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Development and progression of Alzheimer's disease in Sprague-Dawley rats administered with streptozotocin intrahippocampally

Gurmeet Kaur Surindar Singh[1, 2], **Mazzura Wan Chik**[1] and **Nurul Aqmar Mohd Nor Hazalin**[2]
[1]Universiti Teknologi MARA (UiTM)—Puncak Alam Campus, Malaysia
[2]Universiti Teknologi MARA (UiTM)—Shah Alam Campus, Malaysia

Alzheimer's disease (AD) rat model can be reproduced via intrahippocampal (IH) administration of streptozotocin (STZ) in the rat's brain. The hippocampus holds a large amount of insulin receptors (IRs) which are very sensitive to STZ. IRs' exposure to STZ prompted memory impairment and production of amyloid-beta plaques related to AD pathogenesis. The present study is conducted to investigate the effects of IH-STZ administration on the progression of memory impairment and formation of amyloid β (Aβ) at 3, 6 and 12 weeks of STZ-treatment. Sixty male Sprague-Dawley rats (350–450 g) were divided into groups of control (no treatment), sham-operated (received PBS) and IH-STZ treated (G3w, G6w and G12w). STZ (3 mg/kg; 5 µl) was administered bilaterally as a single injection into the dorsal hippocampus of the rats. The memory impairment was studied a week before decapitation using Morris water maze test. The rats were sacrificed at week 3, 6 and 12 after STZ administration and presence of Aβ plaques were studied using the immunohistochemistry. All IH-STZ rats showed significant results in escape latency, total distance travelled and swimming speed ($p<0.05$) when compared to sham indicating memory impairment. In conclusion, STZ when injected intrahippocampally, developed memory impairment as early as two weeks after STZ treatment in the rats.

gurmeet9952@puncakalam.uitm.edu.my

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