HCT 116 ADAM17 Cell Line

Catalogue number: 153209
Sub-type: Continuous
Images:

Contributor

Inventor: Sandra Van Schaeybroeck
Institute: Queen's University Belfast
Images:

Tool details

***FOR RESEARCH USE ONLY**

Name: HCT 116 ADAM17 Cell Line

Alternate name:

Class:
Conjugate:
Description: Chemotherapy (5-fluorouracil) treatment has been shown to result in acute increases in transforming growth factor-a, amphiregulin, and heregulin ligand shedding in vitro and in vivo correlating with significantly increased ADAM-17 activity. Small interfering RNAmediated silencing and pharmacologic inhibition confirmed that ADAM-17 was the principal ADAM involved in this prosurvival response. HCT 116 ADAM17 Cell Line showed that overexpression of ADAM-17 significantly decreases the effect of chemotherapy on tumor growth and apoptosis.
Purpose:
Parental cell: HCT 116
Organism: Human
Tissue: Colon
Model: Knock-In
Gender:
Isotype:
Reactivity:
Selectivity:
Host:
Immunogen:
Immunogen UNIPROT ID:
Sequence:
Growth properties: Invasion, migration
Production details: HCT116 cells were cotransfected with 10 Ä?Â?Ä???g of a plasmid containing

hemagglutinin (HA)-tagged full-length mouse ADAM-17 (HA-ADAM-17) and a construct expressing a puromycin resistance gene. Stably transfected cells were selected and maintained in medium supplemented with 1 Ä?Â?Ä???g/mL puromycin.
Formulation:
Recommended controls: HCT 116 parental line
Bacterial resistance:
Selectable markers:
Additional notes: Offered under licence from the Spanish National Research Council (CSIC)

Target details

Target: mouse ADAM17 (TACE)

Target alternate names:

Target background:

Molecular weight:

Ic50:

Applications

Application:
Application notes:

Handling

Format: Frozen
Concentration:
Passage number:
Growth medium: McCoy's 5a Medium (GIBCO # 16600) + 10% FBS + 100 units/ml penicillin+ 100 ?g/ml streptomycin
Temperature:
Atmosphere:
Volume:
Storage medium:
Storage buffer:
Storage conditions:
Shipping conditions: Dry ice

Related tools

Related tools:

References

References: Dunne et al. 2016. Clin Cancer Res. 22(1):230-42. PMID: 26283684. ; EphA2 Expression Is a Key Driver of Migration and Invasion and a Poor Prognostic Marker in Colorectal Cancer. ; Dunne et al. 2014. Clin Cancer Res. 20(1):164-75. PMID: 24170546. ; AXL is a key regulator of inherent and chemotherapy-induced invasion and predicts a poor clinical outcome in early-stage colon cancer.