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The distinct role of estrogen receptor-α for the Yak-kong and soybean treatment induced proliferation of MG-63 cells.

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Phytoestrogens are receiving great scrutiny as a food supplement for preventing postmenopausal osteoporosis. The beneficial effect of phytoestrogens are caused by functioning as partial against or antagonists of estrogens. Yak -kong (*Rhynchosia Nolubilis* 鼠目太) contains large amounts of the phytoestrogens and has been used as supplements of estrogen for preventing postmenopausal osteoporosis in oriental medicine. Based on our previous study which reported that Yak-kong treatment increases estrogen receptor-α(ERα) expression and proliferation of MG-63 osteoblastic cells, the role of estrogen receptor-α (ERα) in the Yak-kong treatment induced proliferation of MG-63 cells was investigated in this study. MG-63 cells stably expressing ERα or antisense ERα (AsERα) were established by DNA-calcium phosphate coprecipitation of either pcDNA3-ERα or pcDNA3-AsERα plasmids. Constitutive expression of ERα itself increased MG-63 cell proliferation by 137% (of control : 100%) but did not affect the expression of estrogen receptor-β (ERβ), demonstrating that constitutive expression of ERα did not alter basal expression of other ER isozyme. Yak-kong treatment in ERα expressing MG-63 cells (ERα-MG-63 cells) resulted in a further increase of cell proliferation to 174%, which paralleled with the enhanced expression of IGF-1. Decreasing ERα by expression of antisense ERα RNA in MG-63 cells (AsERα-MG-63 cells) exerted the opposite effect, decreasing cell proliferation to 82% of control and IGF-1 expression. Yak-kong treatment in AsERα-MG-63 cells did not increase cell proliferation further. Based on these results, we conclude that ERα plays an important and active role in Yak-kong treatment mediated proliferation of MG-63 cells.