HSP70/HSP72 monoclonal antibody (C92F3A-5)

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.

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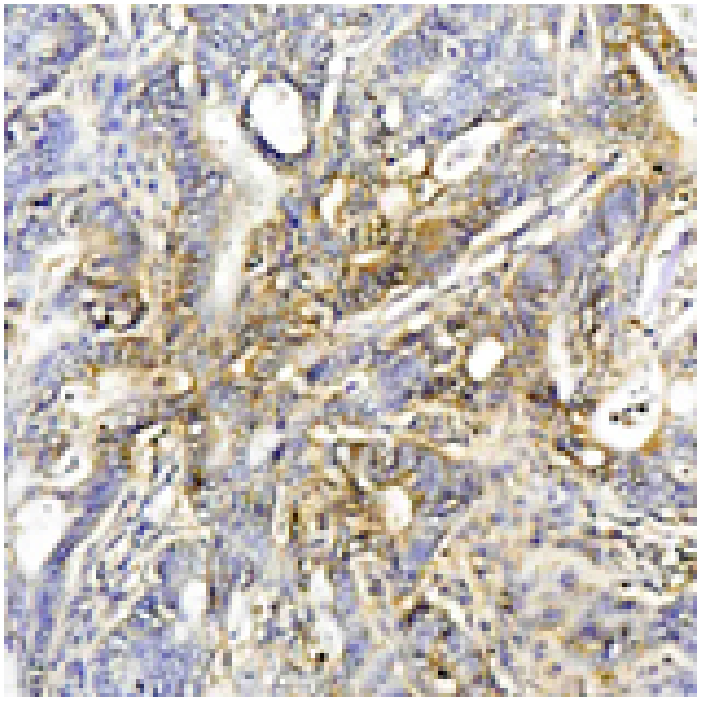
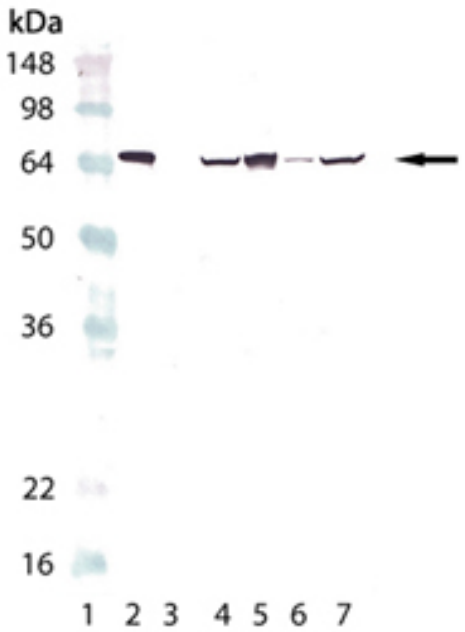
ADI-SPA-810-J	1mg
ADI-SPA-810-D	50µg
ADI-SPA-810-F	200µg

Manuals, SDS & CofA View Online »

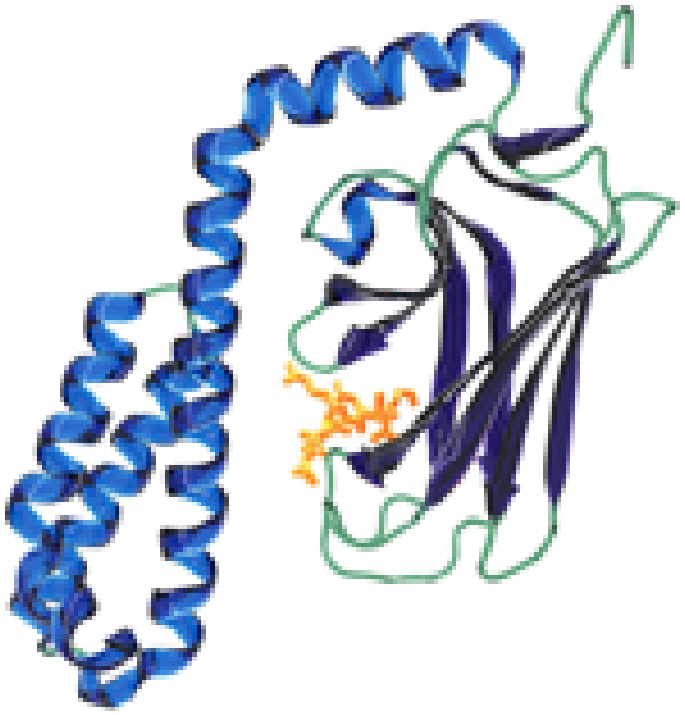
- Validated for multiple applications
- Multi-species reactive
- Widely cited



