HSP90 monoclonal antibody (AC88)

The Hsp90 family of heat shock proteins represents one of the most abundantly expressed and highly conserved families of cellular chaperones whose expression can be upregulated under conditions of cellular stress, and includes cytoplasmic (Hsp90-alpha/beta), ER (grp94), and mitochondrial (TRAP1) localized members. Structurally, Hsp90 is characterized by an N-terminal ATP-binding domain, a medial substrate-binding domain, and a C-terminal dimerization motif. Hsp90 dimers function in cooperation with cochaperones (e.g. Hsp40, Hsp70, Hop, p23) to stabilize a multitude of client protein substrates, including steroid hormone receptors, protein kinases, and transcription factors. The essential binding and hydrolysis of ATP by Hsp90 is inhibited by ansamycin drugs (e.g. geldanamycin, 17-AAG) which occupy the N-terminal Hsp90 nucleotide-binding pocket. Many Hsp90 client proteins such as erbB2/Her-2, c-raf, bcr-abl, p53, and hTERT, are members of well characterized oncogenic pathways, making Hsp90 inhibitors useful anticancer agents.

This antibody is covered by our Worry-Free Guarantee.

Citations: 113 View Online »

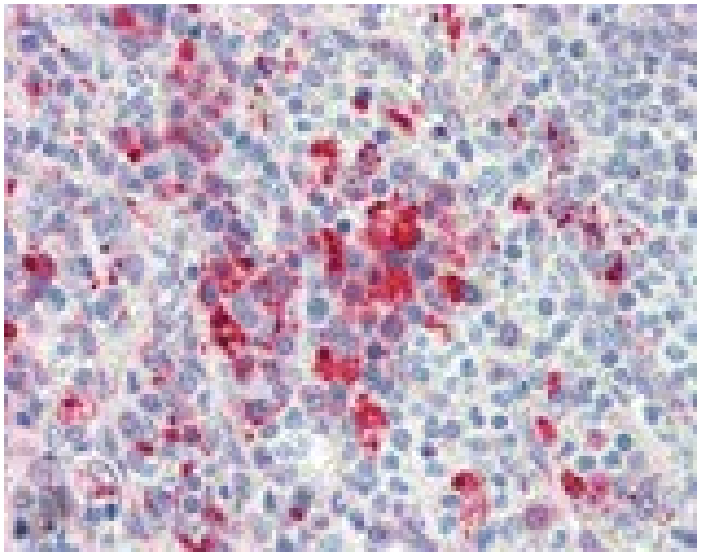
Ordering Information

Order Online »

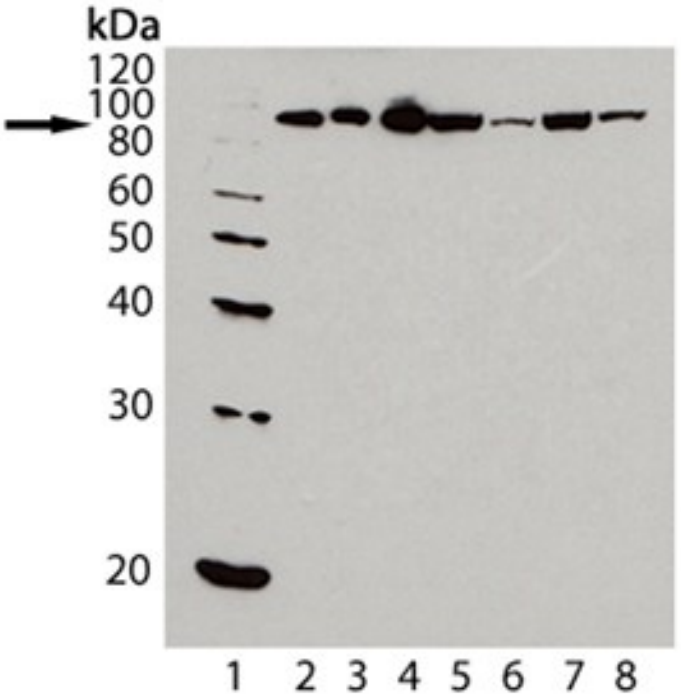
ADI-SPA-830-J	1mg
ADI-SPA-830-D	50µg
ADI-SPA-830-F	200µg

