INTERNATIONAL SYMPOSIUM OF EXPERTS 2020

Under the patronage of
Her Highness Sheikha Madiyah Dalmook Bin Juma Al Maktoum

BOOK OF ABSTRACTS

REGENERATIVE MEDICINE and AGEING

February 01- 02 / 2020
Dubai, UAE

THE EFFECT OF KED TRIPEPTIDE ON NEURONAL DIFFERENTIATION OF HUMAN GINGIVAL MESENCHYMAL STEM CELLS

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Relevance: The generation of new neurons from stem cells could potentially become one of the promising treatment options for the patients with brain disorders such as neurodegenerative diseases. Human gingival mesenchymal stem cells (hGMSCs) are easy to assemble and isolate. This type of cells has a high proliferative ability, homogeneity and a stable phenotype. As has been shown, short peptides regulate functional activity, proliferation, apoptosis and differentiation of human, animal and plant cells. The KED peptide stimulated the restoration of synaptic transmission during neurodegeneration by increasing the number of mushroom spines in hippocampal neuron cultures in a model of Alzheimer's disease (wild-type C57BL/6 mice under conditions of amyloid synaptotoxicity). The aim of the study is to determine the effects of the KED peptide on the hGMSCs differentiation in the neural direction.

Materials and methods: hGMSCs were divided into 2 groups: 1 – control (PBS without addition of a peptide), 2 – addition of the AEDG peptide (10 ng in 1 ml of PBS). Real-time PCR quantitatively analyzed the expression of genes encoding Nestin proteins, β-tubulin III, GAP43, Doublecortin. MRNA expression was analyzed using GraphPad Prism 6.0 software. Qualitatively, the expression of neurogenesis proteins was evaluated by immunofluorescence reaction using confocal microscopy. Tukey criterion were applied for pairwise comparisons. A value of $p<0.01$ was considered statistically significant in all tests.

Results: Neurogenic-related genes were upregulated in hGMSCs treated with KED peptide for one week. KED peptide increased Nestin, GAP43, β-tubulin III and Doublecortin mRNA expression by 1.75, 1.65, 1.60 and 1.67 times in hGMSC culture in comparison with untreated cells. After the treatment period, the cells were observed by means of confocal microscopy to evaluate the modulation in marker expression related to neurogenic differentiation. HGMSCs treated with KED peptide also showed upregulation of all the studied markers: Nestin, GAP43, β-tubulin III and Doublecortin.

Conclusion: Since a decrease in the expression of Nestin, GAP43, β-tubulin III and Doublecortin markers was detected under various pathological conditions of the nervous system, it seems appropriate to study KED peptide as the modulator of neurogenesis in neurodegenerative diseases.