

HSP70/HSP72

polyclonal antibody

The Hsp70 family of heat shock proteins contains multiple homologs ranging in size from 66-78 kDa, and are the eukaryotic equivalents of the bacterial DnaK. The most studied Hsp70 members include the cytosolic stress-induced Hsp70 (Hsp72), the constitutive cytosolic Hsc70 (Hsp73), and the ER-localized BiP (Grp78). Hsp70 family members contain highly conserved N-terminal ATP-ase and C-terminal protein binding domains. Binding of peptide to Hsp70 is assisted by Hsp40, and stimulates the inherent ATPase activity of Hsp70, facilitating ATP hydrolysis and enhanced peptide binding. Hsp70 nucleotide exchange and substrate binding coordinates the folding of newly synthesized proteins, the re-folding of misfolded or denatured proteins, coordinates trafficking of proteins across cellular membranes, inhibits protein aggregation, and targets the degradation of proteins via the proteasomal pathway.

This antibody is covered by our [Worry-Free Guarantee](#).

Citations: 146

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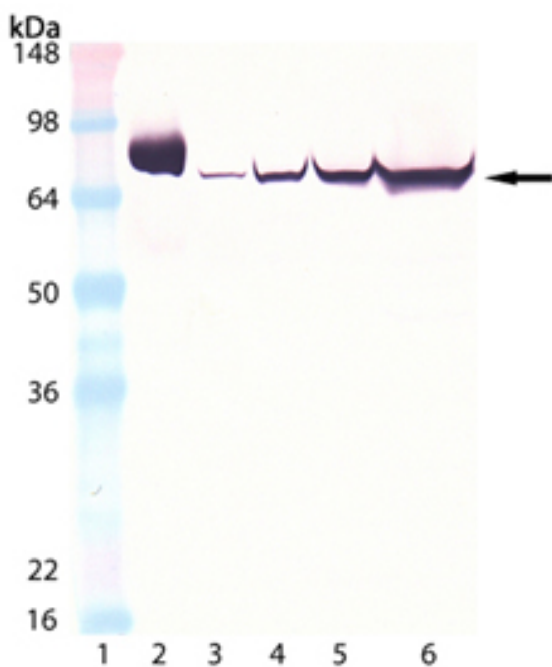
Ordering Information

[Order Online »](#)

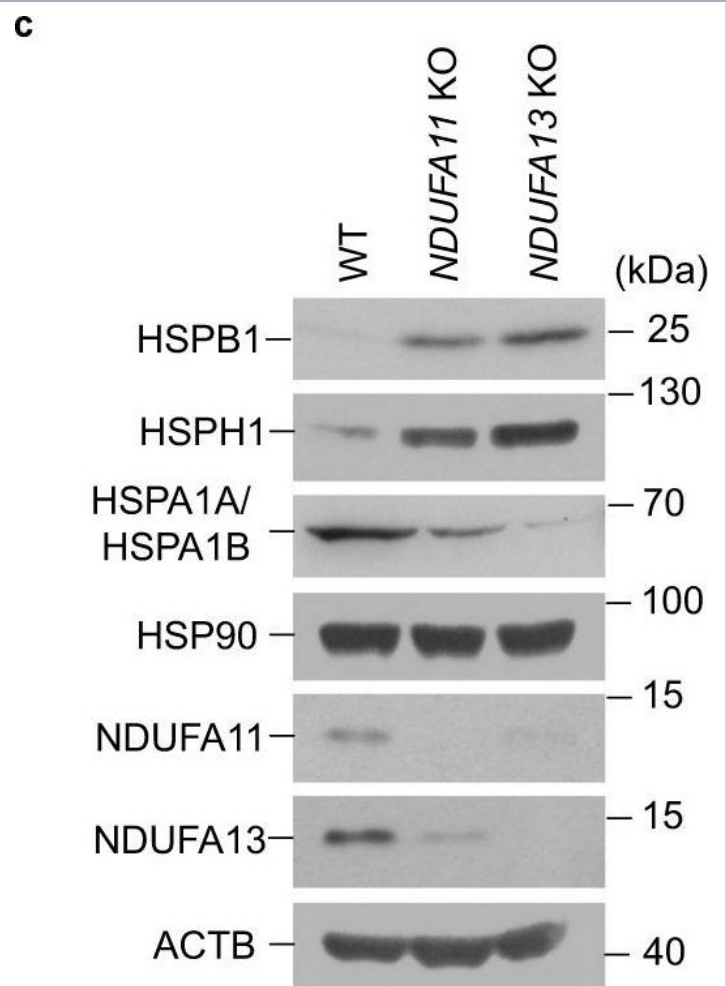
| | |
|---------------|-------|
| ADI-SPA-812-D | 50µg |
| ADI-SPA-812-F | 200µg |

