

THE EFFECT OF GENISTEIN ON TWO RAT PROSTATE CANCER CELL LINES WITH DIFFERENT METASTATIC POTENTIAL

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Genistein (4,5,7-trihydroksyisoflavone) derived from soybean is a potential chemopreventive agent against various types of cancer. The mechanism underlying the chemopreventive action of genistein is not clear. Genistein suppresses tyrosine kinases, topoizomerases, angiogenesis, growth factor-stimulated responses, oncogene product activity and prostaglandin synthesis. It has been reported that genistein induces cell cycle arrest and apoptosis in different cancer cell lines *in vitro*. On the other hand, the effect of genistein on the metastatic activity of tumour cells remains unclear. Tumour cell migration plays an important role in metastases formation because the cells have to migrate from primary sites, intra and extra vasa, and finally invade the target tissue to establish secondary tumours.

In this study, we investigated the effect of genistein on the motile activity, cell proliferation and apoptosis of two rat prostate cancer cell lines, AT-2 and MAT-LyLu, differing markedly in their metastatic ability (MAT-LyLu metastasises in more than 80% of cases when injected into rats, while AT-2 in less than 10%).

To estimate the level of apoptosis, cells were exposed to genistein, and after their DNA had been stained with propidium iodide, they were analysed by flow cytometry. The morphology of apoptotic cells was observed using a fluorescent microscope after staining with Hoechst 33342.

The movements of strongly metastatic MAT-LyLu and weakly metastatic AT-2 cells were recorded under an inverted microscope. Recordings were performed for 4 hours (control) and for 4 hours after adding genistein (100 μ M).

The study demonstrated that: 1) the motility of both investigated cell lines markedly decreased after the addition of 100 μ M genistein; 2) genistein at a concentration of 100 μ M induced apoptosis in two rat prostate cell lines: AT-2 and MAT-LyLu; 3) genistein at a concentration of 100 μ M arrested the cell cycle at the G2/M phase; and 4) the relative arrest of AT-2 cells at the G2/M phase was greater than that of the highly metastatic MAT-LyLu cells.