CESAR program (Cognitive, Emotional and psycho-Social Avatar Reinforcement program).
Results: The results have been very positive and this is attributed to the transdisciplinary network around each patient, family inclusion and the multi-pronged psychotherapeutic approach based on functional analysis of every patient's situation.
Conclusions: In short, The Sun Project has shown that these approaches and interventions give excellent and rapid outcomes in pre-teens and adolescents suffering from suicide related thoughts and acts.

Disclosure of Interest: None Declared

O0083

The role of single nucleotide polymorphisms within genes for oxytocin and vasopressin receptors in the presentation and severity of autistic traits

K. M. Wilczyński[1,2*], A. Auguściak-Duma[3], A. Stasik[2], L. Cichoń[1,2], A. Sieroń[3] and M. Janas-Kozik[1,2]

[1]Department of Psychiatry and Psychotherapy of Developmental Age, Medical University of Silesia, Katowice; [2]John Paul II Paediatric Center in Sosnowiec, Sosnowiec and [3]Department of Molecular Biology, Medical University of Silesia, Katowice, Poland
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.288

Introduction: Autism spectrum disorder is a heterogeneous group of disorders that affects virtually every population, regardless of their ethnic or socioeconomic origin. The pathogenesis of ASD is probably multifactorial, based on interactions between genetic and environmental factors. Their key elements are disorders in the field of social communication, establishing and maintaining relationships and the so-called stereotypical and repetitive patterns of interests and activities. However, of the above- mentioned symptoms, the most important are communication disorders, which are the basis for many of the functional difficulties observed in these patients.
Objectives: The aim of the presented study was to analyze the clinical picture of social cognition deficits in males with autism spectrum disorders, and to link its elements with the frequency of alleles of selected polymorphisms within the OXTR and AVPR1A genes.
Methods: The study included 132 people, 77.5% of whom were male (n = 100). 113 participants (85.6%) were diagnosed with autism spectrum disorders confirmed by the ADOS-2 test conducted by a certified diagnostician. In this group, men constituted 76.1% of the population (n = 77). The remaining 28 people did not have a diagnosis of autism spectrum disorders, and in the ADOS-2 study they obtained the result below the cut-off level. The mean age in the whole group was 14.4 years (95% CI: 13.92-14.93).
Results: A higher frequency of the rs53576 A allele and the rs10877969 C allele could be observed than expected on the basis of the European / world population. In the case of the rs7294536 and rs2254298 polymorphisms, no differences in the distribution of alleles in relation to the expected values were observed. In the network analysis reference allele (T) of SNPs rs10877969 was linked to the higher outcome of the "social affect" domain of ADOS-2 and through it influenced ADOS-2 outcome. All other SNPs did not significantly affect neither domain of ADOS-2. Reference allele (A) of rs53576 was linked with higher odds ratio of clinical diagnosis of ASD in logistic regression. Similarly the rs10877969 polymorphism within the AVPR1a gene significantly shaped the risk of autism spectrum disorders, while in the combined analysis with rs7294536 within the haplotype, the observed effect was significantly stronger.
Conclusions: The studied polymorphisms may constitute an element of larger haplotypes which, depending on the number of mutated alleles, may determine the severity of autism spectrum traits, from the neurotypical population, through people with a broad autism phenotype, to people diagnosed with ASD. Further research is required on the potential clinical application of genotype analysis of the studied polymorphisms and on the exact mechanism of their impact on the risk of ASD and the development of social cognition disorders.

Disclosure of Interest: None Declared

Neuroimaging and Neurobiology

O0084

Apelin-13 and Asprosin in Adolescents with Anorexia Nervosa and Their Association with Psychometric and Metabolic Variables

K. Jowik[1*], M. Dmitrzak-Węglarz[2], N. Pytlińska[1], A. Jasińska-Mikołajczak[3], A. Słopień[1] and M. Tyszkiewicz-Nwafor[1]

[1]Department of Child and Adolescent Psychiatry; [2]Department of Psychiatric Genetics and [3]Department of Adult Psychiatry, Poznan University of Medical Sciences, Poznań, Poland
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.289

