SP045

Cardiovascular health and cognitive function in autism and intellectual disability

K. Krysta

Department of Rehabilitation Psychiatry, Medical University of Silesia, Katowice, Poland
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Abstract: Cardiovascular health plays a critical and often underappreciated role in shaping cognitive function across the lifespan in individuals with autism spectrum disorder (ASD) and intellectual disability (ID). From early development onward, the heart–brain connection exerts a powerful influence on neurocognitive trajectories. Children born with congenital heart defects are at increased risk of neurodevelopmental delays, including ASD and ID, partly due to impaired cerebral oxygenation during critical periods of brain maturation. These effects may be further amplified by shared genetic pathways that influence both cardiac development and neural connectivity, leading to long-term alterations in cognition, attention, and executive functioning.

As individuals with ASD/ID transition into adulthood, cardiovascular vulnerability often increases rather than diminishes. Adults in this population are more likely to develop cardiovascular diseases at a younger age compared with the general population, driven by a combination of biological susceptibility, chronic low-grade inflammation, metabolic dysregulation, and lifestyle-related risk factors such as physical inactivity, obesity, and medication effects. These cardiovascular conditions can accelerate cognitive decline through mechanisms including chronic cerebral hypoperfusion, endothelial dysfunction, and neuroinflammatory processes, all of which negatively affect brain structure and function.

Importantly, cognitive changes related to cardiovascular health in ASD/ID may remain underrecognized, as they are frequently attributed solely to neurodevelopmental factors rather than to potentially modifiable somatic conditions. This highlights the need for a lifespan-oriented model of care. Early neurodevelopmental surveillance in children with cardiac conditions should be integrated with routine cognitive and behavioral monitoring. In adulthood, proactive cardiovascular screening, risk reduction strategies, and individualized lifestyle interventions should become standard components of care.

Disclosure of Interest: None Declared

SP046

Characterization of primary and secondary cognitive impairment in schizophrenia

A. Vita*, Gabriele Nibbio, Jacopo Lisoni, Giacomo Deste and Stefano Barlati

Department of Clinical and Experimental Sciences, University of Brescia, Brescia, Italy
*Corresponding author.
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Abstract: Cognitive impairment associated with schizophrenia (CIAS) affects over 80% of patients and represents a core feature of the disorder, demonstrating greater predictive value for functional outcomes, more than positive symptoms. Understanding the conceptual framework distinguishing "primary" from "secondary" cognitive impairment is essential for targeted therapeutic interventions.

Primary cognitive impairment reflects the intrinsic neurodevelopmental pathology of schizophrenia, manifesting across multiple domains including processing speed, attention, working memory, verbal learning, executive functions, and social cognition. These deficits typically predate psychosis onset, are observed in unaffected first-degree relatives, and remain relatively stable throughout the illness course regardless of symptom remission, with global cognitive performance averaging one standard deviation below general population.

Secondary cognitive impairment results from modifiable extrinsic factors that further compromise cognitive performance beyond the primary deficit. Key contributors include anticholinergic medication burden, first-generation antipsychotic side effects, benzodiazepine use, metabolic syndrome, sleep disturbances, sedentary lifestyle, substance abuse (particularly cannabis), and social deprivation.

Distinguishing primary from secondary cognitive impairment has critical therapeutic implications. While primary deficits require evidence-based interventions, such as cognitive remediation and optimized pharmacological treatment, secondary impairments demand systematic identification and mitigation of contributing factors including medication rationalization, metabolic management, aerobic exercise programs, substance abuse treatment, and social and work inclusion programs.

This conceptual framework provides clinicians and researchers with a structured approach to assessment and personalized intervention strategies, ultimately improving functional recovery in schizophrenia.

Disclosure of Interest: None Declared

SP047

Cognition and inflammation

G. Sachs[1]* and A. Erfurth[1,2,3]

[1]Medical University of Vienna; [2]1st Department of Psychiatry and Psychotherapeutic Medicine, Klinik Hietzing, Vienna, Austria and [3]University of Muenster, Muenster, Germany
*Corresponding author.
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Abstract: The association between elevated inflammation levels and cognitive alterations in inflammation-associated diseases supports the notion that inflammation plays a role in modulating cognition under physiological circumstances. Dysregulation of the immune system could therefore contribute to the cognitive changes observed in severe psychiatric disorders such as psychotic, unipolar and bipolar affective disorders (1).

