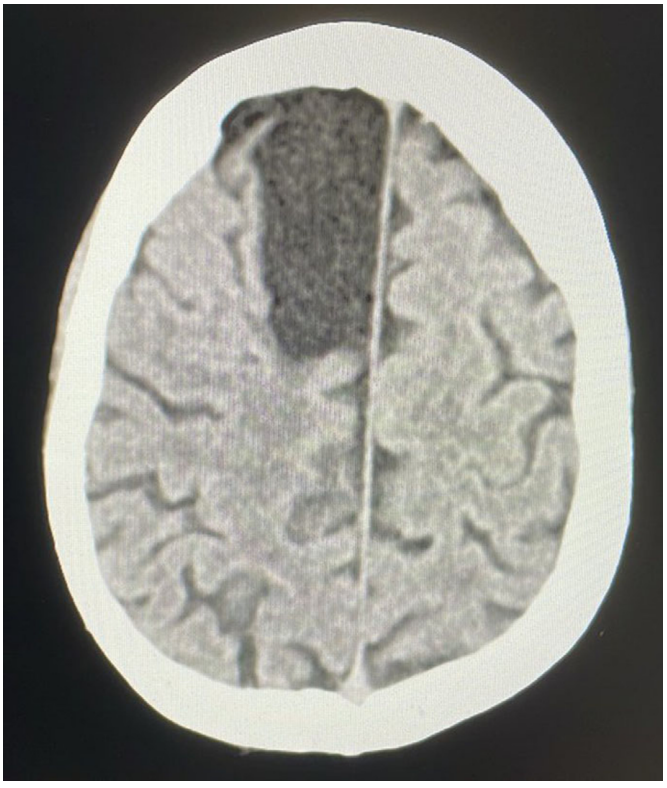
Image 2:



Conclusions: Organic findings on neuroimaging often guide diagnosis. In this case, disinhibition aligns with a frontal lesion. However, the timing of onset suggests the symptoms may be unrelated to the imaging, which suggests a longstanding lesion. The patient was functioning normally until three months ago, and the cause of symptom onset remains unclear.

Disclosure of Interest: None Declared

EPV906

White Matter Integrity of the Corpus Callosum and Dissociative Tendencies: A Cross-Sectional Analysis

P. Podwalski[1]*, B. Dawidowski[1], L. Franczak[1], B. Misiak[2] and J. Samochowiec[1]

[1]Department of Psychiatry, Pomeranian Medical University, Szczecin and [2]Department of Psychiatry, Wroclaw Medical University, Wrocław, Poland

*Corresponding author.

doi: 10.1192/j.eurpsy.2026.11625

Introduction: Dissociative symptoms are common in clinical populations exposed to trauma and are hypothesized to involve disrupted interhemispheric connectivity. Diffusion tensor imaging (DTI) studies have implicated alterations in the corpus callosum (CC), but evidence remains inconsistent. We examined whether fractional anisotropy (FA) in CC regions is associated with self-reported dissociative tendencies.

Objectives: The objective of this study was to examine whether FA in the CC —both globally and within a specific segment—is associated with dissociative tendencies. Advanced statistical procedures and sensitivity analyses with covariates were applied to assess the reliability of effects and to determine the extent to which callosal microstructure explains variability in dissociative symptoms.

Methods: Eighty-two adults (clinical and healthy controls) completed the Dissociative Experiences Scale (DEST). Mean FA across the corpus callosum (CC_MEAN_DTI) and segment 6 (CC_6_MEAN_DTI) were extracted from DTI. Linear regressions were performed with DEST as the dependent variable, after Box–Cox transformation selected by AIC ($\lambda = -1$). Assumption checks guided inference: in case of heteroscedasticity or non-normal residuals, heteroskedasticity-robust SEs and bootstrap (wild or residual, B=2000) were applied. Multiple testing was controlled per marker using Benjamini–Hochberg FDR. Sensitivity analyses included bootstrap stability and stepwise models with covariates (Group, BMI, education).

