

Biochanin A stimulates osteoblastic differentiation and inhibits hydrogen peroxide-induced production of inflammatory mediators in MC3T3-E1 cells

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Phytoestrogens, which have in part some structural similarity to 17β -estradiol, are reported to act as agonist/antagonist of estrogen in animals and humans. We examined the effects of biochanin A on the function of osteoblastic MC3T3-E1 cells and the production of local factors in osteoblasts. Biochanin A (1~50 μ M) significantly increased the growth of MC3T3-E1 cells ($P<0.05$). Biochanin A (1~50 μ M) caused a significant elevation of alkaline phosphatase (ALP) activity, collagen content, and osteocalcin secretion in the cells. The effect of biochanin A (10 μ M) in increasing ALP activity and collagen content was completely prevented by the presence of 1 μ M cycloheximide and 10 μ M tamoxifen, suggesting that biochanin A's effect results from a newly synthesized protein component and might be partly involved in estrogen action. We then examined the effect of biochanin A on the H_2O_2 -induced apoptosis and production of inflammatory mediators in osteoblasts. Treatment with biochanin A (50 μ M) prevented apoptosis induced by 0.2 mM H_2O_2 in osteoblastic cells. Moreover, biochanin A (1~10 μ M) decreased the 0.2 mM H_2O_2 -induced production of TNF- α , IL-6, and NO in osteoblasts. These results suggest that biochanin A may be useful as potential phytoestrogens, which play important physiological roles in the prevention of postmenopausal osteoporosis.

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