Manuals, SDS & CofA

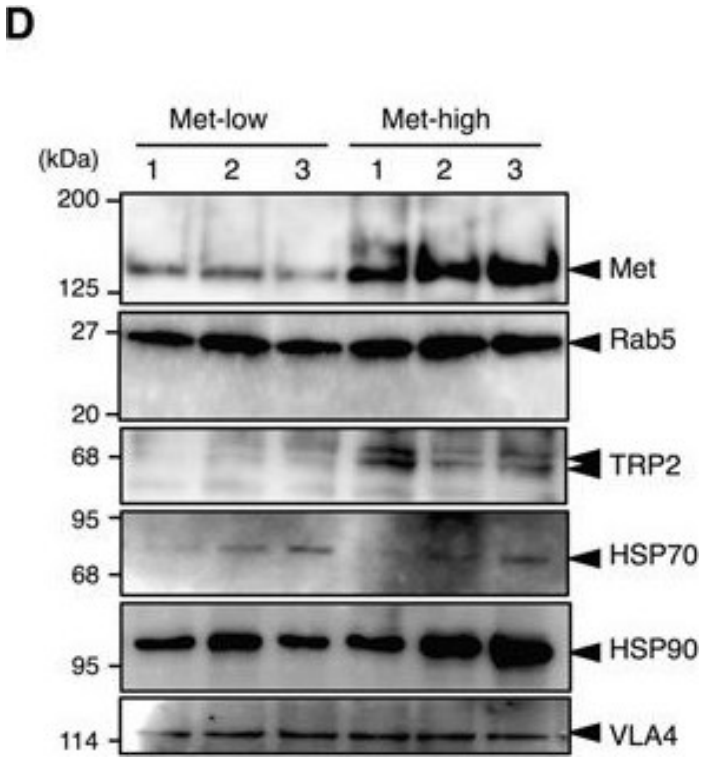
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Immunohistochemistry analysis of human spleen tissue stained with HSP90, mAb (AC88) at 10µg/ml.

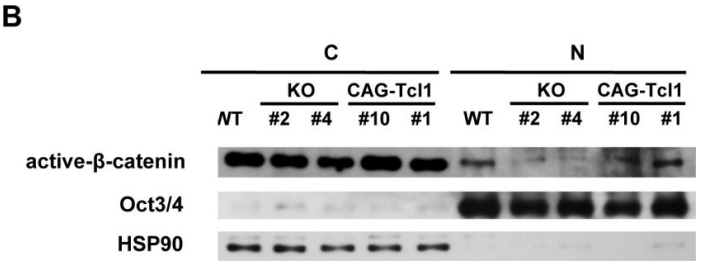


Western blot analysis of HSP90, mAb (AC88) (Prod. No. ADI-SPA-830): Lane 1: MW Marker; Lane 2: HSP90 (human), (native) (Prod. No. ADI-SPP-770); Lane 3: HSP90β, (human) (recombinant) (Prod. No. ADI-SPP-777); Lane 4: HSP90α (human), (recombinant) (Prod. No. ADI-SPP-776); Lane 5: HeLa (heat shocked) (Prod. No. ADI-LYC-HL101); Lane 6: 3T3 (heat shocked) (Prod. No. ADI-LYC-3T101; Lane 7: PC-12 (heat shocked) (Prod. No. ADI-LYC-PC101); Lane 8: CHO-K1 (heat shocked).



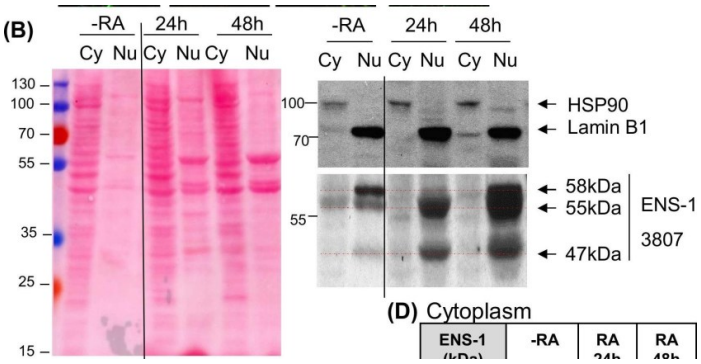
Lung metastasis of Met-low and Met-high cells injected into tail veinA. Appearance of metastases in lungs. Scale bars, 5 mm. B. The number of metastases in lungs. Met-low (n = 15), Met-high (n = 15), and Met-high cells expressing non-target shRNA (Non-target) (n = 13) or Met-targeting shRNA (sh-Met) (n = 14) were used. **p < 0.01 and *p < 0.05 by Tukey's test. In independently performed three sets of same experiments using a smaller number of animals (n = 2 – 6 for each experimental group), substantially the same results were obtained, and values were combined. C. Different pigmentation in metastatic tumors in the lung. Scale bars, 200 μm. D, Protein levels in exosomes derived from Met-low and Met-high cells. Protein levels were analyzed by Western blot and Rab5 was used as markers to indicate the amount of exosomes. Substantially same results were obtained in an independently performed experiment using independently prepared exosomes. E. The effect of Met-high cell-derived exosomes on metastasis of Met-low cells to the lung. Mice were injected with saline or Met-high cell-derived exosomes for three weeks. Met-low cells were injected two weeks after the treatment with exosomes and lung metastasis was analyzed three weeks post-inoculation. n = 6 in each experimental group. **p < 0.01 and *p < 0.05 by Tukey's test.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Different growth and metastatic phenotypes associated with a cell-intrinsic change of Met in metastatic melanoma. *Oncotarget* (2016)



