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

pivotal tool in refining diagnostic accuracy, complementing both psychiatric and neurological perspectives, and enabling therapeutic strategies to improve patients' quality of life.

Disclosure of Interest: None Declared

EPV1070

Depression as the main manifestation of central pontine myelinolysis : A case report

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Introduction: Centropontine myelinolysis (CPM) is an acute demyelinating neurological disorder that primarily affects the central bridge and is frequently associated with rapid correction of hyponatremia. Common clinical manifestations of CPM include spastic quadriparesis, dysarthria, pseudobulbar palsy and encephalopathy of varying degrees. In addition, CPM could be accompanied by neuropsychiatric manifestations, such as personality changes, thymic symptoms, acute psychosis, paranoia, hallucinations or catatonia, usually associated with additional brain damage, described as extrapontine myelinolysis (EPM).
Objectives: Study the nature of comorbidity between CPM and mood disorders, particularly depression.
Methods: We present the case of a patient hospitalized in the psychiatry department B of the Hedi Chaker hospital in Sfax in July 2024. He was been admitted at the request of a third party for behavioral disorders such as agitation and refusal of treatment.
Results: This is Mr. S.BH aged 68, with a psychiatric family history of follow-up for unspecified psychiatric disorders in a niece. He has no psychiatric history, but has a somatic history, unmonitored high blood pressure as well as chronic constipation causing hyponatremia, which was quickly corrected 1 month ago. The latter was responsible for CPM objectified on the brain MRI requested by a free practice neurologist who consulted him for agitation. Our patient is married. He has been retired for a few months and previously worked as a farmer for 35 years. According to the family, the history of his illness dates back to March 2024, following professional stressors when he began to present multiple somatic complaints, with anorexia and weight loss as well as a tendency towards isolation. Since June 2024, following the CPM, he believed that the police wanted to harm him. As a result, he became anxious and agitated. At the interview: Slowed down on the psychomotor level, the contact was superficial, the mood was sad, his speech was provoked, poor and conveyed in a low voice verbalizing anhedonia, he presents congestive disorders and he refuses treatment and diet at the beginning. The patient obtained a score of 12 on < < the Geriatric Depression Scale GDS >> and a score of 12 on < <the mini-mental state examination MMSE >>.
Conclusions: This case demonstrates that depression might represent the main manifestation of CPM, especially in the early stages of the disease, which should be taken into consideration when evaluating patients with acute abnormalities of sodium metabolism.

Disclosure of Interest: None Declared

EPV1071

The Mediating Role of Cortical Atrophy on the Relationship between the Resilience Index and Cognitive Function: Findings from the Healthy Brain Initiative

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Introduction: Background: Lifestyle factors are linked to differences in brain aging and risk for Alzheimer's disease, underscored by concepts like 'cognitive reserve' and 'brain maintenance'. The Resilience Index (RI), a composite of 6 factors (cognitive reserve, physical and cognitive activities, social engagement, diet, and mindfulness) provides such a holistic measure.
Objectives: This study aims to examine the association of RI scores with cognitive function and assess the mediating role of cortical atrophy.
Methods: Baseline data from 113 participants (aged 45+, 68% female) from the Healthy Brain Initiative were included. Life course resilience was estimated with the RI, cognitive performance with Cognivue®, and brain health using a machine learning derived Cortical Atrophy Score (CAS). Mediation analysis probed the relationship between RI, cognitive outcomes, and cortical atrophy.
Results: In age and sex adjusted models, the RI was significantly associated with CAS (β= -0.25, p = 0.006) and Cognivue® scores (β= 0.32, p < 0.001). The RI-Cognivue® association was partially mediated by CAS (β= 0.07; 95% CI [0.02, 0.14]).
Conclusions: Findings revealed that the collective effect of early and late-life lifestyle resilience factors on cognition are partially explained by their association with less brain atrophy. These findings underscore the value of comprehensive lifestyle assessments in understanding the risk and progression of cognitive decline and Alzheimer's disease in an aging population.

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EPV1072

Specific learning difficulties and intelligence in children with RASopathies. The Grey Matter project

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