However, functional improvements (FAST) were not statistically significant (see table 3).
Conclusions: Esketamine demonstrated substantial effectiveness in reducing both depressive and anxiety symptoms in DTD patients over three months. More than half of the patients achieved a significant reduction in depression severity, with nearly a third reaching remission. The presence of late responders suggests that esketamine may benefit those initially unresponsive to treatment. These findings support esketamine as a valuable therapeutic option for DTD in real-world clinical settings.

Disclosure of Interest: None Declared

O094

Polygenic risk for depression and career performance

A. Hazak[1,2,3,4]*, J. Liuhanen[1,5,6], K. Kantojärvi[1,5,6], S. Sulkava[1,5,7], T. Jääskeläinen[1], V. Salomaa[1], S. Koskinen[1], M. Perola[1,8] and T. Paunio[1,5,6]

[1]Department of Public Health and Welfare, Finnish Institute for Health and Welfare; [2]SleepWell Research Program, University of Helsinki, Helsinki; [3]Department of Finance, Aalto University, Helsinki area, Finland; [4]Department of Economics and Finance, Tallinn University of Technology, Tallinn, Estonia; [5]Department of Psychiatry / SleepWell Research Program, University of Helsinki; [6]Department of Psychiatry; [7]Department of Psychiatry / Department of Clinical Genetics, Helsinki University Central Hospital and [8]Research Program for Clinical and Molecular Metabolism, University of Helsinki, Helsinki, Finland
*Corresponding author.
doi: 10.1192/j.eurpsy.2025.348

Introduction: Major depression not only impairs individual health and labour market performance but also imposes significant economic burdens on society. It is linked to substantial costs through healthcare use, lost productivity and absenteeism. Recent genome-wide association studies have identified genetic markers associated with major depression, offering new insights into its genetic risk factors. However, the potential association of these genetic risks with educational attainment and career outcomes remains underexplored. Understanding this connection is crucial for addressing the broader public health and socio-economic implications of depression risk beyond clinical populations.
Objectives: This study aims to investigate the relationship between genetic risk for depression and individual career performance in the general population of Finland from 1992 to 2017.
Methods: We utilised pooled data from the Finnish Finrisk (1992-2012) and FinHealth (2017) studies, which together include a population representative sample of individuals aged 25-64 (N=20,121). Genetic, survey and socio-economic registry data were integrated for this analysis. Using probit and semi-structural regression models, we examined various career performance indicators, with polygenic scores for depression (Howard *et al.* Nat Neurosci 2019; 22 343-352) as the main explanatory variable. Socio-demographic characteristics and genetic principal components were included as controls.
Results: Our study revealed a negative association between higher genetic risk for depression and the likelihood of attaining higher education—an essential predictor of career success. Additionally, our study provides novel insights into how elevated polygenic risk for depression was linked to employment and self-employment rates, both directly and via educational pathways.
Conclusions: These findings highlight that genetic predispositions for depression can adversely affect career prospects in the general population, suggesting that the economic burden of depression extends beyond those clinically diagnosed. As effect sizes are modest, our results imply that supportive measures and compensatory behaviours could mitigate some of the educational and career disadvantages associated with higher genetic risk for depression.

Disclosure of Interest: A. Hazak: None Declared, J. Liuhanen: None Declared, K. Kantojärvi: None Declared, S. Sulkava: None Declared, T. Jääskeläinen: None Declared, V. Salomaa: None Declared, S. Koskinen: None Declared, M. Perola: None Declared, T. Paunio Consultant of: Idorsia Pharmaceuticals and Biogen (unrelated to the present work)

O095

Associations Between Vascular Endothelial Growth Factor, Major Depressive Episode and Response to Electroconvulsive Therapy: A Meta-Analysis

I. Borja De Oliveira[1], G. A. M. Alves[2], D. Soler Lopes[3], A. V. de Vasconcelos[4], A. L. Stephany[5], F. Reis Soares[6], M. Kraide Piedade de Abreu[5], M. C. Carbajal Tamez[7], J. Quevedo[8] and M. Teranishi[9]*

