

GAPDH Antibody

Rabbit mAb Catalog # AP90018

Product Information

Application Primary Accession Reactivity Clonality Other Names	WB, IHC, IF, FC, ICC, IP, IHF <u>P04406</u> Rat, Human, Mouse, Zebrafish, Monkey, Chicken Monoclonal aging-associated gene 9 protein; G3P; G3PD; GAPD; GAPDH; glyceraldehyde 3-phosphate dehydrogenase; Glyceraldehyde-3-phosphate dehydrogenase; MGC88685;
Isotype	Rabbit IgG
Host	Rabbit
Calculated MW	36053

Additional Information

Dilution Purification Immunogen Description	WB 1:5000~1:50000 IHC 1:100~1:500 ICC/IF 1:100~1:250 IP 1:50 FC 1:50 Affinity-chromatography A synthesized peptide derived from human GAPDH Glyceraldebyde 3 phosphate debydrogenase (GAPDH) is well known as one	
	the key enzymes involved in glycolysis. GAPDH is constitutively abundant expressed in almost cell types at high levels, therefore antibodies against GAPDH are useful as loading controls for Western Blotting. Some pathology factors, such as hypoxia and diabetes, increased or decreased GAPDH	
Storage Condition and Buffer	expression in certain cell types. Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.	

Protein Information

Name	GAPDH {ECO:0000303 PubMed:2987855, ECO:0000312 HGNC:HGNC:4141}
Function	Has both glyceraldehyde-3-phosphate dehydrogenase and nitrosylase activities, thereby playing a role in glycolysis and nuclear functions, respectively (PubMed: <u>11724794</u> , PubMed: <u>3170585</u>). Glyceraldehyde-3-phosphate dehydrogenase is a key enzyme in glycolysis that catalyzes the first step of the pathway by converting D- glyceraldehyde 3-phosphate (G3P) into 3-phospho-D-glyceroyl phosphate (PubMed: <u>11724794</u> , PubMed: <u>3170585</u>). Modulates the organization and assembly of the cytoskeleton (By similarity). Facilitates the CHP1- dependent microtubule and membrane associations through its ability to stimulate the binding of CHP1 to microtubules (By similarity). Component of the GAIT (gamma interferon-activated inhibitor of translation) complex which mediates

interferon-gamma-induced transcript-selective translation inhibition in inflammation processes (PubMed:23071094). Upon interferon-gamma treatment assembles into the GAIT complex which binds to stem loop-containing GAIT elements in the 3'-UTR of diverse inflammatory mRNAs (such as ceruplasmin) and suppresses their translation (PubMed:23071094). Also plays a role in innate immunity by promoting TNF-induced NF-kappa-B activation and type I interferon production, via interaction with TRAF2 and TRAF3, respectively (PubMed:23332158, PubMed:27387501). Participates in nuclear events including transcription, RNA transport, DNA replication and apoptosis (By similarity). Nuclear functions are probably due to the nitrosylase activity that mediates cysteine S-nitrosylation of nuclear target proteins such as SIRT1, HDAC2 and PRKDC (By similarity). Cytoplasm, cytosol. Nucleus {ECO:0000250|UniProtKB:P04797}. Cytoplasm, **Cellular Location** perinuclear region. Membrane Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:P04797} Note=Translocates to the nucleus following S-nitrosylation and interaction with SIAH1, which contains a nuclear localization signal (By similarity). Postnuclear and Perinuclear regions (PubMed:12829261) {ECO:0000250 | UniProtKB:P04797, ECO:0000269 | PubMed:12829261 }

Images

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Western blot analysis of GAPDH expression in (1) Hela cell lysate; (2)Jurkat cell lysate; (3)Mouse kidney lysate; (4) Mouse spleen lysate; (5) RAW 264.7 cell lysate; (6) Rat brain lysate with GAPDH Antibody.



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All lanes use GAPDH Antibody at 1:50000 dilution for 1 hour at room temperature.

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LncRNA AK023391 promotes tumorigenesis and invasion of gastric cancer through activation of the PI3K/Akt signaling pathway. -Journal of Experimental & Clinical Cancer Research

Image not found : 202311/AP90018-wb5.jpg	Arsenic circumvents the gefitinib resistance by binding to P62 and mediating autophagic degradation of EGFR in non-small cell lung cancerCell Death and Disease
Image not found : 202311/AP90018-wb6.jpg	Prostaglandin E1 Inhibited Diabetes-Induced Phenotypic Switching of Vascular Smooth Muscle Cells Through Activating AutophagyCellular Physiology and Biochemistry

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