

Anti-p62 (Thr269, Ser272) Antibody

Our Anti-p62 (Thr269, Ser272) rabbit polyclonal phosphospecific primary antibody from PhosphoSolutio
Catalog # AN1506

Specification**Anti-p62 (Thr269, Ser272) Antibody - Product Information**

Primary Accession	Q13501
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	47687

Anti-p62 (Thr269, Ser272) Antibody - Additional Information

Gene ID **8878**

Other Names

A170 antibody, DMRV antibody, EBI 3 associated protein of 60 kDa antibody, EBI 3 associated protein p60 antibody, EBI3 associated protein of 60 kDa antibody, EBI3 associated protein p60 antibody, EBI3-associated protein of 60 kDa antibody, EBIAP antibody, FTDALS3 antibody, MGC127197 antibody, ORCA antibody, OSF-6 antibody, Osi antibody, OSIL antibody, Oxidative stress induced like antibody, p60 antibody, p62 antibody, p62B antibody, Paget disease of bone 3 antibody, PDB 3 antibody, PDB3 antibody, Phosphotyrosine independent ligand for the Lck SH2 domain of 62 kDa antibody, Phosphotyrosine independent ligand for the Lck SH2 domain p62 antibody, Phosphotyrosine-independent ligand for the Lck SH2 domain of 62 kDa antibody, PKC-zeta-interacting protein antibody, Protein kinase C-zeta-interacting protein antibody, Sequestosome 1 antibody, Sequestosome-1 antibody, SQSTM 1 antibody, SQSTM_HUMAN antibody, Sqstm1 antibody, STAP antibody, STONE14 antibody, Ubiquitin binding protein p62 antibody, Ubiquitin-binding protein p62 antibody, ZIP 3 antibody, ZIP antibody, ZIP3 antibody

Target/Specificity

The protein scaffold and signaling regulator p62 (also known as sequestosome1 (SQSTM1)) is important in critical cellular functions, including bone homeostasis, obesity, and cancer, because of its interactions with various signaling intermediaries. p62 is overexpressed in many human cancers and is induced during cell transformation. cdk1 phosphorylates p62 in vitro and in vivo at Thr-269 and Ser-272, which is necessary for the maintenance of appropriate cyclin B1 levels and the levels of cdk1 activity necessary to allow cells to properly enter and exit mitosis (Moscat et al., 2011). The lack of cdk1-mediated phosphorylation of p62 leads to a faster exit from mitosis, translating into enhanced cell proliferation and tumorigenesis in response to Ras-induced transformation (Moscat et al., 2011).

Format

Antigen Affinity Purified from Pooled Serum

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Anti-p62 (Thr269, Ser272) Antibody is for research use only and not for use in diagnostic or

therapeutic procedures.

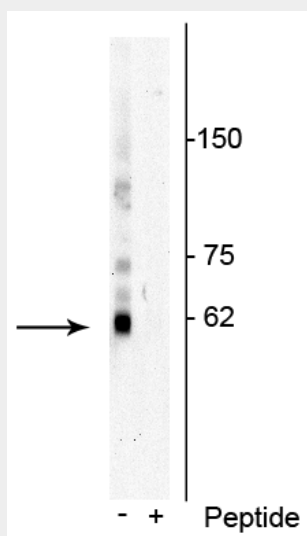
Shipping
Blue Ice

Anti-p62 (Thr269, Ser272) Antibody - Protocols

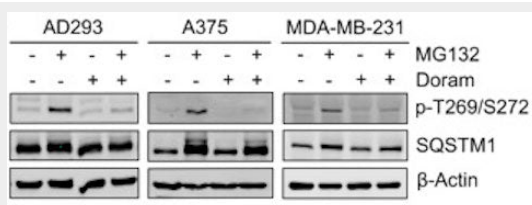
Provided below are standard protocols that you may find useful for product applications.

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

Anti-p62 (Thr269, Ser272) Antibody - Images



Western blot of Jurkat cell lysate showing specific immunolabeling of the ~62 kDa p62 phosphorylated at Thr269/Ser272 in the first lane (-). Phosphospecificity is shown in the second lane (+) where immunolabeling is blocked by preadsorption of the phosphopeptide used as antigen, but not by the corresponding non-phosphopeptide (not shown).



AD293, A375 and MDA-MB-231 cells were treated with DMSO (control), or MG132 (2 μ M), and Doramapimod (50 μ M), alone or in combination for 14 h. The whole-cell lysates were subjected to western blot analysis with indicated antibodies. Image from publication CC-BY-4.0. PMID: 35840557

Anti-p62 (Thr269, Ser272) Antibody - Background

The protein scaffold and signaling regulator p62 (also known as sequestosome1 (SQSTM1)) is important in critical cellular functions, including bone homeostasis, obesity, and cancer, because of its interactions with various signaling intermediaries. p62 is overexpressed in many human cancers and is induced during cell transformation. cdk1 phosphorylates p62 in vitro and in vivo at Thr-269 and Ser-272, which is necessary for the maintenance of appropriate cyclin B1 levels and the levels of cdk1 activity necessary to allow cells to properly enter and exit mitosis (Moscat et al., 2011). The lack of cdk1-mediated phosphorylation of p62 leads to a faster exit from mitosis, translating into enhanced cell proliferation and tumorigenesis in response to Ras-induced transformation (Moscat et al., 2011).