

**TIGAR Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP19202a****Specification**

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**TIGAR Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [Q9NQ88](#)**TIGAR Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 57103**Other Names**

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

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**TIGAR Antibody (N-term) Blocking Peptide - 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>, PubMed:<a href="http://www.uniprot.org/citations/19713938" target="\_blank">19713938</a>, PubMed:<a href="http://www.uniprot.org/citations/22887998" target="\_blank">22887998</a>, PubMed:<a href="http://www.uniprot.org/citations/23726973" target="\_blank">23726973</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 (N-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

## TIGAR Antibody (N-term) Blocking Peptide - Images

## TIGAR Antibody (N-term) Blocking Peptide - Background

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].

## TIGAR Antibody (N-term) Blocking Peptide - References

Bensaad, K., et al. EMBO J. 28(19):3015-3026(2009) Trevino, L.R., et al. Nat. Genet. 41(9):1001-1005(2009) Lopez-Guerra, M., et al. Haematologica 93(12):1843-1851(2008) Matsuoka,

S., et al. Science 316(5828):1160-1166(2007) Bensaad, K., et al. Cell 126(1):107-120(2006)