[1]School of Medicine, University of São Paulo, São Paulo, Brazil; [2]Humanitas University, Milan, Italy; [3]Lancaster University, Lancaster, United Kingdom; [4]Afya College of Medical Sciences of Santa Inês, Santa Inês; [5]São Paulo Association for the Development of Medicine, São Paulo; [6]Pontifical Catholic University of Goias, Goiania, Brazil; [7]Department of Psychiatry and Behavioral Sciences at McGovern Medical School; [8]Center for Interventional Psychiatry, Faillace Department of Psychiatry and Behavioral Sciences, McGovern Medical School, The University of Texas Health Science Center at Houston, Houston, United States and [9]Università degli Studi di Milano, Milan, Italy
*Corresponding author.
doi: 10.1192/j.eurpsy.2025.349

Introduction: Major depressive episodes (MDEs) occur in mood disorders such as major depressive disorder (MDD) and bipolar disorder (BD), affecting nearly one in four U.S. adults over their lifetime. The neurotrophic hypothesis suggests that disruptions in growth factor signaling contribute to MDEs. While brain-derived neurotrophic factor (BDNF) is well-studied, vascular endothelial growth factor (VEGF) may also play a crucial role, though evidence of its association with MDEs is inconsistent. Understanding VEGF is important for identifying predictors of treatment outcomes, such as those related to electroconvulsive therapy (ECT). This study explores the relationship between VEGF and MDEs, focusing on implications for ECT effects.
Objectives: To consolidate evidence from studies evaluating the association between VEGF and ECT outcomes in patients experiencing an MDE.

https://doi.org/10.1192/j.eurpsy.2025.349 Published online by Cambridge University Press

Methods: A systematic search for published clinical trials and cohort studies was conducted on August 13, 2024, using keywords including ECT, VEGF, MDE, and mood disorders, with no language or publication date restrictions. We selected studies enrolling patients in a current MDE related to MDD or BD, excluding those focused on manic episodes. A fixed-effects or random-effects model was applied. Subgroup analyses were performed to investigate the data further.
Results: Seven studies involving 621 participants (61.9% female; mean age: 50.2 years) were preselected. Six studies measured plasma VEGF levels; one assessed cerebrospinal fluid (CSF) levels. Plasma VEGF levels did not differ significantly between healthy controls and MDE patients, either before (SMD = 0.02 [−0.17; 0.21], p = 0.84, I^2 = 0%) or after ECT (SMD = 0.11 [−0.21; 0.44], p = 0.50, I^2 = 0%). Of the five studies reporting post-ECT VEGF levels, three found a significant increase from baseline. A significant correlation was observed between baseline plasma VEGF levels and depression response to ECT (r = 0.34, Z = 4.92, p < 0.0001, I^2 = 0%). Of the five studies examining increased VEGF levels after ECT and symptom reduction, only one found a significant association. A sensitivity analysis indicated substantial heterogeneity when including the CSF study.
Conclusions: Plasma VEGF levels were not significantly different in MDE patients compared to healthy controls, either before or after ECT. Baseline plasma VEGF levels positively correlated with ECT treatment response, suggesting they may provide neurotrophic support and predict outcomes. Despite robust findings and minimal heterogeneity, this analysis was limited by the low number of studies and small sample sizes. Further research is needed to explore the association between MDEs and VEGF, especially in the CSF, and to clarify the role of baseline VEGF in ECT treatment response.

Disclosure of Interest: I. Borja De Oliveira: None Declared, G. A. M. Alves: None Declared, D. Soler Lopes: None Declared, A. de Vasconcelos: None Declared, A. Stephany: None Declared, F. Reis Soares: None Declared, M. Kraide Piedade de Abreu: None Declared, M. Carbajal Tamez: None Declared, J. Quevedo Shareolder of: Instituto de Neurociencias Dr. Joao Quevedo, Grant / Research support from: LivaNova; and receives copyrights from Artmed Editora, Artmed Panamericana, and Elsevier/Academic Press, Consultant of: EMS, Libbs, and Eurofarma, Speakers bureau of: Myriad Neuroscience and AbbVie., M. Teranishi: None Declared

