Volume xx • Supplement x
JUNE 2025

Acta Neuropsychiatrica

An International Journal of Translational Neuropsychiatry

ABSTRACTS

Educational Symposium in Psychiatry
19 June 2025

CAMBRIDGE
UNIVERSITY PRESS
journals.cambridge.org/neu

OFFICIAL PUBLICATION OF THE
SCANDINAVIAN COLLEGE OF
NEUROPSYCHOPHARMACOLOGY (SCNP)

Poster 1

Dendritic spines and their role in the pathogenesis of neurodevelopmental and neurological disorders

Aisan Akhgari[1], Tanja Maria Michel[2], Manouchehr Seyedi Vafaee[2]

[1]Student Research Committee, Tabriz University of Medical Sciences, Iran

[2]Research Unit for Psychiatry, Odense University Hospital, Denmark

aisan@clin.au.dk

Abstract: Since Cajal introduced dendritic spines in the 19th century, they have attained considerable attention, especially in neuropsychiatric and neurologic disorders. Multiple roles of dendritic spine malfunction and pathology in the progression of various diseases have been reported. Thus, it is inevitable to consider these structures as new therapeutic targets for treating neuropsychiatric and neurologic disorders such as autism spectrum disorders, schizophrenia, dementia, Down syndrome, etc. Therefore, we attempted to prepare a narrative review of the literature regarding the role of dendritic spines in the pathogenesis of aforementioned diseases and to shed new light on their pathophysiology.

Poster 2

The effects of cannabidiol on neurotrophic tyrosine kinase receptor 2 localization and trafficking

Eva Sofie Bovbjerg[1], Magnus Kjærgaard[1,2], Caroline Biojone[3], Sâmia Joca[2,3]

[1]Department of Molecular Biology and Genetics, Aarhus University,

[2]Department of Biomedicine, Aarhus University,

[3]Translational Neuropsychiatry Unit, Aarhus University Hospital

au681984@uni.au.dk

Abstract: This abstract has been omitted due to confidentiality of unpublished data. For further information, please contact the authors.

Poster 3

Targeting the resolution of inflammation to improve the treatment of depression

Chaves, Y.[1]; Andersen, E.[1]; Cecchi, C. [1]; Müller, H. K. [1]; Joca, S.2; Wegener, G.[1]

[1]Translational Neuropsychiatry Unit, Department of Clinical Medicine, Aarhus University, Aarhus, Denmark

[2]Department of Biomedicine, Aarhus University, Aarhus, Denmark

yach@clin.au.dk

Abstract: Depression is a complex and heterogeneous disorder affecting millions worldwide, with current antidepressants showing limited efficacy and delayed onset in many patients [1,2]. Among several mechanisms, low-grade inflammation has been consistently associated with depressive symptoms and poor treatment response. Both clinical and preclinical studies demonstrate elevated levels of pro-inflammatory mediators and neuroinflammation in depression models [3]. Recent findings suggest that impaired resolution of inflammation—due to dysfunction in specialized pro-resolving mediators (SPMs) such as annexins, resolvins, and lipoxins—may contribute to this imbalance [4,5]. However, the specific role of SPMs in mood regulation and antidepressant response remains unclear.

This study tested the hypothesis that dysregulation of pro-resolving pathways involving alpha-melanocyte-stimulating hormone (α-MSH), Annexin A1 (AnxA1), and glucocorticoid-induced leucine zipper (GILZ), which are functionally interconnected, contributes to depressive-like behavior and reduced treatment responsiveness. Male Flinders Sensitive Line (FSL) rats, a validated model of gene × environment-driven low-grade inflammation and depression, were treated with vehicle, ketamine (15 mg/kg, i.p.), or imipramine (15 or 30 mg/kg, i.p.), either acutely or for seven consecutive days. Behavioral outcomes were assessed using the Open Field Test (OFT) and Forced Swim Test (FST), followed by hippocampal protein analysis (Western blot and ELISA). Targets included AnxA1 (intact, cleaved, total), FPR2 (AnxA1 receptor), GILZ, ChemR23 (Resolvin E1 receptor), and MC3R (α-MSH receptor). Molecular ratios (e.g., AnxA1/FPR2) were calculated to explore functional balance within resolution signaling pathways. Data were analyzed using GraphPad Prism 10 and R software (one-way ANOVA with Šidák's post-hoc tests and Pearson correlations).

Treatment with ketamine and repeated imipramine (7 days) significantly reduced immobility time in the FST, reflecting antidepressant-like effects. One-way ANOVA confirmed a significant treatment effect ($F(6, 59) = 7.339$, $p < 0.0001$, $R^2 = 0.4274$). Post-hoc Šidák's test showed that immobility was significantly lower in the FSL/Ket ($p = 0.0302$), FSL/IMI 30 ($p = 0.0003$), and FSL/IMI 7 days ($p < 0.0001$) groups compared to FSL/Vehicle. Acute and low-dose imipramine did not significantly affect immobility. FSL/Vehicle rats also displayed higher immobility than non-depressed FRL controls ($p < 0.0001$), confirming the depressive-like phenotype.

At the molecular level, untreated FSL rats showed reduced hippocampal ChemR23 expression, which was restored by both treatments. ANOVA revealed treatment-induced changes in AnxA1 isoform ratios, with ketamine selectively increasing the AnxA1/FPR2 ratio. Pearson correlations showed that in FSL/Vehicle animals, AnxA1 and FPR2 levels were positively correlated with immobility ($r > 0.75$; $p < 0.01$). In the FSL/Ket group, strong molecular correlations among FPR2, GILZ, and AnxA1/FPR2 persisted, but only

GILZ remained correlated with immobility. The FSL/IMI 7 days group showed robust intra-pathway correlations but remained distinct from FRL animals.

