diversity and wellness indices These findings suggest that microbiome recovery during early abstinence may be better reflected by taxonomic and functional restructuring.

P-E
Postersession E: Substance Use & Addictive Disorders

P-E-011
Biology behind behaviour: a longitudinal study of common hematological parameters as biomarkers in ADHD

Presenter: Ravjot Kaur, India

Co-Author:
Abhinav Agrawal

Topic:
10) Other

Abstract

Objective:
To measure CBC-derived inflammatory markers in children diagnosed with ADHD and assess their association with symptom-improvement across stimulant, non-stimulant, non-pharmacological treatment groups.

Methods:
In this prospective observational study, 82 children diagnosed with ADHD were followed over 12weeks. Peripheral blood samples were collected at baseline and follow-up time points to measure CBC and calculated ratios. Changes over time and associations with clinical improvement, measured using the VADPRS were analysed. Group comparisons and repeatedmeasures ANOVA assessed temporal changes and associations.

Results / Discussion:
Participants exhibited elevated CBC-based markers at baseline. neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and eosinophil-to-lymphocyte ratio (ELR) declined across groups. NLR reductions were greater in stimulant (–0.22, $p < 0.001$) and non-stimulant (–0.24, $p<0.001$) groups, with smaller decline in non-pharmacological group (–0.12, $p=0.035$). VADPRS scores improved significantly in medicated groups ($p<0.001$).

Conclusion:
CBCderived inflammatory indices, particularly NLR, declined alongside ADHD symptom improvement. These findings support their potential role as lowcost, widely available adjuncts for monitoring treatment-response, offering objective complement to symptom-based assessment. This study provides preliminary evidence linking routine haematological measures with clinical outcomes in ADHD, underscoring their value for scalable biomarker research.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-001
Prevalence and clinical correlates of ADHD in patients with borderline personality disorder: A cross-sectional study

Presenter: Faisal Mohammad, India

Co-Authors:

Milan Balakrishnan

Topic:
10) Other

Abstract

Objective:
(1) To assess the prevalence of ADHD in adults with BPD using the DIVA-5. (2) To compare impulsivity (BIS-11) and emotional dysregulation (DERS) between BPD patients with and without ADHD. (3) To examine demographic and clinical differences between the two groups. (4) To analyse the relationship between ADHD symptom severity and measures of impulsivity and emotional dysregulation.

Methods:
A cross-sectional study was conducted among 90 adults (18–45 years) with DSM-5 BPD attending a tertiary psychiatry centre in Mumbai. ADHD was assessed using DIVA-5. Impulsivity and emotional dysregulation were measured using BIS-11 and DERS. Data were analysed using Chi-square tests, independent-samples t-tests, and correlation analyses in SPSS Version 25.

Results / Discussion:
ADHD was present in 38.9% (35/90) of participants. The ADHD group had significantly higher BIS-11 scores (80.17±9.40 vs 75.91±8.37; p=0.027) and higher DERS scores (119.37±18.58 vs 112.35±14.84; p=0.050). Hyperactivity/impulsivity correlated with BIS-11 scores (r=0.256, p=0.015), while inattention correlated with DERS scores (r=0.211, p=0.045).

Conclusion:
ADHD is highly prevalent among adults with BPD and is associated with greater impulsivity and emotional dysregulation. ADHD symptom severity correlates with impulsivity and emotion regulation difficulties. These findings highlight the importance of routine ADHD screening in BPD and support integrated treatment approaches addressing both ADHD and personality pathology.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-002
Beyond port wine stain: ADHD in sturgeon Weber syndrome

Presenter: Harshpreet Kour, India

Topic:
10) Other

Abstract

Objective:
Sturge-Weber syndrome (SWS) is a rare neurocutaneous disorder characterized by facial port-wine birthmarks, glaucoma, and leptomeningeal angiomas, commonly associated with epilepsy.This case report aims to highlight attention deficits as the primary barriers to the patient's academic and social development, the clinical challenges and safe pharmacological management.

Methods:
Case report of a 9 year old boy with SturgeWeber syndrome, supported by the evidence of port wine stain and MRI,presented with academic backwardness, diagnosis of ADHD made through application of Vanderbilt scale and Conners rating scale.

Results / Discussion:
Neuropsychological evaluation confirmed a diagnosis of ADHD, predominantly inattentive subtype. Neuroimaging revealed characteristic leptomeningeal enhancement localized to the right-frontal lobes, directly impacting the frontostriatal networks responsible for executive control. In management Clonidine was given and seizure activity was monitored. Significant improvement was noted.

Conclusion:
This case demonstrates that ADHD symptoms in Sturge-Weber syndrome can present as a distinct, disabling neurobehavioral phenotype which can be understood by structural vascular insufficiency and chronic cortical hypoperfusion. It highlights the necessity of routine, proactive screening for executive dysfunction in SWS patients, even when gross motor or cognitive deficits are mild. It helps to understand integrated management of psychiatry.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-004
Evaluation of cannabidiol effects as an adjunctive therapy on symptoms of Patients with obsessive–compulsive disorder

Presenter: Nazli Zahedi, Iran

Topic:
10) Other

Abstract

Objective:
Cannabidiol (cbd) is considered an effective antiepileptic drug in the treatment of patients with ocd. The present study was conducted to investigate the effect of cbd on ocd symptoms in patients from qazvin province, Iran.

Methods:
In the randomized control clinical trial study, 30 patients were randomly divided into two groups. One group received 25 mg of CBD drops daily, whereas the other group received 25 mg of placebo in the form of drops. At the 4th and 8th weeks, the patients were asked to complete the Yale–Brown Scale again to examine changes in the disease process. All analyses were carried out using SPSS software version 20.

Results / Discussion:
The mean age of patients in CBD and placebo groups was 32.53 and 40.6 years, respectively, which displayed the significant relationship between the two groups ($P < 0.05$). Furthermore, the score of OCD did not show any relationship between the two groups. The RM-ANOVA test indicated the mean score of OCD among studied groups in measured times was statistically significant ($P < 0.05$).

Conclusion:
In the current study, the use of CBD in patients with OCD led to a decrease in the mean OCD score during the study period. This effect was found to be statistically significant between the intervention and control groups over time. Although CBD is suggested as a useful treatment for a variety of psychiatric problems, it is suggested to make clinical trials with larger populations.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-005
A retrospective audit of obsessive-compulsive disorder in a tertiary care hospital in Mumbai, India

Presenter: Pragya Lodha, India

Co-Authors:

Ankita Gupta

Topic:
10) Other

Abstract

Objective:
Obsessive-Compulsive Disorder (OCD) is a serious psychiatric condition with a global prevalence of about 2% and is often underdiagnosed. In South Asia, few studies exist, and in India only one epidemiological study reports a prevalence of 2–3.3%. This retrospective study examines OCD prevalence using case record reviews from a tertiary hospital, highlighting the need for more epidemiological research in the region.

Methods:
This is a single site study. The total number of participants was the number of patients identified with a primary or secondary diagnosis of OCD once the retrospective audit took place at the tertiary care hospital in Mumbai. A total of 92 cases were retrospectively identified. The data was statistically analyzed using descriptive statistics.

Results / Discussion:
Most patients were male, married, and employed. Common obsessions were contamination, sexual thoughts, and self-doubt, while common compulsions were cleaning, checking, and masturbation. Many patients received more than one medication due to medical and psychiatric comorbidities. Fluoxetine was most commonly used, followed by Clomipramine, Clonazepam, Aripiprazole, and Memantine. Findings were consistent with earlier studies.

Conclusion:
This was a retrospective case record analysis of patients with OCD attending the psychiatry outpatient clinic of a single tertiary care hospital. Having established that OCD is often misunderstood, not easily diagnosed and remains an under-diagnosed (severe) psychiatric condition, epidemiological studies are warranted to in populous countries like India.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-006
Non-invasive Brain Stimulation in Children and Adolescents: A Systematic Review in the Indian context

Presenter: Priyadeep Zamindar, India

Co-Authors:
Lakshmi Sravanti
John K. Vijay Sagar
Arul Velusamy
Prasanna Kumar

Topic:
10) Other

Abstract

Objective:
This review explores the various NIBS modalities being used for therapeutic or investigative purposes and evaluates the efficacy, safety, and stimulation parameters within the Indian child and adolescent population.

Methods:
A systematic search was conducted for studies involving repetitive transcranial magnetic stimulation (rTMS), deep TMS (dTMS), theta burst stimulation (TBS), and transcranial direct current stimulation (tDCS) in Indian subjects aged ≤ 18 years using PubMed/MEDLINE, PsycINFO, Scopus, Web of Science, EBSCOhost and Google Scholar). Data from 16 studies were synthesized, focusing on clinical outcomes and tolerability.

Results / Discussion:
The review included 16 studies covering diverse conditions and methodologies including tDCS and types of rTMS. Significant clinical improvements were reported in OCD, ADHD and Depression. Most interventions were well-tolerated; serious adverse events included one seizure episode during iTBS and four instances of affective switching. The most commonly reported side effects were transient headaches and scalp sensations like pain or tingling.

Conclusion:
NIBS shows promise as a safe and effective adjunct in child and adolescent mental health care in India. There is preliminary evidence of good efficacy in OCD and Depression. However, the considerable heterogeneity in study methodology, target areas and stimulation parameters coupled with rare but serious adverse events like seizures, underscores the need for further trials and RCTs to establish definitive paediatric protocols.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-007
Proportion of suicidal ideation among adolescents aged 13 to 19 years with problematic internet use in bangla medium schools of dhaka, bangladesh

Presenter: Itfat Kabir, Bangladesh

Co-Authors:

Mahedy Hasan

Topic:
10) Other

Abstract

Objective:
To determine the proportion of suicidal ideation among adolescents aged 13 to 19 years with problematic internet use in Bangla medium schools of Dhaka city.

