Study of Cellular Effects of Quercetin for Non-canonical Autophagy Induction in dff45 Knockdown Breast Cancer Cells (MCF-7 Cell Line)

Negin Rasouli 1,*

*Corresponding Author: Negin Rasouli, E-mail: raoli.negin@yahoo.com

Abstract

Introduction: Quercetin is a member of flavonoids having antioxidant and apoptotic effects. siRNA technology is a potent method for the gene therapy of cancer via down-regulation of some specific genes. Knockdown of dff45 gene could sensitize the cancer cells for apoptosis. Autophagy is another cellular pathway propelling the cells toward cell death in some types of stress conditions.

Materials and Methods: At first, four groups of MCF-7 cells were seeded in a six well plate and then two groups of them were transfected with dff45-siRNA. After 24 hours, siRNA-transfected and the non-transfected groups were treated with Quercetin. Using real time RT-PCR, investigation of the expression levels of the canonical apoptotic genes (Bax, Bcl2, casp3), caspase independent apoptosis (aif), autophagy (atg5, lc3, beclin, dram), cell senescence (p53, p16) and UPR (jnk, rab9, casp7, sqstm1) was carried out.

Results: In the presence of Quercetin, the expression pattern of the genes represents the occurring of cell senescence and canonical autophagy as the two important cellular processes for stopping cell proliferation and cell death. This notion is changed to the non-canonical autophagy and UPR in dff45-siRNA treated cells and is expressed as a mixed criteria for (Quercetin+dff45-siRNA) treatment.

Conclusions: In the Quercetin treated MCF-7 cells, the cause of stopping of cell proliferation and cell death is canonical autophagy (ATG dependent) which is changed to the non-canonical autophagy and UPR in dff45-siRNA treatment.

Keywords:
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Autophagy
Cell Senescence
UPR

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