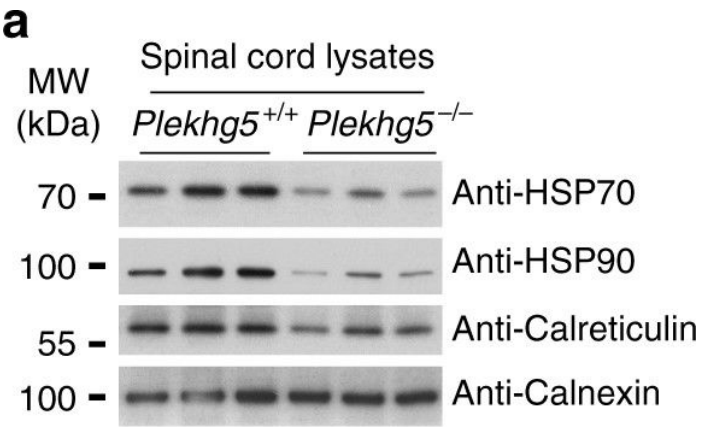
Analysis of Akt and Wnt/β-catenin signaling in Tcl1-deficient and -overexpressing ES cells.(A) Western blot analysis of GSK, Akt, and β-catenin in wild-type (WT), Tcl1−/− (KO) #2 and #4, Tcl1−/−(CAG-Tcl1) #1 and #3, and Tcl1−/−(CAG-EGFP) #5 ES cells. (B) Western blot analysis of active β-catenin, Oct3/4, and HSP90 in the cytoplasmic (C) and nuclear (N) fractions of wild-type (WT), Tcl1−/− (KO) #2 and #4, and Tcl1−/−(CAG-Tcl1) #10 and #1 ES cells. Tcl1−/−(CAG-Tcl1) #10 and #1 were derived from Tcl1−/− (KO) #2 and #4, respectively. Proper fractionation was confirmed by western blotting of Oct3/4 and HSP90, which localize to the nucleus and cytoplasm, respectively. Be8cause active β-catenin levels in the nuclear fractions were much lower than those in the cytoplasmic fractions, active β-catenin in the nuclear fractions was detected by approximately two-fold longer exposure compared with that in the cytoplasmic fractions. (C) TOPflash assay. P values of wild-type ES cells (WT) compared with Tcl1−/−(CAG-Tcl1) #1 and #3 ES cells were less than 0.01. P values of Tcl1−/− (KO) #4 and #5 ES cells compared with Tcl1−/−(CAG-Tcl1) #1 and #3 ES cells were less than 0.02.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Functional analysis of Tcl1 using Tcl1-deficient mouse embryonic stem cells. *PLoS One* (2013)