These findings support that dysregulation of resolution-related signaling—particularly involving AnxA1, FPR2, and GILZ—contributes to depressive-like behavior and influences treatment response. These pathways may represent promising targets for improving antidepressant efficacy.

References

1. Chisholm D, et al. (2016). Scaling-up treatment of depression and anxiety: a global return on investment analysis. Lancet Psychiatry.

2. American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders (5th ed.).

3. Zanoveli JM, et al. (2016). The hypothalamic-ituitary-adrenal axis and neuroinflammation in stress and depression. Current Pharmaceutical Design.

4.Giacobbe J, et al. (2020). Pro-resolving mediators in neuroinflammation and depression. Frontiers in Psychiatry.

5. Ishikawa M, et al. (2017). Omega-3 fatty acid-derived mediators protect against neuroinflammation and depression. Journal of Clinical Investigation.

Poster 4

Primary healthcare use of adolescents with health anxiety: a 3-year follow-up in the Copenhagen Child Cohort 2000

Charlotte Steen Duholm[1,2]; Davíð R. M. A Højgaard[1]; Anders Helles Carlsen[1]; Per Hove Thomsen[1,2]; Martin Køster Rimvall[3,4]; Charlotte Ulrikka Rask[1,2]

[1] Department of Child and Adolescent Psychiatry, Aarhus University Hospital Psychiatry, Denmark

[2]Department of Clinical Medicine, Aarhus University, Denmark

[3]Child and Adolescent Mental Health Center, Mental Health Services, Capital Region of Denmark, Denmark

[4]Department of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark

chaduh@rm.dk

Abstract: Background: Health anxiety (HA) is characterized by excessive worry that bodily sensations may be indicative of serious illness. HA has been identified as a significant driver of healthcare use, placing a substantial burden on healthcare services. This study aims to explore associations between HA at age 16/17 and contacts to the primary sector over a 3-year follow-up period.

Methods: Data from the 16/17-year follow-up (N=2521) from the general population-based Copenhagen Child Cohort 2000 is used. Self-report questionnaires assessing HA and register data on contacts to general practice (GP), specialist doctors, psychologists, physiotherapists, and chiropractors as well as tests performed at the GP will be retrieved. Based on the 90th percentile of the HA score, the sample will be divided into a low and high HA group. First, the difference in number of contacts for each type of contact to the GP (i.e., face-to-face, telephone and email) for both daytime and out-of-hours will be explored. For specialist doctors, psychologists, physiotherapists, and chiropractors the difference in number of first consultations will be examined. Subsequently, differences in number of laboratory and psychometric tests performed at the GP will be investigated. Yearly rates of contacts or tests for each outcome category for the two groups will be calculated together with an incidence rate ratio.

Results and perspectives: A deeper understanding of specific contact patterns with the primary healthcare sector can provide a more nuanced insight into healthcare-seeking behaviors associated with HA in young people. Recognizing potentially maladaptive patterns may inform targeted preventive strategies and help reduce the risk of progression to more severe forms of HA.

Poster 5

Exploring microRNAs and inflammatory cytokines in plasma from female adolescents with major depressive disorder

Kristine Johannessen[1], Michaela Krivošová[2], Dana Dvorská[2], Braný Dušan[2], Betina Elfving[1]

[1]Translational Neuropsychiatry Unit, Department of Clinical Medicine, Aarhus University, Denmark

[2]Biomedical Centre Martin, Jessenius Faculty of Medicine, Comenius University in Martin, Slovakia

kristineejoh@gmail.com

Abstract: Major depressive disorder (MDD) in adolescents is a prevalent psychiatric condition worldwide with severe consequences. Diagnosing adolescent MDD is challenging due to its heterogeneity, highlighting the need for reliable biomarkers to aid in diagnosis or monitoring of a treatment response. Circulating microRNAs (miRNAs) have emerged as promising biomarkers for various diseases. Moreover, growing evidence shows that miRNAs regulate many processes involved in the pathogenesis of MDD, suggesting their suitability as biomarkers of MDD. Numerous hypotheses have been proposed to explain the pathophysiology of MDD, including a bidirectional relationship between MDD and inflammatory processes. Still, more research regarding the inflammatory role in adolescent MDD is needed.

This study applied the NanoString nCounter technology to identify dysregulated miRNAs in plasma from female adolescents with MDD before and after antidepressant treatment(n=27), compared to healthy controls (n=8). A multiplexed immunoassay was also performed to assess

the plasma levels of 27 inflammatory cytokines from female adolescents with MDD before and after antidepressant treatment (n=32) compared to healthy controls (n=14).

Several dysregulated miRNAs were identified in female adolescents with MDD, suggesting their potential as diagnostic biomarkers. Others were regulated with treatment, indicating their potential as biomarkers of an antidepressant response. Some of the dysregulated miRNAs were found to be involved in regulating inflammatory responses. Additionally, the levels of PDGF-BB and IL-7 were increased in female adolescents with MDD compared to healthy controls, while IL-9 and MIP-1β levels decreased with antidepressant treatment.

