prevention program focusing not only stress and symptom management, but also social cognitive domains.
Disclosure of interest Consultant to Boehringer Ingelheim lecture fees by Boehringer Ingelheim, Otsuka travel grant by Servier.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.140

S067

Intervention in early psychosis - Current status and future perspectives

S. Galderisi*, D. Pietrafesa, D. Palumbo
University of Campania "Luigi Vanvitelli", department of psychiatry, Naples, Italy
* *Corresponding author.*

Introduction The delay between psychosis onset and initiation of treatment (duration of untreated psychosis, DUP) is associated with a poorer treatment response and overall functional outcome. In Europe several early detection and intervention programs have been developed to reduce the DUP and promote Phase-specific Treatments (PsTs).
Aims To review the evidence of a) the effectiveness of European Early Interventions (EEIs) in reducing DUP; b) an impact of PsTs on clinical and social outcomes; and c) EEIs cost-effectiveness.
Methods A literature search in PubMed, PsychInfo, Cochrane and individual journals through cross-referencing was performed. All European Randomized Controlled Trials (RCTs) designed to reduce DUP and/or to implement PsTs for people with first-episode psychosis were included in the review.
Results Studies examining early detection programs compared with Standard Care (SC) reported discrepant findings as to their impact on the DUP. PsTs generally reduce hospitalizations and improve service engagement when compared with SC; their impact on other clinical variables, e.g. symptomatology and social functioning, is unclear. Studies assessing EEIs cost-effectiveness in comparison with SC consistently report an advantage for EEIs in the long run.
Conclusions EEIs, as compared to SC, show several advantages that seem to result in an overall reduction in the cost of care. Therefore, the development of EEI is recommended.
On the other hand, some inconsistencies in the reported results suggest that EEIs should include psychosocial interventions targeting unmet needs of schizophrenia patients, such as cognitive dysfunction and negative symptoms.
Disclosure of interest SG received honoraria or Advisory board/consulting fees from the following companies: Lundbeck, Janssen Pharmaceuticals, Hoffman-La Roche, Angelini-Acraf, Otsuka, Pierre Fabre and Gedeon-Richter. All other authors have declared.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.141

Symposium: Childhood trauma across psychopathology: mediators and outcome in clinical samples and molecular mechanistic correlates

S068

Childhood trauma in bipolar disorders: Familial and individual mediators for predicting occurrence and outcome

B. Etain
University Paris Diderot, psychiatry and addictology, Paris, France

Childhood trauma is highly prevalent in patients with bipolar disorder (BD) and has been associated to a more severe/complex expression of the disorder. Little is known about the familial and individual factors that can mediate the occurrence of trauma within families but also influence the outcomes of BD. We will present data from two independent samples of patients with BD in order to identify the potential mediators for occurrence and severity/complexity. In a first sample of 371 patients with BD, 256 relatives and 157 healthy controls, we will show that there is a familial resemblance for emotional and physical abuses. Patients' level of physical abuse was associated with their parental levels of physical abuse, but also with their father's history of alcohol misuse ($p < 0.05$). Second, in a sub-sample of 270 normothymic patients, we have performed a path-analysis to demonstrate that emotional and physical abuses interacted with cannabis misuse to increase the frequency of psychotic features and delusional beliefs. Finally, in an independent sample of 485 euthymic patients from the FACE-BD cohort we used path-analytic models to show that emotional abuse increased all the assessed affective/impulsive dimensions ($p < 0.001$). In turn, affect intensity and attitudinal hostility were associated with high risk for suicide attempts ($p < 0.001$), whereas impulsivity was associated with a higher risk for presence of substance misuse ($p < 0.001$). These results illustrate that childhood trauma might derive from parental characteristics (own childhood trauma and psychopathology) and increase the severity/complexity of BD through individual dimensions of psychopathology.
Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.142

S069

Childhood trauma and structural and functional brain mechanisms linked to psychopathology

M. Aas
Oslo university hospital, Norment- KG Jebsen center for psychosis research- division of mental health and addiction, Oslo, Norway

Background Childhood trauma increases the risk of a range of mental disorders including psychosis. Whereas the mechanisms are unclear, previous evidence has implicated atypical processing of emotions among the core cognitive models, in particular suggesting altered attentional allocation towards negative stimuli and an increased negativity bias. Here we tested if childhood trauma was associated with differentiation in brain responses to negative and positive stimuli. We also tested if trauma was associated with emotional ratings of negative and positive faces.
Methods We included 101 patients with a DSM schizophrenia spectrum or bipolar spectrum diagnosis. History of childhood trauma was obtained using the Childhood Trauma Questionnaire (CTQ). Brain activation was measured with functional MRI during presentation of faces with negative or positive emotional expressions. After the scanner session, patients performed emotional ratings of the same faces. Structural MRI was also measured.
Results Higher levels of childhood trauma were associated with stronger differentiation in brain responses to negative compared to positive faces in clusters comprising the right angular gyrus, supramarginal gyrus, middle temporal gyrus, and the lateral occipital cortex (Cohen's d = 0.72-0.77). In patients with schizophrenia, childhood trauma was associated with reporting negative faces as more negative, and positive faces as less positive (Cohen's d > 0.8).
Conclusions Along with the observed negativity bias in the assessment of emotional valence of faces, our data suggest stronger differentiation in brain responses between negative and positive faces in patients with childhood trauma.

https://doi.org/10.1016/j.eurpsy.2017.01.143 Published online by Cambridge University Press

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.143

S070

Identification of a long lasting stress signatures associated with enhanced vulnerability for depression by using 'omics and cross species approaches

