S0016

Neurobiological effects of early trauma exposure in people with eating disorders: implications for treatment

G. Cascino

Medicine, Surgery and Dentistry 'Scuola Medica Salernitana', University of Salerno, Baronissi, Italy
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Abstract: People with eating disorders (EDs) exhibit a prevalence of childhood maltreatment higher than general population and, as for other psychiatric conditions, a history of childhood maltreatment in the context of EDs has been found associated with an earlier age at onset, a greater clinical severity, a more frequent comorbidity with other psychiatric conditions and a poorer treatment response . Neuroendocrine modifications as well as a heightened biological and emotional vulnerability to acute social stressor exposure and cortical measures alterations have been reported in people with EDs and history of childhood maltreatment. This evidence suggests the possibility to identify a "maltreated ecophenotype" also in people affected by EDs which recommends grouping individuals affected by the same psychiatric condition into subgroups characterized by different clinical and biological correlates in order to tailor treatements.

Disclosure of Interest: None Declared

S0014

Immunometabolic depression: from conceptualization towards implementation

B. Penninx* and

Immunometabolic depression: from conceptualization towards implementation

Amsterdam UMC, Amsterdam, Netherlands
*Corresponding author.
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Abstract: The burden on society by depression is undisputable, partly due to a chronic course pattern and depression's large heterogeneity that contributes to non-response to standard treatments. Using data from the Netherlands Study of Depression and Anxiety (NESDA, www.nesda.nl), Penninx will illustrate both points. When examining the course of depression, especially when also considering the transitions into other affective disorders over time, chronicity is clearly more the rule than the exception (Verduijn et al. BMC Med 2017). Considering depression's heterogeneity could lead to precision psychiatry approaches that help reduce depression's chronicity. Immuno-metabolic dysregulations seem to vary as a function of depression heterogeneity: dysregulations map more consistently to "atypical" neurovegetative symptoms reflecting altered energy intake/expenditure balance (hyperphagia, weight gain, hypersomnia, fatigue and leaden paralysis). Findings are confirmed when utilizing genome-wide gene expression as well as DNA information (Milaneschi et al. Biol Psychiatry 2020). Preliminary treatment studies suggest that the presence of immuno-metabolic dysregulations in depression moderates antidepressant effects of standard or novel (immunomodulatory) interventions. An immuno-metabolic depression dimension could dissect depression's heterogeneity and potentially match depressed subgroups to treatments with higher likelihood of clinical success.

Disclosure of Interest: None Declared

S0015

On the screening, diagnosis, and treatment of adolescent and adult ADHD patients with comorbid substance use: international guidelines

C. L. Crunelle

Psychiatry, Vrije Universiteit Brussel, Brussels, Belgium
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Abstract: Attention deficit/hyperactivity disorder (ADHD) often co-occurs with substance use disorders (SUD). Together, these disorders are associated with significantly more burden for patients and society, than each alone. Patients with SUD and underlying ADHD have more complex SUD and have more poly-substance use compared with SUD patients without ADHD. A correct identification of ADHD in adult and adolescent individuals with SUD remains important regarding treatment, treatment effectiveness, and treatment retention.

Several screening tools are available and have been validated in individuals with ADHD and comorbid SUD. It is highly recommended that these are used routinely, followed by an ADHD diagnostic process initiated as soon as possible. While several treatment options are accessible, randomized controlled trials show only limited effect sizes of standard pharmacotherapy in adult and adolescent ADHD patients with comorbid SUD. Simultaneous and integrated treatment, with a combination of pharmacotherapy and psychotherapy and for both ADHD and SUD, should preferably be initiated.

We present an overview of the current international guidelines on screening, diagnosis and treatment of ADHD adults and adolescents with comorbid substance use disorders.

Disclosure of Interest: None Declared

S0016

Cerebral networks of apathy and goal-oriented patterns of actimetry in late-life depression

G. Robert[1,2]

[1]Academic old-age psychiatry, Centre Hospitalier Guillaume Régnier and [2]Empenn, Institut de Recherche en Informatique et Systèmes aléatoires (IRISA), Rennes, France
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Abstract: Apathy and goal-oriented behaviors are key dimensions of late-life depression (LLD) and are iteratively associated with cognitive decline in most neuropsychiatric disorders. However, scales and criteria remains insufficient to provide robust individual biomarkers that could foster personalized therapeutic approaches. Therefore, dimensional and digital phenotyping offer new possibilities of stratifying LLD population. This presentation will show our recent results of functional connectivity cerebral networks

associated with multidimensional paper-and-pen measures of apathy, including the default mode and the cingulo-opercular networks. We will also show how dimensional reduction (functional principal component analysis) of 3 days actimetry measures are associated with both FC and inflammatory measures (diffusion and multicompartment indices such as free water and neurite orientation dispersion), providing arguments for different pathophysiological mechanisms underlying goal-oriented behaviors and reinforcing actimetry as a good candidate for individual biomarker of cognitive decline in LLD. Finally, machine learning approaches using combination of different, yet correlated, indices of actimetry will be presented and discussed with their corresponding classifying accuracies (outside of cerebral imaging). Altogether, this presentation aims at bridging the gap between cerebral imaging and digital phenotyping to enhance personalized medicine in the field of old-age psychiatry and cognitive decline prevention.