Results: Both FA measures showed negative associations with dissociative tendencies ($\beta \approx -0.67$ for CC_MEAN_DTI; $\beta \approx -0.76$ for CC_6_MEAN_DTI), suggesting lower FA might correspond to higher dissociation. However, after robust bootstrapping, neither effect was statistically significant ($p \approx 0.46$; FDR-adjusted $p \approx 0.46$). Variance explained was modest (R^2 ~6–8%). Stepwise models including group status and covariates further attenuated the associations (β reduced by ~80%), indicating that apparent links were largely confounded. Bootstrap stability was moderate, but confidence intervals overlapped zero when tested adaptively.

Conclusions: This study did not find statistically reliable associations between FA in the corpus callosum and dissociative tendencies. Although trends were in the hypothesized direction (reduced CC integrity linked to higher dissociation), effects were small, unstable, and largely explained by other factors. Results highlight the need for larger samples and longitudinal designs to clarify whether callosal microstructure contributes to dissociative psychopathology.

Disclosure of Interest: None Declared

Neuroscience in Psychiatry

EPV911

Integrating Non-Invasive Neurotherapies in Severe Depression: Where Does ECT Stand?

C. E. Anghel[1,2]* and I. C. Băcilă[1,3]

[1]Faculty of Medicine, The "Lucian Blaga" University; [2]ECT and [3]rTMS, Scientific Research Collective in Neurosciences - The "Dr. Gheorghe Preda" Clinical Hospital of Psychiatry, Sibiu, Romania

*Corresponding author.

doi: 10.1192/j.eurpsy.2026.11624

Introduction: Major depressive disorder (MDD), particularly in its severe and treatment-resistant forms, remains a considerable clinical challenge despite advances in pharmacotherapy and psychotherapy. In recent years, non-invasive neurotherapies—such as repetitive transcranial magnetic stimulation (rTMS) and transcranial direct-current stimulation (tDCS)—have gained increasing

attention due to their favorable safety profiles and expanding evidence. As these modalities become integrated into clinical pathways, the role of electroconvulsive therapy (ECT) requires renewed evaluation.
Objectives: This presentation aims to critically compare ECT with emerging non-invasive neurotherapies and to outline an updated framework for their integration in the management of severe depression.
Methods: This work draws on a narrative review of current clinical guidelines, recent research evidence, and implementation models related to ECT, rTMS, and tDCS published over the past five years. Clinical practice insights are incorporated, focusing on treatment sequencing, combination or stepwise approaches, and decision-making factors in real-world psychiatric settings.
Results: ECT continues to demonstrate superior efficacy and speed of response in severe and treatment-resistant depression, while rTMS and tDCS offer greater tolerability and acceptance, with expanding indications. Differences are highlighted regarding cognitive effects, patient preference, long-term outcomes, and practical considerations for clinical deployment. Ethical and medico-legal aspects—including informed consent, patient autonomy, and shared decision-making—emerge as essential to fostering responsible, patient-centered use of neuromodulatory treatments.
Conclusions: Integrating ECT within modern neurotherapeutic care requires a personalized, evidence-based approach that aligns clinical effectiveness with ethical practice. By combining current evidence, clinical insight, and evolving treatment models, a pragmatic framework is proposed to support clinicians in optimizing recovery, functioning, and quality of life for patients with severe depression. Further research and service development are needed to refine treatment pathways and enhance interdisciplinary neurotherapeutic care.
Keywords: non-invasive neurotherapies, severe depression , ECT, personalized approach.

