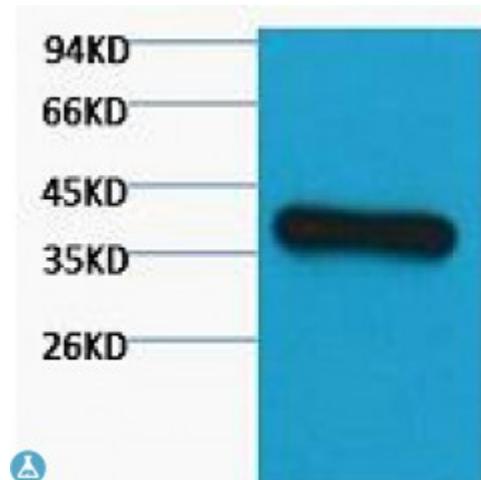


## Anti-GAPDH antibody



<b>Description</b>	Mouse monoclonal to GAPDH.
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<b>Model</b>	STJ96942
<b>Host</b>	Mouse
<b>Reactivity</b>	Bovine, Canine, Hamster, Human, Mouse, Rabbit, Rat, Sheep, Simian, Swine
<b>Applications</b>	WB
<b>Immunogen</b>	Synthetic Peptide
<b>Gene ID</b>	<a href="#">2597</a>
<b>Gene Symbol</b>	<a href="#">GAPDH</a>
<b>Dilution range</b>	WB 1:2000-5000
<b>Specificity</b>	The antibody detects endogenous GAPDH protein.
<b>Purification</b>	The antibody was affinity-purified from mouse ascites by affinity-chromatography using specific immunogen.
<b>Clone ID</b>	2B8
<b>Note</b>	For Research Use Only (RUO).
<b>Protein Name</b>	Glyceraldehyde-3-phosphate dehydrogenase GAPDH Peptidyl-cysteine S-nitrosylase GAPDH
<b>Clonality</b>	Monoclonal
<b>Conjugation</b>	Unconjugated
<b>Isotype</b>	IgG1

<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Storage Instruction</b>	Store at -20°C, and avoid repeat freeze-thaw cycles.
<b>Database Links</b>	<a href="#">HGNC:4141</a> <a href="#">OMIM:138400</a>
<b>Alternative Names</b>	Glyceraldehyde-3-phosphate dehydrogenase GAPDH Peptidyl-cysteine S-nitrosylase GAPDH
<b>Function</b>	Has both glyceraldehyde-3-phosphate dehydrogenase and nitrosylase activities, thereby playing a role in glycolysis and nuclear functions, respectively. Participates in nuclear events including transcription, RNA transport, DNA replication and apoptosis. Nuclear functions are probably due to the nitrosylase activity that mediates cysteine S-nitrosylation of nuclear target proteins such as SIRT1, HDAC2 and PRKDC. Modulates the organization and assembly of the cytoskeleton. Facilitates the CHP1-dependent microtubule and membrane associations through its ability to stimulate the binding of CHP1 to microtubules. 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. Component of the GAIT (gamma interferon-activated inhibitor of translation) complex which mediates interferon-gamma-induced transcript-selective translation inhibition in inflammation processes. 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 ceruloplasmin) and suppresses their translation.
<b>Sequence and Domain Family</b>	The [IL]-x-C-x-x-[DE] motif is a proposed target motif for cysteine S-nitrosylation mediated by the iNOS-S100A8/A9 transnitrosylase complex.
<b>Cellular Localization</b>	Cytoplasm, cytosol Nucleus Cytoplasm, perinuclear region Membrane Cytoplasm, cytoskeleton. Translocates to the nucleus following S-nitrosylation and interaction with SIAH1, which contains a nuclear localization signal. Postnuclear and Perinuclear regions.
<b>Post-translational Modifications</b>	S-nitrosylation of Cys-152 leads to interaction with SIAH1, followed by translocation to the nucleus. S-nitrosylation of Cys-247 is induced by interferon-gamma and LDL(ox) implicating the iNOS-S100A8/9 transnitrosylase complex and seems to prevent interaction with phosphorylated RPL13A and to interfere with GAIT complex activity. ISGylated. Sulphydratation at Cys-152 increases catalytic activity. Oxidative stress can promote the formation of high molecular weight disulfide-linked GAPDH aggregates, through a process called nucleocytoplasmic coagulation. Such aggregates can be observed in vivo in the affected tissues of patients with Alzheimer disease or alcoholic liver cirrhosis, or in cell cultures during necrosis. Oxidation at Met-46 may play a pivotal role in the formation of these insoluble structures. This modification has been detected in vitro following treatment with free radical donor (+/-)-(E)-4-ethyl-2-[(E)-hydroxyimino]-5-nitro-3-hexenamide. It has been proposed to destabilize nearby residues, increasing the likelihood of secondary oxidative damages, including oxidation of Tyr-45 and Met-105. This cascade of oxidations may augment GAPDH misfolding, leading to intermolecular disulfide cross-linking and aggregation.