

EPA-1506 – Impact of brain-derived neurotrophic factor (BDNF) gene polymorphism on cortical inhibition in schizophrenia

W. Strube¹, T. Bunse¹, T. Wobrock², S. Witt³, V. Nieratschker⁴, P. Falkai¹, A. Hasan¹

¹ Psychiatry, Klinikum der LMU München, Munich, Germany

² Psychiatry, Georg-August-University, Göttingen, Germany

³ Genetic Epidemiology in Psychiatry, Central Institute of Mental Health, Mannheim, Germany

⁴ Molecular Psychiatry, University Tübingen, Tübingen, Germany

Introduction

Neuronal plasticity is a characteristic feature of the human central nervous system. Impaired cortical plasticity has been reported to be associated with a val/met gene-polymorphism in the brain-derived neurotrophic factor (BDNF) gene. Based on former studies, impaired cortical plasticity has been described as a common, yet not fully understood phenomenon in patients with schizophrenia.

Objectives

The aim of the study was to investigate the impact of the val/met-BDNF gene-polymorphism on cortical plasticity in patients with schizophrenia.

Materials & Methods

Cortical plasticity was investigated in a cross-sectional sample of schizophrenia patients (SZ) and healthy controls (HC) using anodal and cathodal transcranial direct current stimulation (tDCS) applied to the primary motor cortex inducing long-term potentiation (LTP) and depression (LTD). The after-effect of the applied tDCS was then probed by TMS before and after tDCS stimulation. TMS in all participants was conducted using a standard 70mm-TMS figure-of-eight magnetic coil connected to a MagPro X 100 magnetic stimulator. Analyses were focused on resting-motor threshold (RMT), 1mV motor-evoked potential (MEP), short-interval intracortical inhibition (SICI at 3ms), intracortical facilitation (ICF at 12ms) and cortical silent period (CSP at 120% RMT).

Results

The statistical analyses included n=43 SZ and n=44 HC participants (all numbers presented under reserve of further examination) thus resulting in the largest available sample to probe cortical plasticity parameters in BDNF-genotyped schizophrenia patients. Further results will be presented at the conference.

Conclusion

The findings will give new insights into the influence of plasticity-associated gene polymorphisms and the pathophysiology of schizophrenia.