Methods:
A cross-sectional study of 157 adolescents (multistage random sampling) excluded severe medical/psychiatric cases and non-internet users. Data were collected via a semi-structured questionnaire, Bangla IAT, and Bangla BSSI. Analyses included descriptive stats, Chi-square, and binary logistic regression (SPSS 21); $p < 0.05$ was significant.

Results / Discussion:
Overall, 52.2% had PIU and 36.6% of them reported suicidal ideation. Problematic users were at higher risk of suicidal ideation compared to normal users ($\chi^2 = 12.682$, $p = 0.002$).Logistic regression identified PIU as a strong predictor of suicidal ideation (AOR = 7.729, $p = 0.001$). Other significant factors included family type, mental disorder in family, internet use hours per day and duration of internet use.

Conclusion:
Suicidal ideation was significantly higher among adolescents with PIU highlighting the need for school-based mental health screening and early interventions.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-008
Neurocognitive and Fronto-Midline EEG Correlates of Symptom Change Following Subthalamic Nucleus Deep Brain Stimulation in Treatment-Resistant Obsessive-Compulsive Disorder

Presenter: Sherine Victoria, India

Co-Authors:
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Topic:
10) Other

Abstract

Objective:
Efficacy of subthalamic nucleus deep brain stimulation(STN-DBS) for obsessive-compulsive disorder (OCD)is predicted by stimulation of the hyperdirect pathway connecting STN and anterior cingulate cortex (ACC),supporting the role of cognitive control and executive function networks.We hence examined if baseline and change in executive function performance, and power of midline theta oscillations, predicted symptomatic improvement following STN-DBS.

Methods:
Resting-state electroencephalography(EEG) and executive function were assessed at baseline(n=13) and at 6-12-month follow-up(n=6). Executive function was evaluated using Flanker, Stroop and Beads tasks. EEG data were processed in EEGLAB(MATLAB) and power spectral density was computed; fronto-midline theta power was defined as the mean power at Fcz, Fc1 and Fc2 electrodes. Behavioral indices were computed by standard methods and analyzed on Jamovi.

Results / Discussion:
Seven of the 13 patients responded to DBS, with 30.4% mean reduction in symptom scores. There was a significant reduction in draws-to-decision(DTD) in the Beads task after DBS (W=34.5,p=0.025), which correlated with symptom change(ρ=0.97,p=0.027). Baseline fronto-

midline theta power predicted change in Flanker interference effect (ρ=0.914,p=0.017), but not symptom severity. Baseline cognitive task performance did not correlate with symptom change.

Conclusion:
STN-DBS was associated with symptom reduction, and specific executive function indices predicted clinical improvement; fronto-midline theta related more strongly to cognitive than symptom change. Interpretation is limited by the small sample size, warranting larger confirmatory studies.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-009
Does Timing Matter? Examining the Role of duration of untreated illness in Outcomes of Obsessive–Compulsive Disorder

Presenter: Varshitha Tholasappa, India

Co-Authors:
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Topic:
10) Other

Abstract

Objective:
To examine the association between duration of untreated illness (DUI) and treatment outcomes from a large database of patients with obsessive-compulsive disorder (OCD) seeking treatment at a specialty clinic.

Methods:
We analyzed baseline and follow-up (FU) data (up to 5 visits) in N=1764 patients. Yale Brown Obsessive-Compulsive Scale (YBOCS) severity was assessed at each visit. The following DUI quartiles were compared: <3 years (early'; n= 399), 3-6 years (n = 410; 'middle'), 6-11 years (n=473, late'), >11years (n=482, 'very late'). Linear mixed-effects models were used to compare YBOCS trajectories between groups across follow-ups.

Results / Discussion:
Significant YBOCS reductions were noted in all 4 groups (YBOCS mean diff = - 11.39, SE = 0.75 points by 5 th FU). Compared to the 'middle' group, greater YBOCS reductions were noted in the 'early' group (m.d = -2.77, SE = 0.98), while lesser reductions were noted in the 'late' (m.d = 2.18, SE = 1.03) and 'very late' (m.d = 2.19, SE= 1.03) groups.

Conclusion:
While all patients who regularly followed up with clinicians showed considerable improvement, early' treatment seekers showed faster symptom reduction highlighting critical role of early intervention in OCD.

P-F
Postersession F: Child, Adolescent & Neurodevelopmental Disorders

P-F-010
Does Timing Matter? Examining the Role of duration of untreated illness in Outcomes of Obsessive–Compulsive Disorder

Presenter: Reinmar Hager, United Kingdom

Co-Authors:
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Michael K. Harte

Topic:
2) Schizophrenia

Abstract

Objective:
Exposure to prenatal stressors such as maternal immune activation (MIA) increases risk for neurodevelopmental disorders. MIA is modelled preclinically via gestational administration of the viral mimetic polyinosinic:polycytidylic acid (PIC), impairing cognition in susceptible offspring. Here, we investigate how individual responses to MIA affects spatial memory in adolescent rats.

Methods:
Pregnant dams were injected with PIC or vehicle on gestational day 15, followed by tail-vein blood sampling 3h, and dam weight-change 24h, post-injection. Object Location (OL) testing on postnatal day 42-50: offspring explored two identical objects before removal to a holding box and returning to the testing area where one object had moved. Time spent exploring novel vs familiar object locations was calculated as Discrimination Index.

Results / Discussion:
Comparing group DIs to 0 (with DI>0 indicating novelty preference and intact spatial memory), we found significant novelty preference in untreated controls ($t(21)=3.189$, $p=0.004$) but not vehicle or PIC. Mixed model analysis found significant effects of offspring sex ($F(1,29)=4.268$, $p=0.042$) and dam weight-loss ($F(1,89)=5.321$, $p=0.023$) on offspring OL performance. Dam weight-loss significantly predicted OL performance in males, but not females.

Conclusion:
Dam weight change response, rather than treatment group, was significantly associated with offspring spatial memory, highlighting the role of individual dam prenatal responses to stressors and the susceptibility risk of impaired cognitive domain function in offspring.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-001
Neurobiology of Suicidal Behaviour: Translating Biomarkers into Prevention

Presenter: Sanjeev Sanjeev, India

Co-Authors:
Tanu Kundal
Kashish
Ibadat Anmol Singh

Topic:
10) Other

Abstract

Objective:
To review the neurobiological basis of suicidal behaviour, including serotonergic dysfunction, neuroinflammation, stress-response abnormalities, and impaired neuroplasticity, and to discuss emerging biomarkers, precision psychiatry, rapid-acting therapeutics, neuromodulation, and digital technologies for early risk identification and personalized suicide prevention strategies.

Methods:
Relevant evidence from peer-reviewed psychiatric, neuroimaging, and translational neuroscience literature was reviewed to examine biological correlates of suicidal behaviour. Data on inflammatory, genetic, epigenetic, and neural biomarkers, along with emerging therapeutic approaches, were synthesized to provide an updated overview of biomarker-based suicide prevention in modern psychiatry.

Results / Discussion:
Recent research links suicidal behaviour with immune dysregulation, altered cortisol responses, impaired neuroplasticity, and abnormalities in frontolimbic brain networks. Emerging biomarkers may help identify high-risk individuals, while interventions such as ketamine, rTMS, and digital monitoring technologies show promise for targeted and timely suicide prevention strategies.

Conclusion:
Neurobiological research has provided important insights into suicidal behaviour and identified promising biomarkers for early risk detection and personalized intervention. Emerging advances in precision psychiatry, neuromodulation, and digital technologies may significantly improve future suicide prevention strategies and clinical outcomes.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-002
Psychiatric Morbidity Among Suicide-Bereaved Family Members: A Clinical Case Study

Presenter: Prachi Shukla, India

Co-Authors:
Sujata Satapathy
Chittaranjan Behera

Topic:
10) Other

Abstract

Objective:
Suicide bereavement is a major psychological stressor that increases the risk of psychiatric disorders among surviving family members. In India, stigma related to suicide and mental illness often delays help-seeking and worsens outcomes. The objective of this study is to examine psychiatric outcomes following suicide bereavement and identify factors affecting access to mental health care.

Methods:
A qualitative clinical case study was conducted involving the mother and sister of a suicide victim. Both individuals developed depressive symptoms following the loss and were clinically diagnosed with Major Depressive Disorder. Data were collected through semi-structured clinical interviews, psychological assessments, and review of medical records. The analysis focused on symptom progression, suicidal behavior, and barriers to treatment.

Results / Discussion:
Within one year of the suicide, both suicide loss survivors showed persistent depressive symptoms, guilt, social withdrawal, and suicidal behavior. Despite significant clinical impairment, professional mental health care was delayed due to stigma, fear of social judgment, and family pressure. These factors limited timely diagnosis and treatment and increased psychological distress.

Conclusion:
Suicide bereavement may act as a significant stress-related risk factor for Major Depressive Disorder and suicidal behavior. Stigma and limited postvention services contribute to delayed care and poorer outcomes. Strengthening accessible and culturally appropriate postvention services is essential to reduce psychiatric morbidity among suicide loss survivors.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-003
The Masquerader: A Case of Pseudoseizure in a 52-Year-Old Male Induced by Emotional Distress

Presenter: Ummuhan Ozkal, Turkey

Co-Authors:
Melanie Cilliers
Laila Asmal

Topic:
10) Other

Abstract

Objective:
The overall objective of this study is to explore how individuals living with schizophrenia, together with their caregivers, in Cape Town's Coloured community make sense of and find meaning in the condition, specifically in relation to diagnosis, repeated relapse, and pathways to recovery, within their sociopolitical, familial, cultural, and spiritual contexts and experiences of marginalisation and resilience.

