± 5.6; Years of T1DM [N] 13.7±8.3). The patients filled in a set of questionnaires during their regular visit to the outpatient clinic. Three patients from the whole group were on intensive insulin therapy with Multiple Daily Injections (MDI) and Self-Monitoring of Blood Glucose (SMBG), all the rest were on various types of personal insulin pumps (years on insulin pump [N] 9.1±4.5). All the patients were on regular diabetologist care, with regular visits in a Centre for Advanced Technologies in Diabetes (at least every 6 months).

Results: In QIDS-S 26 patients (33.8%) were screened positive for depression, in PHQ 57.7% of the patients (45 patients) had symptoms of depression (age was negatively correlated with PHQ score (r= -0.26; p=0.023)). In CES-D 16 (20%) of the patients assessed their present affect as depressed. Quality of sleep was highly correlated with depressive symptoms CESD (r=0.61, p=0.001), PHQ Score (r=0.62; p=0.001), QISD (r=0.68; p=0.001).

Conclusions: The prevalence of affective disorders and poor sleep quality in the examined T1DM patients was much higher than in the general population. Even if the patients have in general good glycemic control, their mental health condition should not be neglected. Well organized cooperation between patients, diabetologists, psychiatrists and psychotherapists is needed.

Disclosure of Interest: None Declared

EPP0449

Affective temperament polygenic risk scores predict depression: investigating the role of environmental factors

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Introduction: Depressive disorders are known heterogeneous both in their clinical manifestations and etiopathophysiology. Affective temperaments have a strong biological background and heritability, manifest at early age and remain stable throughout the life span, and have a pathoplastic effect in depression. Thus, they have been suggested as intermediate phenotypes for depression.

Objectives: Our aim was to investigate if polygenic risk scores (PRS) calculated for the five affective temperaments predict depression and to examine their interaction effects of early and recent stressors.

Methods: 1820 nonrelated participants from a general population were genotyped and provided data on current depression (Brief Symptom Inventory-BSI), early (Childhood Trauma Questionnaire, CHA) and recent stressors (List of Threatening Life Events, RLE), and affective temperaments (Temperament Evaluation of Memphis, Pisa, Paris and San Diego, TEMPS-A). Our previously performed TEMPS-A GWAS analysis was used as discovery sample and the NewMood database as target sample for analysing the effects of affective temperament PRS on depression. Linear regression models were used to calculate the interaction effect of early and recent stressors.

Results: PRSs derived from anxious, cyclothymic, depressive, and irritable temperaments had a significant effect on current depression, explaining 2.6-7.1% of variance. PRSs calculated from the anxious, depressive and hyperthymic temperaments significantly predicted current depression in interaction with CHA, explaining 10% of variance. In case of interaction models including both early and recent stressors, a significant effect of depressive PRS was found. Detailed results are shown in Table 1.

		anxious	cyclothymic	depressive	hyperthymic	irritable	
on BSI-depression	R^2	**.0033**	**.0071**	**.0032**	.0016	**.0026**
	p-value	**.011**	**.0002**	**.011**	.076	**.022**
in interaction with CHA	R^2	**.1062**	.1037	**.1029**	**.1015**	.1022
	p-value	**.008**	.551	**.027**	**.038**	.531
in interaction with RLE	R^2	.0365	.0402	.0362	.0369	.0368
	p-value	.396	.140	.483	.227	.480
in interaction with CHA and RLE	R^2	.1387	.1384	**.1395**	.1344	.1348
	p-value	.101	.400	**.0009**	.981	.930

Conclusions: Our results confirm the genetic association between affective temperaments and depressive symptoms, which highlight their role as possible clinically relevant intermediate phenotypes for depression.

Disclosure of Interest: None Declared

EPP0450

Psychopathy and Depression: The moderating role of Psychopathic Personality Traits between Emotional Competence and Cognitive Functioning

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Introduction: Psychopathic personality traits (PPT) are known to deteriorate emotional and cognitive functions, however, little is known about their role in depression. Nevertheless, depressive symptoms have also shown to be associated with emotional problems and worse cognitive functions and could thus also interact with PPT.

Objectives: This study aimed to set up an integrative model by examining the correlative relationships and moderating role of PPT in the association between emotional competence and cognitive functioning in individuals with depression.

Methods: Data from 373 individuals diagnosed with depression (158 males, 215 females) were investigated. Subjects filled out questionnaires surveying PPT and emotional competences. Furthermore, a comprehensive neuropsychological test battery investigating the cognitive domains Attention/Psychomotor Speed, Executive Functions and Verbal Learning/Memory was administered.