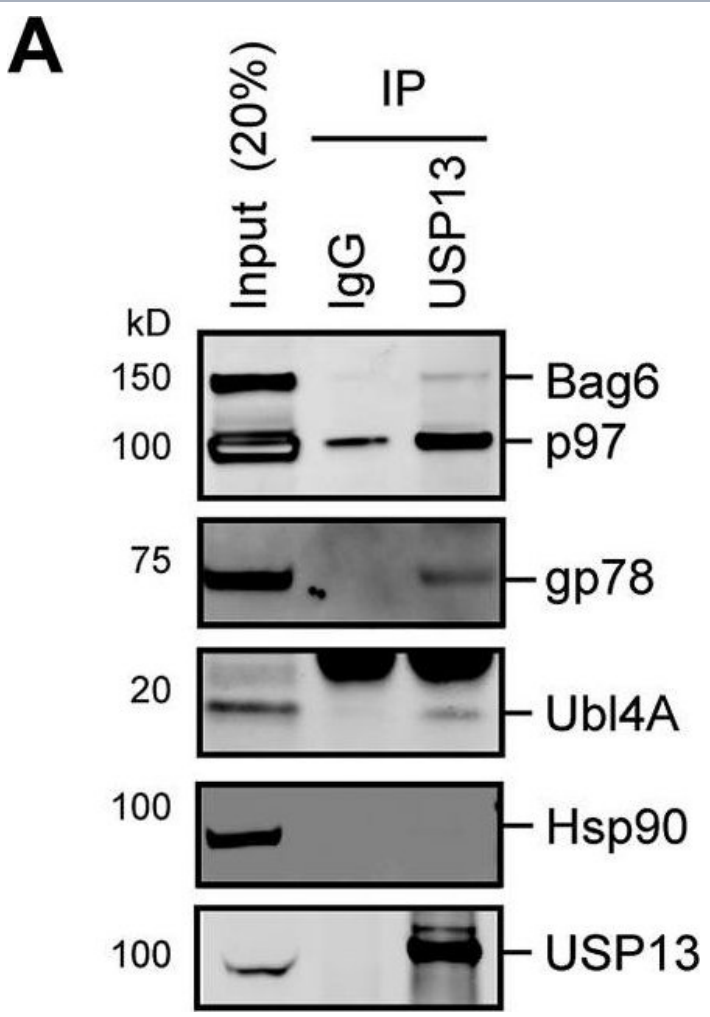
Subcellular distribution of ENS-1 in CES and during differentiation.(A) Immunostaining of ENS-1 (16h4 antibody) and HP1γ (Abcam antibody) in CES (-RA) and CES differentiated with retinoic acid for the indicated period. Nuclei are labeled with Draq5 and are in blue in merging panels. Image acquisition was optimized to observe the distribution of the proteins and does not reflect the real expression level. Scale bar 15 µm. (B) Western blot analysis of ENS-1 in the cytoplasmic (Cy) and in the nuclear (Nu) fractions of undifferentiated CES (-RA) or CES induced to differentiate with retinoic acid for 24h or 48h. Ponceau's red staining serves as a protein loading control between both fractions. In the nucleus soluble and insoluble components were separated and only the precipitating fraction that contains ENS-1 is shown. The volume corresponding to 6×106 cells was loaded for the N fraction and Lamin B1 was used as loading control. In the cytoplasm (Cy) ENS-1 was in the supernatant, 15 µg of proteins were loaded and HSP90 was used as loading control. The vertical line indicates missing lanes between the two presented parts of the same gel. Dotted lines in red indicate the position of three ENS-1 related proteins. (C) Separation of soluble and insoluble components from the cytoplasm. The cytoplasmic fraction separated from the nucleus was subjected to extended centrifugation. The pellet (Cy p) and the supernatant (Cy s) were analyzed for ENS-1 protein and HP1γ. The nuclear fraction corresponding to the whole nuclear proteins is represented (Nu). Protein loading was 10 µg for each fraction. HSP90 identifies the cytoplasmic soluble fraction, Lamin B1 identifies fractions containing membrane proteins and 2MeH3K4 identifies fractions containing chromatin. Results in (B) and (C) were obtained from two independent experiments using distinct fractionation protocols. (D) Summary tables of the ENS-1 forms found in the cytoplasm (upper table) and in the nucleus (lower table) from CES cells (-RA) and from CES induced to differentiate with retinoic for 24 h or 48 h.