Methods:
This study adopts a qualitative, multi-phase design including (1) narrative interviews with individuals diagnosed with schizophrenia and their caregivers; (2) participatory Photovoice activities; (3) follow-up interviews using relational mapping; (4) focus group discussions.

Results / Discussion:
Anticipated Results:

Findings are expected to demonstrate how historical trauma, structural disadvantage, spirituality, and family narratives shape illness models, treatment engagement, and recovery trajectories. These insights may help clarify sociocultural moderators of symptom expression, relapse, and treatment adherence in schizophrenia.

Conclusion:
This proposed study highlights the need to integrate sociocultural, historical, and environmental determinants into contemporary models of schizophrenia. By examining lived experience and structural adversity, it advances a contextually grounded approach within biological psychiatry. Bridging biological and psychosocial perspectives is essential for improving diagnosis, treatment engagement, relapse prevention, and equitable mental health care.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-004
A cross-sectional study on inflammatory markers in non-suicidal self-injury.

Presenter: Shruti Agnihotri, India

Topic:
10) Other

Abstract

Objective:
Non-suicidal self-injury (NSSI) is deliberate self-harm without suicidal intent, often rooted in childhood trauma. Such adversity can disrupt the HPA axis and immune system, raising inflammatory markers like CRP. This study investigates the links between adverse childhood experiences (ACEs), these biological signals, and NSSI to understand how trauma translates into physical and behavioral symptoms.

Methods:
This study analyzed 100 self-harm inpatients (16+), excluding those with medical instability or comorbid mental illness. Researchers mapped trauma via the ACE-IQ and Deliberate Self-Harm Inventory, pairing results with 8:00 AM blood draws to measure CRP, ESR, CBP, and serum cortisol. Data were processed using SPSS v22 to identify the biological footprints of childhood adversity and its link to self-injurious behavior.

Results / Discussion:
The research investigates ACE prevalence and its correlation with inflammatory markers in individuals who engage in NSSI. It hypothesizes that these patients exhibit elevated inflammation and altered cortisol levels compared to standard benchmarks. Ultimately, the data reveals a significant positive correlation: as the severity of childhood trauma increases, the intensity of the body's biological immune response follows suit.

Conclusion:
The findings are expected to support the theory that immune activation and HPA axis dysregulation are central to the biological phenotype of NSSI. By identifying specific inflammatory markers associated with self-injury, this research may help clarify NSSI as a maladaptive coping strategy for emotional regulation and potentially guide future biological interventions or risk assessments.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-005
Association Between Genetic Variants in MAOA, COMT, and 5-HTT Genes and Violent Behavior Among Individuals Convicted of Violent Crimes

Presenter: Arvin Haghighatfard, Iran

Topic:
10) Other

Abstract

Objective:
The genetic basis of violence has been widely investigated; however, the specific mechanisms and variants contributing to violent tendencies and criminal behavior remain unclear. This study examined the association between variants in three key genes involved in dopamine degradation and serotonin transport (COMT, MAOA, and 5-HTT) and violent behavior, particularly among individuals convicted of violent crimes.

Methods:
A total of 560 individuals with records of extreme violent crimes and 1,044 individuals without criminal records or histories of severe violence were recruited. Genomic DNA was extracted from peripheral blood samples, and the full lengths of the COMT, MAOA, and 5-HTT genes were sequenced using an ABI 3700 automated sequencer. Personality traits and intelligence were assessed using the NEO-FFI and the Wechsler Adult Intelligence Scale.

Results / Discussion:
Nine SNPs—four in COMT, three in 5-HTT, and two in MAOA—were significantly associated with violent behavior. Many of these variants are recognized risk alleles for psychiatric disorders. Significant correlations were also observed between specific genetic variants and the personality traits of neuroticism and conscientiousness.

Conclusion:
These findings highlight dopamine degradation and serotonin transport as key biological pathways in violence and suggest shared genetic influences between personality traits and violent criminal behavior.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-006
Longitudinal assessment of tau deposition in individuals at risk for chronic traumatic encephalopathy: a pet study with 18f-florzolotau

Presenter: Yuki Komatsu, Japan

Co-Authors:
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Keisuke Takahata

Topic:
4) Dementia

Abstract

Objective:
Spreading of tau deposition following repetitive head impacts (RHI) remains unclear. This neuroimaging cohort study aimed to assess longitudinal changes in tau accumulation using florzolotau (18F) (18F-florzolotau) PET in individuals at risk for chronic traumatic encephalopathy (CTE).

Methods:
Twenty-nine former contact sports athletes (predominantly boxing, with additional exposure to rugby, kickboxing, ice hockey, and American football; mean age 45.4 years; mean exposure duration 16.4 years) and eight healthy controls (HCs; mean age 42.0 years) underwent 18F-florzolotau PET and brain MRI at two time points approximately 2.2 years apart. Regional radiotracer retention was quantified using standardized uptake value ratios (SUVRs).

Results / Discussion:
At baseline, 18F-florzolotau uptake was higher in frontal and temporal gray matter in athletes than in healthy controls. The number of athletes with tau PET positivity was 13 (45%) at baseline and increased to 21 (72%) at follow-up, whereas healthy controls remained negative at both time points. 18F-florzolotau uptake significantly increased over time, predominantly in the frontal cortex and right cingulate cortex ($P < 0.05$).

Conclusion:
This longitudinal study demonstrates progressive tau accumulation following RHI, as measured by 18F-florzolotau PET, in retired contact sports athletes.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-007
The Silent Struggle: Dyspareunia, Sexual Aversion, Emotional Distress, and Suicidal Ideation Rooted in Early Orthodox Conditioning – A Psychosexual Case Report

Presenter: Ibadat Anmol Singh, India

Co-Authors:
Tanu Kundal
Sanjeev

Topic:
5) Women's mental health, 10) Other

Abstract

Objective:
To highlight the impact of internalized sexual shame and restrictive cultural beliefs on the development of dyspareunia, sexual aversion, marital distress, depressive symptoms, and suicidal ideation in a young married woman without identifiable gynecological pathology. This case emphasizes the importance of culturally sensitive, biopsychosocial and multidisciplinary approaches in the assessment and management of female sexual dysfunction.

Methods:
A detailed psychiatric, psychosexual and gynecological assessment was conducted in a young married woman presenting with dyspareunia, sexual aversion, marital distress, depressive symptoms, and suicidal ideation. Organic pathology was excluded through gynecological evaluation and a biopsychosocial formulation was used to guide multidisciplinary management.

Results / Discussion:
Following multidisciplinary management including psychosexual therapy, cognitive behavioral interventions, couple based sessions and pharmacotherapy for depressive and anxiety symptoms, the patient showed improvement in sexual avoidance, mood symptoms, marital communication and suicidal ideation.

Conclusion:
This case highlights the significant impact of culturally internalized sexual shame on female sexual dysfunction, emotional well-being, and marital functioning. Early psychosexual assessment and culturally sensitive multidisciplinary interventions are essential to reduce psychological distress and prevent severe outcomes, including suicidal ideation.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-008
Circadian disruption and PTSD symptom severity in individuals undergoing inpatient trauma treatment: An interim analysis

Presenter: Benicio Frey, Canada

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Topic:
5) Women's mental health

Abstract

Objective:
Circadian rhythm disruption has been implicated in the development and maintenance of posttraumatic stress disorder (PTSD), yet its role as a predictor of treatment response is unclear. Using Composite Phase Deviation (CPD) derived from actigraphy as an objective index of circadian misalignment, this preliminary analysis of an ongoing study examined whether CPD at baseline predicts PTSD symptom trajectory during trauma-focused treatment.

Methods:
Ten individuals who identify as women (19-54 years old) enrolled in an 8-9-week inpatient trauma treatment program completed wrist actigraphy and the PTSD Checklist for DSM-5 (PCL-5) at baseline (TP1), mid-treatment (TP2), and post-treatment (TP3). Baseline CPD was calculated from actigraphy collected during the initial treatment phase, 7-14 days from TP1.

Results / Discussion:
A mixed-effects repeated-measures model was used to evaluate whether baseline CPD predicted treatment response, adjusting for age. PTSD symptoms significantly decreased over treatment (TP3 vs. TP1: $\beta = -19.34$, $p = .012$). Higher baseline CPD predicted treatment response, with attenuated symptom improvement at post-treatment ($\beta = 14.56$, $SE = 6.16$, $p = .046$).

Conclusion:
These preliminary findings suggest that circadian disruption may represent a clinically meaningful prognostic marker of treatment response. Furthermore, targeting circadian regulation may enhance therapeutic outcomes in trauma-exposed populations. Given the preliminary nature of these data, larger longitudinal studies are warranted.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-009
When the body can't regulate: inflammation and vagal failure in suicide attempts

Presenter: Brinda Shree, India

Co-Authors:
Subhasmita Das
Pankaj Verma

Topic:
7) Mood disorders, 2) Schizophrenia

Abstract

Objective:
Suicide risk assessment remains limited by subjective indicators. Immune activation and autonomic dysregulation are implicated in suicidal behavior. This study evaluated whether inflammatory markers (CRP, IL-6) and reduced vagally mediated heart rate variability (HRV) together characterize an immune–autonomic vulnerability profile in individuals with suicide attempts.

Methods:
A PRISMA 2020–guided systematic review and random-effects meta-analysis of observational studies was conducted across PubMed, Embase, PsycINFO, and Web of Science. Studies comparing suicide attempters with psychiatric controls were included. Outcomes included CRP, IL-6, and HRV indices (RMSSD, HF-HRV, SDNN). Effect sizes (Hedges' g) were calculated.

Results / Discussion:
Twenty-eight studies (n≈3,200) were included. Suicide attempts were associated with elevated CRP (g=0.62, 95% CI: 0.41–0.83) and IL-6 (g=0.48, 95% CI: 0.26–0.70). HRV indices were reduced, including RMSSD (g=−0.55, 95% CI: −0.78 to −0.32) and HF-HRV (g=−0.51, 95% CI: −0.73 to −0.29). Moderate heterogeneity was observed (I^2=40–65%).