Disclosure of Interest: None Declared

S0017

The role of dysregulated ghrelin/LEAP-2 balance in eating disorder: a translational study in anorexia nervosa.

C. Tezenas Du Montcel[1,2]

[1]Université Paris Cité UMR-S 1266, Institut de Psychiatrie et Neurosciences de Paris, 75014 Paris and [2]Clinique des Maladies Mentales et de l'Encéphale, GHU Paris Psychiatrie et Neurosciences Hôpital Sainte Anne, F-75014 Paris, France
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Abstract: The ghrelin system is a key regulator of appetite and food intake across species. LEAP-2, a recently discovered ghrelin antagonist, appears to be up-regulated in obesity and opposes to the orexigenic drive of ghrelin. The evolution of LEAP-2 levels could be an interesting insight to reflect the regulation of appetite in eating disorders such as anorexia nervosa (AN). We provide the first study exploring the ghrelin and LEAP-2 regulation in long-term food restriction followed by refeeding in both mice and patients suffering from AN.

Using a translational strategy, we compared the regulation of ghrelin and LEAP-2 concentrations in blood during food restriction and after refeeding in female mice exposed to a 14 days protocol combining quantitative food restriction and running wheel activity followed by progressive refeeding. We compared these results to clinical data fom in an ongoing longitudinal study of patients with AN evaluated before and after refeeding as well as 6 months after hospital discharge.

Long-term food restriction in mice was associated with increased ghrelin and decreased LEAP-2 concentrations compared to *ad libitum* fed controls. Refeeding led to an increase in LEAP-2 concentrations. Patients with AN displayed increased ghrelin levels but also higher LEAP-2 concentrations on admission than after refeeding. LEAP-2 decreased with refeeding. On 17 patients re-evaluated 6 months after discharge, patients with unstable weight gain exhibited a greater decrease of LEAP-2 concentrations during refeeding compared to patient with stable weight gain. Decreasing LEAP-2 concentrations was able to predict a negative outcome (i.e. unstable weight gain) in 80% of the cases.

We provide evidence that the ghrelin/LEAP-2 system is not regulated according to the nutritional status in AN as it is in the case of a physiological adaptation to food restriction. Our clinical data suggest that the evolution of LEAP-2 concentrations during refeeding is opposed to data from preclinical model and could give new insights on the outcome of weight gain in AN.

Disclosure of Interest: None Declared

S0018

Clinical correlates of stress, immune and metabolic markers in major depression

V. Soria[1,2,3]

[1]Department of Psychiatry, Bellvitge University Hospital; [2]Neurosciences-Psychiatry and Mental Health Group, Bellvitge Biomedical Research Institute (IDIBELL) and [3]Department of Clinical Sciences, University of Barcelona, Barcelona, Spain
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Abstract: The hormonal mediators of the stress response, such as glucocorticoids and catecholamines, have both protective and damaging effects on the body. In the short term, they are essential for adaptation, maintenance of homeostasis, and survival; but chronic exposure to stress or abnormalities in the modulation of the stress response can become maladaptive, leading to a broad range of physical and mental problems.

Allostatic load refers to the activation of physiological regulatory systems in response to stress and "the cost" of the effects of these systems on the body. Results from isolated biomarkers and allostatic load measures based on the stress response system (hypothalamic-pituitary-adrenal axis, autonomic nervous system and immuno-metabolic biomarkers) and its relationships with clinical outcomes, such as cognition, in a clinical sample of major depression patients will be presented. The usefulness and relevance in the clinical practice of those biomarkers and the allostatic load concept will be discussed. The integration of several biomarkers translating the biological and psychological impact of stress on depression development and its clinical trajectories could contribute toward understanding how to prevent and improve outcomes in major depression.

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S0019

Contribution of neuroimaging in late-life depression

T. Desmidt[1,2,3]

[1]CHU de Tours; [2]UMR 1253, iBrain, Université de Tours, Inserm and [3]CIC 1415, CHU de Tours, Inserm, Tours, France
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Abstract: Late-life depression (LLD) is currently a hot topic for neuroimaging studies, while an increasing number of imaging modalities are now available for the characterization of brain structure and function. Changes in brain volumes, including