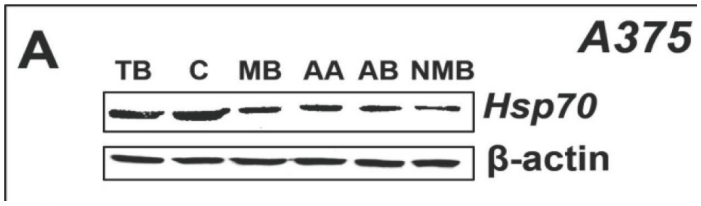
Immunohistochemistry analysis of human colon cancer tissue using HSP70 mAb (C92F3A-5) at a dilution of 1:50.



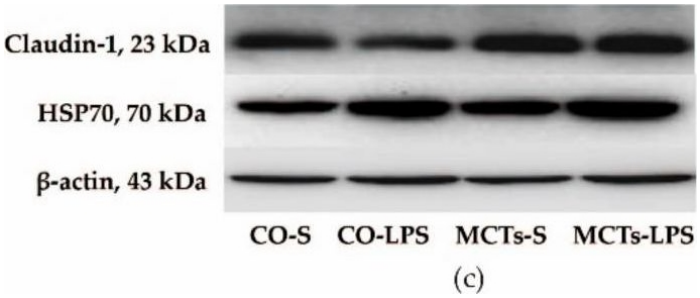
Western blot analysis of HSP70/HSP72, mAb (C92F3A-5) (Prod. No. ADI-SPA-810): Lane 1: MW marker, Lane 2: HSP70/HSP72 (human), (recombinant) (Prod. No. ADI-NSP-555), Lane 3: HSC70/HSP73 (bovine), (recombinant) (Prod. No. ADI-SPP-751) (Negative Control), Lane 4: PC-12 (heat shocked) (Prod. No. ADI-ADI-LYC-PC101), Lane 5: HeLa (heat shocked) (Prod. No. ADI-LYC-HL101), Lane 6: 3T3 (heat shocked) (Prod. No. ADI-LYC-3T101), Lane 7: CHO-K1 (heat shocked).



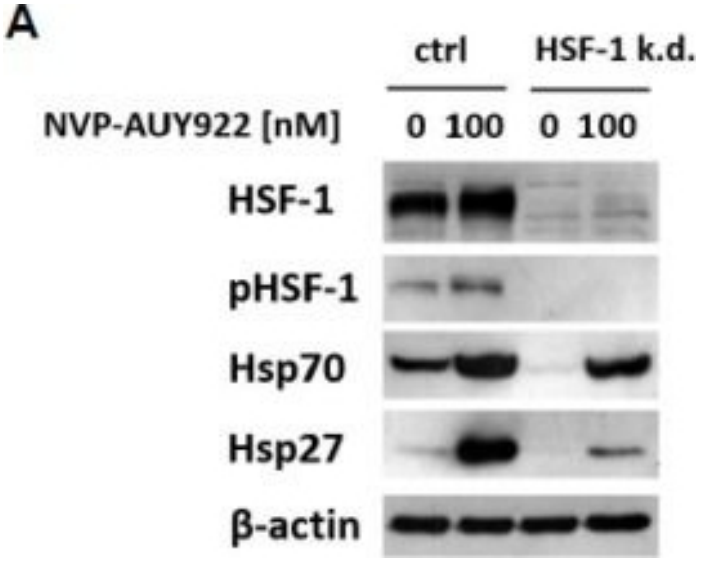
Substrate binding domain of HSP70 in complex with a substrate peptide[10].



