SP0006

Neurobiological markers of early stressful events in psychosis

M. Aas

Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, United Kingdom
doi: 10.1192/j.eurpsy.2024.49

Abstract: New data from the MRC funded project "Integrating psychological models with biological pathways in psychosis" will be presented. The overall objective of this project is to use both environmental and genetic data to understand the biological pathways in patients with schizophrenia and bipolar disorders. Specifically, to find out if polygenic risk and childhood adverse events increase the relative risk of mental illness above that of its individual case-control explained variance, and secondly, the effect of both polygenic risk and childhood adverse events on clinical characteristics and ageing processes. Both data from new unpublished systematic reviews and original data will be presented.

Disclosure of Interest: None Declared

SP0003

The link between early life stress and psycho-cardio-metabolic multi-morbidity: Findings from The EarlyCause Consortium

C. Cecil*

EarlyCause Consortium

Child and Adolescent Psychiatry, Erasmus MC, Rotterdam, Netherlands

*Corresponding author.
doi: 10.1192/j.eurpsy.2024.50

Abstract: In this talk I will present new findings from EarlyCause, a European consortium which aims to better understand the link between early life stress and the development of psycho-cardiometabolic (PCM) comorbidity across the lifespan, leveraging data from large-scale pediatric and adult population studies. I will discuss findings regarding the effect of (prenatal and postnatal) early life stress on PCM health outcomes and their comorbidity, potential moderating and mediating factors, as well as evidence for causality.

Disclosure of Interest: None Declared

SP0004

Childhood trauma in adult depressive and anxiety disorders: immuno-metabolic evidences in the NESDA cohort

Y. Milaneschi

Amsterdam UMC, Amsterdam, Netherlands
doi: 10.1192/j.eurpsy.2024.51

Abstract: Childhood trauma and depression are both associated with increased risk of metabolic disorders, but their joint effects and underlying mechanisms are not well understood. This talk will present recent findings from large-scale epidemiological and biobank studies that explore the metabolic signature of childhood trauma, depression, and their interplay. For example, using longitudinal data from the NESDA cohort, we investigated the association of childhood trauma with metabolic syndrome in ˜3000 adults, including patients with depression and/or anxiety and healthy controls, over 9 years of follow-up. The talk will also describe preliminary results from an individual patient data meta-analysis pooling >160,000 subjects from the Early Cause European Consortium. In this study, we examined the differences in markers of obesity and dyslipidemia across individuals with neither childhood trauma nor depression (controls), those with childhood maltreatment, those with depression, those reporting both of these conditions. The findings described in the talk shed light on the complex interplay between early life stress, mood disorders, and metabolic health.

Disclosure of Interest: None Declared

SP0005

Brain imaging studies in Internet Gaming Disorder (IGD) and problematic social network site use

A. Weinstein

Psychology, Ariel Univesrity, Ariel, Israel
doi: 10.1192/j.eurpsy.2024.52

Abstract: Gaming disorder is characterized by ICD 11 as persistent or recurrent gaming behavior manifested by impaired control over gaming, increasing priority given to gaming over other life interests and daily activities; and continuation or escalation of gaming despite the occurrence of negative consequences. IGD shares to a large extent neurobiological alterations seen in other addictions, such as activation in brain regions associated with reward, reduced activity in impulse control areas and impaired decision-making; and reduced functional connectivity in brain networks that are involved in cognitive control, executive function, motivation and reward. Moreover, there were structural changes, mainly reduction in gray matter volume and white matter density. Comorbidity studies indicate that executive control networks in ADHD may increase the susceptibility to develop IGD. Problematic SNS use has been associated with an increased rate of depression, anxiety, stress, obsessive-compulsive disorder (OCD), attention-deficit/hyperactivity disorder (ADHD), and propensity to excessive alcohol use. It may also lead to vulnerability to aggression, cyberbullying and fear of missing out (FOMO). There is little evidence for cognitive impairments, but there is some preliminary event-related potentials (ERPs) evidence for inefficiency in allocating and monitoring resources and inhibitory control. There is evidence for reduced sleep quality and quantity, longer sleeping latency and more sleep disturbance. Brain imaging studies showed impaired inhibitory-control mechanism, reduced gray matter volumes in the nucleus accumbens, amygdala, and the insula, suggesting rewarding effects of SNS use on the brain.

Disclosure of Interest: None Declared