Manuals, SDS & CofA

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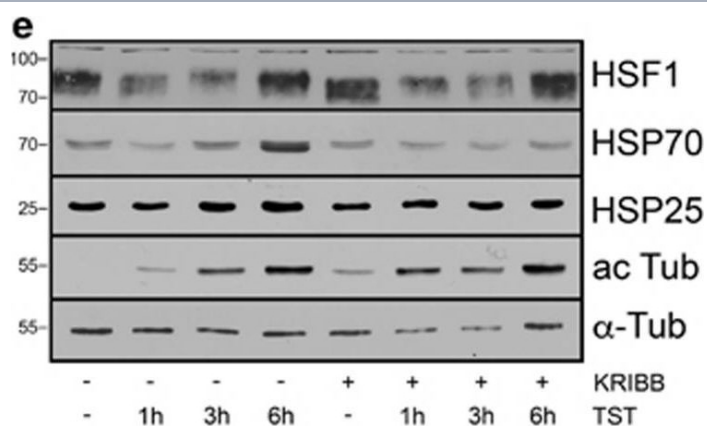


Western blot analysis of HSP70/HSP72 pAb (Prod. No. ADI-SPA-812): Lane 1: MW marker; Lane 2: HSP70/HSP72 (human), (recombinant) (Prod. No. ADI-NSP-555); Lane 3: PC-12; Lane 4: PC-12 (heat shocked); Lane 5: HeLa; Lane 6: HeLa (heat shocked).



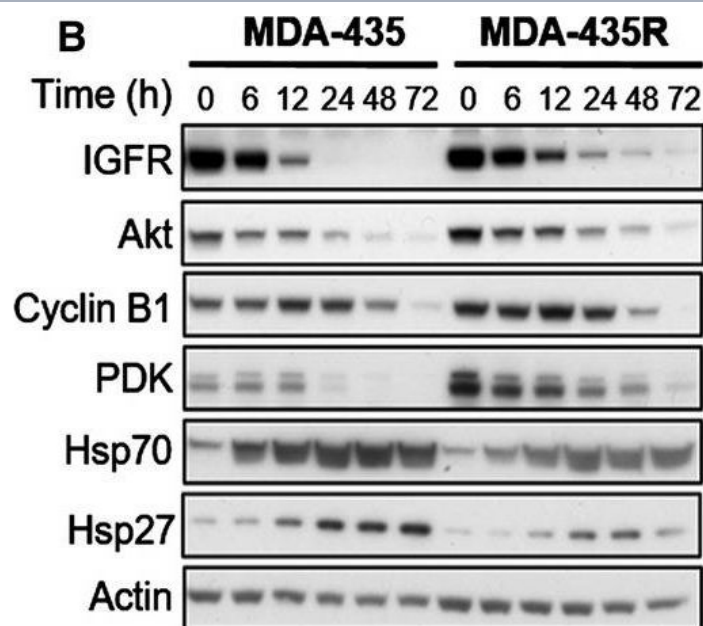
Small HSP and HSP110 families have a tendency to be increased under mitochondrial stress. a RNA-seq analysis of HSPs gene expression log₂ fold changes (log₂FC) in NDUFA11 KO and NDUFA13 KO compared to WT HEK293T cells (n = 4). Up- and down-regulated genes (q-value < 0.05) are shown in green and pink, respectively. The intensity of the color shades depends on the level of expression change. Gray indicates genes with not statistically significant expression changes. b mRNA expression patterns of selected transcripts validated by RT-qPCR. The mRNA levels are presented as fold changes relative to WT. Data shown are mean ± SD (n = 3 biological replicates with two technical replicates). p-value from an ordinary one-way ANOVA with Dunnett's multiple comparisons test using GraphPad Prism. c Western blot analysis of HSPs expression performed in whole cell lysates of NDUFA11 KO, NDUFA13 KO and WT HEK293T cells. ACTB was used as a loading control. Data shown are representative of three independent experiments. d Quantification of HSPs in western blot analysis normalized to ACTB using ImageJ. The protein levels are presented as fold changes relative to WT. Data shown are mean ± SD (n = 3). p-value from two-sided, unpaired t-test using GraphPad Prism. Source data are provided as a Source Data file.