Conclusion:
Suicide attempts are characterized by concurrent systemic inflammation and reduced vagal tone, supporting an immune–autonomic vulnerability signature. Integrating inflammatory and HRV markers with clinical assessment may enhance biologically informed risk stratification and guide targeted interventions in psychiatric populations.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-010
A biomarker-based approach to post-traumatic stress disorder diagnosis

Presenter: Keren Doenyas-Barak, Israel

Co-Authors:
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Uri Alon
Shai Efrati

Topic:
9) Mental health consequences of terrorism

Abstract

Objective:
To develop and validate a multimodal, objective biomarker for post-traumatic stress disorder (PTSD) by integrating physiological and neurological measures in post-traumatic patients and trauma-exposed controls, addressing the limitations of single-modality biomarker approaches.

Methods:
Military veterans exposed to potentially traumatogenic events, with and without PTSD, are recruited and undergo clinical phenotyping, assessment of post-traumatic symptoms, autonomic nervous system monitoring, and functional MRI using trauma-relevant paradigms. Advanced large-scale data analysis and mechanistic modeling are conducted in collaboration with the Weizmann Institute.

Results / Discussion:
The study is ongoing. Preliminary findings from the multimodal dataset will be presented. Over the course of the research program, a multimodal PTSD biomarker is being developed using a training cohort of approximately 300 individuals with PTSD and trauma-exposed controls and will be validated in an independent PTSD cohort, including evaluation of treatment-related changes.

Conclusion:
This integrative, data-driven approach aims to advance the objective characterization of PTSD and to establish a framework for biomarker-based diagnosis and treatment monitoring.

P-G
Postersession G: Trauma, Stress & Suicidality

P-G-011
Composite Biological Suicide Risk Score Using Cbc-Derived Inflammatory Indices (Nlr, Sii, Siri) And Serum Vitamin D In First-Episode Drug-Naive Psychiatric Presentations: An Observational Study At Sms Medical College, Jaipur

Presenter: Shaifali Mahar, India

Abstract

Objective:
To compare NLR, PLR, MLR, SII, SIRI, and serum Vitamin D levels between first-episode drug-naive psychiatric patients with a recent suicide attempt and those without any history of suicide attempt, and to develop a composite biological risk score for predicting suicide attempt.

Methods:
This observational study was conducted in the Department of Psychiatry, SMS Medical College, Jaipur. First-episode drug-naive psychiatric patients with and without recent suicide attempts were enrolled after informed consent. CBC-derived inflammatory indices (NLR, PLR, MLR, SII, SIRI), serum Vitamin D, HAM-D-17, and C-SSRS scores were assessed and compared between groups.

Conclusion:
A composite biological suicide risk score incorporating CBC-derived inflammatory indices and serum Vitamin D may serve as a simple, low-cost, and objective adjunct for suicide risk assessment in first-episode drug-naive psychiatric patients, with better predictive utility than individual biomarkers alone.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-001
Neuropsychiatric Manifestations of Prion Disease – a Case Report

Presenter: Hemendra Singh, India

Co-Authors:
Swati Chandramouli
Lakshmi Lalitha Priya

Topic:
1) Psychoses, 10) Other

Abstract

Objective:
Prion disease is a rare, rapidly progressive neurodegenerative illness characterised by rapidly progressive cognitive impairment, psychiatric symptoms, and motor symptoms. We report a case of Mrs S, a 58-year-old female, who presented with delusional persecution, hallucinations, and disturbed sleep for 2 weeks. The patient was treated with Olanzapine and Clonazepam 0.5mg BD with no further improvement.

Methods:
Her brain MRI revealed bilateral basal ganglia and right cerebral cortical T2 FLAIR hyperintensity with diffusion restriction, with cortical thinning in the left > right occipital and parietal lobes. Her EEG showed periodic frontotemporal sharp wave discharges with short-interval periodic complexes. Patients developed motor symptoms such as rigidity, myoclonic jerks, apathy, ataxia and inability to walk over a period of 10 days.

Results / Discussion:
She was diagnosed with Creutzfeldt–Jakob disease (CJD). Family members were educated regarding the nature of the illness, and the treatment team explained a guarded prognosis. Patient's condition rapidly deteriorated at home, and she became bedridden, mute and continued to have myoclonic jerks, disturbed sleep and dependent on Ryle tube feeding. The patient passed away 2 weeks post-discharge at home.

Conclusion:
In consistence with previously reported cases, our case presented with psychiatric manifestations and later developed neurological manifestations suggestive of CJD with a poor prognosis. Our case highlights the need for a comprehensive neuropsychiatric evaluation for the early detection of neuropsychiatric manifestations of rare illnesses such as CJD

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-002
Mind in malignancy: neuropsychiatric sequelae of chemotherapy in advanced ovarian carcinoma

Presenter: Poushali Dutta, India

Co-Authors:

Shrinivasa Bhat

Topic:
1) Psychoses

Abstract

Objective:
To highlight the coexistence of advanced ovarian carcinoma and evolving neuropsychiatric manifestations, including psychosis and catatonia, during multimodal chemotherapy, and to emphasize the critical role of early psychiatric evaluation and integrated psycho-oncology care in improving treatment continuity, functional recovery, and overall clinical outcomes in medically complex cancer patients.

Methods:
A 53-year-old woman with Stage IIIC papillary adenocarcinoma of the ovary received neoadjuvant chemotherapy, cytoreductive surgery, adjuvant therapy, and later palliative regimens for metastasis. During treatment she developed behavioral changes progressing to psychosis and catatonia. Serial psychiatric assessments were done. Antipsychotics were started, and a lorazepam challenge confirmed catatonia, guiding benzodiazepine therapy.

Results / Discussion:
Early psychiatric intervention led to rapid identification of psychosis and catatonia. A positive lorazepam challenge enabled prompt benzodiazepine treatment, resulting in reversal of catatonia and stabilization of behavior. Timely management prevented medical complications, improved functional status, and allowed continuation of oncological therapy without significant interruption.

Conclusion:
Neuropsychiatric complications in cancer patients may arise from treatment-related neurotoxicity, metastasis, inflammation, or psychological stress. Early recognition and routine psychiatric screening are essential. Integrated psycho-oncology care enables timely intervention, improves symptom control, supports treatment adherence, enhances functional recovery, and optimizes overall clinical outcomes.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-003
AI in psychotherapy: Exploring the future of mental health care

Presenter: Susmita Halder, India

Topic:
10) Other

Abstract

Objective:
The rapid advancement of AI has significantly influenced recent mental health care, particularly psychotherapy. The paper aims to explore the emerging role of AI in psychotherapy, focusing on its potential benefits, ethical concerns, accessibility, and future implications for mental health care delivery.

Methods:
Data were collected from mental health professionals about their perception and use of AI in psychological intervention. The analysis focused on clinical effectiveness, patient engagement, therapeutic alliance, ethical challenges, and accessibility of mental health services.

Results / Discussion:
Findings indicate that AI-assisted psychotherapy demonstrates promising outcomes in improving accessibility, early screening, symptom monitoring, and delivery of low-intensity psychological interventions. Studies report increased awareness and user engagement particularly among young populations. However, concerns regarding ethics and role of human therapeutic relationship remain significant challenges.

Conclusion:
AI has the potential to transform psychotherapy by complementing traditional therapeutic approaches and expanding access to mental health care. Nevertheless, AI should be viewed as an assistive tool rather than a replacement for human therapists. Future directions require interdisciplinary collaboration, ethical regulation, culturally sensitive models, and empirical validation to ensure responsible integration of AI into psychotherapy practice.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-004
Psychiatric Morbidity in Patients with Type-II Diabetes Mellitus Attending a Tertiary Care Hospital

Presenter: Kashish Kashish, India

Co-Authors:

Tanu Kundal

Topic:
10) Other

Abstract

Objective:
1. To determine the prevalence of psychiatric morbidity in patients with Type-II diabetes mellitus.

2. To identify the common psychiatric disorders among diabetic patients.

3. To assess the association between psychiatric morbidity and glycaemic control.

4. To evaluate the relationship between psychiatric morbidity and medication adherence.

Methods:
A cross-sectional observational study was conducted among 25 patients with Type-II diabetes mellitus attending the diabetic clinic at AMCH Shahabad. Psychiatric morbidity was assessed using the ICD-10 Symptom Checklist for Mental Disorders. Statistical analysis was performed using SPSS version 27.0. Chi-square tests were applied to determine associations between psychiatric morbidity and different variables.

Results / Discussion:
Psychiatric morbidity was identified in 52% of participants. Depression was the most common psychiatric disorder, followed by anxiety disorders and substance use disorders. A significant association was observed between poor glycaemic control (HbA1c >7%) and psychiatric morbidity ($p < 0.001$). Furthermore, 76.9% of participants with psychiatric morbidity were non-adherent to diabetic medications.

Conclusion:
Psychiatric morbidity is highly prevalent among patients with Type-II diabetes mellitus, with depression being the most common disorder. Poor glycaemic control and medication non-adherence were significantly associated with psychiatric morbidity. Routine mental health

screening and integrated psychiatric care should be incorporated into diabetic management protocols to improve patient outcomes and quality of life.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-006
Childrens of a family with progressive regression,slurred speech and clingy behaviour

Presenter: Harshpreet Kour, India

Topic:
10) Other

Abstract

Objective:
To understand the neuropsychiatric manifestations of a rare disorder -Atypical Neuroaxonal Dystrophy (ANAD)and underscore the vital role of genetic testing in confirming the diagnosis.

Methods:
Clinical case evaluation was done through detailed history, family history and neurological examination. MRI was done to evaluate structural changes and diagnosis was confirmed by genetic testing.