A meta-analysis of inflammation and cognition in schizophrenia (2) revealed very modest associations between peripheral inflammatory biomarkers, such as C-reactive protein (CRP), and cognition. Elevated CRP levels were associated with impaired performance in all cognitive domains except verbal fluency. Similarly, a meta-analysis of inflammation and cognition in individuals with affective and psychotic disorders (3) revealed a weak association between global cognitive ability and pro-inflammatory indicators. Peripheral markers such as CRP have been shown to be highly state-dependent in mood and psychotic illnesses, suggesting that they are more relevant as

biomarkers for episodic symptoms. A study of patients presenting with their first episode of schizophrenia found that an increased neutrophil-to-lymphocyte ratio was negatively associated with cognitive function (4).
In our ongoing study (5), we assess cognitive impairments in acute psychiatric inpatients diagnosed with psychotic, bipolar or depressive disorders, using the German version of the SCIP (6). Our sample comprises routinely collected clinical data from adult psychiatric patients living in a specific catchment area who were previously indicated for acute, inevitable admission following an emergency assessment. Data examining the effect of CRP values and the neutrophil-to-lymphocyte ratio on cognitive function in our sample of acute patients is shown.
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SP048

Mood and mitochondria: how the endocannabinoid system fuels bipolar disorder

H. Andreu Gracia* and I. Pacchiarotti

Unidad de Trastornos Depresivos y Bipolares, Hospital Clínic de Barcelona, Barcelona, Spain
*Corresponding author.
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Abstract: Bipolar disorder is increasingly recognised as a disorder involving impaired cellular energy regulation and neurobiological resilience. Mitochondrial dysfunction has been consistently associated with mood instability, cognitive impairment, and illness progression. In parallel, the endocannabinoid system has emerged as a key modulator of emotional regulation, synaptic plasticity, and intracellular energy pathways. This presentation will focus on current evidence linking alterations in endocannabinoid signalling to mitochondrial dysregulation, oxidative stress, and inflammatory mechanisms relevant to bipolar disorder. By integrating preclinical and clinical findings, the talk will highlight how the interaction between the endocannabinoid system and mitochondrial function may contribute to affective symptomatology and treatment response. These insights support a more integrative pathophysiological model and may inform the development of novel biomarkers and therapeutic strategies in bipolar disorder.

Disclosure of Interest: None Declared

SP049

The genetic code of bipolar disorder: new frontiers in pharmacogenetics

S. Papiol on behalf of ConLiGen - The International Consortium on Lithium Genetics and ConLiGen - The International Consortium on Lithium Genetics

Clinical Translation, Max Planck Institute of Psychiatry, Munich, Germany
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Abstract: Lithium remains the most effective mood stabilizer for bipolar disorder (BD), significantly reducing suicide risk and overall mortality. However, treatment response is highly heterogeneous and heritable (SNP-h^2 = 0.22–0.29), with familial clustering observed. The Consortium on Lithium Genetics (ConLiGen) was established to identify genetic determinants of lithium response able to disentangle such heterogeneity in lithium response.
ConLiGen Phase 1 conducted a genome-wide association study (GWAS) of 2,563 BD patients from 22 sites, evaluating lithium response both dichotomously and continuously via the Alda Scale. Four linked polymorphisms on chromosome 21 met genome-wide significance criteria for association with lithium response. The response-associated region contains two genes for long, non-coding RNA. ConLiGen Phase 2 is expanding recruitment to new sites, targeting > 6,000 participants, mainly including European cohorts.
We will present the results of Phase 1 ConLiGen study and also interim results adding new Phase 2 cohorts to the study. Likewise, we will also present ongoing analyses that use genetic information to define clusters of patients mapping to response/non-response phenotype.
ConLiGen Phase 2 will substantially increase statistical power and global representation to unravel the genetic basis of lithium response. This collaborative framework will accelerate discovery for personalized BD treatment and suicide prevention. Data sharing and new partnerships are actively encouraged.

Disclosure of Interest: None Declared

SP050

Lived illness experience in fibromyalgia

C. M. Esposito[1,2]*

[1]Department of Neurosciences and Mental Health, Fondazione IRCCS Ospedale Maggiore Policlinico, Milan and [2]Department of Health Sciences, University of Florence, Florence, Italy
*Corresponding author.
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Background: Fibromyalgia (FM) profoundly affects not only physical functioning but also the lived experience of time, space, the body, selfhood, and relationships. Although many qualitative