Introduction: Anorexia nervosa (AN) is a widespread, metabo-psychiatric disorder with high relapse rates, comorbidity, and mortality. Many regulatory proteins and neurohormones studied to date play essential roles in the etiopathogenesis of eating disorders and the maintenance of psychopathological symptoms. Nevertheless, the regulatory and pathophysiological mechanisms of AN are still poorly understood.
Objectives: The present study aimed to investigate the plasma levels of asprosin (ASP) and apelin-13 (APE-13) in malnourished (AN1) and partially cured (AN2) adolescent patients with AN. Correlations between protein levels and several dimensions of AN symptomatology, such as eating disorder, depressive, and obsessive compulsive symptoms, were investigated.
Methods: Sixty-four patients aged 11–18 years admitted to the Department of Child and Adolescent Psychiatry in the acute phase of AN participated in the study. Between the 1st and 3rd days of admission, patients with AN (AN1) underwent psychometric evaluation, height and weight assessment, and 15 mL of blood was drawn. The same procedures were repeated at a second time point about 11.2 ± 2.3 weeks later, after partial normalization of body weight on the day of discharge (AN2). The control group (CG) normal-weight girls with no history of psychiatric disorders, recruited from among the students of a local school. The Eating Attitudes Test (EAT-26), Beck Depression Inventory (BDI) Hamilton Depression Scale (HAMD) and Yale–Brown Obsessive Compulsive Scale (CYBOCS), were used to assess eating disorder

symptoms, depression, obsessions and compulsions. Patients were included in a nutritional rehabilitation program. Daily caloric intake was 2000–2500 kcal and was gradually increased to 3500–4000 kcal depending on weight gain (1.0–1.5 kg per week).
Results: APE-13 levels were higher in the AN1 group than in the post-realimentation and the CG group. APE-13 levels were independent of insulin and glucose levels. Plasma ASP levels increased with increasing body weight in patients with AN, correlating with the severity of eating disorder symptoms in emaciation.
Image:

	Group M ± SD			AN1 vs. AN2		AN1 vs. CG		AN2 vs. CG	
	An1 n = 44	An2 n = 44	Control n = 29	MD (95% CI)	p	MD (95% CI)	p	MD (95% CI)	p
Age	15.50		15.00	-	-	MD (95% CI) = 0.50 (-1.00; 1.00); p = 0.995			
Height (m)	1.61		1.65	-	-	MD (95% CI) = -0.04 (-0.06; < 0.01); **p = 0.024**[3]			
Body weight (kg)	37.00	45.00	53.70	-8.10	**< 0.001**[1]	-16.70	**< 0.001**	-8.70	**< 0.001**
BMI	14.13	17.08	18.91	-3.28)	**< 0.001**[1]	-4.78	**< 0.001**	-1.83	**0.006**
ASP [ng/ml]	8.06	10.08	5.61	-2.04	**0.008**[2]	2.45	0.961	4.47	0.075
APE-13 [pg/ml]	113.56	50.93	68.08	30.99	**0.037**[2]	45.48	**0.046**	-17.15	0.479

Image 2:

	Group M			AN1 vs. AN2		AN1 vs. CG		AN2 vs. CG	
	An1 n = 44	An2 n = 44	Control n = 29	MD (95% CI)	p	MD (95% CI)	p	MD (95% CI)	p
BDI	13.00	11.50	5.00	3.00	**0.012**[2]	8.00	**0.003**	6.50	**0.042**
HAMD	12.00	8.00	0.00	4.50	**0.050**[2]	12.00	**< 0.001**	8.00	**< 0.001**
CYBOCS	8.00	2.00	2.00	6.00	**< 0.001**[2]	6.00	**0.006**	0.00	0.079
EAT-26	22.00	7.00	4.50	11.00	**< 0.001**[2]	17.50	**< 0.001**	2.50	0.079

Image 3:

	AN1				AN2				CG			
	ASP		APE-13		ASP		APE-13		ASP		APE-13	
	rho	p	rho	p	rho	p	rho	P	rho	p	rho	p
BDI	0.23	0.298	-0.46	**0.034**	0.06	0.771	0.28	0.181	-0.26	0.201	-0.09	0.736
HAMD	0.10	0.661	-0.42	0.051	0.06	0.751	0.11	0.579	-0.25	0.214	-0.31	0.242
CY-BOCS	0.23	0.291	< 0.01	0.998	0.04	0.832	0.15	0.461	-0.21	0.315	-0.16	0.542
EAT-26	0.51	**0.025**	-0.50	**0.028**	-0.13	0.588	0.53	**0.030**	0.19	0.420	0.34	0.237

Conclusions: The presented data suggest that APE-13 and ASP may be AN's biomarkers-regulation of eating behavior by APE-13 and ASP, the close relationship between them and emotional behavior, and changes in neurohormone levels in patients with eating and affective disorders seem to support these hypotheses. Moreover, their plasma levels seem to be related to the severity of psychopathological symptoms of eating disorders.

