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NEUROBIOLOGICAL BASIS OF COGNITIVE IMPAIRMENT IN SCHIZOPHRENIA

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Schizophrenia is a severe mental disorder which is characterized by delusions, hallucinations, disorganized speech, catatonic behavior that last for six months or longer affects 1 percent of the population [1]. Some studies categorize the symptoms of schizophrenia into five dimensions; positive, negative, cognitive, aggressive and affective symptoms [2]. Cognitive impairment is often said to be the "core" of schizophrenia, because it affects daily psychosocial functioning. Cognitive has been established as a predictor of real-world community functioning that perform everyday living skills that are related to live independently [1]. Cognitive functions include speed of processing, attention, working memory, verbal and visual learning and memory, verbal skills, social cognition, executive functions, visual function and motor functions [3]. Gene-environmental interaction affects cognition[4]. Brain derived neurotrophic factor (BDNF), disrupted in schizophrenia 1 (DISC 1), neuregulin 1 (NRG 1), "Akt 1", dystrobrevin binding protein 1 (DTNBP 1), ZNF 804A, ITIH3/4, CACNA1C and SDCCAG8 genes influence cognition. BDNF is related to hippocampal neuroplasticity, which is involved in cognitive processing. DISC1 gene regulates neuritic growth and migration. NRG1 play a role in regulating synaptic plasticity [2],[4]. Working memory impairment is associated with dorsolateral prefrontal cortex, medial prefrontal cortex, visual cortex. Dysregulation in dopaminergic, gamma-aminobutyric acid (GABA), glutamatergic in the functional brain areas are responsible for working memory deficits in schizophrenia [4]

References

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Biography

Elmeida Effendy has completed her PhD in 2013. She is the biological psychiatrist consultant, senior lecturer at Department Psychiatry –University as Sumatera Utara –Indonesia, and now she is Head of

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