O097

Response to Ketamine Therapy in Anxious and Non-Anxious Major Depressive Disorder: A Meta-Analysis of Clinical Trials

I. Borja De Oliveira[1], A. L. Stephany[2], M. L. Geremias[3], D. Xavier[4], F. Wagner[5], A. L. Balduino de Souza[6], M. O. Pozzolo Pedro[7], D. Soler Lopes[8], M. C. Carbajal Tamez[9], J. Quevedo[10] and M. Teranishi[11]*

[1]School of Medicine, University of São Paulo, São Paulo; [2]Integrated Medicine Service, Jacareí; [3]University of the Joinville Region, Joinville, Brazil; [4]ECPE - PPCR Program, Harvard T. H. Chan School of Public Health, Boston, United States; [5]School of Medicine, Pontifical Catholic University of Rio Grande do Sul, Porto Alegre; [6]Evangelical University of Goias, Anapolis; [7]Departament and Institute of Psychiatry, University of São Paulo, São Paulo, Brazil; [8]Lancaster University, Lancaster, United Kingdom; [9]Department of Psychiatry and Behavioral Sciences at McGovern Medical School; [10]Center for Interventional Psychiatry, Faillace Department of Psychiatry and Behavioral Sciences, McGovern Medical School, The University of Texas Health Science Center at Houston, Houston, United States and [11]Università degli Studi di Milano, Milan, Italy
*Corresponding author.
doi: 10.1192/j.eurpsy.2025.350

Introduction: Anxious depression (AxD) as an independent diagnostic has been controversial, with many suggesting it as a transient state and others highlighting evidence of a worse outcome, severity, and increased suicide risk. The International Classification of Diseases (ICD-11) lists a related concept under 6A73, Mixed depressive and anxiety disorder. Previous literature on ketamine's efficacy has mainly focused on either anxiety or depression, with limited comparison of both groups. Given their high comorbidity and shared pathophysiology, we aimed to assess ketamine's efficacy in these populations.
Objectives: This meta-analysis aimed to consolidate evidence from clinical trials evaluating ketamine therapy in AxD and Non-Anxious Depression (NAxD).
Methods: A search for published clinical trials in indexed journals and databases was conducted on August 11, 2024. Keywords included ketamine, anxiety, comorbidity, and depression, with no restrictions on language or publication date. Studies on bipolar or psychotic depression were excluded. A random-effects model accounted for variability, and subgroup analyses were performed.
Results: Eight studies involving 536 participants (mean age = 39.0 years) were preselected. Seven studies defined "anxious depression" as a score of 7 or higher on the HAMD-AS, with AxD mean of 8.74 (±0.56) and NAxD mean of 5.83 (±1.9). MADRS scores were 35.18 (±2.22) for AxD and 31.97 (±2.29) for NAxD. The effect size of improvement in depressive symptom severity (as assessed by the MADRS) was not significantly different between the groups either 13 days after treatment (SMD = -0.07[-0.69, 0.55], p = 0.82, I2 = 73%) or 26-28 days after treatment (SMD = -0.30[-0.64, 0.04], p = 0.09, I2 = 21%). The overall depression response also did not significantly differ between the groups (odds ratio = 0.84 [0.50, 1.41], p = 0.52, I^2 = 13%). Insufficient data were available for remission rates.
Conclusions: Ketamine shows comparable efficacy in reducing depressive symptoms and achieving response in both groups. The group classified as AxD parallels previous reports of increased severity when reviewing baseline scores MADRS and other available scores. Thus, ketamine should be considered a viable treatment for patients with AxD, as they may have lower response rates to traditional antidepressants. This analysis was limited by the small number of studies, small sample sizes, and moderate heterogeneity. Differences in baseline depressive symptom severity and varying definitions of MDD with anxiety also constrained our analysis. Given the severity of symptoms in this population, we recommend developing better classification instruments for AxD. Further research is needed to explore remission differences in AxD and refine treatment strategies.

Disclosure of Interest: I. Borja De Oliveira: None Declared, A. Stephany: None Declared, M. Geremias: None Declared,