In conclusion, the findings of this study confirm the potential of specific miRNAs as

biomarkers in female adolescents with MDD and indicate a possible interaction between specific miRNAs and inflammation.

Poster 6

Primary Care Perspectives on Tic Disorders in Children and Adolescents: A Qualitative Study of Clinical Experiences, Challenges, and Reflections on Digital Support

Maria Louise Jöhnk[1,2], Claus Sixtus Jensen[2,3,4], Stina Lou[2,5], Anne Flyvholm Jørgensen[6], Julia Osp Nielsen[6], Filippa Ilakkija Sebastian[6], David Mataix-Cols[7], Per Hove Thomsen[1,2], Judith Becker Nissen[1,2]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital (AUH), Denmark.

[2]Department of Clinical Medicine, Aarhus University (AU), Denmark

[3]Department of Paediatrics and Adolescent Medicine, Unit for Research and Development in Nursing for Children and Young People, (AUH), Denmark

[4]Research Centre for Emergency Medicine, (AUH) and (AU), Denmark

[5]DEFACTUM–Public Health Research, Central Denmark Region, Aarhus, Denmark.

[6]Department of Public Health, Aarhus University

[7]Centre for Psychiatry Research, Department of Clinical Neuroscience, Karolinska Institutet and Stockholm Health Care Services, Region Stockholm, Stockholm, Sweden.

maria.joehnk@viborg.rm.dk

Abstract: This abstract has been omitted due to confidentiality of unpublished data. For further information, please contact the authors.

Poster 7

Profiling the Dynamic of Binge Eating Disorder: A longitudinal study examining the influence of emotion regulation, executive function, and eating pattern on BED and outcome (PRODY-BED)

Marie Lyngdrup Kjeldbjerg[1,2], Loa Clausen[1,2], Gry Kjærsdam Telléus[3,4]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital, Denmark.

[2]Department of Clinical Medicine, Faculty of Health, Aarhus University, Denmark.

[3]Unit for Psychiatric Research, Aalborg University Hospital, Denmark.

[4]Department of Communication and Psychology, Aalborg University, Denmark.

makjpd@rm.dk

Abstract: Background: Binge eating disorder (BED) is an eating disorder marked by recurrent binge eating episodes but without regular compensatory behaviors (e.g. fasting). BED is known to lead to feelings of shame and distress but is also associated with serious mental and physical conditions such as depression, anxiety, diabetes and severe overweight among others. BED is a relatively new eating disorder category in the diagnostic systems DSM-5 and ICD-11. Therefore, research on perpetuating factors and trajectories in remission is scarce. An etiological developmental model posits that problems with emotion regulation, reward responsivity including attention bias towards food and problems with executive functions in general puts an individual at risk for loss of control eating – a precursor to binge eating.

Objectives: The aim of PRODY-BED is to test the developmental model of BED by exploring whether adults in treatment of BED can be divided into subgroups according to eating pattern, attention bias towards food and dysfunctioning executive functions and lastly emotion regulation deficiencies. Furthermore, whether potential subgroups differ in their remission patterns and outcome of group-based psychotherapy.

Methods: The design of PRODY-BED is a longitudinal survey design where participants from three treatment facilities are invited to complete a survey at start of treatment, 8-12 weeks into treatment, at end of treatment and at 6- and 12-month follow-up. All patients aged 18+years accepted for treatment at one of the sites are invited to participate. Inclusion has started and will continue through 2025. Data will be analyzed quantitatively.

Perspectives: More knowledge on defining psychological and behavioral perpetuating factors and their influence on treatment effect can help direct more effective and specialized treatment for BED.

Poster 8

Cytokines in the pathophysiology and treatment of epilepsy and epilepsy syndromes

Yevgeniya Lekomtseva[1], Gregers Wegener[1], Sâmia Joca[2], Mykola Posokhov[3], Tatayna Gorbatch[4], Liudmyla Bilyk[5]

[1]Translational Neuropsychiatry Unit, Aarhus University, Aarhus, Denmark

[2]Department of Biomedicine, Aarhus University, Aarhus, Denmark
[3]Department of Functional Neurosurgery and Paroxysmal States, State Institute of Neurology, Psychiatry and Narcology of the Academy of National Medical Sciences of Ukraine named P. Voloshin, Kharkiv, Ukraine
[4]Department of Biochemistry, Kharkiv National Medical University, Kharkiv, Ukraine
[5]Medical Center Rishon, Kharkiv, Ukraine
yevgeniyalekomtseva@gmail.com

Abstract: Background: There is considerable clinical and experimental evidence that seizures are associated with elevated levels of proinflammatory cytokines, particularly interleukin-1β (IL-1β), IL-6, and tumor necrosis factor-α (TNF-α), which underline the impact of neuroinflammation on brain hyperexcitability and epileptogenesis (B. Semple et al., 2017, 2023; F. Dede et al., 2019; R. Qi et al., 2022).

Methods: A literature review was performed to evaluate whether peripheral levels of pro-inflammatory markers, including IL-1β, IL-6, and TNF-α are significantly changing in epilepsy and epilepsy syndromes. The pathogenesis of epilepsy syndromes due to infections and the role of experimental models in studying mechanisms of epileptogenesis induced by infections were also reviewed.