Results: Correlation analyses revealed a significant positive association between emotional competence and overall cognitive functioning. Further, negative associations between emotional competence and the PPT "Blame Externalisation" and "Careless

Nonplanfulness", as well as positive associations with psychopathic "Social Potency" and "Stress Immunity" were found. Moderation analyses indicated a significant positive influence of psychopathic "Stress Immunity" and "Social Influence" on the relationship between emotional competence and cognitive parameters.
Conclusions: The findings highlight the importance of considering PPT in further research on depression and reflect their impact in therapeutic settings.

Disclosure of Interest: None Declared

Genetics and Molecular Neurobiology

EPP0451

Mycobiota, neuro-cognitif disorders and behavioural impairments: is there a relationship?

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Introduction: The human body carries large and diverse communities of symbiotic microbes that are important for human health and development. While the impact of the bacterial microbiota, which are mostly found in the human gut, on host physiology is relatively well described, much less is known about the interactions between the mycobiota and the host and the resulting effects on human health. At the level of the nervous system, there is increasing evidence implicating the gut microbiota in a variety of neurological disorders. Similar demonstrations of a causal or supportive role of the mycobioma in neurological disorders are still rare, but several studies linking fungal dysbiosis to disease in humans suggest a contribution of symbiotic fungi to neurocognitive and behavioral disorders.
Objectives: We aim through this review to show the role of mycobiota in neurocognitive and behavioral disorders.
Methods: We comprehensively review the scientific literature using Pubmed database and other search platforms such as Google scholar to state the role of mycobiota in neurocognitive and behavioral disorders.
Results: Our bibliographic review revealed that, according to recent studies, Candida species are overrepresented in the stool of individuals with autism spectrum disorders and Rett syndrome compared to healthy controls. Other studies revealed mycobiome signatures specific to cognitive impairment and demonstrated that different diets modulate the mycobiome in association with Alzheimer's disease markers and fungal-bacterial co-regulatory networks in patients with cognitive impairment.
Conclusions: Our understanding of the role of the mycobiota in the biology of neurocognitive disorders-whether causal, consequential, or predisposing-could open up new hypotheses in this area and inspire further research on potential mycobiotic signatures, associated dysbiosis and dysfunction in the neurocognitive developmental-homeostasis spectrum that may contribute to neurocognitive and behavioral developmental disorders and predisposition to cognitive decline, dementia, and progression of neurodegenerative diseases including Parkinson's and Alzheimer's disease in high-risk subjects.

Disclosure of Interest: None Declared

EPP0452

Schizophrenia may be considered as a member of the spectrum of PBAFopathies

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Introduction: Chromatin modifications and epigenetics are important pathogenesis mechanisms leading to various neurologic and psychiatric disorders including epilepsy, drug addictions, depression, autistic spectrum, learning disabilities and schizophrenia. Recently, the disruption of the chromatin remodeling BAF complex has been linked to several neurodevelopmental syndromes, commonly referred to as PBAFopathies.
Objectives: Here, we review the implication of PBAF complex genes in schizophrenia and we outline syndromes caused by mutations in these chromatin-modifying enzymes labelled as PBAFopathies to discuss the functional consequences of reported mutations in the literature.
Methods: We comprehensively review the scientific literature using Pubmed database and other search platforms such as Google scholar to state the role of PBAF complex genes in schizophrenia and to reveal the most frequent genes mutations reported in literature.
Results: Our review revealed that the human analogs of the subfamily of ATP-dependent chromatin remodeling complexes, which are known in eukaryotes as the mammalian SWI/SNF complex (counting a group of proteins that associate and possess a DNA-stimulated ATPase activity that can destabilize histone-DNA interactions in reconstituted nucleosomes providing crucial nucleosome rearrangement and allowing the activation/repression of genes) are crucial for the regulation of genes expression and cells differentiation. They involve two well-known complexes which are SWI/SNF-A (known as BAF complex) and SWI/SNF-B (known as Polybromo-associated BAF or PBAF complex). SWI/SNF is a multisubunit chromatin-remodeling complex that performs fundamental roles in gene regulation, cell lineage specification, and organismal development and mutations that inactivate SWI/SNF subunits are found in nearly 20% of human cancers and in various developmental disorders, forming a continuum or spectrum of diseases. Since the first description of BRG1/BRM mutations in schizophrenia, other mutations of the SWI/SNF subunits have been reported: SMARCA1, SMARCA2, SMARCA4/BRG1, etc. Single nucleotide polymorphisms (SNPs) in these and other genes of PBAF have been also associated with schizophrenia.
Conclusions: This review focuses on the PBAF SWI/SNF subunits to find out if schizophrenia may be considered as a member of the spectrum of PBAFopathies.

Disclosure of Interest: None Declared