

Exenatide Stimulates Hippocampal Autophagic Activity in Alzheimer Rat Model

Eman Abdo¹, Mohamed Saad¹, Alaa El-Din Firgany¹, Youssef mosaad¹, and Sara Eldusoky¹

¹Mansoura University Faculty of Medicine

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Abstract

Purpose: This study was designed to demonstrate potential neuroprotective and autophagic activity of exenatide in rodent AD model. **Experimental Approach:** Thirty adult Sprague-Dawley male rats were divided into 3 groups (10 rats each); Group 1; control normal group, Group 2; AD pathological group, Group 3; exenatide treated group. All drugs were given intraperitoneal (IP) for 42 days. Behavioral changes using Morris water maze test has been evaluated, gene expressions of beclin-1 and the mammalian target of rapamycin (mTOR) in the hippocampus were assessed. Examination of hippocampal tissue using hematoxylin & eosin (H&E) stain and ultrastructural analyses were also done. Data were analyzed by using the statistical package for the social sciences (SPSS). **Key Results:** Exenatide alleviated both behavioral and pathological changes compared to pathological group. Exenatide treated group was found to improve autophagic activity by increasing beclin-1 and decreasing mTOR gene expression. Exenatide treatment significantly prevented hippocampal neuronal degeneration demonstrated by H&E. Moreover, accumulation of autophagic vacuoles in ultrastructure study of hippocampus, alleviated in exenatide group compared to pathological group indicating enhanced autophagic activity by exenatide. **Conclusion and Implications:** The results of the present study clearly indicated exenatide might have beneficial effects on impaired cognitive performance and hippocampal neuronal viability in AD by increasing autophagic activity. Increased beclin-1 seems to be the initiating player in this disease modifying effect and this supports the assumption of a disease modifying activity of exenatide through the autophagic activity.

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