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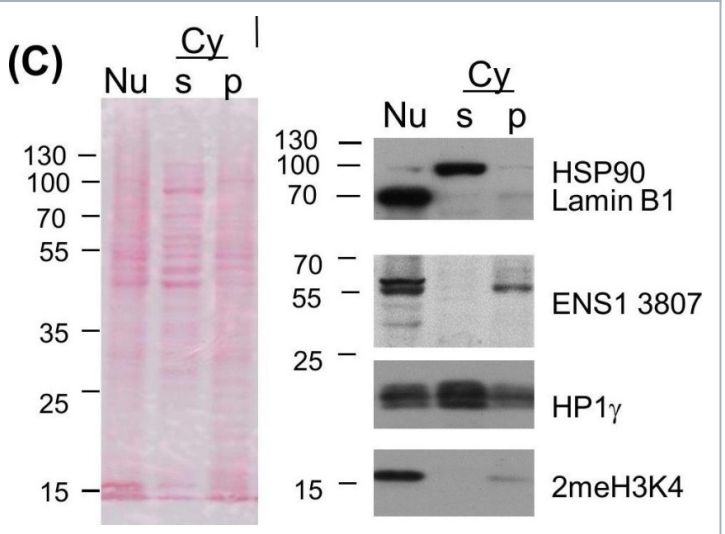
Plekhg5 depletion in SOD1 G93A motoneurons results in elevated ER-stress. a Expression of HSP70, HSP90, Calreticulin, and Calnexin in spinal cord lysates from three animals per genotype. b Quantification of western blot shown in a (each data point represents expression levels of one animal; unpaired t-test; two-tailed). c Expression of IRE1α and Chop1 in spinal cord lysates from three animals per genotype. d Quantification of western blot shown in c (each data point represents expression levels of one animal; unpaired t-test; two-tailed). e SOD1 G93A and non-transgenic motoneurons were depleted of Plekhg5 and several ER-stress markers were examined after 7 days in culture. f Quantification of western blots shown in e (each data point represents one individual experiment; mean ± SEM; unpaired t-test; two-tailed). g Survival of SOD1 G93A motoneurons decreased upon knockdown of Plekhg5 using two independent sh-RNA constructs (each data point represents the % of motoneuron-survival from one individual embryo. At least 50 motoneurons were evaluated from one embryo; mean ± SEM; two-way ANOVA; Bonferroni post-test). Images have been cropped for presentation. Full size images are presented in Supplementary Fig. 7

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Plekhg5-regulated autophagy of synaptic vesicles reveals a pathogenic mechanism in motoneuron disease. *Nat Commun* (2017)

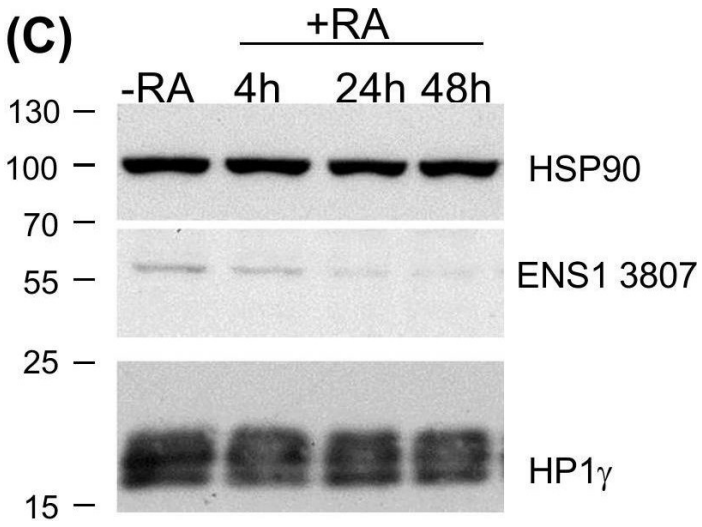


USP13 interacts with the Bag6 complex.(A) Endogenous interaction of USP13 with the Bag6 complex. Extracts from cells were subject to IP by the indicated antibodies followed by immunoblotting. (B) USP13 binds gp78 and Bag6 independent of its DUB activity. Extracts from HEK293 cells-transfected with the indicated plasmids were subject to IP with anti-FLAG beads. Asterisk indicates a non-specific band. (C) USP13 interacts with both the proteasome and Bag6 in a gp78 independent manner. Control (ctrl.) or gp78-depleted cell extracts were incubated with glutathione beads containing either GST or GST-USP13. The precipitated materials were analyzed by immunoblotting (Upper two panels) or by Ponceau S staining (the lower panel). (D) The UBL domain in Bag6 is required for interaction with USP13. A GST pull-down experiment was performed using the indicated Bag6 proteins purified from mammalian cells. (E) Bag6 UBL binds USP13 more tightly than Ubl4A UBL. Recombinant proteins purified from E. coli were used. (F) USP13, gp78 and Bag6 form a multi-protein complex. Sequential IP first by FLAG beads then by gp78 antibody using extracts from either control (−) or FLAG-USP13 (F-USP13)-expressing cells (indicated by '+').DOI:http://dx.doi.org/10.7554/eLife.01369.009

