MCF7/164R-7 Cell Line

Catalogue number: 152103

Sub-type: Continuous

Images:

Contributor

Inventor: Anne Lykkesfeldt

Institute: Danish Cancer Society, Denmark

Images:

Tool details

*FOR RESEARCH USE ONLY

Alternate name: MCF7/164R-7; 164R-7

Class:
Conjugate:
Description **Description:** The MCF7/164R-7 cell line is a breast cancer cell line resistant to fulvestrant (Faslodex). The MCF7/164R-7 cell line is a human breast cancer cell line established from MCF7. The cellular classification is epithelial, and their shape is polygonal. The passage number of this cell line is 430 (AL2678, AL2679). Treatment with the steroidal antiestrogen fulvestrant has proven effective upon progression on tamoxifen therapy and is now approved for second-line treatment after tamoxifen or aromatase inhibitors. As for tamoxifen treatment of advanced breast cancer, resistance will inevitably occur also for fulvestrant. Clarification of the molecular changes associated with the resistant growth is needed to find targeted treatments to resistant tumour cells and treatments that can inhibit or delay the emergence of resistance.

Purpose:

Parental cell: MCF7 S0.5

Organism: Human Tissue: Breast Model: Tumour line Gender: Female

Isotype: Reactivity: Selectivity:

Host:

Immunogen:

Immunogen UNIPROT ID:

Sequence:

Growth properties:

Production details: The MCF7/164R-7 cell line has been established from a clone of MCF7/S0.5 cells surviving long term growth with the pure steroidal antiestrogen ICI 164,384 in 100 nM concentration, see Lykkesfeldt et al 1995. The MCF7/164R-7 cells are also resistant to the pure steroidal antiestrogen fulvestrant (ICI 182,780) and can be maintained continuously in growth medium with 100 nM fulvestrant.

Formulation:

Recommended controls:

Bacterial resistance:

Selectable markers:

Additional notes: Upon withdrawal of fulvestrant, the cells express ER alpha, although at a reduced level compared to the parental MCF7/S0.5 cell line. The MCF7/164R-7 cells do not express progesterone receptor. The MCF7/164R-7 cells express increased level of EGFR, phosphorylated EGFR and phosphorylated ErbB3 and reduced level of ErbB4 compared to the parental MCF7/S0.5 cells. Passage 430 (AL2678, AL2679)

Target details

Cancer Tools.org Target: Oestrogen receptor

Target alternate names:

Target background:

Molecular weight:

Ic50:

Applications

Application: Investigation of signalling pathways involved in fulvestrant resistance Application notes: Upon withdrawal of fulvestrant, the cells express ER alpha, although at a reduced level compared to the parental MCF7/S0.5 cell line. The MCF7/164R-7 cells do not express progesterone receptor. The MCF7/164R-7 cells express increased level of EGFR, phosphorylated EGFR and phosphorylated ErbB3 and reduced level of ErbB4 compared to the parental MCF7/S0.5 cells. Passage 430 (AL2678, AL2679)

Handling

Format: Frozen **Concentration:**

Passage number: Passage 430 (AL2678, AL2679)

Growth medium: Phenol red free DMEM/F12 (1:1) supplemented with 1% FCS, Glutamax 2.5 mM

and 6 ng/ml insulin. Supplemented with 100nM fulvestrant to maintain resistance.

Temperature:

37° C

Atmosphere: 5% CO2

Volume:

Storage medium: Storage buffer: Storage conditions:

Shipping conditions: Dry ice

Related tools

Related tools:

References

References: Thrane et al. 2014. Oncogene. PMID: 25362855.; A kinase inhibitor screen identifies McI-1 and Aurora kinase A as novel treatment targets in antiestrogen-resistant breast cancer cells.; Sonne-Hansen et al. 2010. Breast Cancer Res Treat. 121(3):601-13. PMID: 19697122. ; Breast cancer cells can switch between estrogen receptor alpha and ErbB signaling and combined treatment against both signaling pathways postpones development of resistance. ; Frogne et al. 2009. Breast Cancer Res Treat. 114(2):263-75. PMID: 18409071.; Activation of ErbB3, EGFR and Erk is essential for growth of human breast cancer cell lines with acquired resistance to fulvestrant.; Frankel et al. 2007. Breast Cancer Res Treat. 104(2):165-79. PMID: 17061041.; Protein Kinase C alpha is a marker for antiestrogen resistance and is involved in the growth of tamoxifen resistant human breast cancer cells. ; Frogne et al. 2005. Endocr Relat Cancer. 12(3):599-614. PMID: 16172194.; Antiestrogen-resistant human breast cancer cells require activated protein kinase B/Akt for growth.; Larsen et al. 1997. Int J Cancer. 72(6):1129-36. PMID: 9378550.; Resistance of human breast-cancer cells to the pure steroidal anti-estrogen ICI 182,780 is not associated with a general loss of estrogen-receptor expression or lack of estrogen responsiveness.; Lykkesfeldt et al. 1995. Int J Cancer. 61(4):529-34. PMID: 7759159.; Human breast cancer cell lines resistant to pure anti-estrogens are sensitive to tamoxifen treatment.