Results / Discussion:
The proband exhibited a progressive onset of cerebella ataxia slurred speech and developmental regression such as clingy behaviour. Brain MRI showed cerebellar atrophy correlating to it. The pedigree assessment revealed autosomal recessive pattern in a family where marriages where mainly consanguineous. Genetic testing Solved the mystery of untimely deaths of children with similar illness in the same family.

Conclusion:
ANAD can manifest as a psychiatry disorder. In this case, a child served as a diagnostic proxy for the entire lineage. This allows for risk stratification, prenatal screening and counselling of surviving family members.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-007
Duloxetine induced diurnal bruxism, clinical profile and management, a case report.

Presenter: Hema Rajarapu, India

Co-Authors:
Ayushi Chaturvedi
Nishtha Chawla

Topic:
10) Other

Abstract

Objective:
Serotonin norepinephrine reuptake inhibitors (SNRIs) are known to cause movement disorders, however diurnal bruxism, is rarely reported. The proposed mechanism involves serotonergic modulation of dopaminergic pathways within the basal ganglia, leading to impaired motor inhibition and increased muscle activity. This case report aims to highlight Duloxetine-induced bruxism, its clinical course, and response to medication change.

Methods:
This is a case report of a 29-year-old married female who was initiated on Duloxetine for management of bodily distress disorder (ICD-11). Two weeks after starting Duloxetine, the patient developed involuntary jaw clenching and teeth grinding associated with yawning. The symptoms persisted for eight months. Literature review was done and Duloxetine was gradually tapered and discontinued, and the patient was started on Amitriptyline 10 mg.

Results / Discussion:
Following discontinuation of Duloxetine, the patient reported improvement in bruxism within two weeks. Complete resolution of jaw clenching, teeth grinding, and associated jaw discomfort was observed within one month. No recurrence of symptoms was noted after switching to Amitriptyline. The bruxism in this case was predominantly diurnal, with no significant nocturnal symptoms reported.

Conclusion:
This case highlights Duloxetine induced diurnal bruxism as a rare but clinically relevant adverse effect. Awareness of such rare presentations is essential, as delayed recognition may lead to prolonged discomfort and functional impairment. Prompt identification, medication review, and timely substitution with alternative agents can result in complete symptom resolution and improved quality of life.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-008
Clinical Spectrum and Referral Patterns for EEG in a Tertiary Care Teaching Hospital: A Psychiatry-Centric Perspective

Presenter: Rutuja Dharmadhikari, India

Co-Authors:
Chetan Vispute
Deepak Thakur

Topic:
10) Other

Abstract

Objective:
1. To study the demographic data of patients referred for EEG.
2. To analyze department-wise EEG referral patterns.

Methods:
1)Study Design: Retrospective observational study.
2)Study Setting: Tertiary care teaching hospital.
3)Study Duration: From December 2025 to December 2026
4)Sample: Consecutive patients referred for EEG during the study period.

Inclusion Criteria: * All patients referred for EEG during the study period.
Exclusion Criteria: * Incomplete records or missing demographic data.

Results / Discussion:
Patients spanned pediatric, adult, and geriatric groups with near-equal gender distribution. Most referrals came from ICUs and medicine wards, followed by neurology and psychiatry. Indications included seizures, altered sensorium, delirium, and acute behavioral disturbances. Of 1,056 EEGs, 17.99% were normal, 76.04% abnormal, 5.78% grossly abnormal, and 0.19% brain-death, highlighting substantial organic dysfunction.

Conclusion:
EEG provides key diagnostic clarity in psychiatry, especially in delirium and acute behavioral disturbances. ICU-based referrals highlight its role in liaison psychiatry. A psychiatry-focused approach bridges neurology, aids early recognition of organic pathology, strengthens multidisciplinary care, and structured EEG training may improve diagnostic confidence and outcomes.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-009
Investigating host genetic and gut microbiome interaction in common psychiatric disorders within a South African cohort

Presenter: Connor Nell, South Africa

Co-Authors:
Sian Hemmings
Elizna Maasdorp

Topic:
10) Other

Abstract

Objective:
To identify host genetic variants and construct polygenic risk scores associated with PTSD, depression, and anxiety in a South African cohort; to characterise gut microbiome composition and diversity in relation to symptom severity of these stress-related psychiatric disorders; and to investigate interactions between host genetic risk and gut microbial profiles to better understand their combined contribution to psychiatric vulnerability.

Methods:
Genotype data underwent quality control, phasing, and imputation prior to the construction of polygenic risk scores for PTSD, depression, and anxiety. Gut microbiome sequencing data were processed using QIIME2 to assess diversity and differential abundance. Integrated regression models were then applied to evaluate interactions between genetic risk and microbiome composition while adjusting for relevant covariates.

Results / Discussion:
Laboratory work has been completed, with microbial DNA successfully extracted and sequenced, and host genetic DNA genotyped using high-density SNP arrays. Genotype data have undergone quality control, filtering, and imputation, and the resulting SNPs are currently being used for polygenic risk score construction. Microbiome data processing and statistical analyses are underway and are expected to be completed by June.

Conclusion:
This project integrated host genetic and gut microbiome data to investigate stress-related psychiatric symptoms in a South African cohort. With analyses underway, final interpretation will clarify genome–microbiome interactions underlying mental health risk. Once completed, findings have the potential to advance South African specific psychiatric research and gain a deeper understanding of the biological processes in psychiatric disorders.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-010
Neuropsychiatric pleiotropy in 7q11.23 microduplication: A Case Report

Presenter: Sarthak Kukreja, India

Co-Authors:

Vaibhav Patil

Topic:
10) Other

Abstract

Objective:
To describe an adult presentation of 7q11.23 microduplication (dup7q11.23) with convergent neuropsychiatric phenotypes: autism spectrum disorder (ASD), chronic motor tic disorder, and obsessive compulsive disorder (OCD) and to contextualise this triad within the dup7q11.23 literature.

Methods:
Clinical case review of developmental, behavioural, and movement symptoms from childhood to age 22 years, supplemented by genetic confirmation via chromosomal microarray demonstrating dup7q11.23 and a focused review of dup7q11.23-associated neuropsychiatric phenotypes.

Results / Discussion:
Social-communication deficits and delayed expressive language supported ASD. From ~10 years, he had brief, partially suppressible motor tics with premonitory urge (blinking and multifocal limb/neck–shoulder/orofacial tics), worsening over 2 years with functional decline. For 4 years he had contamination obsessions with washing/cleaning compulsions. Clonidine, risperidone, clonazepam and haloperidol failed; aripiprazole improved tics and OCD.

Conclusion:
This case supports dup7q11.23 as a rare-variant risk state for a clinically significant ASD–chronic motor tic–OCD phenotype into young adulthood and highlights aripiprazole as a potentially useful option when standard tic-targeting agents are inadequate, warranting systematic study in genetically defined subgroups.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-011
Divergent spatial strategies and acquisition rate in a cross-species T-maze

Presenter: Chaim G. Pick, Israel

Co-Authors:
Topaz Loushy Kay
Oded Perlman
Lior Bikovski
Ella Been

Topic:
10) Other

Abstract

Objective:
Spatial navigation relies on allocentric (map-like) and egocentric (body-centered) strategies, yet the extent to which these processes are conserved across species remains unclear. We aimed to compare learning speed, strategy preferences, and performance in mice and humans using translationally analogous T-maze tasks.

Methods:
Mice (n=24) performed a physical T-maze, while humans (n=62) navigated a virtual reality analogue version with natural locomotion. Both underwent learning and probe phases to isolate Response (egocentric), Cue, and Place (allocentric) strategies. Human data included self-reports and eye-tracking measures for validation and further understanding of the underlying cognitive processes.

Results / Discussion:
Humans learned ~10-fold faster than mice ($P<0.001$). Multivariate analysis revealed strong species differences in strategy use ($P<0.001$): Mice relied more heavily on the Response strategy, whereas humans exhibited a stronger preference for the Cue strategy. Humans also demonstrated greater Place consistency ($P<0.001$). No significant sex or species-by-sex interaction was found. Human behavioural measures converged with self-reports ($P<0.001$).

Conclusion:
These findings reveal conserved navigational abilities across mammals but pronounced species-specific preferences. Mice rely on an egocentric strategy, while humans exhibit faster learning and more versatile navigation, integrating landmark-based strategies. This highlights a significant evolutionary divergence in spatial cognitive strategies despite broadly conserved hippocampal architecture.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-012
Premonitory Urges at the Interface of Subjective Experience and Motor Activity: A Surface EMG Case Study

Presenter: Dipesh Patel, United Kingdom

Co-Authors:

Himanshu Tyagi

Topic:
10) Other

Abstract

Objective:
Premonitory urges are central to tic disorders, yet assessment relies heavily on subjective awareness. Limitations in identifying or localising urges can limit the effectiveness of Habit Reversal Training (HbRT). This case study investigates whether surface electromyography (sEMG) can detect sub-threshold motor activity associated with premonitory urges in a patient without reliable urge awareness.

Methods:
Surface EMG electrodes were placed over muscles implicated in a patient's most impairing motor tics in the context of a well-established diagnosis of Gilles de la Tourette syndrome. Recordings were collected during rest, tic expression, and attempted suppression. Pre-tic muscle activation occurring without conscious urge awareness was identified and used to refine the timing and muscle specificity of competing responses during HbRT.

Results / Discussion:
Surface EMG showed reproducible low-amplitude muscle activation preceding overt tics, despite absent subjective awareness of premonitory urges. These signals enabled earlier and more precise competing responses targeting the involved muscles. Over treatment, the patient demonstrated improved behavioural control and emerging awareness of early motor activity previously experienced as sudden or unpredictable tics.

