Our model demonstrated good construct validity since all the variables included behaved as expected. That is, they score significantly worse as the patients' severity increases. Besides, it showed good accuracy, specificity, and sensitivity classification properties. Using staging models in daily clinical practice will help clinicians to better.

Disclosure of Interest: None Declared

JS0003

Precision in bipolar disorders

I. Grande

Hospital Clínic, Barcelona, Spain
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Abstract: In many fields of heath, precision medicine has been integrated in clinical practise, for instance in oncology. In the case of psychiatry, and in particular bipolar disorder, we are in a prior step for the moment. Different proposal of stratification will be described during the talk for instance the classification of staging. Moreover some proposal of precision psychiatry in bipolar disorder will be put forward.

Disclosure of Interest: None Declared

JS0004

Ghrelin at the interface of hunger, reward and obesity

S. L. Dickson on behalf of Iris Stoltenborg, Reneé Poelman, Erik Schéle, Karlijn Kooij and Roger AH Adan

Institute of Neuroscience and Physiology, The Sahlgrenska Academy at the University of Gothenburg, Gothenburg, Sweden
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Abstract: Ghrelin, a stomach-derived hunger and appetite signal, drives behaviors that ensure we seek out and consume foods, not only in situations of energy deficit but also when anticipating palatable foods. Key target pathways for ghrelin include the orexigenic agouti-related peptide (AgRP) neurones in the hypothalamic arcuate nucleus (Arc), that appear to confer the unpleasant feelings of hunger. They also include dopamine neurones in the ventral tegmental area, where ghrelin heightens motivation for food rewards. Recently, we have employed a variety of neural circuit mapping techniques in rodents to help clarify the function of populations of ghrelin-responsive targets. We found that 1) chemogenetic activation of ghrelin-responsive cells in the Arc is sufficient to drive a feeding response and to induce food-motivated behaviour and 2) that dopamine neurones in the VTA are activated when mice are exposed to cues that predict a food reward than to its retrieval, as revealed by fiber photometry recordings from these cells. Further studies aim to determine the role of ghrelin-responsive cells in the parabrachial nucleus of the brainstem. Overall, the brain ghrelin signalling system is well positioned to integrate the response to hunger, enhanced by both intrinsic and external cues and in ways that are relevant to curb over-eating in obesity.

Disclosure of Interest: None Declared

JS0005

Microbiome-gut-brain axis in Nutritional Psychiatry

H. Schellekens[1,2]

[1]Department of Anatomy & Neuroscience, University College Cork and [2]APC Microbiome Ireland, Cork, Ireland
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Abstract: The last decade has seen an enormous expansion on our knowledge of the human gut microbiome and its importance for human physiology. The gastrointestinal inhabitants have taken centre stage as regulators of the bidirectional gut to brain communication. The gut microbiota is not only critical for metabolism, glucose homeostasis and body composition, but increasing evidence is demonstrating the significant effects of the gut microbiota on mood and mental wellbeing and its role in the development of affective disorders, such as anxiety and depression, and other neuropsychiatric conditions. Studies in the field of nutritional neuroscience and nutritional psychiatry are now increasingly including the gut microbiota as a key factor mediating the impact of diet on central nervous system function. Accumulating evidence from cell-based in vitro studies, animal models and preclinical intervention studies are linking the gut microbiota to the effects of diet on brain function, but the precise mechanism are still not fully understood and studies have had limited translation to human intervention studies. Overall, the increase in our understanding of interconnectedness of the gastrointestinal microbiota of human health and disease, has led to a strong focus on the development of microbiota-targeted strategies to influence all host physiological responses, including those that can modulate central nervous system function. In this talk, I will provide an overview of the most recent advances in the nutritional psychiatry–microbiome field, highlighting significant opportunities for future research.

Disclosure of Interest: None Declared

JS0006

Striatal ups or downs? Neural correlates of monetary reward anticipation, cue reactivity and their interaction in gambling disorder and alcohol use disorder

A. Goudriaan

Psychiatry, Amsterdam University Medical Centers, Amsterdam, Netherlands
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Abstract: Striatal dysfunction is a key characteristic of addictive disorders, but neuroimaging studies have reported conflicting

findings. An integrative model of addiction points to the presence or absence of addiction-related cues as an explanation for striatal hypo- or hyperactivations, respectively, but has never been directly tested. Here, we developed a novel paradigm to investigate striatal activation during monetary reward anticipation in the presence versus absence of addiction-related pictures using functional MRI. Across two studies, we compared 24 gambling disorder (GD) patients with 22 matched healthy controls and 46 alcohol use disorder (AUD) patients with 30 matched healthy controls. A behavioral interaction was seen where gambling cues made participants respond faster for bigger, but slower for smaller rewards. During monetary reward anticipation, hypoactivation of the reward system was seen in AUD individuals compared to HCs. However, no striatal differences were seen between the participants with GD or AUD and their matched controls. In sum, these findings suggest that striatal dysfunction is a key but heterogeneous mechanism within both AUD and GD and indicate an important but complex role for addiction-related cues in explaining striatal dysfunction in addiction.

Disclosure of Interest: None Declared

JS0007

Neuroimaging studies of addiction: The need to incorporate real life data and profile heterogeneity

G. Sescousse

INSERM, Lyon Neuroscience Research Centre, Vinatier hospital, Lyon, France

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Abstract: Neuroimaging studies of addiction seek to understand the brain mechanisms that predispose to and support the maintenance of addictive behaviors. Traditional studies are *case-control cross-sectional* studies, i.e. they conceptualized individuals suffering from addiction as a homogenous group, and report lab-based experiments conducted at one particular point in time. In this talk, I will argue that a refined understanding of addictive behaviors requires the use of *dimensional longitudinal* studies. Using dimensions will reveal the existence of *heterogenous profiles* within diagnostic groups, and allow researchers to incorporate *individual variability* in their models. In turn, using longitudinal follow-up measures should allow researchers to determine whether brain-related abnormalities are *predictive* of symptoms in *real-life*. I will illustrate these points using a few example studies from the literature.

Disclosure of Interest: None Declared

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