

Anti-APC [c-APC 28.9]

Catalogue number: 152092

Sub-type: Primary antibody

Images:

Contributor

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Images:

Tool details

***FOR RESEARCH USE ONLY**

Name: Anti-APC [c-APC 28.9]

Alternate name:

CancerTools.org

Class: Monoclonal
Conjugate: Unconjugated
Description: C-APC 28.8 can be used for APC expression and detection of APC mutations.
Purpose:
Parental cell:
Organism:
Tissue:
Model:
Gender:
Isotype: IgG1 kappa
Reactivity: Human
Selectivity:
Host: Mouse
Immunogen: human APC C-terminal fusion with MBP (maltose binding protein)
Immunogen UNIPROT ID:
Sequence:
Growth properties:
Production details:
Formulation:
Recommended controls: Colon cell line HCT116
Bacterial resistance:
Selectable markers:
Additional notes:

Target details

Target: APC

Target alternate names:

Target background: The adenomatous polyposis coli tumor suppressor gene is mutated (often deletion of the C-terminal portion of APC) in the inherited disease, familial adenomatous polyposis (FAP), and over 80% of colorectal cancers. Clone c-APC 28.9 is useful for the detection of APC expression and detection of mutations. This antibody has been used routinely for Western blotting on colon cell line HCT116 extract which expresses full-length APC.

Molecular weight: 320 kDa

Ic50:

Applications

Application: IHC ; IF ; IP ; WB

Application notes:

Handling

Format: Liquid

Concentration: 1 mg/ml

Passage number:

Growth medium:

Temperature:

Atmosphere:

Volume:

Storage medium:

Storage buffer: PBS with 0.02% azide

Storage conditions: Store at -20° C frozen. Avoid repeated freeze / thaw cycles

Shipping conditions: Shipping at 4° C

Related tools

Related tools:

References

References: Thrane et al. 2015. Oncogene. 34(32):4199-210. 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. ; Madsen et al. 1997. Cancer Res. 57(4):585-9. PMID: 9044830. ; Estrogen receptor messenger RNA splice variants are not involved in antiestrogen resistance in sublines of MCF-7 human breast cancer 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.