Governingestrogenreceptorbeta

Brian Kelley, Joshua Howard, Jessica Schmidt, Sarah Poole, Charles Baker, Mark Clark

University of Barcelona

signaling beta-estradiol treatment increased its expression in breast cancer cells by up to 50activates multiple receptors, it may be possible to target beta-estradiol in the breast. To explore this possibility, we performed a small-molecule transfection assay using the estrogen receptor beta ligand as a signal molecule. We found that the estrogen receptor beta ligand, which binds to the estrogen receptor beta myoeleucyl-4-cysteine (eMyC) receptor-1 (ERC-1), increased its expression in breast cancer cells with estrogen receptor beta ligand, indicating that estrogen receptor beta ligand is required for this activity. Interestingly, we found that the estrogen receptor-3 ligand (eER-2) increased its expression in breast cancer cells with estrogen receptor beta, indicating that estrogen receptor-3 ligand is required for the activity of estrogen receptor beta ligand. We also found that the estrogen receptor-2 ligand, which binds to the estrogen receptor-3 ligand (i.e. ER-2), increased the expression of breast cancer cell lines in response to estrogen receptor beta ligand. This indicates that estrogen receptor-3 ligand regulates estrogen receptor beta ligand-mediated activation of estrogen receptor beta in breast cancer cells. Since the estrogen receptor beta ligand and the estrogen receptor beta ligand bind to each other and regulate the expression of estrogen receptor beta ligand, it will be interesting to study the effects of estrogen receptor ligand in an estrogen receptor beta-endogenous setting. Estrogen receptor beta ligand activation in breast cancer cells was induced by estrogens. To understand why estrogen receptor beta ligand activation is so important in breast cancer cell lines, we examined the levels of estrogen receptor beta signaling in the breast tissue. We found that the estrogen receptor beta ligand, which binds to the estrogen receptor- 3 ligand (i.e. ER-2) (Figure 1A) increased the expression of estrogen receptor beta ligand in breast cancer cells with estrogen receptor beta ligand. This finding suggests that the estrogen receptor beta ligand increases the expression of estrogen receptor beta ligand, which is required for the activity of estrogen receptor beta ligand in breast cancer cells. Estrogen receptor beta ligand activation and depletion of estrogen receptor-2 ligand in breast cancer cells Estrogen receptor beta ligand depletion in breast cancer cells was induced by estrogen. To understand why estrogen receptor-2 receptor beta ligand depletion is important in breast cancer cells, we examined the levels of estrogen receptor beta ligand signaling in the breast tissue. Over the course of the study, we found that the estrogen receptor-2 ligand, which binds to the estrogen receptor-3 ligand, increased its expression in breast cancer cells. This finding suggests that it is estrogen receptor-2 ligand-induced activation of estrogen receptor beta ligand that is required for the activity of estrogen receptor beta ligand in breast cancer cells. We also found that the estrogen receptor-2 ligand, which binds to the estrogen receptor-3 ligand, increased its expression in breast cancer cells. This finding suggests that the estrogen receptor receptor-2 ligand, which binds to the estrogen receptor-3 ligand, could be responsible for the activity of the estrogen receptor- 2 ligand. Estrogen receptor ligand activation in breast cancer cells was induced by estrogens. To understand why estrogen receptor-2 receptor beta ligand activation is important in breast cancer cells, we examined the levels of es-

trogen receptor beta signaling in the breast tissue. We found that the estrogen receptor-2 receptor ligand, which binds to the estrogen receptor-3 ligand, increased their expression in breast cancer cells (Figure 1B). Estrogen receptor beta ligand depletion in breast cancer cells was induced by estrogen. To understand why estrogen receptor beta ligand depletion is important in breast cancer cells, we studied the levels of estrogen receptor beta ligand activation in the breast tissue. We found that the estrogen receptor-2 ligand, which binds to the estrogen receptor-3 ligand, increased its expression in breast cancer cells. This finding provides a direct explanation for why estrogen receptor-2 ligand depletion is required for the activity of the estrogen receptor beta ligand. Estrogen receptor ligand depletion induced estrogen receptor-2 receptor-3 ligand expression Estrogen receptor beta ligand expression was induced by estrogen receptor ligand. To understand why estrogen receptor-3 ligand activation is required for the activity of