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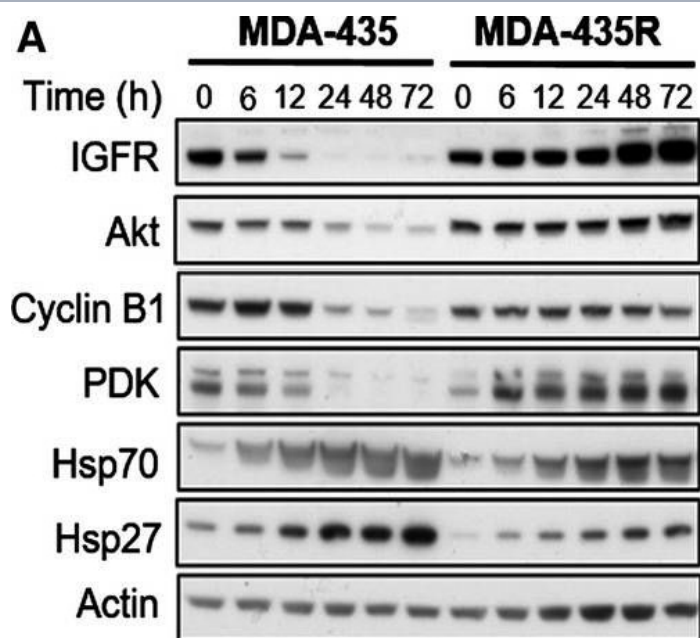
Tubastatin A induces heat-shock protein expression by activating heat-shock factor 1. 661W cells were treated with 1, 5 and 10 μM of tubastatin A (TST) for 24 h (a), or in (b) with 10 μM TST for 8 h, or with 200 μM H₂O₂ for 6 h or were preincubated with 10 μM TST for 2 h followed by incubation with 200 μM H₂O₂ for 6 h. ac Tub, acetylated tubulin. α -Tub, α -tubulin. Co, untreated control. Quantitative evaluation of immunoblot analysis revealed a significant increase in heat-shock protein (HSP) 70 level after 8 h (c), while HSP25 was significantly enhanced after 24 h (d); n=4. (e) Heat-shock factor 1 (HSF1) activity was investigated using immunoblot analysis of 661W cell extracts that were treated 10 μM TST for 1, 3 and 6 h, or with 5 μM KRIBB11 (KR) for 6.5 h alone, or preincubated with 5 μM KR for 30 min, followed by incubation with 10 μM TST for 1–6 h. (f) Cell viability MTT assay. Cells were treated as indicated. TST (10 μM) for 8 h or KR (5 μM) for 8.5 h did not influence 661W cell number. H₂O₂ (200 μM) for 6 h led to a strong decrease in cell viability, which was enhanced by pre-incubation with TST for 2 h (TST+H₂O₂). Pre-incubation with KR for 30 min followed by incubation with TST for 2 h followed by treatment with H₂O₂ for 6 h (KR+TST+H₂O₂) did not diminish the protective effect of TST. Experiments were carried out three times with similar results. Data represent the mean \pm S.D. of one representative experiment with eight replicates and are expressed as the percent of the untreated control, which was set at 100%