Results: Chronic microglia-mediated neuro-inflammation involves multiple pathways covering different interactions of proinflammatory cytokines, which act as critical regulators of inflammatory processes, activation of innate immunity receptors, and the complement system. Activated signaling pathways include MAPK-NFκB, JAK-STAT, and PI3-AKT pathways. Processes underlying seizure precipitation include a dynamic network of pathogenic changes overshooting brain homeostasis, where chronic neuroinflammation contributes to neuronal damage, its functional deficits, and neuroplasticity. There are a certain number of well-established studies confirming that multifunctional cytokines could directly induce neuronal injury through various mechanisms: excitotoxicity, oxidative stress, secretion of matrix metalloproteinases, apoptosis. Available evidence is consistent with suggesting that cytokine levels did not change after partial seizures, but IL-2 and IL-6 increased in cases of generalized tonic-clonic seizures. The brainstem levels of IL-1β and TNF-α after audiogenic seizures were significantly lower than in the background. Previous experimental and clinical studies reported that IL-6, a pro-convulsive and neurotoxic cytokine, and IL-10, an anti-inflammatory and neuroprotective cytokine, are crucial inflammatory mediators in the brain. A sluggish anti-inflammatory system also contributes to neuroinflammation.

Conclusion: Neuroinflammation and proinflammatory cytokines contribute to epileptogenesis, where anti-inflammatory therapies might be a promising strategy for preventing epileptogenesis and its related neurobehavioral comorbidities. Future studies are needed to clarify the role of proinflammatory mediators in the pathophysiology of chronic neuroinflammation in epilepsy.

Poster 9

Associations Between Functional Somatic Symptoms and Problematic Eating Behaviour

Paula Magdalene Maier Madsen[1,2], Lina Münker[1,2], Else Marie Olsen[3], Nadia Micali[4,5,6], Charlotte Ulrikka Rask[1,2]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital, Denmark
[2]Department of Clinical Medicine, Aarhus University Hospital, Denmark
[3]Center for Clinical Research and Prevention, Frederiksberg Hospital, the Capital Region, Denmark.
4Center for Eating and feeding Disorders research, Mental Health Services of the Capital Region of Denmark, Psychiatric Centre Ballerup, Denmark
[5]Institute of Biological Psychiatry, Psykiatrisk Center, Denmark
[6]Great Ormond Street Institute of Child Health, University College London, London, UK
paumad@rm.dk

Abstract: Functional somatic symptoms (FSS), encompassing physical symptoms without well-defined medical causes, are common in childhood and adolescence. A specific subtype, functional gastrointestinal symptoms (FGS), has been linked to problematic eating behaviors in adults, but little is known about these connections in younger populations. This study explores the association between FSS and problematic eating behaviors, specifically restrictive and emotional eating, in preadolescents. The role of body dissatisfaction and emotional problems in these associations was also investigated.

Using data from the Copenhagen Child Cohort, we analyzed self-reported measures of FSS, eating behaviors, body dissatisfaction, emotional problems, puberty stage, and BMI, supplemented by sociodemographic information from national registers. Results revealed significant associations between FSS and both restrictive and emotional eating, with similar patterns observed for FGS. These associations persisted after accounting for body dissatisfaction and emotional problems.

The findings suggest that FSS may be a risk factor for problematic eating behaviors in preadolescents, highlighting the need for early identification and intervention in this group. Future research should examine the long-term development and underlying mechanisms of these associations to support prevention efforts and mitigate risks of severe psychopathology.

Poster 10

Bodily Distress, Psychotic Experiences, and Low-Grade Inflammation in Adolescents at Familial High Risk of Schizophrenia or Bipolar Disorder: The Danish High Risk and Resilience Study – VIA

Sanciya Mano Perfalk[1], Charlotte Ulrikka Rask[2], Aja Greve[3], Ole Köhler-Forsberg[4]

[1,2]Department of Child and Adolescent Psychiatry, Aarhus University Hospital Psychiatry, Denmark

[3]Department of Clinical Medicine, Aarhus University, Denmark

[4]Department of Psychoses, Aarhus University Hospital Psychiatry, Denmark

sanman@rm.dk

Abstract: Background: Children of parents with schizophrenia (SZ) or bipolar disorder (BD) have a tenfold increased risk of developing the same disorder, as well as other psychiatric conditions (e.g., anxiety, behavioral disorders, ADHD). Familial high-risk (FHR) studies offer crucial insights into developmental trajectories and opportunities for early intervention. The Danish High Risk and Resilience Study – VIA follows 522 children born to parents with SZ, BD, or neither (controls), assessed at ages 7 (VIA7), 11 (VIA11), and 15 (VIA15), with VIA19 ongoing since 2024.

Objective: Using blood samples from VIA11 and VIA15 and questionnaire data from VIA15, this study aims to:

1. Determine the prevalence and symptom profile of bodily distress at age 15.

2. Investigate the association between bodily distress and psychotic experiences.

3. Examine whether low-grade inflammation is associated with bodily distress and psychotic experiences.

Methods: Participants were identified via national registries and recruited at age 7: 202 with a parent diagnosed with SZ, 120 with a parent diagnosed with BD, and 200 controls. Groups were matched by postal code, age, and sex. Assessments were conducted by clinicians blinded to group status. The present analysis includes data from VIA11 and VIA15, focusing on self-reported bodily distress and psychotic experiences at age 15 and inflammatory biomarkers measured at ages 11 and 15.

Expected Outcomes: We hypothesize that:

• FHR adolescents will report more bodily distress symptoms than controls.

• Bodily distress will be positively associated with psychotic experiences.

• Elevated inflammatory markers will correlate with greater bodily distress and psychotic experiences in FHR adolescents.

