

# MCF7/182R-7 Cell Line

**Catalogue number:** 152106

**Sub-type:** Continuous

**Images:**

## Contributor

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**Images:**

## Tool details

**\*FOR RESEARCH USE ONLY**

**Name:** MCF7/182R-7 Cell Line

**Alternate name:** MCF-7/182R-7; 182R-7

**Class:**

**Conjugate:**

**Description:** The MCF7/182R-7 cell line is a breast cancer cell line resistant to fulvestrant (Faslodex). The MCF7/182R-7 cell line is a human breast cancer cell line established from a clone of MCF7/S0.5 cells surviving long term growth with the pure steroidal antiestrogen ICI 182,780 (fulvestrant) in 100 nM concentration. The cellular classification is epithelial, and their shape is polygonal. MCF7/182R-7 cells express oestrogen receptor alpha and do not express progesterone receptor. The passage number of MCF7/182R-7 is 427, 430. Treatment with the steroidal antioestrogen 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 MCF7/182R-7 cell line has been established from a clone of MCF7/S0.5 cells surviving long term growth with the pure steroidal antiestrogen ICI 182,780 in 100 nM concentration, Lykkesfeldt et al (1995). The MCF7/182R-7 cells 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. The MCF7/182R-7 cells do not express progesterone receptor. The MCF7/182R-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.

## Target details

**Target:** Oestrogen receptor

**Target alternate names:**

**Target background:**

**Molecular weight:**

**Ic50:**

## Applications

**Application:** Investigation of molecular changes

**Application notes:** Upon withdrawal of fulvestrant, the cells express ER alpha, although at a reduced level. The MCF7/182R-7 cells do not express progesterone receptor. The MCF7/182R-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 427(AL3419), 430 (AL3779)

## Handling

**Format:** Frozen

**Concentration:**

**Passage number:** Passage 427(AL3419), 430 (AL3779)

**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% CO<sub>2</sub>

**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 Mcl-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. ; Nabha et al. 2005. *Oncogene*. 24(19):3166-76. PMID: 15735693. ; Upregulation of PKC-delta contributes to antiestrogen resistance in mammary tumor cells. ; 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.