

regard, Machine Learning (ML) could represent a turning point in the field precision psychiatry, given its capability of making single-subject level predictions.

Objectives: In the present work, we aimed at providing a biomarker-based differential diagnosis between MDD and BD. To that end, we implemented: i) a structural MRI-based ML model; ii) a combined ML model, trained on MRI data and Polygenic Risk Scores (PRS) for different psychiatric disorders.

Methods: 168 depressed patients (73 MDD, 95 BD) were recruited at the IRCCS San Raffaele Scientific Institute. All patients underwent T1-weighted and Diffusion Tensor Imaging scans. Voxel-Based Morphometry (VBM) measures were extracted with Computational Anatomy Toolbox 12 (CAT12). Fractional Anisotropy (FA), Axial Diffusivity (AD), Mean Diffusivity (MD), and Radial Diffusivity (RD) were extracted with Tract-Based Spatial Statistics (TBSS). PRS for MDD, BD, Schizophrenia, Attention Deficit/Hyperactivity Disorder, Anorexia Nervosa and Autism were computed for a subsample of 155 patients (67 MDD; 88 BD) through Infinium PsychArray 24 BeadChip. We trained a Multiple Kernel Learning (MKL) algorithm with voxel-wise VBM and DTI features, subsequently combining them with the extracted PRS.

Results: The neuroimaging model achieved a Balanced Accuracy (BA) of 71.65% and an Area Under the Curve (AUC) of 0.77 (85.44% sensitivity, 57.86% specificity). All the features contributed to the prediction, with AD (63%) and MD (26%) as the most predictive. Adding PRS to neuroimaging resulted in an improved performance, reaching 74.18% BA and 0.77 AUC (90.97% sensitivity, 57.38% specificity). The most predictive features of the neuroimaging-PRS model were MD (56%) and AD (27%).

Conclusions: Structural MRI discriminated between MDD and BD, and adding PRS to neuroimaging features improved the performance of the ML model. These results highlight the predictive power of structural neuroimaging for the differential diagnosis between MDD and BD, as well as prompting multimodal classifiers as a promising tool in the context of precision psychiatry.

Disclosure of Interest: None Declared

Neuroimaging

O021

Neural Associations of Preclinical Alzheimer's Disease in Individuals with Low Anxiety Scores

U. Cikrikcili^{1*}, B. H. Schott^{2,3}, U. Ay⁴, T. N. Jockschat⁵ and E. Düzel^{1,6}

¹German Center for Neurodegenerative Diseases (DZNE), Magdeburg;

²German Center for Neurodegenerative Diseases (DZNE);

³Department of Psychiatry and Psychotherapy, University Medical Center Goettingen, Goettingen, Germany; ⁴Hulusi Behçet Life Sciences Research Laboratory, Neuroimaging Unit, Istanbul University, Istanbul, Türkiye; ⁵Department of Psychiatry and Psychotherapy and ⁶Institute of Cognitive Neurology and Dementia Research, Otto-von-Guericke University, Magdeburg, Germany

*Corresponding author.

doi: 10.1192/j.eurpsy.2025.287

Introduction: Preclinical Alzheimer's disease (AD) is characterized by subtle cognitive changes that precede the onset of clinical symptoms. Neuropsychiatric symptoms such as anxiety have been

increasingly recognized for their potential role in accelerating disease progression. Although various theories have been proposed, anxiety may exacerbate cognitive decline through mechanisms involving stress-induced neurochemical dysregulation, affecting brain regions vulnerable to AD pathology.

Objectives: This study examines the neural correlates of preclinical AD in individuals with low anxiety scores, employing MRI to explore potential early biomarkers and elucidate the complex role of anxiety in the progression of AD.

Methods: A total of 172 participants from the German Center for Neurodegenerative Diseases Longitudinal Cognitive Impairment and Dementia Study (DELCODE) were categorized into three groups: Healthy Controls (HC, n=59), Subjective Cognitive Decline (SCD, n=77), and Mild Cognitive Impairment (MCI, n=36). Anxiety levels were assessed using the Geriatric Anxiety Inventory-Short Form (GAI-SF), and neural responses to novelty were examined using 3-Tesla MRI. Statistical models were adjusted for relevant covariates, including age, education and study site. The differences of the three groups were analysed by one-way ANOVA contrasts and post-hoc analyses were performed with two sample t-tests.

Results: Significant neural differences were observed across groups, particularly in the precuneus, right angular gyrus, and right cerebellum exterior ($p < 0.001$, $p = 0.001$, and $p = 0.002$ respectively). The SCD group demonstrated greater activation in the right angular gyrus compared to HC ($p = 0.008$), while the MCI group exhibited more pronounced differences L-R precuneus, right cerebellum exterior, right angular gyrus, and right middle frontal gyrus regions indicating further cognitive decline ($p < 0.001$, $p < 0.001$, $p = 0.001$, $p = 0.017$ respectively).

Conclusions: This study identifies critical brain regions, with a particular emphasis on the right angular gyrus, associated with the early stages of AD in individuals with low anxiety scores. The activation in these areas likely correlates with an early inhibition deficit at the systems level in individuals with preclinical memory impairment. However, the role of anxiety in preclinical AD is complex and variable among individuals. Anxiety may serve as an early response to subtle cognitive changes in some, while in others, it might emerge as a consequence of these changes. Moreover, the relationship between anxiety and neural alterations in AD could be bidirectional, where anxiety both influences and is influenced by the disease's progression. These findings highlight the importance of considering anxiety when identifying early biomarkers for AD and suggest that targeted interventions addressing anxiety may help slow cognitive decline.

Disclosure of Interest: None Declared

Precision Psychiatry

O022

Toward precision psychiatry using HD-EEG and normative modeling

M. Hassan^{1,2*}, A. Ebadi¹, A. Mheich^{1,3}, J. Tabbal¹, A. Kabbara¹, G. Robert⁴, A. Lefebvre⁵, A. Iftimovici^{6,7}, B. Rodríguez-Herreros⁸, N. Chabane⁸ and S. Allouch¹

¹MINDIG, Rennes, France; ²School of Science and Engineering, Reykjavik University, Reykjavik, Iceland; ³Service des Troubles du Spectre de l'Autisme et apparentés, Département de psychiatrie,