

**HSP90AB1 Antibody**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO1577a**

**Specification**

**HSP90AB1 Antibody - Product Information**

Application	<b>E, WB, IHC, IF, FC</b>
Primary Accession	<a href="#">P08238</a>
Reactivity	<b>Human, Mouse, Rat, Monkey</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>IgG1</b>
Calculated MW	<b>84kDa KDa</b>

**Description**

HSP90 proteins are highly conserved molecular chaperones that have key roles in signal transduction, protein folding, protein degradation, and morphologic evolution. HSP90 proteins normally associate with other cochaperones and play important roles in folding newly synthesized proteins or stabilizing and refolding denatured proteins after stress. There are 2 major cytosolic HSP90 proteins, HSP90AA1 (MIM 140571), an inducible form, and HSP90AB1, a constitutive form.

**Immunogen**

Purified recombinant fragment of human HSP90AB1 expressed in E. Coli. <br />

**Formulation**

Ascitic fluid containing 0.03% sodium azide.

**HSP90AB1 Antibody - Additional Information**

**Gene ID** 3326

**Other Names**

Heat shock protein HSP 90-beta, HSP 90, Heat shock 84 kDa, HSP 84, HSP84, HSP90AB1, HSP90B, HSPC2, HSPCB

**Dilution**

E~~1/10000  
WB~~1/500 - 1/2000  
IHC~~1/500 - 1/2000  
IF~~1/200 - 1/1000  
FC~~1/200 - 1/400

**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**

HSP90AB1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## HSP90AB1 Antibody - Protein Information

Name HSP90AB1 ([HGNC:5258](#))

### Function

Molecular chaperone that promotes the maturation, structural maintenance and proper regulation of specific target proteins involved for instance in cell cycle control and signal transduction. Undergoes a functional cycle linked to its ATPase activity. This cycle probably induces conformational changes in the client proteins, thereby causing their activation. Interacts dynamically with various co-chaperones that modulate its substrate recognition, ATPase cycle and chaperone function (PubMed:[16478993](http://www.uniprot.org/citations/16478993), PubMed:[19696785](http://www.uniprot.org/citations/19696785)). Engages with a range of client protein classes via its interaction with various co-chaperone proteins or complexes, that act as adapters, simultaneously able to interact with the specific client and the central chaperone itself. Recruitment of ATP and co-chaperone followed by client protein forms a functional chaperone. After the completion of the chaperoning process, properly folded client protein and co-chaperone leave HSP90 in an ADP-bound partially open conformation and finally, ADP is released from HSP90 which acquires an open conformation for the next cycle (PubMed:[26991466](http://www.uniprot.org/citations/26991466), PubMed:[27295069](http://www.uniprot.org/citations/27295069)). Apart from its chaperone activity, it also plays a role in the regulation of the transcription machinery. HSP90 and its co-chaperones modulate transcription at least at three different levels. They first alter the steady-state levels of certain transcription factors in response to various physiological cues. Second, they modulate the activity of certain epigenetic modifiers, such as histone deacetylases or DNA methyl transferases, and thereby respond to the change in the environment. Third, they participate in the eviction of histones from the promoter region of certain genes and thereby turn on gene expression (PubMed:[25973397](http://www.uniprot.org/citations/25973397)). Antagonizes STUB1- mediated inhibition of TGF-beta signaling via inhibition of STUB1- mediated SMAD3 ubiquitination and degradation (PubMed:[24613385](http://www.uniprot.org/citations/24613385)). Promotes cell differentiation by chaperoning BIRC2 and thereby protecting from auto-ubiquitination and degradation by the proteasomal machinery (PubMed:[18239673](http://www.uniprot.org/citations/18239673)). Main chaperone involved in the phosphorylation/activation of the STAT1 by chaperoning both JAK2 and PRKCE under heat shock and in turn, activates its own transcription (PubMed:[20353823](http://www.uniprot.org/citations/20353823)). Involved in the translocation into ERGIC (endoplasmic reticulum-Golgi intermediate compartment) of leaderless cargos (lacking the secretion signal sequence) such as the interleukin 1/IL-1; the translocation process is mediated by the cargo receptor TMED10 (PubMed:[32272059](http://www.uniprot.org/citations/32272059)).

### Cellular Location

Cytoplasm. Melanosome Nucleus. Secreted. Cell membrane. Dynein axonemal particle {ECO:0000250|UniProtKB:Q6AZV1}. Cell surface. Note=Identified by mass spectrometry in melanosome fractions from stage I to stage IV (PubMed:17081065) Translocates with BIRC2 from the nucleus to the cytoplasm during differentiation (PubMed:18239673). Secreted when associated with TGFB1 processed form (LAP) (PubMed:20599762).

