10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

TIGAR Antibody

Catalog # ASC10505

Product Information

Application	WB, E, IHC-P
Primary Accession	Q9NQ88
Other Accession	NP_065108, 9966849
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	30063
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	TIGAR antibody can be used for detection of TIGAR by Western blot at 0.5 - 2 g/mL. Antibody can also be used for immunohistochemistry starting at 2.5 g/mL.

Additional Information

Gene ID	57103
Other Names	TIGAR Antibody: FR2BP, TIGAR, C12orf5, Fructose-2, 6-bisphosphatase TIGAR, TP53-induced glycolysis and apoptosis regulator, chromosome 12 open reading frame 5
Target/Specificity	C12orf5;
Reconstitution & Storage	TIGAR antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	TIGAR Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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).

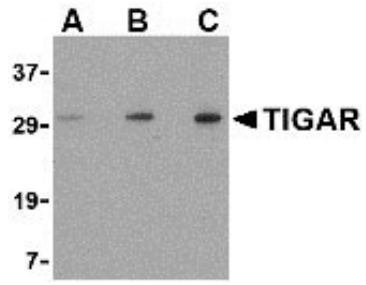
Background

TIGAR Antibody: The p53 tumor-suppressor gene integrates numerous signals that control cell life and death; loss of its functions contributes to the development of most cancers. Recent studies have demonstrated the ability of p53 to regulate the expression of several proteins involved in glycolysis and oxidative phosphorylation, such as TIGAR, SCO2, and phosphoglycerate mutase. TIGAR is a recently discovered protein that functions to regulate glycolysis and protect cells against oxidative stress. TIGAR is similar in structure to proteins in the phosphoglycerate mutase family, most notably 6-phosphofructo-2-kinase, suggesting TIGAR may function as a fructose bisphosphatase. Expression of TIGAR in transfected cells correlated with an inhibition of glycolysis and decreased levels of reactive oxygen species and p53-induced apoptosis, indicating that TIGAR may act to modulate the apoptotic response to p53, thereby allowing cells to survive mild or transient stresses.

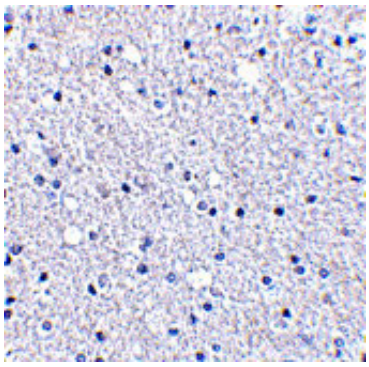
References

Guimaraes DP and Hainaut P. TP53: a key gene in human cancer. Biochimie2002; 84:83-93.
Corcoran CA, Huang Y, and Sheikh MS. The regulation of energy generating pathways by p53. Cancer Biol. Ther.2006; 5:1610-3.
Bensaad K, Tsuruta A, Selak MA, et al. TIGAR, a p53-inducible regulator of glycolysis and apoptosis. Cell2006; 126:107-20.

Images



Western blot analysis of TIGAR in EL4 cell lysate with TIGAR antibody at (A) 0.5, (B) 1 and (C) 2 µg/mL.



Immunohistochemistry of TIGAR in human brain tissue with TIGAR antibody at 2.5 µg/mL.

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