Conclusion: Findings may enhance our understanding of the biological and psychological processes underlying emerging psychopathology in high-risk youth and support earlier detection and targeted preventive strategies.

Poster 11

Disassembling Brain Plasticity Brakes: Targeting the TrkB-NOS1 Interaction

Anne Munk Svendsen[1], Nicole R. da Silva[1,2], Samia Joca[1,2], Gregers Wegener[1], Eero Castren[3], Caroline Biojone[1,2]

[1]Translational Neuropsychiatry Unit

[2]Department of Biomedicine, Aarhus University

[3]Neuroscience Center, University of Helsinki

202004000@post.au.dk

Abstract: Background: Major Depressive Disorder (MDD) remains a leading cause of disability, with current treatments often insufficient. The BDNF/TrkB signaling pathway is crucial for antidepressant (AD)-induced critical-period plasticity, but global TrkB activation poses risks of adverse effects. Thus, identifying more targeted, druggable mechanisms is clinically relevant. Nitration, a post-translational modification driven by neuronal nitric oxide (NO), acts as a molecular brake on TrkB. Since NO sustains negative emotional responses in the fear conditioning (FC) model and its inhibition mimics AD effects in preclinical models, we aim to explore nitrated TrkB (nitroTrkB) as a potential therapeutic target.

Hypothesis: We hypothesize that: 1- TrkB nitration occurs specifically when TrkB forms a complex with nitric oxide synthase 1 (NOS1) and its adaptor protein CAPON; 2- This complex is enriched in lipid rafts (LRs), specialized neuronal membrane microdomains; 3- TrkB's localization within this complex and in LRs influences negative emotional responses and stress coping.

Methods: Using the contextual FC model, we assessed whether the behavioral effects of the NOS1 inhibitor n-propyl-L-arginine (NPA, p.o.) in male C57Bl6J mice (WT and BDNF+/−) depend on brain TrkB signaling. Data mining and RaptorX-based in silico modeling identified CAPON as a potential adaptor linking TrkB and NOS1. We validated this by examining TrkB nitration and its interactions with CAPON and NOS1 via co-immunoprecipitation and ELISA in LR and non-LR fractions from naïve mouse brains, isolated using detergent-resistant membrane fractionation. We further isolated these fractions from the prefrontal cortex, hippocampus, and amygdala of mice subjected to FC, aiming at linking behavioral outcomes to TrkB nitration and protein complex formation.

Preliminary Results: NPA induced an antidepressant-like effect in WT mice in the FC model, which was abolished in BDNF+/− mice. We validated a novel protocol for investigating protein-protein interactions in

LRs and showed, for the first time, that TrkB interacts with CAPON and NOS1: the interactions were significantly enriched in LRs compared to non-raft domains, along with a trend toward increased TrkB nitration in LRs. LR isolation was consistent in the hippocampus, though more challenging in the amygdala and prefrontal cortex.

Perspectives: Future work includes comparing limbic regions between FC and control mice to identify region-specific changes in protein interactions and TrkB nitration, and further validating raft purity using additional markers. Importantly, we plan to assess TrkB nitration in brain samples from depressed patients versus healthy controls to better understand the biological relevance of our findings.

Poster 12

Functional somatic symptoms in preadolescence as risk factor of psychiatric disorders during early adulthood - a prospective cohort study

Lina Münker[1], Charlotte Steen Duholm[1,2], Lisbeth Frostholm[2,3], Oleguer Plana-Ripoll[4,5], Anders Helles Carlsen[1], Pia Jeppesen[6,7,8], Judith Rosmalen[9], Martin Køster Rimvall[6,7], Charlotte Rask[1,2]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital, Psychiatry, Denmark

[2]Department of Clinical Medicine, Aarhus University, Denmark

[3]Department of Functional Disorders and Psychosomatics, Aarhus University Hospital, Denmark

[4]Department of Clinical Epidemiology, Aarhus University and Aarhus University Hospital

[5]National Centre for Register-based Research, Aarhus University

[6]Department of Child and Adolescent Psychiatry, Copenhagen University Hospital - Psychiatry Region Zealand, Roskilde, Denmark

[7]Child and Adolescent Mental Health Centre, Copenhagen University Hospital - Mental Health Services CPH, Copenhagen, Denmark

[8]Department of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark

[9]University of Groningen, University Medical Center Groningen, Departments of Psychiatry and Internal medicine, Groningen, Netherlands

linamu@rm.dk

Abstract: Background: Adolescence represents a particularly vulnerable life stage, marked by extensive physical and psychosocial transformations. The presence of severe Functional Somatic Symptoms (FSS) may represent as an early risk factor for psychiatric disorders later on. We aim to provide a comprehensive description of the temporal associations between FSS in preadolescence and a range of psychiatric disorders diagnosed through adolescence until age 20 years. Methods: FSS self-report questionnaire at 11-12 years were obtained from the population-based Copenhagen Child Cohort (CCC2000). These data were linked to each participant's psychiatric diagnostic status from mental health services over the 8 years thereafter. We categorized ICD-10 diagnostic codes into Hierarchical Taxonomy of Psychopathology (HiTOP) spectra, and added additional clusters representing neurodevelopmental and unspecific disorders as outcome categories. Time-to-event analyses (i.e. unadjusted Cox proportional hazard regression models) were performed to investigate the risk between those with high (top 10%) versus low (bottom 90%) FSS levels at age 11-12 on the HiTOP spectra. Preliminary results indicate that preadolescent severe FSS are associated with an increase in the risk for being diagnosed with any psychiatric diagnosis. More specifically, we found a significant association between FSS at age 11 with internalizing-, externalizing- and unspecific psychiatric diagnoses at age 21. Final results will be presented at the research meeting. Conclusion: Findings could inform targeted healthcare approaches for young people with severe FSS, including new avenues for early disease prevention and improved clinical treatment.