A. Cattaneo
King's college London, psychological medicine, London, United Kingdom

Depression results from the interplay of vulnerability genes with environmental factors, a phenomenon named as 'gene-environment (GxE) interaction'. To date, GxE interaction studies have been limited to hypothesis-based candidate genes, since genome-wide (GWAS)-based GxE interaction studies would require enormous datasets with genetics, environmental and clinical variables. We used a novel, cross-species and cross-tissues "*omics*" approaches to identify genes predicting depression in response to stress in GxE interactions. We integrated the transcriptome and miRNome profiles from the hippocampus of adult rats exposed to prenatal stress (PNS) with transcriptome data obtained from blood mRNA of adult humans exposed to early life trauma, using a stringent statistical analyses pathway. Network analysis of the integrated gene lists identified the Forkhead box protein O1 (*FOXO1*), Alpha-2-Macroglobulin (*A2 M*) and Transforming Growth Factor Beta 1 (*TGFB1*) as candidates to be tested for GxE interactions, in two GWAS samples of adults either with a range of childhood traumatic experiences (Grady Study Project, Atlanta, USA) or with childhood emotional abuse only (Helsinki Birth Cohort Study, Finland). Six *FOXO1* SNPs showed significant GxE interactions with emotional abuse in the Grady Study that survived stringent permutation analyses and were all replicated in the Helsinki study. In addition, other SNPs in all the three genes showed significant GxE interactions with emotional, physical and sexual abuse in the Grady Study. We therefore provide a successful 'hypothesis-free' approach for the identification and prioritization of candidate genes for GxE interaction studies that can be investigated in GWAS datasets.

Disclosure of interest The author has not supplied his declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.144

S071

Epigenetic signatures of early life adversities in animal models: A role for psychopathology vulnerability

M.A. Riva
University of Milan, Department of Pharmacological and Biomolecular Sciences, Milan, Italy

Stressful experiences early in life (ELS) represent one of the most relevant factors for the vulnerability to psychopathologies. Epigenetic changes, such as DNA methylation, have emerged as a major mechanism through which ELS can alter adult behaviour leading to persistent changes of gene regulation.

We performed DNA methylation analyses in the hippocampus and prefrontal cortex of adult rats exposed to stress during gestation (PNS), a model that is associated with persistent behavioral alterations relevant for psychiatric disorders.

Using an epigenome-wide analysis, an overlap of 893 differentially methylated genes was observed between hippocampus and prefrontal cortex of adult male and female rats exposed to PNS. The list includes several genes previously associated with schizophrenia and other psychiatric conditions, such as calcium and potassium voltage operated channels as well as GABA and glutamate receptor subunits. By restricting the overlap to genes that were modulated in the same direction, we identified miR-30a as being less methylated in PNS rats. Interestingly one of the targets for this miRNA is the neurotrophin BDNF, whose expression was indeed reduced as a consequence of the prenatal manipulation. Interestingly chronic treatment of PNS rats with the multi-receptor modulator lurasidone during adolescence was able to prevent the changes in miR30a and BDNF expression.

These results highlight the importance for the identification of methylation signatures through which stress exposure early in life could engrave on the outcome of the adult phenotype, and may allow the identification of novel genes and pathways that are affected as a consequence of ELS.

Disclosure of interest M.A.R. has received compensation as speaker/consultant from Lundbeck, Otzuka, Sumitomo Dainippon Pharma and Sunovion. He has received research grants from Lundbeck, Sumitomo Dainippon Pharma and Sunovion.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.145

Symposium: Intergenerational transmission of parenting: Epigenetic, genetic, and psychological mechanisms

S072

Intergenerational transmission of well being–genetic and epigenetic mechanisms

E. Unternaehrer[1,*], K. Greenlaw[2], S. Hari Dass[1], L.M. Chen[1], A.A. Bouvette-Turcot[1], K. Cost[3], K.J. O'Donnell[1], H. Gaudreau[1], L. McEwen[4], J. MacIsaac[4], M.S. Kobor[4], A.S. Fleming[5], L. Atkinson[6], J.E. Lydon[7], M. Steiner[8], A. Ciampi[2], C.M.T. Greenwood[2], M.J. Meaney[1]
[1] *McGill University, Douglas Mental Health University Institute, Montreal, Canada*
[2] *Lady Davis Institute, Centre for Clinical Epidemiology, Montreal, Canada*
[3] *Sick Kids, Department of Psychiatry, Toronto, Canada*
[4] *University of British Columbia, Centre for Molecular Medicine and Therapeutics, Vancouver, Canada*
[5] *University of Toronto Mississauga, Department of Psychology, Toronto, Canada*
[6] *Ryerson University, Department of Psychology, Toronto, Canada*
[7] *McGill University, Department of Psychology, Montreal, Canada*
[8] *McMaster University, Department of Psychiatry & Behavioral Neurosciences, Hamilton, Canada*
[*] *Corresponding author.*

Introduction Maternal mental well being influences offspring development. Research suggests that an interplay between genetic and environmental factors underlies this familial transmission of mental disorders.

Objectives To explore an interaction between genetic and environmental factors to predict trajectories of maternal mental well being, and to examine whether these trajectories are associated with epigenetic modifications in mothers and their offspring.

Method We assessed maternal childhood trauma and rearing experiences, prenatal and postnatal symptoms of depression and stress experience from 6 to 72 months postpartum, and genetic and epigenetic variation in a longitudinal birth-cohort study ($n = 262$) (Maternal adversity, vulnerability and neurodevelopment project). We used latent class modeling to describe trajectories in maternal depressive symptoms, parenting stress, marital stress and general stress, taking polygenetic risk for major depressive disorder (MDD),