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Cognitive Enhancement Effects of BRAHMIi On Novel Object Recognition and Neuronal Density in the Prefrontal Cortex, Striatum and Hippocampus of Schizophrenia Rat Model

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Introduction: Cognitive deficit is a significant problem in schizophrenic patients. It cannot be attenuated by any antipsychotic drugs. It has been known that changes of neuronal density is correlated with learning and memory deficits. *Bacopa monnieri* (Brahmi), popularly known as a cognitive enhancer, might be a novel therapeutic agent for cognitive deficit in schizophrenia by changing cerebral neuronal density.

Objectives: To determine the effects of Brahmi on attenuation at cognitive deficit and on the neuronal density in the prefrontal cortex, striatum and cornu ammonis subfield 1 (CA1) and 2/3 (CA2/3) of hippocampus in sub-chronic phencyclidine (PCP) rat model of schizophrenia.

Methods: Rats were assigned to three groups; **Group-1**: Control, **Group-2**: PCP administration and **Group-3**: PCP + Brahmi. Rats were tested for cognitive ability by using novel object recognition test. Neuronal density from a serial Nissl stain sections of the prefrontal cortex, striatum and CA1-3 of all rats were measured by using Image ProPlus software and manual counting.

Results: Sub-chronic PCP administration resulted in cognitive deficits in novel object recognition task. This occurred alongside significantly increased neuronal density in CA1. The cognitive deficit was recovery to normal in PCP + Brahmi group and this occurred alongside significantly decreased neuronal density in CA1.

Conclusion: Brahmi can be a potential cognitive enhancer against Schizophrenia. It reduces density of neuron in CA1. The reduced neuron is possibly glutamatergic neuron, which results in neuronal toxicity and cognitive deficit. Therefore, Brahmi eliminates glutamatergic excitotoxicity in CA1 and cognitive function is then recovery.

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