Poster 13

Beyond Diagnostics: A Dimensional Approach to Mental Profiling

Niels Mørch[1], Andrés Barrena Calderón[1], Timo Kvamme[2], Filip Ottoson[3], Simon Durand[1], Julie Donskov[1], Madeleine Ernst[3], Kristian Sandberg[2], Anders Børglum[1] & Per Qvist[1]

[1]Department of Biomedicine, Aarhus University, Aarhus 8000, Denmark

[2]Department of Clinical Medicine - Center of Functionally Integrative Neuroscience, Aarhus University, Aarhus 8000, Denmark

[3]Statens Serum Institut (SSI) - Danish Center For Neonatal Screening, 2300 Copenhagen S, Denmark

ncmo@biomed.au.dk

Abstract: Mental health extends beyond the absence of mental illness (MDx). It arises from a complex interplay between hereditary factors and exposures, encompassing emotional, psychological, and social dimensions. However, ambiguity between pathological and non-pathological states challenges traditional diagnostic classifications, necessitating a dimensional approach for precision psychiatry. This study aimed to explore data-driven methods for stratifying mental health risk based on psychometric and biological markers.

Here we analyze data from a large, young, representative and deep-phenotyped European sample. Data included psychometric assessments (e.g., personality, cognition, emotional regulation), brain-imaging, circulatory OMICs markers, and polygenic scores (PGSs) for a wide range of of psychiatry-related traits. Archetypal (soft-clustering) analyses was applied to psychometrics and PGSs to display participants on the phenotype spectrum as convex combinations of extreme observations.

Associations between archetype scores, OMICs and imaging were assessed using standard statistical tests.

Deep psychometric archetypal profiling effectively stratified participants into risk clusters defined by personal and familial MDx history. Highlighting their biological basis, clusters exhibited distinct biological features, with individual PGSs (e.g., well-being, MDx, and brain MRI measures) predicting liability to psychometry-based archetypes. Intriguingly, psychometric and PGS-based archetypes significantly overlapped, identifying high-risk subsets with multiple MDx diagnoses and elevated genetic risk.

Our findings demonstrate the feasibility of data-driven risk stratification of the general population the relevance of multimodal archetypal analyses for uncovering latent psychopathology structures. By integrating psychometric and biological data, we refine risk stratification and trait specification, revealing biosignatures associated with particular mental health traits. While replication in larger, longitudinal cohorts is warranted, our study advances the understanding of mental health by embracing a dimensional perspective, laying the foundation for personalized mental health care strategies.

Poster 14

Study protocol: The CI-CAP Youth project on Cognitive Bias and Interoception in Chronic Abdominal Pain in Youth

Sigrid Agersnap Bom Nielsen[1,2], Charlotte Ulrikka Rask[1,2], Karen Hansen Kallesøe[1,2], Micah Allen[2,3], Tanja Hechler[4]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital

[2]Department of Clinical Medicine, Faculty of Health, Aarhus University

[3]Center of Functionally Integrative Neuroscience (CFIN)

[4]Department of Clinical Psychology and Psychotherapy for Children and Adolescents, Institute of Psychology, University of Münster, Germany

sigrnl@rm.dk

Abstract: BACKGROUND: Functional abdominal pain disorders (FAPD) and inflammatory bowel disease (IBD) are increasingly common in youth. Despite differing pathologies, both involve chronic abdominal pain. Predictive coding models propose that persistent symptoms may arise from how the brain attends to and interprets both external stimuli (via cognitive biases) and internal bodily signals (via interoception).

OBJECTIVE: The study examines whether cognitive biases (in attention, interpretation, and memory) and altered interoception (altered perception, processing and regulation of internal bodily signals) represent mechanisms underlying chronic abdominal pain in youth across disorders.

DESIGN: We will include N = 180 participants aged 8–17: N = 60 with FAPD, N = 60 with IBD, and N = 60 healthy controls. Cognitive bias will be assessed by the Bias in Youth toward GastroIntestinal related Stimuli (BY-GIS task) using words and pictures. Interoceptive accuracy will be measured by the heart rate discrimination task (HRD) and the respiratory resistance sensitivity task (RRST). Interoceptive sensations (and if these provoke a fearful response) will be measured by an abdominal tensing task. Finally, gastric sensitivity will be measured by the water load test II (WLT-II). All participants will complete the BY-GIS and HRD tasks; a subset (15–30 per group, ≥13 years) will complete all five tasks. Abdominal symptoms, symptom load, emotional distress, self-perceived interoception, and related parent-reported variables will be assessed via validated questionnaires.

PERSPECTIVES: The study excels in a novel design which assess cognitive bias towards relevant stimuli and altered interoception across constructs (accuracy, sensations, and metacognition) and bodily domains (cardiac, respiratory, and gastric) in a young clinical sample. Results may inform a transdiagnostic understanding and, eventually, new treatments for chronic abdominal pain in youth.

