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Int J Oncol. 2001 Oct; 19(4):837-44.

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## Induction of cell cycle arrest and apoptosis in human breast cancer cells by quercetin.

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Quercetin, a widely distributed bioflavonoid, has been shown to induce growth inhibition in certain cancer cell types. In the present study we have pursued the mechanism of growth inhibition in MCF-7 human breast cancer cells. Quercetin treatment resulted in the accumulation of cells specifically at G2/M phase of the cell cycle. Mitotic index measured by MPM2 staining clearly showed that cells were transiently accumulated in M phase, 24 h after treatment. The transient M phase accumulation was accompanied by a transient increase in the levels of cyclin B1 and Cdc2 kinase activity. However, 24 h or longer treatment caused a marked accumulation of cells in G2 instead of M phase. Levels of cyclin B1 and cyclin B1-associated Cdc2 kinase activity were also decreased. We also found that quercetin markedly increased Cdk-inhibitor p21CIP1/WAF1 protein level after treatment for 48 h or longer, and the induction of p21CIP1/WAF1 increased its association with Cdc2-cyclin B1 complex, however, up-regulation of p53 by quercetin was not observed. Quercetin also induced significant apoptosis in MCF-7 cells in addition to cell cycle arrest, and the induction of apoptosis was markedly blocked by antisense p21CIP1/WAF1 expression. The present data, therefore, demonstrate that a flavonoid quercetin induces growth inhibition in the human breast carcinoma cell line MCF-7 through at least two different mechanisms; by inhibiting cell cycle progression through transient M phase accumulation and subsequent G2 arrest, and by inducing apoptosis.