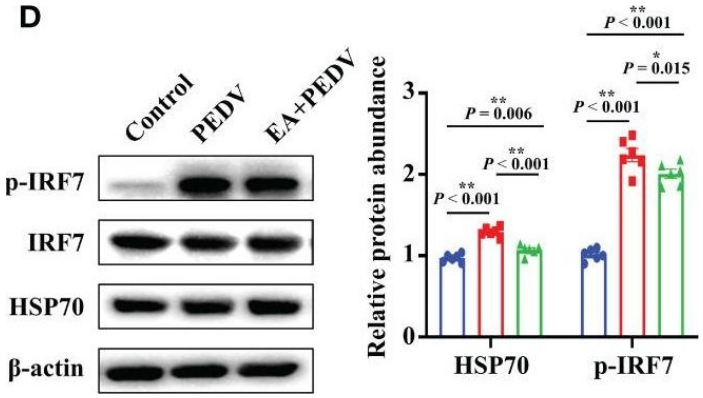
Comparative activity of phenothiazine-derivatives on melanoma cell viability and Hsp70 expression. (A) Hsp70 modulation as assessed by immunoblot analysis in A375 melanoma cells exposed to phenothiazine derivatives (10 μM, 24 h; methylene blue (MB), toluidine blue (TB), azure A (AA), azure B (AB), “new methylene blue” (NMB)) as assessed by immunoblot analysis. β-actin: loading control; (B) Viability analysis by flow cytometric analysis (all test compounds as used in (A)); (C) Hsp70 modulation as assessed by immunoblot analysis in G361 melanoma cells exposed to phenothioazine derivatives as specified in (A); (D) Viability analysis of G361 melanoma cells by flow cytometric analysis showing annexinV-PI panels; test compounds as used in (A); The numbers indicate viable (AV-negative, PI-negative) in percent of total gated cells (mean ± SD, n = 3). (E) Molecular structures of phenothiazine test compounds; abbreviations as in (A).



Effect of medium-chain triglyceride (MCT) supplementation on claudin-1 (a) and heat shock protein 70 (HSP70) (b) protein expression in the liver after 4-h LPS challenge in pigs. The bands are the representative Western blot images (c). Values are mean and SE, n = 6 (1 pig/pen). Means with different letters differ significantly ($P < 0.05$). All data for protein expression were acquired using Western blot. Values for relative claudin-1 and HSP70 expression were normalized for β-actin. CO-S, pigs fed the control diet and injected with saline; MCTs-S, pigs fed MCTs and injected with saline; CO-LPS, pigs fed the control diet and challenged with LPS; MCTs-LPS, pigs fed MCTs and challenged with LPS.



HSF-1 k.d. reduces the expression of Hsp70 and Hsp27 and the transcriptional activity of HSF-1. (A) Representative immunoblot showing the expression of HSF-1, HSF-1 phospho S326 (pHSF-1), Hsp70, Hsp27, and β-actin in H1339 cells transfected with control (ctrl) or HSF-1 shRNA (HSF-1 k.d.). Cells were treated with NVP-AUY922 (100 nM) for 24 h. (B) Transcriptional activity of an HSF-1 responsive firefly luciferase construct in H1339 ctrl and HSF-1 k.d. cells. Cells were treated with NVP-AUY922 (100 nM) for 24 h. Significance * $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$. (C) Intracellular (ic) Hsp70 protein concentrations assessed by ELISA in H1339 ctrl and HSF-1 k.d. cells treated with NVP-AUY922 (100 nM) for 24 h. Significance * $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$.



Expression of antiviral-related genes and proteins in the jejunum. (A) mRNA levels of PEDV marker genes; (B) mRNA level for genes involved in the interferon pathway; Protein abundance of MX1 and ISG15 (C), HSP70 and p-IRF7 (D), p-JAK2 and p-STAT3 (E). Data are presented as means ± SEMs (n = 8). *P < 0.05, **P < 0.01. IFN, interferon β; MX1, myxovirus resistant 1; IFIT1, interferon-induced protein with tetratricopeptide repeats 1; IFITM3, interferon induced trans-membrane proteins 3; ISG15, interferon-stimulated gene 15; HSP70, heat shock protein 70; IRF7, interferon regulatory factor 7; JAK2, Janus tyrosine kinase 2; STAT3, signal transducer and activator of transcription 3.

