MCF7/TAMR-1 Cell Line

Catalogue number: 152089
Sub-type: Continuous
Images:

Contributor

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Images:

Tool details

***FOR RESEARCH USE ONLY**

Name: MCF7/TAMR-1 Cell Line

Alternate name: MCF-7/TAMR-1; MCF7/TAMR-1; MCF7 TAMR-1; MCF-7/TAM(R)-1; MCF7-TAMR; TAMR-1; TamR-1; TamR1; AL-1

Class:
Conjugate:
Description: MCF7/TAMR-1 cell line is a stable tamoxifen-resistant subline. This cell line has been established in tissue culture after long term treatment with 1 uM tamoxifen. Tamoxifen (Nolvadex) is a widely used drug for hormone-dependent cancer. Tamoxifen resistance (either primary or acquired) makes oestrogen receptor-positive breast cancer much more difficult to treat. This cell line was produced from the parental cell line MCF7/S0.5, as a model cell system to study the effects of tamoxifen resistant cancer growth. MCF7/TAMR-1 cells are oestrogen receptor positive and progesterone receptor negative. They are growth inhibited by the pure antioestrogen fulvestrant.Previous applications of this cell line include treatment with steroidal antiestrogens. MCF7/TAMR-1 enables identification of new hormone therapies and greater understanding of the signalling pathways/methods behind tamoxifen resistance. Additionally MCF/TAMR-1 can aid in identifying new predictive markers for response to hormonal therapy.
Purpose:
Parental cell: MCF7/S0.5 Cell Line
Organism: Human
Tissue: Breast
Model: Tumour line
Gender: Female
Isotype:
Reactivity:
Selectivity:
Host:

Immunogen:
Immunogen UNIPROT ID:
Sequence:
Growth properties:
Production details: The parental cell line for the MCF7/TAMR-1 cells is MCF7/S0.5, which was adapted to grow at low serum concentration in order to study the effect of estradiol and tamoxifen. MCF7/TAMR-1 has been established from a clone of cells that survived long term treatment with 1 uM tamoxifen. The establishment of the MCF7/TAMR-1 cell line, originally named AL-1, was first described in Lykkesfeldt et al (1986). Tamoxifen-resistant cells are passaged continuously in presence of 1 uM tamoxifen, which is le...
Formulation:
Recommended controls: MCF7-S0.5 parental line
Bacterial resistance:
Selectable markers:
Additional notes: During the establishment process the treatment of MCF7/S0.5 cells with tamoxifen was started in passage 351. Few colonies of cells survived the treatment and after 28 days of tamoxifen treatment, tamoxifen was omitted from the medium for 22 days. After 19 passages without tamoxifen (passage 372) the cells underwent a second treatment with tamoxifen which initially reduced cell growth rate, but around 390-400 the growth rate of the tamoxifen resistant cell lines was close to the growth rate of...

Target details

Target: Oestrogen receptor

Target alternate names:

Target background:

Molecular weight:

Ic50:

Applications

Application: New hormone therapies identification; Elucidating signaling pathways involved in tamoxifen-resistant cancer growth
Application notes: Points of Interest Estrogen receptor-positive breast cancer is the most common form of breast cancer, with approximately 80% of all breast cancers expressing the estrogen receptor (ER). They depend on the estrogen hormone to facilitate the growth and expansion of cancer cells. Hormone therapy (e.g. tamoxifen) can limit the growth of ER breast cancers by blocking the actions of estrogen. Tamoxifen resistance (either primary or acquired) makes ER+ breast cancer much more difficult to treat. MCF7/TAMR-1 cell line is able to survive with tamoxifen in growth medium, allowing the resistance to be understood and prospective new treatment options to be discovered. MCF7/TAMR-1 cells are oestrogen receptor positive and progesterone receptor negative. MCF7/TAMR-1 cells are

growth inhibited by the pure antioestrogen fulvestrant. The oestrogen receptor is a major driver of growth of MCF7/TAMR-1 cell. Treatment targeting the Aurora kinase A restores sensitivity to tamoxifen treatment. The TAMR lines were established from the MCF7/S0.5 cell line, which was adapted to grow with 0.5% fetal calf serum in phenol red containing DMEM/F12 medium. Treatment with tamoxifen was started in passage 351. Few colonies of cells survived the treatment and after 28 days of tamoxifen treatment, tamoxifen was omitted from the medium for 22 days. After 19 passages without tamoxifen (passage 372) the cells underwent a second treatment with tamoxifen which initially reduced cell growth rate, but around 390-400 the growth rate of the tamoxifen resistant cell lines was close to the growth rate of the parental MCF7/S0.5 cells. Passage 431 (AL3502, AL3503) Concentration Vial has between 1-5 million cells as standard, however this may vary.

Handling

Format: Frozen
Concentration:
Passage number: 431 (AL3502, AL3503)
Growth medium: Phenol red-free DMEM/F-12 containing 1% Fetal bovine serum, 2 mM Glutamax and 6 ng/ml Insulin. To maintain high-level resistance, the medium should be supplemented with 1 uM Tamoxifen
Temperature: 37° C
Atmosphere: 5% CO2
Volume:
Storage medium:
Storage buffer:
Storage conditions: Liquid Nitrogen
Shipping conditions: Dry ice

Related tools

Related tools: MCF7/S0.5 Cell Line

References

References: Joshi et al. 2016. Oncotarget. :. PMID: 27528030. ; Integrative analysis of miRNA and gene expression reveals regulatory networks in tamoxifen-resistant breast cancer. ; Elias et al. 2015. Oncogene. 34(15):1919-27. PMID: 24882577. ; 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. ; Gene expression profiling identifies FYN as an important molecule in tamoxifen resistance and a predictor of early recurrence in patients treated with endocrine therapy. ; Thrane et al. 2013. Breast Cancer Res Treat. 139(1):71-80. PMID: 23609470. ; Estrogen receptor a is the major driving factor for growth in tamoxifen-resistant breast cancer and supported by HER/ERK signaling. ; Cutrupi et al. 2012. Oncogene. 31(40):4353-61. PMID: 22249258. ; Targeting of the adaptor protein Tab2 as a novel approach to revert tamoxifen resistance in breast cancer cells. ;

Millour et al. 2010. Oncogene. 29(20):2983-95. PMID: 20208560. ; FOXM1 is a transcriptional target of ERalpha and has a critical role in breast cancer endocrine sensitivity and resistance. ; Pancholi et al. 2008. Endocr Relat Cancer. 15(4):985-1002. PMID: 18824559. ; ERBB2 influences the subcellular localization of the estrogen receptor in tamoxifen-resistant MCF-7 cells leading to the activation of AKT and RPS6KA2. ; Sarwar et al. 2006. Endocr Relat Cancer. 13(3):851-61. PMID: 16954434. ; Phosphorylation of ERalpha at serine 118 in primary breast cancer and in tamoxifen-resistant tumours is indicative of a complex role for ERalpha phosphorylation in breast cancer progression.