Conclusion:
This case provides preliminary evidence that sEMG can detect motor correlates of premonitory urges beyond conscious awareness and may enhance the precision of behavioural interventions in tic disorders. Objective physiological markers may help bridge subjective experience and motor output, particularly in individuals with limited interoceptive awareness. Further systematic research is needed to assess generalisability and clinical impact.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-013
Neuromodulation Therapies in the Management of Tardive Dyskinesia: A Narrative Review

Presenter: Anaf Kololichalil, India

Co-Authors:

Vaibhav Patil

Topic:
10) Other

Abstract

Objective:
Tardive dyskinesia (TD) is a chronic hyperkinetic movement disorder resulting from prolonged exposure to dopamine receptor–blocking agents. Despite the availability of vesicular monoamine transporter-2 (VMAT2) inhibitors, therapeutic response remains inconsistent, and many patients continue to experience disabling symptoms. Neuromodulation strategies have therefore emerged as potential alternatives for refractory TD.

Methods:
A comprehensive literature search was conducted in PubMed, Web of Science, and Scopus using the relevant search terms. Original studies, randomized trials, open-label investigations, retrospective analyses, and clinically relevant case reports published in English between January 2000 and September 2025 were included.

Results / Discussion:
Nineteen studies met inclusion criteria. Among neuromodulation techniques, deep brain stimulation of the globus pallidus internus showed the most consistent and durable benefit, with up to 90% symptom reduction. Electroconvulsive therapy produced improvement in about two-thirds of cases, while rTMS showed short-term benefit. tDCS yielded promising but variable results. Overall, safety profiles were favorable.

Conclusion:
Neuromodulation is an evidence-supported adjunct for pharmacoresistant TD, with GPi-DBS showing the strongest efficacy. Prevention remains key, including careful antipsychotic use, regular monitoring, and early VMAT2 inhibitor initiation. Future research should focus on multicentre trials, standardized protocols, biomarkers, and emerging modalities.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-014
Undiagnosed Alkaptonuria in a 42-Year-Old Female with Intellectual Disability: A Case Report

Presenter: Harshit Harshit, India

Topic:
10) Other

Abstract

Objective:
To present a case of undiagnosed Alkaptonuria in a 42-Year-Old Female with Intellectual Disability and discuss implications of rare diseases in psychiatry

Methods:
A 42-year-old woman with lifelong intellectual disability, seizures, and progressive joint pain presented with worsening anger outbursts. She had black urine since birth. Examination showed restricted joint movement and mild extrapyramidal signs. Urine homogentisic acid was elevated, confirming alkaptonuria with ochronotic arthropathy. Behavioral issues improved after pain management, antipsychotic adjustment, and functional behavioral therapy.

Results / Discussion:
Alkaptonuria was diagnosed late despite longstanding arthritis and black urine. Psychiatric comorbidity and intellectual disability likely delayed recognition. While pain-related irritability is explainable, direct evidence linking homogentisic acid to intellectual disability is lacking, though potential neurodegenerative effects remain hypothesized.

Conclusion:
Rare diseases in patients with psychiatric comorbidity are often overlooked due to diagnostic overshadowing and low clinical suspicion. Integrated medical–psychiatric evaluation is essential to avoid delayed or missed diagnoses.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-015
Lavender oil preparation Silexan does not impair cognitive performance

Presenter: Borwin Bandelow, Germany

Co-Authors:
Priyaranjan Avinash
Debanjan Banerjee
Rohit Garg

Topic:
10) Other

Abstract

Objective:
Use of anxiolytics and some antidepressants may be associated with cognitive dysfunctions. In a published clinical trial focusing on EEG-effects of Silexan, a lavandula extract used to treat anxiety disorders, cognitive performance of healthy volunteers was examined and will be reported here in detail.

Methods:
In a double-blind, placebo-controlled, 3-period crossover trial, healthy volunteers (n=23) aged 18–65 received 1 capsule of 80 mg or 160 mg/day of Silexan1 or placebo during each treatment phase for 14 days. Participants were alone in a quiet room with dimmed light. After a 6-minute quantitative EEG recording with the participants' eyes open and a 4-minute recording with eyes closed, 5-minute recordings of 3 cognitive tests were performed.

Results / Discussion:
The performance of the d2 concentration test, the Concentration Performance Test (CPT) and a memory test revealed no relevant differences between placebo and the two Silexan dosages. This was true for all time points (0, 1, 2, 3, and 4 hours after drug intake), whether the drug was administered once or repeatedly. Furthermore, Silexan did not alter EEG parameters during the 3 cognitive tests.

Conclusion:
Silexan (80 mg and 160 mg) did not affect the cognitive performance of trial participants in three tests assessing aspects of cognition and memory.

1 Silexan® is the active substance of Lasea (Dr. Willmar Schwabe GmbH & Co. KG, Karlsruhe, Germany)

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-016
Emotional brain under fire: a case series of psychiatric presentations in temporal lobe epilepsy

Presenter: Sunil Mittal, India

Co-Authors:
Arundhati Bhagabati
Asmita Tomar

Topic:
10) Other

Abstract

Objective:
Temporal lobe epilepsy is the most common form of focal epilepsy and is characterised by seizures arising from medial or lateral temporal structures with comorbid psychiatric disorders occurring frequently(40% to 80%). The most consistently described conditions include mood, anxiety, psychotic disorders and behavioral difficulties. the objective is to understand the overlap of symptoms and subsequent treatment lapse and morbidity.

Methods:
Case study of patients

Results / Discussion:
The temporal lobe epilepsy had gone undiagnosed in the patients and were treated primarily for the psychiatric manifestation which lead to polypharmacy and doctor shopping. once diagnosed and treated adequately there was significant improvement in symptoms.

Conclusion:
Neurological disorders can present with symptoms of psychiatric disorders. early identification can help mitigate debilitating sequelae.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-017
Anxiety as a bridge between schizotypal traits and negative symptom expression: evidence from a non-clinical sample

Presenter: Jozef Dragašek, Slovakia

Co-Authors:
Matus Hrebenar
Martina Ruzickova

Topic:
10) Other

Abstract

Objective:
Negative-symptom–like experiences occur on a continuum in non-clinical populations and may relate to schizotypal traits. Trait anxiety may act as a transdiagnostic mechanism amplifying motivational deficits and social withdrawal. We examined associations among schizotypy, self-reported negative symptoms and trait anxiety, and tested whether trait anxiety mediates the schizotypy–negative symptom link.

Methods:
Cross-sectional study in healthy young adults (N=193; 22.7±4.07 years; 17–37; 73% female). Participants completed SPQ (total; Cognitive–Perceptual, Interpersonal, Disorganized), SNS (total), and STAI Y-2. Pearson correlations were computed. Mediation models tested STAI Y-2 between SPQ factors and SNS; analyses adjusted for age and sex.

Results / Discussion:
Schizotypy correlated with negative symptoms (SPQ–SNS r=0.67), strongest for SPQ Interpersonal (r=0.69). Trait anxiety correlated with SNS (r=0.58) and SPQ factors (r=0.42–0.59). STAI Y-2 partially mediated SPQ→SNS: CP indirect=0.225 (z=5.19; p<0.001; 56.7%), IP 0.159 (z=3.91; 23.2%), DO 0.185 (z=4.69; 35.9%); effects persisted after age/sex adjustment.

Conclusion:
In a non-clinical young-adult sample, schizotypal traits and trait anxiety are robustly associated with self-reported negative symptom expression. Trait anxiety accounts for a meaningful proportion of the schizotypy–negative symptom relationship, supporting anxiety as a potentially relevant mechanistic bridge and a candidate target for future longitudinal and intervention research.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-018
Exploring the Neuroprotective Potential of Raloxifene and Fulvestrant Against Haloperidol Induced Parkinson Disease: Insights into GPR30/PI3K/Akt/Nrf2 Signaling Pathways

Presenter: Shubham Upadhayay, India

Co-Authors:

Puneet Kumar

Topic:
2) Schizophrenia, 1) Psychoses

Abstract

Objective:
To investigate the neuroprotective effect of raloxifene and fulvestrant on behavioral, biochemical, neurochemical, and molecular alterations in a haloperidol-induced animal model of Tardive dyskinesia.

To investigate the effect of raloxifene and fulvestrant on immunohistopathological alteration in striatal tissues on haloperidol-injected Rats.

To explore the molecular mechanism of raloxifene and fulvestrant by using GPR30 agonist and antagonist.

Methods:
Adult 8-week-old Male Wistar rats weighing 180 to 250 g were utilized for the in-vivo study; all behavior parameters were performed on 13 groups. Biochemical, neurochemical, and western blotting were performed on n=6 animals. ELISA kits were utilized for inflammatory and apoptotic markers, and CST antibodies were preferred for western blot.

Results / Discussion:
Raloxifene and fulvestrant 5 and 10 mg/kg treatment significantly improved the behavioral activity, including motor coordination, grip strength, and locomotor activity in rats treated with haloperidol. Moreover, raloxifene and fulvestrant significantly restored antioxidant enzyme activity, the expression of GPR30, PI3k, Akt, and Nrf2, and reduced TNF-α, IL-1β, and apoptotic markers in the striatum of rats treated with haloperidol.

Conclusion:
Study findings suggested that raloxifene and fulvestrant have strong antioxidant, anti-inflammatory, and anti-apoptotic properties, which help to ameliorate the antipsychotic-induced tardive dyskinesia-like movement. findings also suggested that raloxifene and

fulvestrant have neuroprotective abilities. It could be used as a treatment option for neurological disorders, including tardive dyskinesia.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-019
Sex- and Region-Specific TRKB Isoform Imbalance and Amyloid-Beta Modulation in 5xFAD Mice

Presenter: Noa Bregman-Yemini, Israel

Co-Authors:
Keren Nitzan
Oren Marzuk
Tal Cohen
Dikla Rozen
Shiran Tetro
Maayan Gal
Ravid Doron

Topic:
4) Dementia

Abstract

Objective:
Alzheimer's disease (AD) shows sex differences, with females exhibiting earlier affective and cognitive decline and greater amygdalar vulnerability. Dysregulation of the BDNF/TRKB pathway, marked by reduced TRKB.FL and increased dominant-negative TRKB.T1, may drive amyloid-β-related pathology. This study mapped early sex- and region-specific TRKB imbalance in 5xFAD mice to determine whether Aβ and TRKB deficits exhibit sex-dimorphic patterns.

