17-B-estradiol inhibits tumore necrosis factor-a induced nuclear factor kB actiation by increasing nuclear factor kB p105 in MCF-7 breast cancer cells

陳彥州
Hsu SM;Chen YC and Jiang MC

Abstract

Tumor necrosis factor-a (TNF-a) exerts many cytological effects on a wide range of cells. TNF-a can activate nuclear factor-kB (NF-kB). Activation of NF-kB by TNF-a mediates many functions of TNF-a. The NF-kB inhibitor, IkBa, negatively regulates the activity of NF-kB. In MCF-7 cells (an estrogen and TNF-a receptor positive cell line), treatment with 17bestradiol (E2) inhibited TNF-a-induced NF-kB DNA binding activity in the gel retardation assays. But, the level of the IkBa and the TNF-a receptor, TNF-R1, were not obviously affected. The NF-kB precursor, NF-kB p105, has been shown to be associated with NF-kB in the cytoplasm and efficiently blocks its nuclear translocation and activation. Treatment of MCF-7 cells with E2 increased the level of NF-kB p105 protein. The anti-estrogen, 4OH-tamoxifen, treatment inhibited E2-induced NF-kB p105 expression. Our findings indicate that NF-kB p105 plays a role in modulating the functions of TNF-a in the estrogen receptor positive breast cancer cells.© 2000 Academic Press Key Words: 17b-estradiol; TNF-a; IkBa; NF-kB p105; NF-kB; nuclear translocation; 4OH-tamoxifen; MCF-7 breast cancer cells.