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doi: 10.1192/j.eurpsy.2025.171

Abstract: Clinical stageing models are well-established in many areas of medicine, most notably in oncology. These models are used to determine personalised treatment options and indicate prognosis. In psychiatry, staging models have been developed most prominently for psychosis and mood disorders. These models are being used to guide treatment and service provision, e.g. through development of high-risk and early intervention services and stage-appropriate treatment delivery. Clinical staging models for obesity, incorporating medical history, clinical and functional assessments and routine diagnostic investigations also exist and appear to show improved clinical utility in assessing obesity- related risk and prioritising treatment. Recent generic frameworks for staging in mental health have highlighted that there is much individual variability in people's progression through different illness stages, and therefore there is a need to also specify stage modifiers (disease progression factors (e.g. cognitive or neural factors) and extension factors (e.g. physical complications)). Several staging models for eating disorders have been developed. Most of these focus on anorexia nervosa (AN). Criteria for stage differentiation are mostly based on combinations of cognitive, behavioural and physcial features, and illness duration. As yet most of these models are untested. In my talk I will summarise the available evidence on illness stages, stage modifiers and extension factors and their utility in AN and present evidence on what is known about stage-appropriate interventions. I will discuss implications for research and clinical practice.

Disclosure of Interest: None Declared

SP090

ADHD and the Inflammatory Pathway: from infections to inflammations

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doi: 10.1192/j.eurpsy.2025.172

Abstract: Attention-Deficit/Hyperactivity Disorder (ADHD) has been reported as a risk factor for COVID-19 infection and severity of illness. This presentation outlines the research trajectory that began during COVID-19 studies and has since demonstrated that ADHD is not only associated with an increased risk of infection but also with long-term COVID-19 syndrome (LCS). The chain of studies supporting these findings includes the association of ADHD with infections and inflammatory/autoimmune disorders, such as Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency and Familial Mediterranean Fever (FMF). These links, alongside globally reported associations between ADHD and other autoimmune disorders, such as Systemic Lupus Erythematosus (SLE), underscore the potential involvement of inflammatory and immune dysregulation in the pathophysiology of ADHD. The emerging data linking ADHD with inflammatory conditions—at both clinical and genetic levels—represents a significant new direction in ADHD research. Further investigation into these connections may provide deeper insights into the underlying mechanisms of ADHD and contribute to developing novel therapeutic strategies.

Disclosure of Interest: I. Manor Consultant of: Last three years: Teva Israel Ltd., Madison Israel Ltd., Takeda Israel Ltd., Peri Ltd., Mindtension Ltd.,

SP091

Immune mechanisms and medical comorbidity in ADHD: a narrative review

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doi: 10.1192/j.eurpsy.2025.173

Abstract: Introduction: The potential role of inflammatory and immune pathways in ADHD pathophysiology and pathogenesis has received considerable recent attention. Some of these pathways are long-studied in ADHD because of known or suspected mechanisms of high frequency comorbidities, such as obesity, sleep disorders, cardiovascular system disorders/migraine, atopic illnesses, and several autoimmune diseases. Aberrant cellular functioning, aberrant glial functioning, and/or aberrant mitochondrial dysfunction have all been implicated in mechanistic studies. While we separate these mechanisms conceptually, in reality they are intertwined and communicate with each other in a multi-directional complex of mechanisms.
Objectives: To introduce the relationships among inflammation, immune mechanisms and ADHD, and provide a high level overview of current and cutting edge research in this area. We will review data linking ADHD with inflammation and immunologic mechanisms, recent findings on the genetic underpinnings of medical comorbidity in ADHD, and the relationship between maternal inflammation and genetic risk for ADHD in offspring.
Methods: This is a focused, narrative review of recent literature in the field.
Results: Epidemiological, genetic, and biomarker data converge to illuminate the pleiotropic role of immune mechanisms in the pathophysiology of ADHD. Suspected mechanisms include both innate and adaptive pathways. Epigenetic vulnerabilities interact with environmental factors to confer greater risk for health problems among individuals with ADHD. Likewise, many of these same risk factors also carry increased risk for ADHD.
Conclusion: This information exemplifies the close relationship between psychiatric and medical conditions and/or their risk factors. The high heritability of the immune system and associated immunological dysfunctions provides a novel approach to understand the mechanisms through which gene-environment interactions may contribute to the occurrence and clinical presentation of ADHD.

Disclosure of Interest: None Declared