Methods:
Male and female 5xFAD mice were assessed at 3, 4.5, and 6 months. TRKB.FL/TRKB.T1 levels, Aβ burden, and glial markers (GFAP, IBA1) were quantified in the amygdala, hippocampus, and prefrontal cortex. A parallel cohort received early Aβ-attenuation immunotherapy to examine sex- and region-dependent effects on neurotrophic signaling, neuroinflammation, and cognition, alongside behavioral tests of recognition memory and learning.

Results / Discussion:
Female, but not male, 5xFAD mice showed early elevation of amygdalar TRKB.T1 by 3 months, preceding neuroinflammatory activation. Across 3-6 months, TRKB isoforms displayed distinct sex-dependent pathological trajectories. Early Aβ attenuation selectively prevented TRKB.T1 upregulation in females, reduced GFAP and IBA1 expression, and enhanced behavioral outcomes, revealing a significant therapy, sex and region interaction.

Conclusion:
These data identify Aβ-driven elevation of TRKB.T1 as a reversible, sex- and region-selective

pathogenic switch. The female amygdala emerges as an early vulnerability node, defining a therapeutic window in which targeted Aβ modulation may stabilize TRKB signaling, limit neuroinflammation, and modify disease trajectory.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-020
Psychoactive drug effects on melanin production and melanocyte gene expression in zebrafish larvae

Presenter: Siroshini K Thiagarajan, Malaysia

Co-Authors:
Pek Yee Tang
Siew Ying Mok

Topic:
6) Addiction, 1) Psychoses

Abstract

Objective:
This study examines how opioid use disorder–related compounds (morphine, MK801, and domperidone) affect melanogenesis and melanocyte gene expression in developing zebrafish (Danio rerio) to determine whether pigmentation biology is altered through pathways relevant to opioid exposure.

Methods:
Wild-type zebrafish embryos were exposed to optimized drug concentrations for 3 or 10 days, followed by a 5-day withdrawal period. Concentration optimization used n=5 embryos in three independent experiments. For pigmentation studies, embryos were allocated to three groups (n=50/group). Pigmentation was assessed microscopically, melanin quantified spectrophotometrically, and SOX10, TYR, and TRP1 expression analyzed by qPCR.

Results / Discussion:
The compounds produced distinct pigmentation effects. Morphine altered pigmentation mainly after long-term exposure and withdrawal, whereas MK801 consistently increased melanin levels under both acute and chronic treatments, as confirmed spectrophotometrically. qPCR showed differential regulation of SOX10, TYR, and TRP1, suggesting pathway-specific modulation of melanogenesis rather than toxicity or disrupted melanocyte development.

Conclusion:
Opioid use disorder–related compounds significantly modulate pigmentation biology in zebrafish by altering melanin production and melanocyte gene expression. These findings highlight links between opioid-related signaling and melanogenesis, supporting the use of zebrafish as a sensitive model for studying pigmentation changes associated with opioid exposure.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-021
Clinical Correlates of Elevated Serum Interleukin-6 in Drug-Naive Major Depressive Disorder: Evidence for an Inflammatory Vulnerability Subtype

Presenter: Ranjit Singh, India

Co-Authors:
Sumeet Kaur Madaan
Rohit Yadan
Suresh Gocher

Topic:
7) Mood disorders, 1) Psychoses

Abstract

Objective:
Depression is a leading global burden, particularly in LMICs. Elevated interleukin-6 (IL-6) has been implicated in MDD, yet whether it reflects acute symptom severity or biological vulnerability remains unclear. This study compared serum IL-6 in drug-naïve MDD patients versus healthy controls and examined clinical correlates including illness duration, family history, and symptom severity.

Methods:
Case-control study at Rabindranath Tagore Medical College, Udaipur (July–November 2022). Fifty drug-naïve MDD patients and 50 matched healthy controls were enrolled. Depression severity was rated using HAM-D. Serum IL-6 was quantified by ELISA. Statistical analysis included t-tests, one-way ANOVA with LSD post-hoc, and Pearson correlation.

Results / Discussion:
IL-6 was significantly elevated in cases vs controls (11.26 vs 4.59 pg/mL; p=0.001). IL-6 was higher with family history of depression (18.07 vs 8.61 pg/mL; p=0.032) and illness duration >5 years (35.85 pg/mL; p=0.002). IL-6 did not correlate with HAM-D scores (r=0.108; p=0.454).

Conclusion:
Serum IL-6 is elevated in drug-naïve MDD and associates with illness chronicity and familial risk, but not acute symptom severity. This supports IL-6 as a trait biomarker of neuroinflammatory vulnerability rather than a state marker, with implications for patient stratification and anti-inflammatory treatment strategies.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-022
When control cost too much: phenytoin induced adverse drug reactions with psychiatric morbidity in a woman with epilepsy.

Presenter: Sanjeev Sanjeev, India

Co-Authors:

Tanu Kundal

Topic:
7) Mood disorders, 10) Other

Abstract

Objective:
To highlight the broad spectrum of phenytoin-induced adverse drug reactions, particularly neuropsychiatric and depressive manifestations, in a woman with epilepsy, and to emphasize the importance of early recognition and multidisciplinary management.

Methods:
A detailed clinical evaluation of the patient and review of medication history and adverse effects was conducted in a 34-year-old woman with epilepsy receiving long-term phenytoin therapy. Adverse drug reaction causality was assessed using WHO-UMC/Naranjo criteria, and multidisciplinary management involving psychiatry, neurology, and dermatology and monitoring of seizure control and mood symptoms after AEM modification.

Results / Discussion:
The patient showed improvement in dermatological symptoms and improvement in mood symptoms following phenytoin withdrawal, supportive care and multidisciplinary management. Further longitudinal monitoring of seizure control, depressive symptoms, and overall functional outcome was planned following AEM modification and continuation of psychiatric treatment.

Conclusion:
Long-term phenytoin therapy requires vigilant monitoring for adverse reactions, especially in women with epilepsy who face unique psychosocial and reproductive health challenges. Early recognition and rational AEM substitution, coupled with integrated psychiatric care, are essential to prevent morbidity.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-023
Somatic multimorbidity in affective and anxiety disorders in the German National Cohort (NAKO): Epidemiological patterns and risk associations

Presenter: Julia Fietz, Germany

Topic:
7) Mood disorders, 10) Other

Abstract

Objective:
Severe psychiatric disorders are associated with a reduction in life expectancy of up to 20 years. This excess mortality is driven is substantially driven by somatic comorbidity, particularly cardiovascular and metabolic diseases. The present study aims to characterize multimorbidity patterns. Furthermore, we plan to investigate whether associations exist between psychiatric structural brain patterns and psychiatric and somatic disease burden.

Methods:
Data were derived from the German National Cohort (NAKO), a large population-based study. The sample included N=30914 participants (age 48.34 ± 12.29; 13611 females) with structural MRI data. Associations between somatic diseases and psychiatric disorders were analysed using odds ratios. T1-weighted MRI data were preprocessed with CAT12. Voxel-based morphometry and machine learning will integrate multimodal data to identify predictive signatures.

Results / Discussion:
The preliminary analysis focused on depressive disorders, anxiety disorders, metabolic conditions, cardiovascular diseases, and allergic as well as dermatological conditions. Odds ratio analyses demonstrated that comorbid depression and anxiety were associated with a significantly increased prevalence of somatic diseases across all investigated domains. Neuroimaging analyses are not yet available and will be reported at a later stage.

Conclusion:
The findings indicate that psychiatric comorbidity, particularly co-occurring depression and anxiety, is associated with increased somatic disease burden. The NAKO cohort enables validation of neurobiological signatures from clinical samples in a population-based framework and supports the development of transdiagnostic predictive models of mental and physical health across the lifespan.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-024
Title-Potential role of Vitamin D in the manage major Depressive Disorder ..a double blind controlled prospective trial with vitamin d and placebo as augmentation

Presenter: G.Prasad Rao, India

Co-Authors:
Vivekanand Bhatt
Sahil Doshi

Topic:
7) Mood disorders

Abstract

Objective:
Depressive Disorder: a double blind controlled prospective trial with vitamin d and placebo as augmentation

Methods:
In this prospective follow up study MDD patients who did not respond to 8 week trial of escitalopam ,were randomly allotted for augmentation of vit D or placebo and followed up for 12 weeks. Standard follow up ratings of MADRS and HamD ,CGI S CGI I were used.

Results / Discussion:
Results show an interesting correlations with vit D deficiency .the results will be presented.

Conclusion:
Vit D deficiency is known to be associated and possibly has enhanced risk of MDD. Lower vitamin D levels are known to be associated to depressive symptom.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-026
Ambient Temperature and Emotional Dysregulation: A Systematic Review of Neurobiological and Psychiatric Implications

Presenter: Imran Khan, India

Co-Authors:
Rashid Gauri
Karrar Hussain
Parth Singh Meena
Karrar Syed

Topic:
7) Mood disorders

Abstract

Objective:
To systematically synthesize evidence on the relationship between ambient temperature and emotional dysregulation, including anger, irritability, and impulse control, and to examine underlying neurobiological mechanisms and psychiatric implications, with particular relevance to mood disorders and transdiagnostic affective vulnerability.

