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Increased Novel Object Recognition by Reduced VGLUT3 in CA1-3 of Schizophrenia Rat Model: Effects of Bacoside a and B

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Introduction: It has been reported that vesicular glutamate transporter 1 (VGLUT1) and 2 (VGLUT2) are implicated in cognitive deficit in schizophrenia rat model. However, vesicular glutamate transporter 3 (VGLUT3) has not been investigated yet. *Bacopa monnieri* (Brahmi) has been popularly known as a cognitive enhancer in elderly and patients with Alzheimer's disease. It consists mainly of bacoside A and B, which may increase cognitive function in schizophrenia by changing VGLUT3 density in the brain.

Objectives: To study effects of bacoside A and B (bacoside A+B) on attenuation at cognitive deficit and cerebral VGLUT3 density in sub-chronic phencyclidine (PCP) rat model of schizophrenia.

Methods: Rats were administered PCP or vehicle. Half of the PCP-group was treated with bacoside A+B. Discrimination ratio (DR) representing cognitive ability was obtained from novel object recognition test. VGLUT3 immunodensity was measured in prefrontal cortex, striatum and CA1-3 of hippocampus using immunohistochemistry.

Results: DR in PCP-group was significantly decreased compared with control. This occurred alongside VGLUT3 up-regulation in CA1-3, but not in prefrontal cortex and striatum. PCP with bacoside A+B showed a significant increase in DR score compared with PCP alone. This occurred alongside significant decrease in VGLUT3 immunodensity in CA1-3. No significant difference in cerebral VGLUT3 immunodensity was observed between PCP with bacoside A+B and control.

Conclusions: VGLUT3 up-regulation in CA1-3 could produce glutamate excitotoxicity, which then induce cognitive deficit in schizophrenia rat model. Interestingly, bacoside A+B can recover this cognitive deficit by reducing excessive VGLUT3 in CA1-3 to normal level.

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