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Subcellular distribution of ENS-1 in CES and during differentiation.(A) Immunostaining of ENS-1 (16h4 antibody) and HP1γ (Abcam antibody) in CES (-RA) and CES differentiated with retinoic acid for the indicated period. Nuclei are labeled with Draq5 and are in blue in merging panels. Image acquisition was optimized to observe the distribution of the proteins and does not reflect the real expression level. Scale bar 15 μm. (B) Western blot analysis of ENS-1 in the cytoplasmic (Cy) and in the nuclear (Nu) fractions of undifferentiated CES (-RA) or CES induced to differentiate with retinoic acid for 24h or 48h. Ponceau's red staining serves as a protein loading control between both fractions. In the nucleus soluble and insoluble components were separated and only the precipitating fraction that contains ENS-1 is shown. The volume corresponding to 6×106 cells was loaded for the N fraction and Lamin B1 was used as loading control. In the cytoplasm (Cy) ENS-1 was in the supernatant, 15 μg of proteins were loaded and HSP90 was used as loading control. The vertical line indicates missing lanes between the two presented parts of the same gel. Dotted lines in red indicate the position of three ENS-1 related proteins. (C) Separation of soluble and insoluble components from the cytoplasm. The cytoplasmic fraction separated from the nucleus was subjected to extended centrifugation. The pellet (Cy p) and the supernatant (Cy s) were analyzed for ENS-1 protein and HP1γ. The nuclear fraction corresponding to the whole nuclear proteins is represented (Nu). Protein loading was 10 μg for each fraction. HSP90 identifies the cytoplasmic soluble fraction, Lamin B1 identifies fractions containing membrane proteins and 2MeH3K4 identifies fractions containing chromatin. Results in (B) and (C) were obtained from two independent experiments using distinct fractionation protocols. (D) Summary tables of the ENS-1 forms found in the cytoplasm (upper table) and in the nucleus (lower table) from CES cells (-RA) and from CES induced to differentiate with retinoic for



Western blot analysis of the protein ENS-1.(A) Proteins (15 µg) of whole cell lysates from murine (MES) and chicken (CES) cells were analyzed by western blot using the anti-ENS-1 3807 antibody. Protein loading was equivalent in both conditions as illustrated by the Ponceau's red staining of the blot. (B) Proteins lysates (20 µg) from CES cells transfected with an HA-tagged ENS-1 protein (lanes 4,5,6) or with an empty vector (lanes 1,2,3) were compared with untransfected cells (WT, lane 7). Triplicates were from three independent transfection experiments. The HA-tagged protein was detected by the 3807 antibody and by the anti-HA antibody at a molecular weight of 60 kDa. The anti-ENS1 antibody also detected the endogenous protein at 55 kDa. Both proteins differed in size by 3 kDa corresponding to the 33 additional amino acids added at the C-terminal part of ENS-1 in the transgenic protein in addition to the two HA tags (2.4 kDa). (C) Western blot analysis of ENS-1 and HP1γ (Chemicon antibody) in whole cell lysates (12 µg) from undifferentiated CES (-RA) or CES induced to differentiate with retinoic acid (RA, 10−6M) for 4 to 48 h as indicated. HSP90 was used as protein loading control.

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Handling & Storage

Handling	Avoid freeze/thaw cycles.
Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	Heat shock protein 90
Application	IHC (PS), WB
Application Notes	Detects a band of ~90kDa by Western blot.
Clone	AC88
Formulation	Liquid. In PBS containing 50% glycerol and 0.09% sodium azide.
Host	Mouse
Immunogen	Achlya ambisexualis (water mold) Hsp90.
Isotype	IgG1
Purity Detail	Protein G affinity purified.
Recommendation Dilutions/Conditions	Western Blot (1:1,000, ECL)Suggested dilutions/conditions may not be available for all applications.Optimal conditions must be determined individually for each application.
Source	Purified from ascites
Species Reactivity	Beluga, C. elegans, Chicken, Dog, E. coli, Fish, Fusobacterium nucleatum, Guinea pig, Hamster, Human, Monkey, Mouse, Mussel, Mycobacterium leprae, Porcine, Rabbit, Rat, Scallop, Sheep, Water mold
Technical Info / Product Notes	**Cited samples:** *For an overview on cited samples please click here.*

UniProt ID Q8LLI6

Worry-free Guarantee **This antibody is covered by our Worry-Free Guarantee .**

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