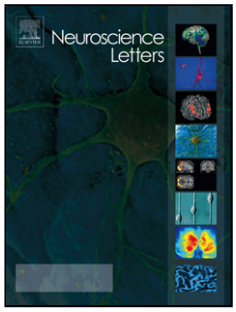

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Exendin-4 promotes proliferation of adipose-derived stem cells through PI3K/Akt-Wnt signaling pathways

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Highlight:

- It's the first time to evidence that Exendin-4 is effective in promoting Adipose-derived stem cell (ADSC) proliferation via PI3K-Akt-Wnt pathway;
- Exendin-4 activates the PI3K-Akt-GSK3β signaling pathway;
- Exendin-4 activates the Wnt signaling pathway.

Abstract

Adipose-derived stem cell (ADSC) transplantation has emerged as a potential tool for the treatment of cardiovascular disease. However, with a limited renewal capacity and the need for mass cells during the engraftment, strategies are needed to enhance ADSC proliferative capacity. In this study, we explored the effects of Exendin-4, a glucagon-like peptide-1 analog, on the growth of ADSCs, focusing in particular on phosphatidylinositol 3-kinase (PI3K)-protein kinase B (Akt) and Wnt signaling pathways. Firstly, ADSCs were isolated and cultured *in vitro*. Then, flow cytometry demonstrated that ADSCs were positive for CD44, CD90 and CD29 but negative for CD31, CD34, and CD45. Exendin-4 (0-200 nM) treatment increased ADSC proliferation. In order to examine specific signaling pathways, a western blotting assay was performed. Our results demonstrate that after treated with 50 nM Exendin-4 for 48 hours, the phosphorylation of PI3K, Akt, and GSK3β were increased and phosphorylation of β-catenin was decreased. From these results, we concluded that PI3K-Akt and Wnt-β-catenin signaling pathways mediate Exendin-4 induced ADSC proliferation, the function of which might contribute to the regulation of ADSC proliferation. Our findings provided new insights into the function of the mechanisms underlying Exendin-4 of ADSCs.