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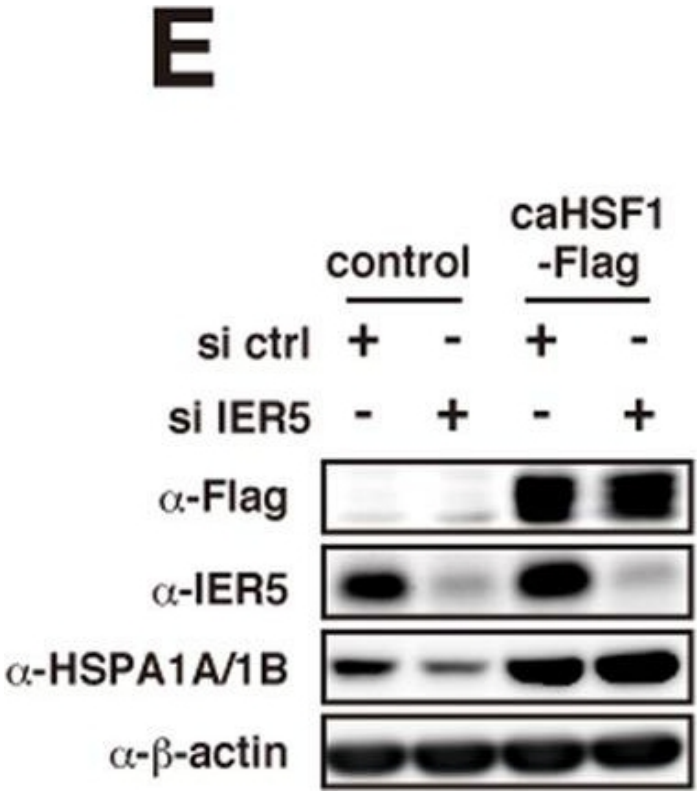
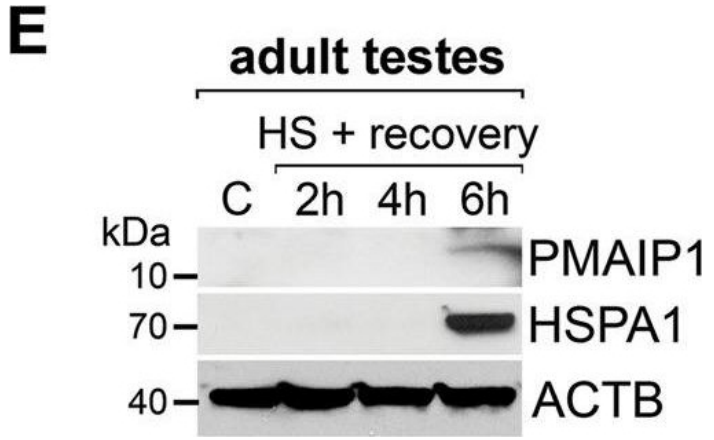
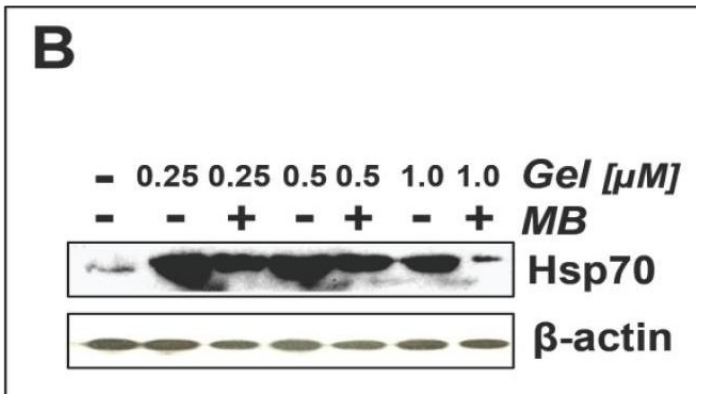


