Objectives: The purpose of this study is to evaluate the effectiveness of an add-on postbiotic to Aps on metabolic disturbances and psychopathological variables in patients diagnosed with FEP or schizophrenia spectrum disorder (SSD). , as well as to determine whether the addition of postbiotics can improve biomarkers related to compensatory immunity and the endocannabinoid system.
Methods: A randomized, double-blind, placebo-controlled clinical trial, in which postbiotic or placebo will be administered for 12 weeks as add-on APs. The study comprises two branches: FEP branch, patients recently diagnosed with first psychotic episode; and SSD branch, patients with long-standing psychotic disorder. Five follow-up appointments will be conducted along the 12 weeks to carry on clinical assessments. Patients will be monitoring with a glucose sensor, and blood and microbiota will be analysed.
Results: This is a study protocol that is currently underway. No results are available at this time.
Conclusions: Over the past few decades, it has been abundantly evident how important the human microbiota is to both short-term and long-term human health. In this regard, postbiotics seem to have higher benefitial effects and lower risk than probiotics and they offer a promising approach to improve metabolic disturbances and amelioration of psychopathological symptoms in FEP and SSD patients.

Disclosure of Interest: None Declared

EPV1067

Emotion recognition and self-versus-other referential learning in mood disorders and schizophrenia

C.-D. Chiu

Department of Psychology, Chinese University of Hong Kong, North Territory, Hong Kong
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Introduction: Patients of depression and psychotic disorders are often troubled by unsatisfactory interpersonal relationships. While an inability to maintain a stable sense of self restricts one's understanding another's emotional state, whether disrupted self-versus-other referential processing is a transdiagnostic predictor of increased emotion misreading across diagnostic groups has not been explicated.
Objectives: We tested whether weakened differential learning between self and other may account for impoversihed emotion recognition accross mood and psychotic disorders.
Methods: Inpatients admitted for major depressive disorder (MDD), bipolar disorder (BD), and schizophrenia (SCZ; ns = 59, 32, and 43) and 40 healthy controls were recruited. Aside from ratings of depressive and schizophrenic symptoms by psychiatrists, participants were assessed on self- versus other- referential learning, emotion recognition, emotion sharing.
Results: Regression analysis indicates lower effectiveness of self-other tagging to be a predictor independent from symptom severity for increased emotion misrecognition across MDD, BD and SCZ ($F(8, 160) = 8.52$, $p < 0.001$). Clinical groups showed lower accuracy for other-referential recall and emotion recognition, but comparable emotion sharing and self-prioritization to healthy controls.

Image:

	Emotion recognition		
	β	*t*	*p*
Age	0.114	1.470	0.144
Sex	0.250	3.530	0.001
Years of education	0.086	1.080	0.282
Intellectual quotient	0.231	2.480	0.014
Depression (T2)	0.053	0.690	0.490
Positive symptoms (T2)	-0.025	-0.190	0.847
Negative symptoms (T2)	-0.122	-0.970	0.335
Efficiency of SOT	-0.066	-0.970	0.334
Effectiveness of SOT	0.309	3.290	0.001

Conclusions: Heightened emotion misrecognition in MDD, CD, and SCZ patients can be traced back to the weakened ability in coordinating self- and other-representations according to task-demands. Future examinations on whether interventions on brain regions pertaining to self-versus-other learning might enhance emotion recognition in different patient groups would be clinically relevant.

Disclosure of Interest: None Declared

EPV1068

Molecular Mechanisms of Hypericin and Hyperforin in Modulating Mammalian Neurotransmitter Systems: A Review

D. J. Cox[1]*, R. Maldonado-Puebla[1] and B. Carr[1]

[1]Psychiatry, University of Florida, Gainesville, United States
*Corresponding author.
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Introduction: Hypericin and hyperforin, key secondary metabolites of *Hypericum spp.*, commonly known as St. John's Wort, are known for their ability to modulate neurotransmitter systems in the mammalian brain. These compounds, which evolved as plant defense chemicals, have significant implications for their interaction with mammalian neurobiology, particularly concerning serotonin, dopamine, and norepinephrine pathways.
Objectives: This review aims to elucidate the precise molecular mechanisms by which hypericin and hyperforin influence mammalian brain function. The focus is on understanding how these compounds interact with neurotransmitter transporters and receptors, and how these interactions may lead to both therapeutic and adverse neurobiological outcomes.