

Product datasheet

Human APC knockout K562 cell line ab277914

Overview

Product name	Human APC knockout K562 cell line
Description	APC KO K562 cell line
Parental Cell Line	K562
Organism	Human
Passage number	<20
Biosafety level	1
General notes	<p>Recommended control: Human wild-type K562 cell line (ab275469). Please note a wild-type cell line is not automatically included with a knockout cell line order, if required please add recommended wild-type cell line at no additional cost using the code WILDTYPE-TMTK1.</p> <p>Cryopreservation cell medium: Cell Freezing Medium-DMSO Serum free media, contains 8.7% DMSO in MEM supplemented with methyl cellulose.</p> <p>Culture medium: IMDM + 10% FBS</p> <p>Initial handling guidelines: Upon arrival, the vial should be stored in liquid nitrogen vapor phase and not at -80°C. Storage at -80°C may result in loss of viability.</p> <ol style="list-style-type: none"> 1. Thaw the vial in 37°C water bath approximately 1-2 minutes. 2. Transfer the cell suspension (0.8 ml) to a 15 ml/50 ml conical sterile polypropylene centrifuge tube containing 8.4 ml pre-warmed culture medium, wash vial with an additional 0.8 ml culture medium (total volume 10 ml) to collect remaining cells, and centrifuge at 201 x g (rcf) for 5 minutes at room temperature. 10 ml represents minimum recommended dilution. 20 ml represents maximum recommended dilution. 3. Resuspend the cell pellet in 5 ml pre-warmed culture medium and count using a haemocytometer (Click here to view haemocytometer protocol) or alternative cell counting method. Based on cell count, seed cells in an appropriate cell culture flask at a density of 2×10^4 cells/cm². This should allow for confluency within 48 hours. Seeding density is given as a guide only and should be scaled to align with individual lab schedules. 4. Incubate the culture at 37°C incubator with 5% CO₂. Cultures should be monitored daily. <p>Subculture guidelines:</p> <p>All seeding densities should be based on cell counts gained by established methods. A guide seeding density of 2×10^4 cells/cm² is recommended for confluency (80-90% confluence) within 48 hours.</p> <p>A partial media change 24 hours prior to subculture may be helpful to encourage growth, if required.</p> <p>Cells should be passaged when they have achieved 80-90% confluence.</p>

[Click here to view the Mammalian cell tissue culture protocol](#)

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Properties

Number of cells	1 x 10 ⁶ cells/vial, 1 mL
Viability	~90%
Adherent /Suspension	Suspension
Tissue	Blood
Cell type	erythroleukemia
Disease	Chronic Myelogenous Leukemia
Gender	Female
Mycoplasma free	Yes
Storage instructions	Shipped on Dry Ice. Store in liquid nitrogen.
Storage buffer	Constituents: 8.7% DMSO, 2% Cellulose, methyl ether

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-polyposis syndrome 1 (BTSP1). 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:114550]. 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.

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