Disclosure of Interest: None Declared

EPV912

The Energetic Roots of Mental Illness: Mitochondrial Dysfunction in Psychiatry

M. Arts[1,1,2*], M. Zeilstra[3], K. M. Zeilstra-Kalt[3] and L. D. Jonge[4]
[1]Geriatric Psychiatry and Neuropsychiatry; [2]Lifestyle Medicine, GGZ WNB, Halsteren; [3]Integrative Medicine, Center de Wetering, Leeuwarden and [4]Integrative Medicine, Leonardo Scientific Research Institute, Groningen, Netherlands
*Corresponding author.
doi: 10.1192/j.eurpsy.2026.11623

Introduction: Psychosocial stress and early-life adversity (ELA) increase lifetime risk for psychiatric and medical morbidity. Mitochondria, key organelles for energy, signaling, and steroidogenesis, are tightly coupled to stress mediators (glucocorticoids, catecholamines, cytokines) and are abundant in the brain. Acute stress adapts mitochondrial dynamics; chronic stress induces dysfunction with oxidative stress, altered mitochondrial DNA (mtDNA), and impaired bioenergetics, potentially embedding adversity biologically and shaping mood and cognition.
Objectives: To synthesize evidence on (a) mitochondrial mechanisms in stress responding and allostasis, (b) associations between mitochondrial biomarkers and psychopathology, and (c) implications for prevention and treatment.
Methods: Selective literature review integrating human and animal studies across neuroscience, endocrinology, and immunology. Focus on endocrine, inflammatory, and epigenetic pathways; neuroimaging; and peripheral biomarkers (mtDNA copy number, cell-free mtDNA, mitochondrial health index).
Results:
- Stress activates SAM/HPA axes and immune signaling, increasing energetic demand and ROS; prolonged exposure fragments mitochondria and damages mtDNA.
- Mitokines (ROS, calcium, cell free mtDNA) signal mitochondrial distress and promote inflammation; cell free mtDNA rises after psychosocial stress.
- ELA and lifetime psychopathology correlate with higher leukocyte mtDNA copy number and shorter telomeres in several cohorts; findings vary by tissue, chronicity, and medication.
- Animal models show that corticosterone or stress paradigms impair mitochondrial function and induce anxiety/depression-like behavior; brain studies implicate cortico-limbic circuits.
- Mitochondrial dysfunction occurs across disorders (major depressive disorder, anxiety, PTSD, bipolar disorder, schizophrenia, autism, neurodegeneration) with altered ETC activity and metabolism.
- Measurement challenges (cell heterogeneity, frozen samples, assay variability) limit comparability.

Conclusions: Mitochondria lie at the nexus of stress biology and psychiatric outcomes, linking endocrine, immune, and metabolic pathways to neural circuits and behavior. Evidence supports a contributory role of mitochondrial dysfunction and stress-related "mitochondrial allostatic load." Standardized, longitudinal studies with functional assays are needed to resolve heterogeneity, clarify reversibility, and test targeted interventions (e.g., anti-oxidative, metabolic, psychotherapeutic) as adjuncts to improve mental health.

Disclosure of Interest: None Declared

EPV913

Mitochondrial Dysfunction as a Therapeutic Target in Neurodegenerative and Psychiatric Disorders

M. Arts[1,1,2*], M. Zeilstra[3], K. M. Zeilstra-Kalt[3] and L. D. Jonge[4]
[1]Geriatric Psychiatry and Neuropsychiatry; [2]Lifestyle Medicine, GGZ WNB, Halsteren; [3]Integrative Medicine, Center de Wetering, Leeuwarden and [4]Integrative Medicine, Leonardo Scientific Research Institute, Groningen, Netherlands
*Corresponding author.
doi: 10.1192/j.eurpsy.2026.11622

Introduction: Mitochondria are central to cellular bioenergetics, signaling, and apoptosis. Dysfunction in these organelles is increasingly recognized as a common pathway in neurodegenerative and psychiatric disorders. Aberrant mitochondrial dynamics, impaired oxidative phosphorylation, calcium dyshomeostasis, and oxidative stress contribute to neuronal vulnerability, synaptic failure, and altered brain connectivity. Understanding these mechanisms opens novel therapeutic avenues beyond symptomatic treatment.
Objectives: This review synthesizes current evidence on (a) mitochondrial pathophysiology in neurodegenerative and psychiatric disorders, and (b) emerging therapeutic strategies including dietary, pharmacological, and transplantation-based interventions.

https://doi.org/10.1192/j.eurpsy.2026.11624 Published online by Cambridge University Press