Genetics and Molecular Research

Epigallocatechin-3-gallate protects retinal vascular endothelial cells from high glucose stress *in vitro* via the MAPK/ERK-VEGF pathway

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Genet. Mol. Res. 15 (2): gmr.15027874
Received October 21, 2015
Accepted January 15, 2016
Published June 10, 2016
DOI http://dx.doi.org/10.4238/gmr.15027874

ABSTRACT. Diabetic retinopathy (DR) is a frequent microvascular complication of diabetes, and one of the most common causes of legal blindness in the world. Epigallocatechin-3-gallate (EGCG) produces an anti-oxidative and anti-inflammatory effect against various human diseases. In this study, we determined the effect of EGCG on a human retinal endothelial cell (HREC) line. The cell viability was determined by a standard MTT assay, while the cell cycle and apoptosis rate were analyzed by flow cytometry. Inflammatory marker expression was detected by enzyme-linked immunosorbent assay. Treatment of HRECs with EGCG (20 and 40 μM) led to a significant decrease in the apoptosis rate (2.35 ± 0.56 and 1.24 ± 0.32%). The culture supernatant of cells treated with high glucose concentrations showed significantly higher levels of TNF-α (598.7 ± 89.7 *vs* 193.2 ± 38.5 pg/mL; $P < 0.001$), IL-6 (6.16 ± 0.51 *vs* 1.61 ± 0.21 ng/mL; $P < 0.001$),

and ICAM-1 (31.6 ± 4.4 *vs* 14.8 ± 2.9 ng/mL; $P < 0.001$) compared to the cells in the control group. EGCG decreased the expression level of phosphorylated p38-mitogen activated protein kinase (MAPK) and extracellular regulated kinase (ERK)1/2. Moreover, EGCG was shown to significantly inhibit the expression of vascular endothelial growth factor (VEGF). Therefore, EGCG treatment ameliorated the negative effect of high glucose concentrations on the cell viability and apoptotic rate. The protective effects of EGCG under high glucose conditions may be attributed to the regulation of inflammatory cytokines and inhibition of the MAPK/ERK-VEGF pathway.

Key words: Diabetic retinopathy; Epigallocatechin-3-gallate; Human retinal endothelial cell