## HSP90AB1 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](#)

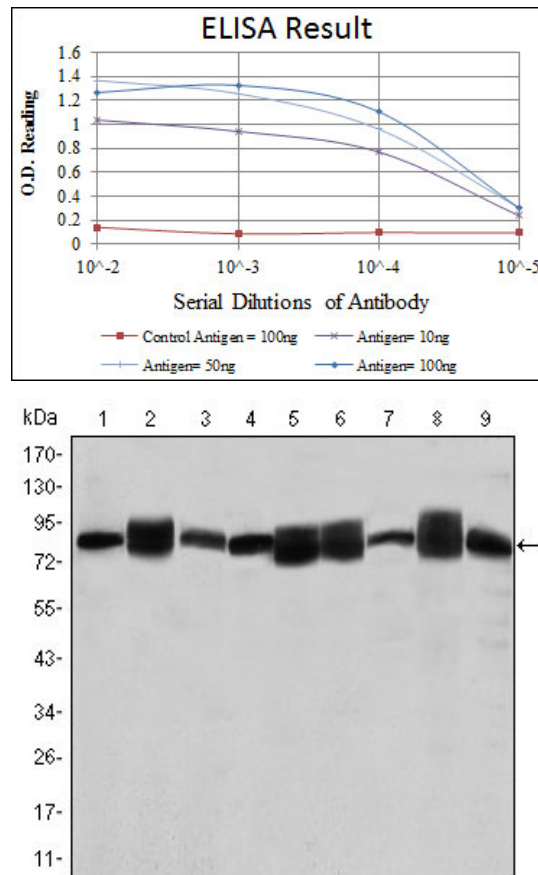


Figure 1: Western blot analysis using HSP90AB1 mouse mAb against Jurkat (1), A431 (2), HeLa (3), A549 (4), HEK293 (5), K562 (6), NIH/3T3 (7), PC-12 (8) and Cos7 (9) cell lysate.

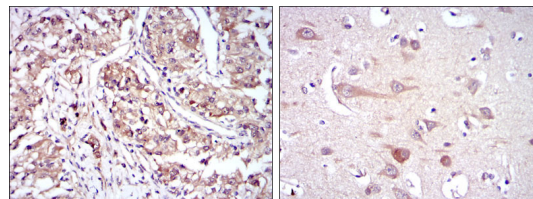


Figure 2: Immunohistochemical analysis of paraffin-embedded kidney cancer tissues (left) and brain tissues (right) using HSP90AB1 mouse mAb with DAB staining.

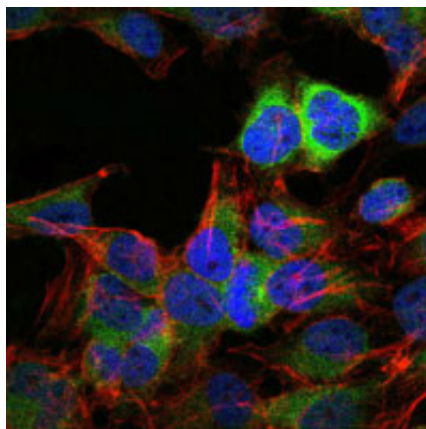


Figure 3: Immunofluorescence analysis of HeLa cells using HSP90AB1 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor-555 phalloidin.

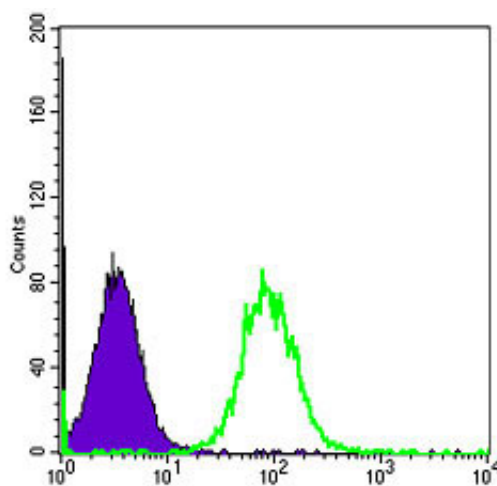


Figure 4: Flow cytometric analysis of HeLa cells using HSP90AB1 mouse mAb (green) and negative control (purple).

#### HSP90AB1 Antibody - References

1. J Biol Chem. 2009 Dec 18;284(51):35381-9.
2. Int J Biol Macromol. 2009 Oct 1;45(3):310-4.