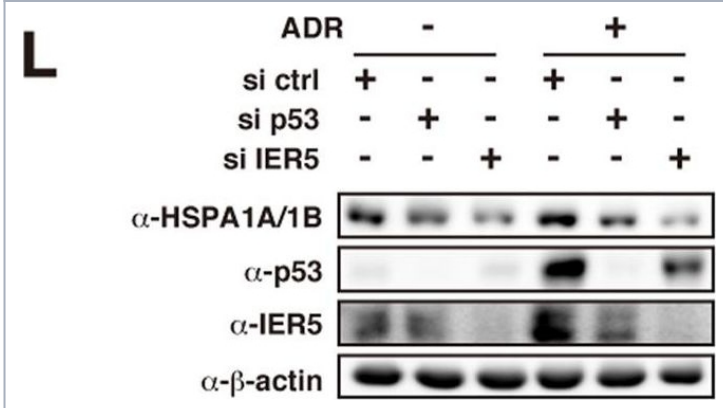
IER5/HSF1/HSP family gene axis in cancer and in stressed cells.(A–D) OE33 cells (2 × 103 cells) were plated in adherent (A) or suspension (B) 96 well culture plates, and control, IER5-targeting or HSF1-targeting siRNAs were introduced. Cell growth assays were performed on the indicated days. Relative cell numbers were analyzed with CellTiter-Glo reagents from four wells and the mean cell numbers ±SD are shown. IER5 and HSF1 mRNA expression was analyzed by quantitative RT-PCR 48 hrs post-transfection (C,D). (**p < 0.01, #p < 0.0001). (E) OE33 cells were stably transfected with caHSF1, and the indicated siRNAs were introduced. Cell lysates were prepared 48 hrs post-transfection, and expression of caHSF1-Flag, IER5, HSPA1A/1B were analyzed by Western blotting. (F) Cell growth assays were performed as in (B) using OE33 cells expressing caHSF1. (**p < 0.01). (G,H) Expression of IER5 (G) and HSPA6 (H) and prognosis in cancer patients. Disease-specific survival of patients with bladder cancer (Transitional cell carcinoma, dataset GSE13507) was analyzed using the PrognoScan database. (I–K) Expression of IER5 (I), HSPA6 (J) and HSPA1A (K) mRNA in cells treated with Adriamycin. Cell lines carrying wild-type p53 were treated with Adriamycin (1 μM) for 24 hrs (MRC5, MCF7, U2OS) or 19 hrs (A549). (*p < 0.05, **p < 0.01). (L) Expression of IER5, p53 and HSPA1A protein level in U2OS cells. Control, p53 and IER5 targeting siRNAs were introduced. Cells were treated with Adriamycin (1 μM) for 24 hrs and harvested 48 hrs post siRNA-transfection. (M) IER5 is transiently induced downstream of p53 and activates HSF1 in stressed cells, while IER5 is overexpressed and constitutively activates HSF1 in



Heat shock-induced HSF1 binding in the introns of the Pmaip1 gene correlates with upregulation of its expression and enhanced apoptosis in mouse spermatogenic cells.a Chromatin binding of HSF1 assessed by ChIP-Seq in isolated spermatocytes. Organization of mouse and human genes is shown below peaks of ChIP-Seq tags: bars—exons (darker bars—coding regions), lines—introns; corresponding start and stop codons are linked by light-gray dashed or solid lines, respectively; the positions of HSE or HSE-like motifs are indicated by the closed and open arrows, respectively. Right panel shows the magnitude of HSF1 binding in intronic HSE of the Pmaip1 gene in comparison to Hsph1 promoter based on data from ChIP-Seq extracted from GSE56735. b HSF1 binding in Pmaip1 introns analyzed by ChIP-PCR in isolated spermatocytes. Binding to the Hsph1 promoter is shown as a positive control. C control, physiological temperature of testes (32 °C); 38° and 43°, heat shock at 38 or 43 °C, respectively; M marker; − +, negative and positive PCR controls. c Induction of Pmaip1 transcription assayed by RT-PCR and RT-qPCR in isolated spermatocytes after heat shock in vitro at 43 °C and d in testes of mice after heat shock in vivo. 18S rRNA and Hspa1 were used as transcript level controls for loading and the heat shock response, respectively; C control, HS heat shock. e Accumulation of PMAIP1 protein after heat shock in vivo in mouse testes demonstrated by western blot. ACTB and HSPA1 were used as controls for loading and the heat shock response, respectively. f Induction of Pmaip1 transcription assayed by RT-PCR and RT-qPCR in testes of transgenic mice expressing constitutively active mutated HSF1 (aHSF1) during postnatal development; wt wild type, tg transgenic. Asterisks on the graphs indicate statistical significance of differences: *p < 0.05, **p < 0.001. g Accumulation of PMAIP1 in transgenic mouse testes demonstrated by western blot. ACTB was used as a control for loading. h Detection of PMAIP1 or HSF1 by immunofluorescence (green) and apoptotic DNA breaks (by TUNEL assay, red; DNA



