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TIGAR Rabbit pAb

Catalog # AP97803

Product Information

Application	WB, IHC-P, IHC-F, IF, E
Primary Accession	Q9NQ88
Other Accession	Q9NQ88
Predicted	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	30063
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human TIGAR
Epitope Specificity	161-270/270
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the phosphoglycerate mutase family.
SUBUNIT	Monomer.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	This gene is regulated as part of the p53 tumor suppressor pathway and encodes a protein with sequence similarity to the bisphosphate domain of the glycolytic enzyme that degrades fructose-2,6-bisphosphate. The protein functions by blocking glycolysis and directing the pathway into the pentose phosphate shunt. Expression of this protein also protects cells from DNA damaging reactive oxygen species and provides some protection from DNA damage-induced apoptosis. The 12p13.32 region that includes this gene is paralogous to the 11q13.3 region. [provided by RefSeq, Jul 2008]

Additional Information

Gene ID	57103
Other Names	C12orf5; FR2BP; TIGAR_HUMAN; TIGAR; TP53-induced glycolysis and apoptosis regulator; TP53-induced glycolysis regulatory phosphatase; 3.1.3.46; TP53 induced glycolysis regulatory phosphatase; chromosome 12 open reading frame 5
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500,ELISA=1:5000-10000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	TIGAR {ECO:0000303|PubMed:16839880}
Synonyms	C12orf5
Function	Fructose-bisphosphatase hydrolyzing fructose-2,6-bisphosphate as well as fructose-1,6-bisphosphate (PubMed:19015259). Acts as a negative regulator of glycolysis by lowering intracellular levels of fructose-2,6-bisphosphate in a p53/TP53-dependent manner, resulting in the pentose phosphate pathway (PPP) activation and NADPH production (PubMed:16839880, PubMed:22887998). Contributes to the generation of reduced glutathione to cause a decrease in intracellular reactive oxygen species (ROS) content, correlating with its ability to protect cells from oxidative or metabolic stress-induced cell death (PubMed:16839880, PubMed:19713938, PubMed:22887998, PubMed:23726973, PubMed:23817040). Plays a role in promoting protection against cell death during hypoxia by decreasing mitochondria ROS levels in a HK2- dependent manner through a mechanism that is independent of its fructose-bisphosphatase activity (PubMed:23185017). In response to cardiac damage stress, mediates p53-induced inhibition of myocyte mitophagy through ROS levels reduction and the subsequent inactivation of BNIP3. Reduced mitophagy results in an enhanced apoptotic myocyte cell death, and exacerbates cardiac damage (By similarity). Plays a role in adult intestinal regeneration; contributes to the growth, proliferation and survival of intestinal crypts following tissue ablation (PubMed:23726973). Plays a neuroprotective role against ischemic brain damage by enhancing PPP flux and preserving mitochondria functions (By similarity). Protects glioma cells from hypoxia- and ROS- induced cell death by inhibiting glycolysis and activating mitochondrial energy metabolism and oxygen consumption in a TKTL1- dependent and p53/TP53-independent manner (PubMed:22887998). Plays a role in cancer cell survival by promoting DNA repair through activating PPP flux in a CDK5-ATM-dependent signaling pathway during hypoxia and/or genome stress-induced DNA damage responses (PubMed:25928429). Involved in intestinal tumor progression (PubMed:23726973).
Cellular Location	Cytoplasm. Nucleus Mitochondrion. Note=Translocated to the mitochondria during hypoxia in a HIF1A-dependent manner (PubMed:23185017). Colocalizes with HK2 in the mitochondria during hypoxia (PubMed:23185017). Translocated to the nucleus during hypoxia and/or genome stress-induced DNA damage responses in cancer cells (PubMed:25928429). Translocation to the mitochondria is enhanced in ischemic cortex after reperfusion and/or during oxygen and glucose deprivation (OGD)/reoxygenation insult in primary neurons (By similarity). {ECO:0000250|UniProtKB:Q8BZA9, ECO:0000269|PubMed:23185017, ECO:0000269|PubMed:25928429}
Tissue Location	Expressed in the brain (PubMed:22887998). Expressed in breast tumors (PubMed:21820150). Expressed in glioblastomas (PubMed:22887998).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.