

Anti-APC antibody [ALi 12-28] ab58

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Overview

Product name	Anti-APC antibody [ALi 12-28]
Description	Mouse monoclonal [ALi 12-28] to APC
Host species	Mouse
Tested applications	Suitable for: Flow Cyt
Species reactivity	Reacts with: Human Does not react with: Mouse
Immunogen	Amino acids 1-433 N-terminal fragment of human APC (Adenomatous Polyposis Coli gene Chr 5q) fused to maltose binding protein.
Epitope	ALI-12-28 was epitope mapped by differential in vitro expression of the N-terminal region of the APC gene by using the protein truncation test and was found to bind to APC in the region between nucleotides 135 and 422 (exons 2-3) (Efsthathiou et al.).
General notes	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As</p>

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.02% Sodium azide Constituent: 99.98% PBS
Purity	Protein A purified
Clonality	Monoclonal
Clone number	ALi 12-28
Myeloma	unknown

Isotype	IgG1
Light chain type	unknown

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab58 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
Flow Cyt		Use 1µg for 10 ⁶ cells. ab170190 - Mouse monoclonal IgG1, is suitable for use as an isotype control with this antibody.

Target

Function Tumor suppressor. Promotes rapid degradation of CTNNB1 and participates in Wnt signaling as a negative regulator. APC activity is correlated with its phosphorylation state. Activates the GEF activity of SPATA13 and ARHGEF4. Plays a role in hepatocyte growth factor (HGF)-induced cell migration. Required for MMP9 up-regulation via the JNK signaling pathway in colorectal tumor cells. Acts as a mediator of ERBB2-dependent stabilization of microtubules at the cell cortex. It is required for the localization of MACF1 to the cell membrane and this localization of MACF1 is critical for its function in microtubule stabilization.

Tissue specificity Expressed in a variety of tissues.

Involvement in disease Defects in APC are a cause of familial adenomatous polyposis (FAP) [MIM:175100]; which includes also Gardner syndrome (GS). FAP and GS contribute to tumor development in patients with uninherited forms of colorectal cancer. FAP is characterized by adenomatous polyps of the colon and rectum, but also of upper gastrointestinal tract (ampullary, duodenal and gastric adenomas). This is a viciously premalignant disease with one or more polyps progressing through dysplasia to malignancy in untreated gene carriers with a median age at diagnosis of 40 years. Defects in APC are a cause of hereditary desmoid disease (HDD) [MIM:135290]; also known as familial infiltrative fibromatosis (FIF). HDD is an autosomal dominant trait with 100% penetrance and possible variable expression among affected relatives. HDD patients show multifocal fibromatosis of the paraspinal muscles, breast, occiput, arms, lower ribs, abdominal wall, and mesentery. Desmoid tumors appears also as a complication of familial adenomatous polyposis. Defects in APC are a cause of medulloblastoma (MDB) [MIM:155255]. MDB is a malignant, invasive embryonal tumor of the cerebellum with a preferential manifestation in children. Although the majority of medulloblastomas occur sporadically, some manifest within familial cancer syndromes such as Turcot syndrome and basal cell nevus syndrome (Gorlin syndrome). Defects in APC are a cause of mismatch repair cancer syndrome (MMRCS) [MIM:276300]; also known as Turcot syndrome or brain tumor-polypoid syndrome 1 (BTPS1). MMRCS is an autosomal dominant disorder characterized by malignant tumors of the brain associated with multiple colorectal adenomas. Skin features include sebaceous cysts, hyperpigmented and cafe au lait spots. Defects in APC are a cause of gastric cancer (GASC) [MIM:613659]; also called gastric cancer intestinal or stomach cancer. Gastric cancer is a malignant disease which starts in the stomach, can spread to the esophagus or the small intestine, and can extend through the stomach wall to nearby lymph nodes and organs. It also can metastasize to other parts of the body. The term

gastric cancer or gastric carcinoma refers to adenocarcinoma of the stomach that accounts for most of all gastric malignant tumors. Two main histologic types are recognized, diffuse type and intestinal type carcinomas. Diffuse tumors are poorly differentiated infiltrating lesions, resulting in thickening of the stomach. In contrast, intestinal tumors are usually exophytic, often ulcerating, and associated with intestinal metaplasia of the stomach, most often observed in sporadic disease. Defects in APC are a cause of hepatocellular carcinoma (HCC) [MIM:14550]. This defect includes also the disease entity termed hepatoblastoma.

Sequence similarities

Belongs to the adenomatous polyposis coli (APC) family.
Contains 7 ARM repeats.

Domain

The microtubule tip localization signal (MtLS) motif; mediates interaction with MAPRE1 and targeting to the growing microtubule plus ends.

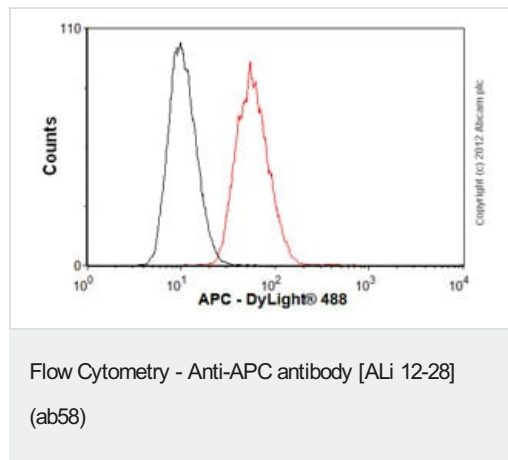
Post-translational modifications

Phosphorylated by GSK3B.
Ubiquitinated, leading to its degradation by the proteasome. Ubiquitination is facilitated by Axin. Deubiquitinated by ZRANB1/TRABID.

Cellular localization

Cell junction > adherens junction. Cytoplasm > cytoskeleton. Cell projection > lamellipodium. Cell projection > ruffle membrane. Cytoplasm. Cell membrane. Associated with the microtubule network at the growing distal tip of microtubules. Accumulates in the lamellipodium and ruffle membrane in response to hepatocyte growth factor (HGF) treatment. The MEMO1-RHOA-DIAPH1 signaling pathway controls localization of the phosphorylated form to the cell membrane.

Images



Overlay histogram showing HCT116 cells stained with **ab58** (red line). The cells were fixed with 80% methanol (5 min) and then permeabilized with 0.1% PBS-Tween for 20 min. The cells were then incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions followed by the antibody (**ab58**, 1 µg/1x10⁶ cells) for 30 min at 22°C. The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (**ab96879**) at 1/500 dilution for 30 min at 22°C. Isotype control antibody (black line) was mouse IgG1 [ICIGG1] (**ab91353**, 2 µg/1x10⁶ cells) used under the same conditions. Acquisition of >5,000 events was performed.

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