

including cerebrospinal fluid (CSF) levels of A β 42, phosphorylated tau (p-tau), and total tau (t-tau). Additionally, we examined associations between brain fluidity and cognitive performance (Mini-Mental State Examination (MMSE)). Statistical analyses included between-group comparisons and regression models to determine the predictive value of fluidity in tracking disease severity.

Results

Fluidity analysis across frequency bands (theta, alpha, beta, gamma) revealed significant differences in AD patients (Fig. 1a). In the theta band (4–8 Hz), fluidity was higher in AD compared to controls, while in the beta band (14–30 Hz), fluidity was lower. Correlation analyses showed no significant associations between theta fluidity and clinical measures. However, beta fluidity negatively correlated with tTau and pTau (Fig. 1c), suggesting a link to neurodegeneration. Notably, no significant associations were found between fluidity and A β levels. Using a multilinear regression model we also found that adding fluidity calculated in the beta band significantly improved the predictive power for clinical disability.

Discussion

This results could imply that changes in the ability of the brain to flexibly switch between different dynamic states are associated with neurodegenerative processes, specifically tau-related damage. Reduced brain fluidity in beta may reflect underlying neurodegenerative processes, providing insights into the functional consequences of neuronal loss. Given its sensitivity to AD-related changes, brain fluidity may serve as a promising biomarker for tracking disease progression and evaluating treatment efficacy in clinical settings.

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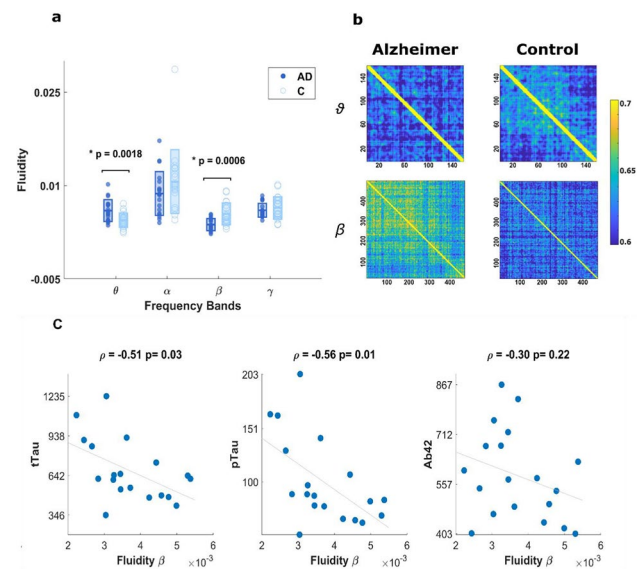


Figure 1: a) Fluidity for each frequency band in AD and control groups. b) dFC matrices averaged across AD (left) and control (right), computed in theta (top) and beta (bottom). c) Correlation between beta-band fluidity and tTau (left), pTau (center), A β (right), with significant links to tTau ($p = 0.03$) and pTau ($p = 0.01$), but not A β 42.

P192 Identifying Cell-Type-Specific Alterations Underlying Schizophrenia-Related EEG Deficits Using a Multi-scale Model of Auditory Thalamocortical Circuits

Scott McElroy^{1,2}, James Chen^{1,2}, Nikita Novikov^{1,2,3}, Pablo Fernández-López⁴, Carmen Paz Suárez-Araújo⁴, Christoph Metzner⁵, Daniel Javitt³, Sam Neymotin³, and Salvador Dura-Bernal^{1,2,3}

¹Global Center for AI, Society and Mental Health, SUNY Downstate Health Sciences University, Brooklyn, United States of America

²Department of Physiology and Pharmacology, SUNY Downstate Health Sciences University, Brooklyn, United States of America

³Center for Biomedical Imaging & Neuromodulation, Nathan Kline Institute, Orangeburg, United States of America

⁴Instituto Universitario de Cibernética, Empresa y Sociedad, Universidad de Las Palmas de Gran Canaria, Gran Canaria, España

⁵Technische Universität Berlin, Berlin, Germany

*Email: scott.mcelroy@downstate.edu

Introduction

Schizophrenia is associated with cognitive deficits, including disruptions in sensory processing. Electroencephalography (EEG) studies have identified abnormalities in event-related potentials and cortical oscillations, particularly within the auditory system. Among the most well-established EEG biomarkers are the reduced 40 Hz Auditory Steady-State Response (ASSR) and impaired mismatch negativity (MMN). Understanding the neural mechanisms underlying these EEG deficits is critical for linking molecular and circuit-level alterations to cognitive dysfunctions in schizophrenia.

Methods

We extended our computational model of auditory thalamocortical circuits to investigate the circuit-level mechanisms underlying schizophrenia-related EEG abnormalities [1]. The model simulates a cortical column with over 12,000 neurons and 30 million synapses, incorporating experimentally derived neuron densities, laminar organization, morphology, biophysics, and connectivity across multiple scales. Auditory inputs to the thalamus were modeled using a phenomenological cochlear representation, allowing for the reproduction of realistic physiological responses. Additionally, a more systematic approach to providing background network activity was implemented using Ornstein-Uhlenbeck (OU) processes to model time-varying, statistically independent somatic conductance injections.

Results

Our refinements enhance the physiological fidelity of EEG simulations, enabling improved replication of schizophrenia-related biomarkers. The integration of OU-modeled background activity ensures smoother, correlated variations in network input, leading to more biologically realistic fluctuations in neuronal dynamics. The OU process's mean and standard deviation are expressed as input conductance percentages for each cell type, linking them to intrinsic cellular properties. Additionally, we are developing an adaptive algorithm to dynamically calibrate population-specific OU parameters, ensuring model flexibility as it evolves.

Conclusion

By incorporating experimentally observed molecular and genetic alterations, our model provides deeper insights into the neural basis of auditory processing deficits in schizophrenia and strengthens the link between cellular dysfunctions and EEG biomarkers.

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P193 Brief Neurofeedback Training Increases Midline Alpha Activity in Default Mode Network

Matthew McGowan¹, Alison Crilly¹, Rongxiang Tang², and Yi-Yuan Tang*¹

¹College of Health Solutions, Arizona State University, Tempe, United States

²Department of Psychological & Brain Sciences, Texas A&M University, College Station, United States

*Email: yiyuan@asu.edu

Introduction

EEG Neurofeedback trains individuals to voluntarily modulate brainwave activity, promoting cognitive, emotional, and behavioral improvements by modulating large-scale brain networks and inducing neural plasticity [1]. While traditional neurofeedback protocols often require 20–40 sessions over several weeks or months, this study investigated whether a brief neurofeedback intervention—10 sessions over 2 weeks—could achieve similar neural regulation, particularly within the Default Mode Network (DMN).

Methods

To maximize the effects of neurofeedback, we selected a protocol designed to reward frontal midline Theta (4–8 Hz) to enhance executive function and emotional balance, and central sensorimotor Rhythm (SMR, 12–15 Hz) to promote focus and calmness, while inhibiting posterior midline Beta (16–35 Hz) to reduce stress and improve sensory clarity. This protocol aims to enhance self-regulation, resilience, and overall brain efficiency, thereby facilitating neurofeedback learning and benefits. Twenty participants with mild alcohol, tobacco, and/or cannabis use were recruited, and 19 provided usable data. Participants were instructed to complete each neurofeedback session with minimal effort to achieve the training goals. The NASA Task Load Index (NASA-TLX), a subjective workload assessment tool, was administered to 12 participants (11