Disclosure of Interest: None Declared

O0085

The role of dysregulated ghrelin/LEAP-2 balance in eating disorder: a translational study in anorexia nervosa

C. Tezenas Du Montcel[1,2*], P. Duriez[3,4], J. Cao[3], N. Ramoz[5], O. Viltart[6,7], P. Gorwood[8,9] and V. Tolle[6]

[1]Université Paris Cité UMR-S 1266, Institut de Psychiatrie et Neurosciences de Paris, 75014 Paris; [2]Clinique des Maladies Mentales et de l'Encéphale, GHU Paris Psychiatrie et Neurosciences Hôpital Sainte Anne, F-75014 Paris; [3]Université Paris Cité UMR-S 1266 INSERM, Institut de Psychiatrie et Neurosciences de Paris - Inserm U1266, 75014 Paris; [4]Clinique des Maladies Mentales et de l'Encéphale, GHU Paris Psychiatrie et Neurosciences Hôpital Sainte-Anne, F-75014; [5]Université Paris Cité, UMR-S 1266 INSERM, Institut de Psychiatrie et Neuroscience de Paris, 75014; [6]Université Paris Cité, UMR-S 1266 INSERM, Institut de Psychiatrie et Neuroscience de Paris, 75014 Paris; [7]Faculté de Lille, Faculté des Sciences et Technologies, SN4, F-59650 Villeneuve d'Ascq; [8]1Université Paris Cité, UMR-S 1266 INSERM, Institut de Psychiatrie et Neuroscience de Paris, 75014 Paris and [9]Clinique des Maladies Mentales et de l'Encéphale, GHU Paris Psychiatrie et Neurosciences, Hôpital Sainte-Anne, F-75014 Paris, France
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.290

Introduction: Anorexia nervosa (AN) is a complex psychiatric disorder characterized by a persistant decrease in food intake leading to dramatic weight loss and energy deficit. The ghrelin system is a key regulator of appetite and food intake across species. LEAP-2, a recently discovered ghrelin antagonist, appears to be up-regulated in obesity and opposes to the orexigenic drive of ghrelin. The evolution of LEAP-2 levels could be an interesting insight to reflect the regulation of appetite in eating disorders such as anorexia nervosa (AN).
Objectives: We tested this hypothesis and here provide the first study exploring the ghrelin and LEAP-2 regulation in long-term food restriction followed by refeeding in both mice and patients suffering from AN.
Methods: Using a translational strategy, we compared the regulation of ghrelin and LEAP-2 concentrations in blood during food restriction and after refeeding i/ in female mice exposed to a 14 days protocol combining quantitative food restriction and running wheel activity followed by 10 days of progressive refeeding; ii/ in an ongoing longitudinal study of patients with AN evaluated before and after refeeding (n=30) as well as 6 months after hospital discharge to evaluate if the weight gain was stable (n=7) or unstable (n=10). Plasma concentrations of ghrelin and LEAP-2 were measured with selective immunoassays.
Results: Long-term food restriction in mice was associated with increased ghrelin ($p<0.001$) and decreased LEAP-2 concentrations ($p=0.006$) compared to *ad libitum* fed controls. Refeeding led to a decrease in ghrelin ($p<0.01$) and increase in LEAP-2 concentrations ($p<0.01$). Patients with AN displayed increased ghrelin levels ($p<0.01$) but also higher LEAP-2 concentrations on admission than after refeeding ($p=0.04$). LEAP-2 decreased with refeeding. On 17 patients re-evaluated 6 months after discharge, patients with unstable weight gain exhibited a greater decrease of LEAP-2 concentrations during refeeding compared to patient with stable