

ASK-1 and p38 MAPK Mediate the Induction of Apoptosis by Genistein in Human Breast Cancer MCF-7 Cells

Hye Young Shim, Jong-Hwa Park and Ye Sun Han

Department of Advanced Technology Fusion and Bio/Molecular Informatics

Center, Konkuk University, Seoul 143-701, Korea

TEL: +82-2-450-3764, FAX: +82-2-3437-6106

Abstract

The effects of genistein on the cell proliferation in human breast cancer MCF-7 cells were investigated. Genistein inhibited the cell proliferation of MCF-7 cells and the concentration required to produce 50% growth inhibition at 24 h was approximately 27.5 M. Genistein-induced cell death was characterized with changes in DNA fragmentation. DNA ladder produced by a genistein-induced apoptosis was observed in dose- and time-dependent manners. The molecular mechanism of genistein-induced apoptosis was also investigated by the Western blot analysis of apoptosis-related proteins, such as caspase-3, 8, 9, p38 mitogen-activated protein kinase (MAPK) and apoptosis signal-regulating kinase 1 (ASK-1). MCF-7 cells treated with genistein induced the activation of caspase-3 by the cleavage of procaspase-3 and the phosphorylation of p38 MAPK and ASK-1. However, the cleavage of procaspase-8 and 9, which is known as mediators of procaspase-3 activation, was not observed in genistein treated MCF-7 cells. We are currently investigating the relation of p38 MAPK, ASK-1 and genistein in MCF-7 cells.

References

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