Methylene blue sensitizes A375 melanoma cells to etoposide-, doxorubicin-, and geldanamycin-induced cell death and attenuates geldanamycin-induced HSPA1A upregulation. (a) MB-induced (10 μM cotreatment; 24 h) sensitization to etoposide, doxorubicin, and geldanamycin (GA) cytotoxicity (CellTiter-Glo™ luminescence analysis); (b) MB attenuation (10 μM; 1h pretreatment) of GA-induced (≤1.0 μM, 24 h) Hsp70 upregulation (immunoblot analysis); (c) MB attenuation (10 μM; 1 h pretreatment) of GA-induced (0.5 μM, 24 h) HSPA1A mRNA upregulation; (d) MB-sensitization (10 μM; 1 h pretreatment) to GA-induced cell death (0.5 μM, 24 h) (flow cytometric analysis; left panels). Numbers indicate viable (AV-negative, PI-negative) in percent of total gated cells (mean ± SD, n = 3); right panels: Representative light microscopy pictures (24 h).



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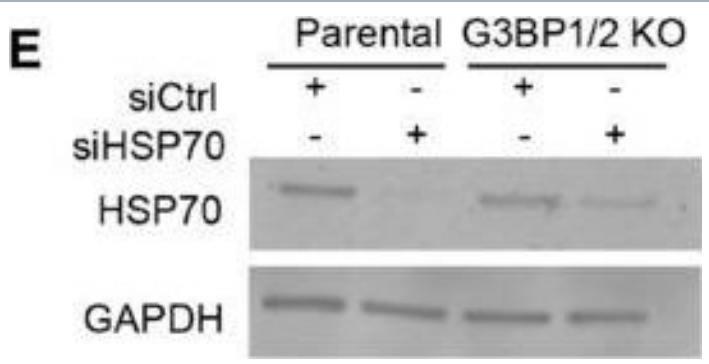
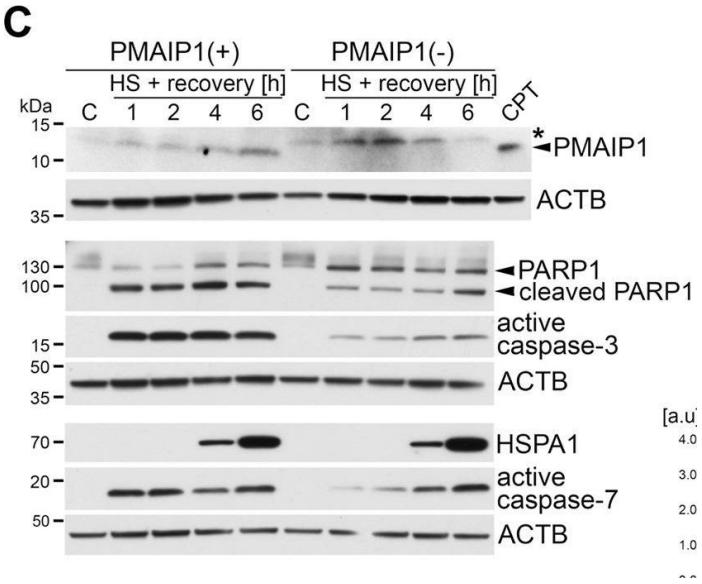
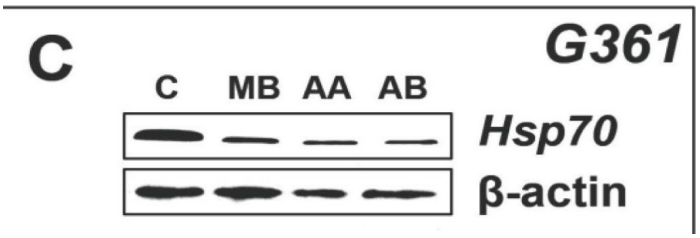