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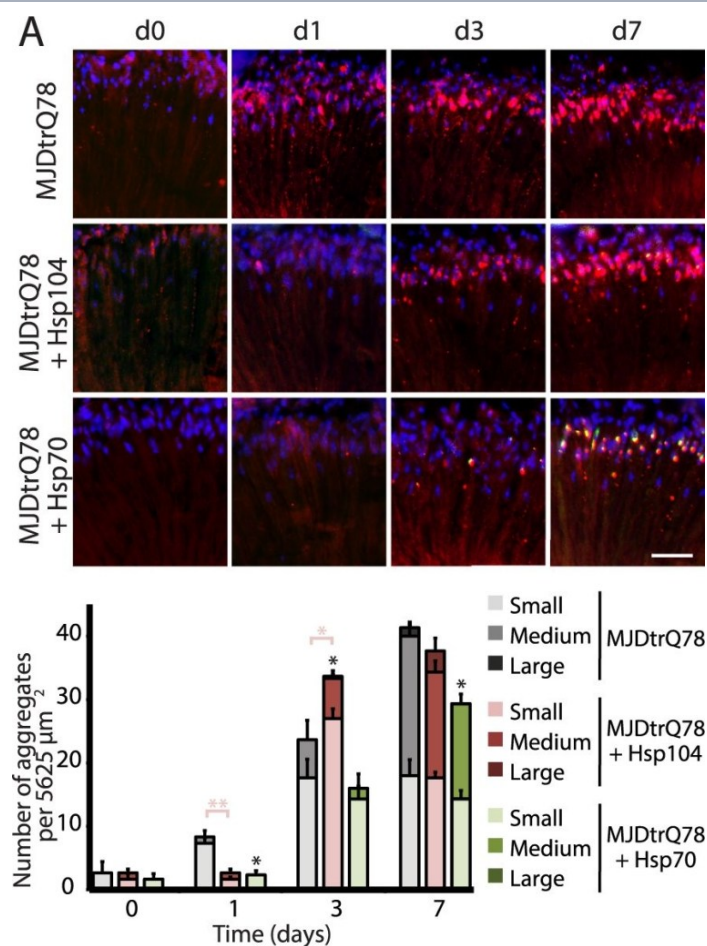
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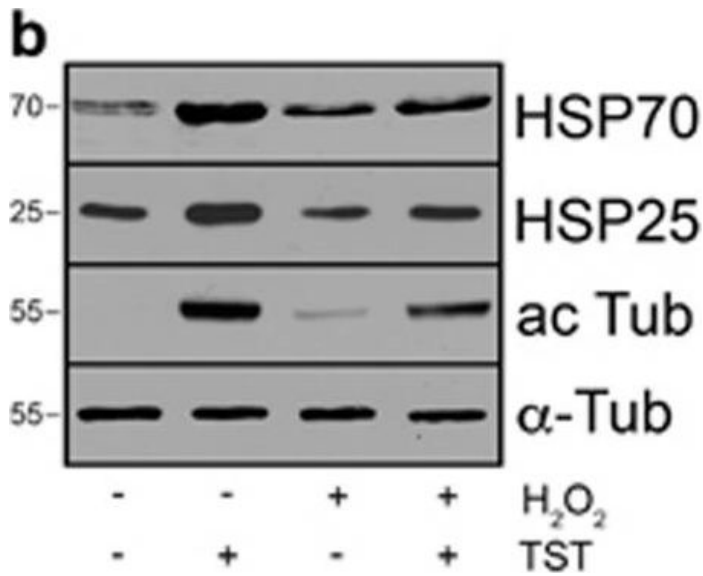


Molecular effects of HSP90 inhibition by 17 β AAG on HSP90 client proteins in parental and resistant cell lines. MDA β 435 parental and resistant MDA β 435R cells treated with 0.1 μ M of 17 β AAG ($\sim 5 \times 17\beta$ AAG IC₅₀ concentrations of the parental cell line) (A) and with 30 μ M of 17 β AAG ($\sim 5 \times 17\beta$ AAG IC₅₀ concentrations of the resistant cell line) (B). MDA β 231 parental and resistant MDA β 231R cells treated with 5.0 μ M of 17 β AAG ($\sim 5 \times 17\beta$ AAG IC₅₀ concentrations of the parental cell line) (C) and with 50 μ M of 17 β AAG ($\sim 5 \times 17\beta$ AAG IC₅₀ concentrations of the resistant cell line) (D). Total cell lysates were collected at the indicated time points and analysed by western blotting.

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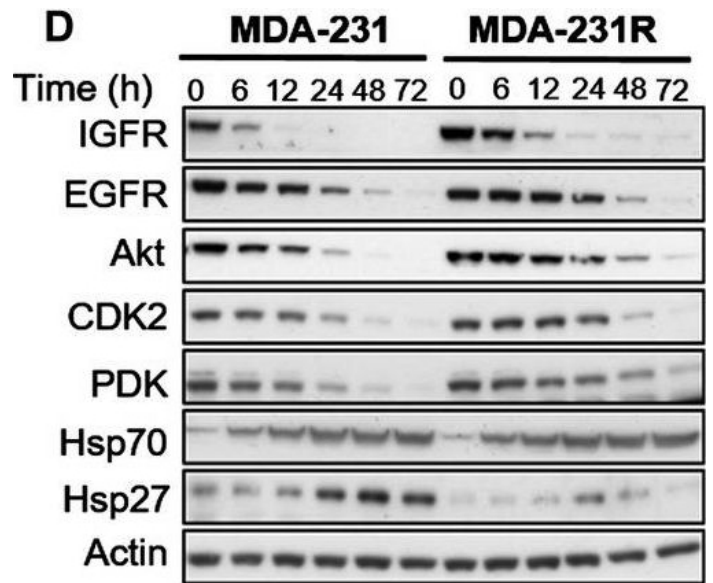