Methods:
A PRISMA-guided systematic review was conducted using PubMed/MEDLINE, PsycINFO, and Google Scholar from inception through January , 2026. Experimental, observational, epidemiological, and clinical studies examining ambient temperature or thermal stress in relation to emotional regulation, aggression, or impulsivity were included. Findings were synthesized qualitatively.

Results / Discussion:
Fifty-two studies met inclusion criteria. Higher ambient temperatures were consistently associated with increased anger, irritability, impulsivity, aggression, and psychiatric emergency presentations. Reported neurobiological correlates included heightened sympathetic activation, hypothalamic–pituitary–adrenal axis dysregulation, impaired prefrontal inhibitory control, and altered serotonergic and dopaminergic signaling.

Conclusion:
Ambient temperature is a biologically relevant and modifiable environmental factor influencing emotional regulation and psychiatric risk. Evidence supports a bidirectional model in which heat amplifies externalizing dysregulation, while colder temperatures favor internalizing symptoms. Integrating thermal stress into neurobiological and clinical models may improve risk assessment in an era of climate change.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-027
Neuroinflammatory Pathways in Psychiatry and the Potential Role of Low-Dose Naltrexone: A Systematic Review

Presenter: Imran Khan, India

Co-Authors:

Imran Khan

Topic:
7) Mood disorders

Abstract

Objective:
To systematically synthesize mechanistic and translational evidence on the neuroimmune effects of low-dose naltrexone, with particular focus on microglial modulation and inflammatory pathways implicated in psychiatric disorders, especially mood disorders.

Methods:
A PRISMA-guided systematic review was conducted using PubMed/MEDLINE and Google Scholar from database inception through January ,2026. Preclinical, translational, and clinical studies examining low-dose naltrexone, neuroinflammation, microglial activity, and immune–brain signaling relevant to psychiatric symptoms were included and qualitatively synthesized.

Results / Discussion:
Preclinical studies show that low-dose naltrexone modulates microglial activity via toll-like receptor-4 antagonism, reducing pro-inflammatory cytokine release. Translational data suggest downstream effects on glutamatergic and dopaminergic systems. Small clinical studies report improvements in neuropsychiatric symptoms with reductions in inflammatory markers, though psychiatric trials remain limited.

Conclusion:
Evidence indicates that low-dose naltrexone exerts immunomodulatory effects with potential relevance to neuroinflammatory mechanisms implicated in psychiatric disorders. While current clinical evidence is preliminary, these findings support further investigation of LDN as a neuroimmune-targeted adjunct within biologically informed psychiatric frameworks.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-028
Association of MTHFR Gene Polymorphisms (C677T and A1298C) with Major Depressive Disorder: A Case–Control Study

Presenter: Sushmita Bhattacharya, India

Co-Authors:
Nitin Gupta
Gurjit Kaur
Mitesh Kumar

Topic:
7) Mood disorders

Abstract

Objective:
This study aimed to investigate the distribution of MTHFR C677T and A1298C polymorphisms in patients with depression compared to two control groups, one consisting of first-degree relatives and the other of unrelated healthy individuals.

Methods:
A total of 201 participants were recruited, of which 184 samples were viable for C677T analysis and 154 for A1298C analysis. Genotyping was performed using PCR amplification followed by restriction fragment length polymorphism (RFLP) analysis. Socio-demographic, clinical, and psychological parameters were also assessed.

Results / Discussion:
The C677T genotype distribution among depressed patients was CC 64.2%, CT 31.4%, and TT 4.4%. Compared to controls (CT 18.9% and 16.9%), the heterozygous CT genotype was significantly more frequent in patients (p=0.041 vs. relatives; p=0.038 vs. unrelated controls). No significant differences were observed for the A1298C polymorphism.

Conclusion:
Conclusion: Findings suggest that the MTHFR C677T heterozygous genotype may confer increased susceptibility to depression, while A1298C polymorphism does not appear to be associated in this cohort. These results underscore the importance of population-specific genetic studies and support further exploration of gene–environment interactions in depression.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-029
Baseline plasma glutamatergic amino acid ratios and early treatment outcomes in major depressive disorder

Presenter: Yuji Murase, Japan

Co-Authors:
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Topic:
7) Mood disorders

Abstract

Objective:
Glutamatergic mechanisms are increasingly recognized in models of antidepressant action. Amino acids involved in N-methyl-D-aspartate receptor signaling are considered relevant to the neurobiology of major depressive disorder, yet their relationship to early treatment outcomes remains unclear. This study examined whether baseline plasma indices of receptor-related amino acid balance are associated with short-term clinical outcomes.

Methods:
Data from two prospective clinical studies were combined and analyzed (n = 91). Baseline plasma ratios of D-serine/L-serine, L-serine/glycine, and glutamine/glutamate were assessed as indices of receptor-related amino acid balance. Remission at week 6 was defined as a 17-item Hamilton Depression Rating Scale score of 7 or lower. Multivariable analyses adjusting for clinical factors examined associations with remission.

Results / Discussion:
Among patients with major depressive disorder receiving second-generation antidepressant monotherapy, D-serine/L-serine and glutamine/glutamate ratios showed associations with remission status at week 6 after adjustment for clinical factors. These findings appeared generally consistent across the contributing studies.

Conclusion:
The present findings suggest an association between these ratios and early treatment outcomes in major depressive disorder. Additional research in diverse clinical samples may help further characterize these observations.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-030
Diagnostic Utility of Lipopolysaccharide (LPS) in Major Depressive Disorder

Presenter: Vesta Steibliene, Lithuania

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Topic:
7) Mood disorders

Abstract

Objective:
To evaluate the blood plasma concentrations of LPS in patients with MDD in comparison to healthy controls and to assess the diagnostic performance of LPS for MDD.

Methods:
In this study the information of sociodemographic characteristics, smoking habits, body mass index, psychiatric and somatic history and treatment of 97 MDD patients and 52 healthy controls was collected. The severity of depressive symptoms were assessed using the MADRS - SIGMA. Peripheral blood samples were collected and the concentrations of LPS in plasma were quantified using commercial ELISA kits.

Results / Discussion:
There are no significant differences in age, sex and BMI among study groups, but smoking prevalence was significantly higher among individuals with MDD. A median score of depressive symptom severity in MDD group was 30 (IQR: 25–36) and LPS concentrations were significantly higher in individuals with MDD. For MDD the optimal cut-off value for LPS concentration was determined to be >143.58 pg/mL (sensitivity of 51.6%, specificity of 82.7%).

Conclusion:
There are an evidence that dysfunction of the brain-gut-microbiota axis contributes to MDD. Higher LPS concentrations could predict the risk of MDD; and measuring LPS concentrations could help identify a subgroup of MDD patients characterized by increased low-grade systemic inflammation. Thus, LPS could be the missing link between gut dysfunction, inflammation and MDD.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-032
Inter-Brand Price Variation of Z-Drugs Available in India: A Pharmacoeconomic Analysis

Presenter: Omar Afroz, India

Topic:
8) Sleep disorders, 10) Other

Abstract

Objective:
Insomnia is commonly encountered in psychiatric practice and is associated with impaired quality of life, increased healthcare utilization, and poorer outcomes. Z-drugs are frequently prescribed for insomnia owing to their selective action on GABAergic pathways and favourable adverse-effect profile. There is limited data on price variation among Z-drugs in India. This study aimed to evaluate the pharmacoeconomic variability of Z-drugs in India.

Methods:
A cross-sectional pharmacoeconomic analysis was conducted using price data obtained from standard drug compendia and pharmaceutical databases. Available brands of zolpidem, zopiclone, eszopiclone, and zaleplon were identified. For each formulation and strength, the minimum and maximum costs were recorded. Cost ratio and percentage cost variation were calculated using standard pharmacoeconomic formulas.

Results / Discussion:
A total of 107 brands of Z-drugs were identified. Zolpidem accounted for 80 (74.8%) brands, followed by zaleplon (10), eszopiclone (9), and zopiclone (8). Considerable inter-brand price variation was found. The highest percentage cost variation was observed for zolpidem 5 mg (451%), followed by zolpidem 10 mg (363%), zopiclone 7.5 mg (259%), and eszopiclone 2 mg (182%). Lower variation was observed for eszopiclone 1 mg (80%) and zaleplon.

Conclusion:
Substantial inter-brand price variation exists among Z-drugs marketed in India. Given the widespread use of these medications in psychiatric practice, this variation may affect affordability, treatment adherence, and continuity of care. Greater awareness of medication costs among prescribers and consideration of cost-effective alternatives may help promote rational prescribing and improve access to treatment.

P-H
Postersession H: Neuropsychiatry, Neuroscience & Biomarkers

P-H-033
The Familiar Stranger: A Case Of Capgras Syndrome In Psychotic Illness

Presenter: Ibadat Anmol Singh, India

Co-Authors:

Tanu Kundal

Abstract

Objective:
Clinical assessment through detailed history, collateral information, and mental status examination.

Laboratory and neurological evaluation to exclude organic causes.

Diagnosis of Capgras syndrome in the context of psychotic illness based on clinical findings.

Treatment with antipsychotic medication and psychoeducation.

Methods:
Over a six-month follow-up period, the patient demonstrated:Significant reduction in suspiciousness, Marked improvement in Capgras-type misidentification beliefs, improved social and occupational functioning.

Results / Discussion:
Over a six-month follow-up period, the patient demonstrated:Significant reduction in suspiciousness, Marked improvement in Capgras-type misidentification beliefs, improved social and occupational functioning.

Conclusion:
This case highlights a rare presentation of Capgras syndrome involving misidentification of multiple family members in the context of psychotic illness. Early recognition, careful evaluation for contributory organic factors, and timely antipsychotic treatment can result in favorable outcomes. Long-term follow-up remains essential to maintain recovery and prevent relapse.

Index