

**TIGAR Antibody (Internal)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS11394****Specification****TIGAR Antibody (Internal) - Product Information**

Application	WB, IHC
Primary Accession	<a href="#">O9NQ88</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	30kDa KDa

**TIGAR Antibody (Internal) - Additional Information****Gene ID** 57103**Other Names**

Fructose-2, 6-bisphosphatase TIGAR, 3.1.3.46, TP53-induced glycolysis and apoptosis regulator, TIGAR, C12orf5

**Target/Specificity**

18 amino acid peptide from near the center of human TIGAR.

**Reconstitution & Storage**

Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles. Store undiluted.

**Precautions**

TIGAR Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

**TIGAR Antibody (Internal) - 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:<a href="http://www.uniprot.org/citations/19015259" target="\_blank">19015259</a>). 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:<a href="http://www.uniprot.org/citations/16839880" target="\_blank">16839880</a>, PubMed:<a href="http://www.uniprot.org/citations/22887998" target="\_blank">22887998</a>). 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:<a href="http://www.uniprot.org/citations/16839880" target="\_blank">16839880</a>).

target="\_blank">16839880</a>, PubMed:<a href="http://www.uniprot.org/citations/19713938" target="\_blank">19713938</a>, PubMed:<a href="http://www.uniprot.org/citations/23726973" target="\_blank">23726973</a>, PubMed:<a href="http://www.uniprot.org/citations/22887998" target="\_blank">22887998</a>, PubMed:<a href="http://www.uniprot.org/citations/23817040" target="\_blank">23817040</a>). 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:<a href="http://www.uniprot.org/citations/23185017" target="\_blank">23185017</a>). 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:<a href="http://www.uniprot.org/citations/23726973" target="\_blank">23726973</a>). 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:<a href="http://www.uniprot.org/citations/22887998" target="\_blank">22887998</a>). 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:<a href="http://www.uniprot.org/citations/25928429" target="\_blank">25928429</a>). Involved in intestinal tumor progression (PubMed:<a href="http://www.uniprot.org/citations/23726973" target="\_blank">23726973</a>).

#### 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).

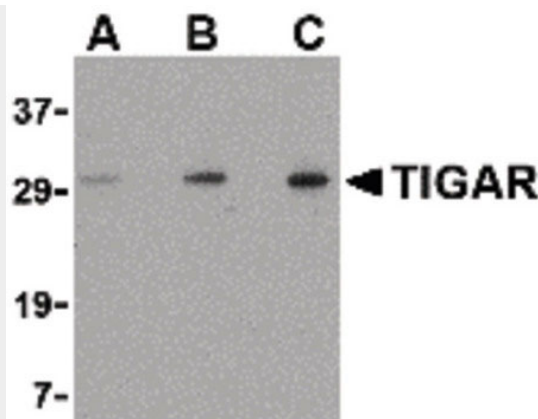
#### TIGAR Antibody (Internal) - Protocols

Provided below are standard protocols that you may find useful for product applications.

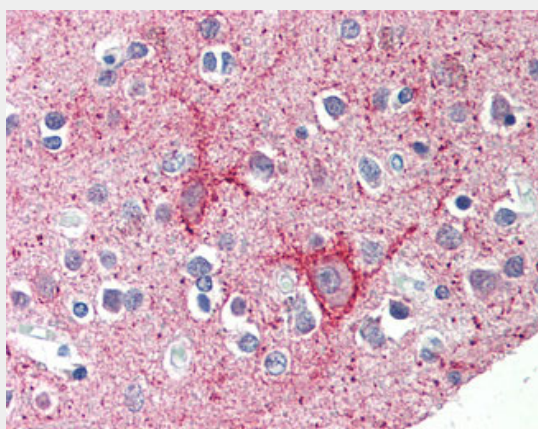
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### TIGAR Antibody (Internal) - Images





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



Anti-C12orf5 / TIGAR antibody IHC of human brain, cortex.

#### **TIGAR Antibody (Internal) - Background**

Fructose-bisphosphatase hydrolyzing fructose-2,6- bisphosphate as well as fructose-1,6-bisphosphate. Inhibits glycolysis by reducing cellular levels of fructose-2,6- bisphosphate. May protect cells against reactive oxygen species and against apoptosis induced by tp53.

#### **TIGAR Antibody (Internal) - References**

White K.E.,et al.Nat. Genet. 26:345-348(2000).  
Cheng J.,et al.Submitted (SEP-2003) to the EMBL/GenBank/DDBJ databases.  
Ota T.,et al.Nat. Genet. 36:40-45(2004).  
Mural R.J.,et al.Submitted (SEP-2005) to the EMBL/GenBank/DDBJ databases.  
Bensaad K.,et al.Cell 126:107-120(2006).