Hsp104 delays aggregation of truncated MJD, but enhances aggregation of full-length MJD. (A and B) With the rh1-GAL4 driver at indicated time points, cryosections and IHC demonstrate accumulations of MJDtrQ78 and MJDnQ78 (red) over time, using anti-HA and anti-myc antibodies, respectively. Sections were co-stained with anti-Hsp104 or anti-Hsp70 (green) as indicated, and nuclei were labeled by Hoechst (blue). Hsp104 delayed but did not suppress aggregation of MJDtrQ78, but Hsp104 enhanced accumulation formation of MJDnQ78. Hsp70 suppressed aggregation of both MJD proteins. The size of the aggregates was quantified using ImageJ, with delineations for large inclusions (>5 μ m across), medium inclusions (2.5–5 μ m), or small inclusions (<2.5 μ m) (n = 3 (mean \pm SEM)). Scale bar = 20 μ m. *p<0.05, **p = 0.001–0.01, ***p<0.001; Statistics indicate comparison to the disease protein alone for total number of inclusions at each timepoint (black asterisks). Additional statistical comparisons for inclusion size divisions are indicated by color, e.g., dark red asterisk indicates significant change in large inclusions. (C and D) With the rh1-GAL4 driver at indicated time points, SDD-AGE and immunoblot analysis show the progression of amyloid formation of MJDtrQ78 and MJDnQ78 proteins over time. Hsp104 did not greatly affect the aggregation profile of MJDtrQ78 but enhanced formation of SDS-insoluble amyloid aggregates of MJDnQ78. Hsp70 suppressed aggregation of both MJD proteins. The formation of



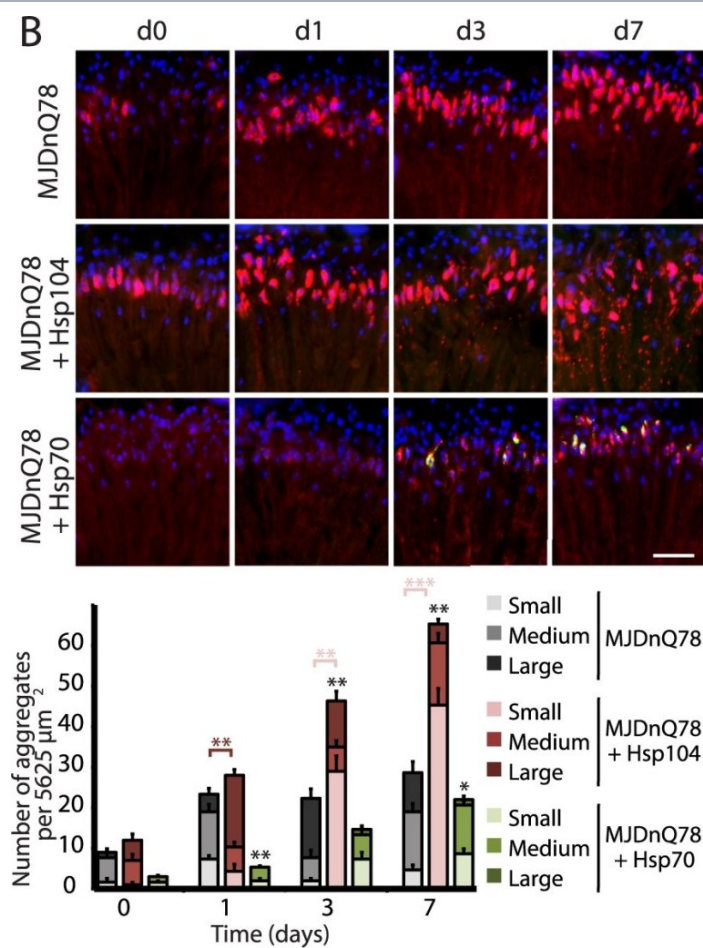
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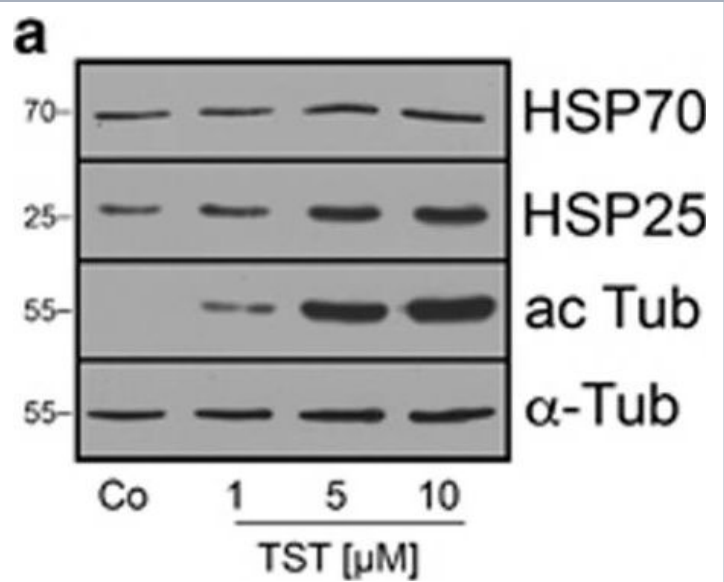