Poster 15

Chemogenetic modulation of hippocampal circuits reveals links between fear memory and depressive-like behaviour

Lia Parada Iglesias[1], Michel Christian van den Oever[2], Gregers Wegener[1], Samia Joca[1,3]

[1]Translational Neuropsychiatry Unit, Clinical Medicine, Aarhus University, Denmark

[2]Department of Molecular and Cellular Neurobiology, Center for Neurogenomics and Cognitive Research, Amsterdam Neuroscience, Vrije Universiteit Amsterdam, The Netherlands

[3]Department of Biomedicine, Aarhus University, Denmark

iglesias_lp@clin.au.dk

Abstract: This abstract has been omitted due to confidentiality of unpublished data. For further information, please contact the authors.

Poster 16

Epigenetic Regulatory Signatures in Cognition and Mental Health

Manuel Peral-Vázquez[1,2], Anna Starnawska[1], Per Qvist[1]

[1]Department of Biomedicine, Aarhus University, 8000 Aarhus, Denmark

[2]Department of Molecular Biology and Genetics, 8000 Aarhus, Denmark

manuel@biomed.au.dk

Abstract: Mental health is a complex and heterogeneous trait shaped by the interplay of genetic, epigenetic, and environmental factors. It encompasses neurocognitive, neurostructural, emotional, psychological, and social dimensions, extending far beyond the absence of mental disorders. Studying intermediate phenotypes—simpler, more proximal traits—can offer deeper insights into the root causes of mental health conditions. Here, we introduce a novel approach that utilizes a large, deeply phenotyped sample to explore the relationship between DNA methylation patterns and mental health intermediate phenotypes in the general young population. Our future objectives include generating high-resolution methylation maps, identifying phenotype-linked methylation markers, and exploring their potential to explain variability in brain structure and psychological function. This work aims to advance our understanding of the molecular mechanisms contributing to mental health, and inform early detection and intervention strategies.

Poster 17

Spinal motoneurone excitability from pre-symptomatic to symptomatic stages in mice with C9orf72 repeat expansions

Rehák ová, Viktória; Dræby, Sandra Duval; Kadlecová, Marion; Zhao, Zhenxiang; Kolmos, Mie Gunni; Jensen, Dennis Bo; Meehan, Claire Francesca
University of Copenhagen
viktoria.rehakova@sund.ku.dk

Abstract: An increased excitability of the motor system has consistently been observed in Amyotrophic Lateral Sclerosis (ALS) at the cortical, spinal and peripheral level. Research from our laboratory and others has shown that the core pathological feature of this disease - nuclear depletion and cytoplasmic aggregation of TDP-43 protein – is sufficient to drive such changes. However, the majority of research investigating hyperexcitability in ALS uses mouse models with an atypical form of the disease not expressing TDP-43 pathology. In our current experiments, we have started exploring excitability in a relatively new ALS mouse model expressing TDP-43 pathology, based on the most common mutations found in both familial and sporadic ALS: C9orf72 repeat expansions (C9orf72RE). As excitability changes in peripheral axons are more pronounced in C9orf72RE carriers with ALS than C9orf72RE carriers without, we focused our experiments on C9orf72RE mice showing a slowly progressing motor phenotype. These mice all showed a clasping behaviour when suspended by the tail. Axon initial segments of the motoneurones appeared structurally relatively normal, however, the nodes of Ranvier showed many structural abnormalities. The changes seen in this model are therefore less extreme than those seen in SOD1 models at more advanced disease stages and more consistent with our observations in SOD1 mice at earlier stages. In vivo intracellular recordings from spinal motoneurones were performed at around 250 and 400 days of age in C9orf72RE mice and WT littermates. Most basic excitability parameters were unchanged, except motoneurones from C9orf72RE mice (both ages) showed signs of increased persistent inward currents. A progressive increase in Ih currents, that have been observed in motoneurons of ALS patients and are thought to contribute to neuronal dysfunction, were indicated by a greater sag response and reduced input resistance at 400 days. We hypothesized that as mice age, they would exhibit more signs consistent with an advancing ALS phenotype, including the loss of corticospinal motoneurons, spasticity, muscle weakness, and reductions in neuronal excitability, characterized by decreased rheobase and increased I/F gain. As expected, we observed these changes in 600-day-old mice.

Poster 18

Parental distress and Symptom severity in referred youths with Functional Abdominal Pain Disorders: A cross-sectional study prior to internet based Cognitive Behavioral Therapy

Eva Skovslund Nielsen[1,2], Karen Hansen Kallesøe[1,2], Anne Sofie Hansen[3,4], Lisbeth Frostholm[2,5], Maria Lalouni[6,7], Marianne Bonnert[8], Charlotte Ulrikka Rask[1,2]

[1]Department of Child and Adolescent Psychiatry, Aarhus University Hospital Psychiatry, Palle Juul-Jensens Boulevard 175, 8200 Aarhus N, Denmark

[2]Department of Clinical Medicine, Aarhus University, Palle Juul-Jensens Boulevard 82, 8200 Aarhus N, Denmark

[3]Research Unit for Child and Adolescent Psychiatry, Psychiatry, Aalborg University Hospital, Aalborg, Denmark.

[4]Department of Clinical Medicine, Aalborg University, Aalborg, Denmark.

