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

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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

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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.

Methods: A comprehensive review of neurobiological and pharmacological literature was conducted, focusing on studies that detail the molecular interactions of hypericin and hyperforin with key components of neurotransmitter systems in mammals. in vitro binding assays, in vivo neuropharmacological studies, and molecular dynamics simulations were reviewed to understand these compounds' binding affinities, receptor modulation, and downstream signaling effects.
Results: Hypericin, with its planar polycyclic structure, exhibits a strong affinity for serotonin transporters (SERT), where it acts as a non-competitive inhibitor, leading to increased synaptic levels of serotonin. This mechanism mirrors that of selective serotonin reuptake inhibitors (SSRIs) but also introduces the potential for serotonin syndrome when combined with other serotonergic agents. Additionally, hypericin's ability to generate reactive oxygen species (ROS) under light exposure contributes to neurotoxicity, particularly in regions of the brain exposed to higher oxidative stress.
Hyperforin, characterized by its phloroglucinol core and multiple prenyl groups, exerts its effects primarily through modulation of synaptic vesicle function and ion channel activity. It non-selectively inhibits the reuptake of several neurotransmitters, including serotonin, dopamine, and norepinephrine, through a mechanism involving transient receptor potential (TRP) channels. This broad-spectrum inhibition can lead to significant changes in synaptic plasticity and neurotransmission, impacting mood regulation, anxiety, and cognition.
Conclusions: The interaction of hypericin and hyperforin with mammalian neurotransmitter systems underscores their potential as both therapeutic agents and neurotoxins. The molecular mechanisms by which these compounds modulate neurotransmitter transporters and receptors reveal a delicate balance between beneficial and adverse effects. Understanding these mechanisms is critical for evaluating the safety and efficacy of *Hypericum* extracts in clinical contexts, particularly regarding their impact on brain function and the potential for neurotoxicity.

Disclosure of Interest: None Declared

EPV1069

Mental Chronic Disease: from psychiatry to neurology

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Introduction: Chronic mental illness is a significant risk factor for developing neurocognitive disorders. Advanced molecular imaging techniques, such as amyloid PET and FDG-PET, provide critical insights into the neurobiological mechanisms that link psychiatry and neurology, enhancing our understanding of the continuum between these fields.
Objectives: This study aims to describe the clinical history of chronic mental illness in a sample of patients with diagnosis of dementia, using molecular imaging to investigate the relationship between psychiatric history and neurodegenerative pathology.
Methods: We conducted a retrospective, descriptive analysis of patients who underwent amyloid PET imaging at the Neurology Department of Infanta Leonor University Hospital from January 2019 to October 2024. Inclusion criteria mandated a documented history of chronic mental illness, irrespective of psychiatric hospitalization. Collected data included demographic variables (age, sex), cardiovascular risk factors, psychiatric diagnoses according to DSM-5, years of mental illness, neurological diagnoses, and results from FDG and amyloid PET imaging. Ethical approval was obtained, and statistical analyses were performed using SPSS 22.0.
Results: A total of 25 patients were included. The main characteristics of the sample are shown in Table 1.
Among those with a chronic mental illness history exceeding ten years (N=8), the diagnostic distribution was as follows: 20% Alzheimer's disease, 20% Lewy body dementia, 20% major depressive disorder, 10% post-traumatic stress disorder, and 10% indeterminate. Notably, 75% of Alzheimer's patients and 66.6% of those with Lewy body dementia had a history of major depressive disorder. Patients with frontotemporal dementia often presented with neurocognitive behavioral disorders or obsessive-compulsive disorder. Among four patients with psychiatric hospitalization, only one received a definitive neurological diagnosis (frontotemporal dementia).

Image:

Total of patients	**25 patients**	
Mean of age	**62.6 years**	
Gender	Male	34 %
	Female	64 %
Vascular Risk Factors	High Blood Pressure	52 %
	Diabetes Mellitus	20 %
	Dyslipidemia	64 %
Mean years of mental chronic disease	11.64 years old	
Psychiatric Diagnosis (DSM V)	Major Depressive Disorder	60 %
	Neurocognitive Disorder	8 %
	Adjustment Disorder	16 %
	Anxiety	4 %
	TOC / Agoraphobia	4 %
	Delusional Disorder	4 %
Psychiatric Hospitalization	20 %	
Neurological Diagnosis	Alzheimer's disease	16 %
	Frontotemporal dementia	16 %
	Lewy Body Dementia	24 %
	Non conclusive diagnosis	16 %
FDG- PET Scan Pattern	Alzheimer-like	12 %
	Non-conclusive	24 %
AP Scan	Positive results	16 %

Table 1

Conclusions: These findings highlight the importance of considering chronic mental illness histories in the cognitive assessment of patients. The neurobiological links between depressive disorders and Alzheimer's disease emphasize the need for interdisciplinary approaches in clinical practice. Molecular imaging serves as a