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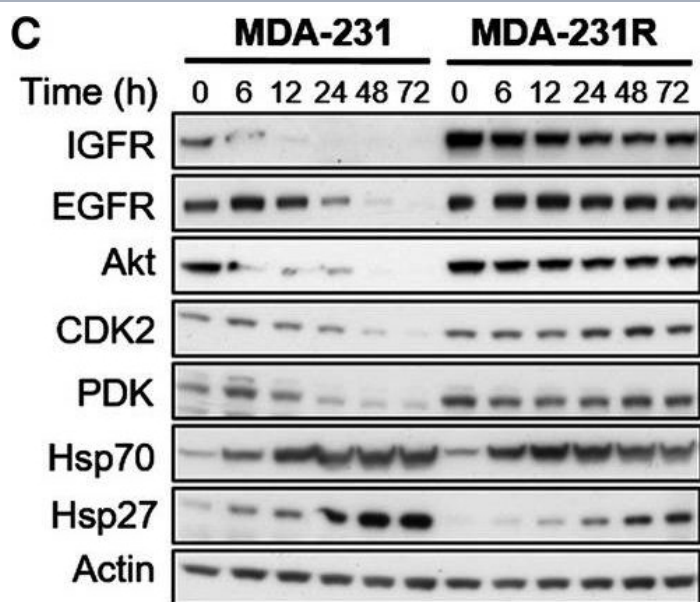


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Handling & Storage

Handling Avoid freeze/thaw cycles.

Long Term Storage -20°C

Shipping Blue Ice

Regulatory Status RUO - Research Use Only

Product Details

Alternative Name Hsp70, HspA1A, Heat shock protein 70, HspA1B, Hsp72

Application ICC, IF, IHC, IP, WB

Application Notes Detects a band of ~70kDa by Western blot.

Formulation Liquid. In PBS, pH 7.2, containing 50% glycerol and 0.09% sodium azide.

GenBank ID M11717

Gene/Protein Identifier NP_005336.3 (RefSeq), NM_005345 (RefSeq), 3303 (Entrez GeneID), 140550 (OMIM)

Host Rabbit

Immunogen Recombinant human Hsp70.

Purity Detail Protein A affinity purified.

Recommendation Dilutions/Conditions Immunoprecipitation (1:100)Western Blot (1:1,000)Suggested dilutions/conditions may not be available for all applications.Optimal conditions must be determined individually for each application.

Source Purified from rabbit serum.

Species Reactivity Beluga, Bovine, Dog, Drosophila, Fish, Guinea pig, Hamster, Human, Monkey, Mouse, Porcine, Rat, Sheep

Technical Info / Product Notes Recommended by the Human Protein Atlas Organization for IHC (Ensembl No. ENSG00000204389).

UniProt ID

P0DMV8 (HSPA1A), P0DMV9 (HSPA1B)

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