Universität Basel

Research Project

Brain derived neurotrophic factor as a biomarker of insomnia

Third-party funded project

Project title Brain derived neurotrophic factor as a biomarker of insomnia
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Brain-derived neurotrophic factor (BDNF) is a member of a family of growth factors located in the brain and peripheral tissues and plays an essential role in the neuronal cell differentiation, growth and survival. In the last decade, BDNF has become increasingly accepted as a central mediator of the effects of stress on neuronal plasticity. In a recent pilot study, our research group was the first to show that patients suffering from insomnia showed significantly decreased serum BDNF levels compared to sleep healthy controls. In the proposed project, we aim at assessing, whether BDNF may serve as a biomarker for insomnia on a larger scale across different diagnostic entities of sleep disorders and sleep healthy controls to verify the results of our exploratory investigation (Giese at al. 2013) and to elucidate the underlying mechanisms more closely. Moreover, given the relationship between sleep and cognitive performance, our research is also aimed in a second line, to extend our knowledge on the relationship between sleep and cognitive disturbances with reference to potential mediators. For that purpose, 62 male and female patients aged between 18 and 65 years suffering from insomnia according to DMS-IV criteria as well as a control group of 62 healthy, age and gender-matched controls will be enrolled in a prospective sleep center study performed at the Psychiatric University Clinics in Basel. Our main working hypothesis is that higher severity of insomnia is related to lower BDNF serum levels in patients suffering from insomnia (primary study outcome). As secondary objectives, the relationship between serum BDNF levels and objective sleep EEG parameters of sleep continuity (sleep onset latency, sleep duration, number of awakenings, wakefulness after sleep onset and sleep efficiency) and sleep EEG parameters of sleep architecture (stage 1 and 2, slow wave sleep and REM sleep), BDNF serum levels, BDNF signalling and cognition will be investigated in an explorative approach. Thus, we expect the following secondary outcomes: i.) Objective EEG sleep parameters indicating abnormalities of sleep continuity and sleep architecture are related to lower serum BDNF levels. ii.) Higher severity of insomnia is associated with decreased cognitive performance in attentional as well as memory-related cognitive performance. iii.) Lower BDNF serum levels are associated with lower cognitive performance in the cognitive test battery. iv.) Lower BDNF serum levels are associated with reduced pattern separation ability, thus indicating a hippocampal-dependent memory dysfunction. v.) Lower BDNF serum levels are associated with disturbed BDNF signalling pathways. If we can substantiate the role of BDNF as a marker of insomnia in a larger group of patients and across different diagnostic entities this has important implications for our understanding of the pathophysiology of insomnia. Once validated as a reliable marker of insomnia

BDNF may serve as a research and diagnostic tool for a dimensional approach to insomnia that better reflects the underlying neurobiological mechanisms than mere categorical diagnostic attributions.

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