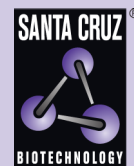


# TIGAR (E-2): sc-166290



The Power to Question

## BACKGROUND

TIGAR (TP53 (tumor protein 53)-induced glycolysis and apoptosis regulator), also known as C12orf5, is a 270 amino acid protein induced by the p53 tumor suppressor pathway that functions to protect against oxidative stress. TIGAR shares sequence similarity with the bisphosphate domain of the fructose-2,6-bisphosphate degrading enzyme (fructose bisphosphatase or FBPase) of the glycolysis pathway and can thus lower the intracellular levels of fructose-2,6-bisphosphate. TIGAR specifically functions to block glycolysis, leading the pathway to the pentose phosphate shunt and decreasing the intracellular concentration of reactive oxygen species. This suggests a role for TIGAR in protecting cells from reactive oxygen species that can be DNA damaging and lead to apoptosis.

## CHROMOSOMAL LOCATION

Genetic locus: TIGAR (human) mapping to 12p13.32; Tigar (mouse) mapping to 6 F3.

## SOURCE

TIGAR (E-2) is a mouse monoclonal antibody raised against amino acids 61-269 mapping at the C-terminus of TIGAR of mouse origin.

## PRODUCT

Each vial contains 200 µg IgG<sub>2b</sub> kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

TIGAR (E-2) is available conjugated to agarose (sc-166290 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-166290 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-166290 PE), fluorescein (sc-166290 FITC), Alexa Fluor® 488 (sc-166290 AF488), Alexa Fluor® 546 (sc-166290 AF546), Alexa Fluor® 594 (sc-166290 AF594) or Alexa Fluor® 647 (sc-166290 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-166290 AF680) or Alexa Fluor® 790 (sc-166290 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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## APPLICATIONS

TIGAR (E-2) is recommended for detection of TIGAR of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for TIGAR siRNA (h): sc-76662, TIGAR siRNA (m): sc-76663, TIGAR shRNA Plasmid (h): sc-76662-SH, TIGAR shRNA Plasmid (m): sc-76663-SH, TIGAR shRNA (h) Lentiviral Particles: sc-76662-V and TIGAR shRNA (m) Lentiviral Particles: sc-76663-V.

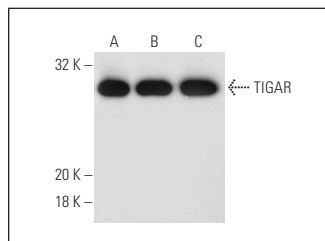
Molecular Weight of TIGAR: 30 kDa.

Positive Controls: Saos-2 cell lysate: sc-2235, Hep G2 cell lysate: sc-2227 or Jurkat whole cell lysate: sc-2204.

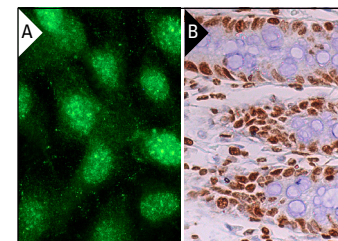
## STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## DATA



TIGAR (E-2): sc-166290. Western blot analysis of TIGAR expression in Jurkat (A), Hep G2 (B) and Saos-2 (C) whole cell lysates.



TIGAR (E-2): sc-166290. Immunofluorescence staining of methanol-fixed HeLa cells showing nuclear and cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human rectum tissue showing nuclear staining of glandular cells (B).

## SELECT PRODUCT CITATIONS

- Dai, C., et al. 2013. Negative regulation of the acetyltransferase TIP60-p53 interplay by UHRF1 (ubiquitin-like with PHD and RING finger domains 1). *J. Biol. Chem.* 288: 19581-19592.
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- Feng, J., et al. 2018. TP53-induced glycolysis and apoptosis regulator is indispensable for mitochondria quality control and degradation following damage. *Oncol. Lett.* 15: 155-160.
- Tang, Z. and He, Z. 2019. TIGAR promotes growth, survival and metastasis through oxidation resistance and Akt activation in glioblastoma. *Oncol. Lett.* 18: 2509-2517.
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- Chen, D., et al. 2021. iPLA2β-mediated lipid detoxification controls p53-driven ferroptosis independent of GPX4. *Nat. Commun.* 12: 3644.
- Yin, K., et al. 2021. Mitophagy protein PINK1 suppresses colon tumor growth by metabolic reprogramming via p53 activation and reducing acetyl-CoA production. *Cell Death Differ.* 28: 2421-2435.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.