[5]The Department of Functional Disorders, Aarhus University Hospital, Palle Juul-Jensens Boulevard 11, 8200 Aarhus, Denmark

[6]Division of Neuro, Department of Clinical Neuroscience, Karolinska Institutet, 17177 Stockholm, Sweden, Sweden

[7]Center for epidemiology and community medicine, Health Care Services Stockholm County, Solnavägen 1E, 10431 Stockholm, Sweden

[8]Centre for Psychiatry Research, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden & Stockholm Health Care Services, Stockholm County Council, Norra Stationsgatan 69, 113 64 Stockholm, Sweden

eniels@rm.dk

Abstract: Background: Functional abdominal pain disorders (FAPDs) affect 10–15% of children and adolescents, often co-occurring with anxiety, depression, and school absenteeism. These conditions significantly burden parents, who frequently experience elevated anxiety and depression compared to parents of healthy children. Parental distress not only exacerbates

challenges in managing the child's condition but is also linked to poorer child outcomes, including greater pain severity and reduced functioning in children and adolescents with chronic pain. Despite these findings, few studies have simultaneously explored multiple parental factors—such as emotional distress, protective responses, and health anxiety by proxy—and their relationships with child symptoms and functional outcomes in FAPDs. Understanding these relationships is critical for developing interventions that address parental well-being alongside child-focused treatments.

Aim: This study aims to describe parental characteristics at the time of inclusion in Internet-delivered Cognitive Behavioral Therapy (ICBT) for children and adolescents with FAPDs, explicitly focusing on parental distress (SCL-8), health anxiety by proxy (HAPYS), and parental responses (ARCS). Additionally, it explores how these factors correlate with child-reported symptoms and functional outcomes, such as gastrointestinal symptoms, health-related quality of life, avoidance and control behavior and illness worries.

Methods: Baseline data from 48 parent-child dyads and 27 parent-adolescent dyads enrolled in ICBT for FAPDs were analyzed. Spearman correlations were computed to assess associations between parent-, child/adolescent-, and dyadic variables. Hierarchical multiple linear regressions examined parental predictors of child outcomes, including gastrointestinal symptoms, pain intensity, quality of life, GI-specific anxiety, and avoidance/control behaviors. Parental distress, health anxiety by proxy, and symptom response were entered in Step 1, followed by child/adolescent sex in Step 2.

Results: The majority of participating parents were mothers, highly educated, employed, living with the child's other parent, and reported a middle to high household income. Parental reports indicated low emotional distress (median SCL-8 score of 2.0), moderate health anxiety by proxy (mean HAPYs score of 19.3) and monitoring behavior (mean ARCS Monitor score of 10.7), and low levels of protective behavior (mean ARCS Protect score of 7.9).

In adjusted analyses, higher parental emotional distress was associated with lower child/adolescent quality of life ($\beta = -0.93$, $p = 0.022$) and increased gastrointestinal-specific anxiety ($\beta = 0.63$, $p = 0.008$). Monitoring behavior was associated with greater pain intensity ($\beta = 0.22$, $p = 0.010$) Health anxiety by proxy and protective behavior were not independently associated with adverse child outcomes, although protective behavior was inversely related to gastrointestinal symptoms in adolescents ($\beta = 2.84$, $p = 0.015$). No significant sex differences were observed.

Conclusion: Parental emotional distress and monitoring behavior emerged as potentially important clinical targets in the context of pediatric FAPDs. These findings underscore the relevance of addressing parental factors in treatment but require replication in larger and more diverse samples to guide clinical applications.

Poster 19

Candidate pathways and biomarkers in OGT-CDG - a novel intellectual disability syndrome

Marie Sønderstrup[1,2], Per Qvist[1], Daan van Aalten[2]
[1]Department of Biomedicine;
[2]Department of Molecular Biology and Genetics
masoje@biomed.au.dk

Abstract: O-GlcNAc Transferase Congenital Disorder of Glycosylation (OGT-CDG) is a recently discovered intellectual disability (ID) syndrome caused by missense mutations in the OGT gene. OGT catalyses O-GlcNAcylation, a dynamic post-translational modification affecting thousands of proteins. OGT-CDG patients present with extensive phenotypic heterogeneity, including ID, developmental delay, hypotonia, and dysmorphic features. Despite some overlap between patients, the clinical presentation is highly variable, complicating diagnosis and impeding the identification of underlying mechanisms.

To address this, we enrolled a large cohort of OGT-CDG patients and family members to systematically map the heterogeneity of OGT-CDG. We tested all the patient variants in a pathogenicity assay to measure disruption of OGT and OGA protein levels. The results of this test and standardised clinical data were used to cluster and subset patients based on shared and distinct symptom profiles. Blood samples and shredded epithelial cells from patients, healthy family members, and OGT-CDG mouse models carrying patient mutations were analysed using various OMICs approaches for biological profiling, identifying potential biomarkers and revealing potential molecular mechanisms underlying OGT-CDG pathology.

Preliminary data from three different OGT-CDG mouse models showed upregulated Ogt mRNA levels and downregulated Oga mRNA levels compared to wild type (wt) mice in whole brain samples, in leukocytes, and in whole blood, respectively, suggesting that altered O-GlcNAc homeostasis may contribute to disease pathophysiology. In bulk RNA sequencing data from whole blood, we also saw differences at the transcriptional level when comparing one of the OGT-CDG mouse lines to the corresponding wt.

By integrating clinical, genetic, and biomarker data, this study aims to provide a comprehensive characterisation of OGT-CDG. Defining clinical profiles and associated biological signatures will offer novel insights into the aetiological processes underlying this syndrome, paving the way for improved diagnostic tools and potential therapeutic interventions for affected patients and their families.