DRiPs and proteasome activity in thermally stressed G3BP1/2 knockout cellsRepresentative confocal images of immunofluorescent staining of the nucleolar marker nucleolin and puromycin□labeled proteins in U2OS cells pretreated with or without 4 μM Actinomycin D for 3 h, subjected to a heat shock. Scale bar is 20 μm.U2OS G3BP1/2 knockout cells were treated with or without 25 μM ivermectin for 2 h. The ataxin1 was visualized by immunostaining. Scale bar is 20 μm.U2OS G3BP1/2 knockout cells were treated with or without 25 μM ivermectin for 2 h before being exposed to 43°C heat shock in a 5 μg/ml puromycin□containing medium. The DRiPs and nucleoli were visualized by immunostaining for puromycin and nucleolin, respectively. Scale bar is 20 μm.Immunoblot of ubiquitylated DRiPs by TUBE pulldown of lysates from parental U2OS and G3BP1/2 knockout cells after heat shock.Immunoblot of HSP70 in parental and G3BP1/2 knockout U2OS cells transfected with HSP70 siRNA for 72 h.Parental U2OS and G3BP1/2 knockout cells were either left untreated (− HS), or exposed to 43°C for 30 min and followed for 4 h after heat shock (HS + Rec.). The SUMO2 deconjugase activity was detected by following the conversion of the fluorogenic SUMO2□AMC substrate over 1 h. As a control, 20 mM NEM was added to the reaction mixture to inhibit SUMO2 deconjugases. Data represent the mean ± SD (n = 3 independent experiments, Student's unpaired t□test).Parental U2OS and G3BP1/2 knockout cells were either left untreated (− HS), or exposed to 43°C for 30 min and followed for 4 h after heat shock (HS + Rec.). The chymotrypsin□like activity (β5 subunit) of the proteasome was detected by following the conversion of the fluorogenic Suc□LLVY□AMC substrate over 1 h. As a control, 100 nM epoxomicin (EPX) was added to the reaction mixture to inhibit proteasome activity. Data represent the mean ± SD (n = 3 independent experiments, Student's unpaired t□test).Representative images of parental U2OS and G3BP1/2 knockout cells transiently transfected with α4□GFP, which were either left untreated (− HS), or exposed to 43°C for 30 min and followed for 4 h after heat shock (HS + 4 h Rec.). Scale bar is 20 μm.



PMAIP1 deficiency reduces heat-induced death and delays caspase-3 and caspase-7 activation.a Apoptosis and b necrosis of PMAIP1(+) and PMAIP1(−) HECa10 cells monitored between 2 and 24 h after heat shock or during bortezomib (Bort) or camptothecin (CPT) treatments. Shown are mean values ± SD from one (representative) of three independent experiments; the statistically significant difference between treated and untreated samples or PMAIP1(+) and PMAIP1(−) samples is marked with an asterisk (*p < 0.05, **p < 0.001). c PMAIP1(+) and PMAIP1(−) cells were heat-shocked for 1 h at 43 °C and protein extracts were analyzed by western blot up to 6 h of recovery. ACTB was used as a loading control. C untreated cells, CPT camptothecin treatment of wild-type HECa10 cells for 6 h (positive control for PMAIP1 induction); unspecific protein band recognized by anti-PMAIP1 Ab is marked with an asterisk. The graphs show the results of densitometric analyses from three independent experiments; *p < 0.05, **p < 0.001.



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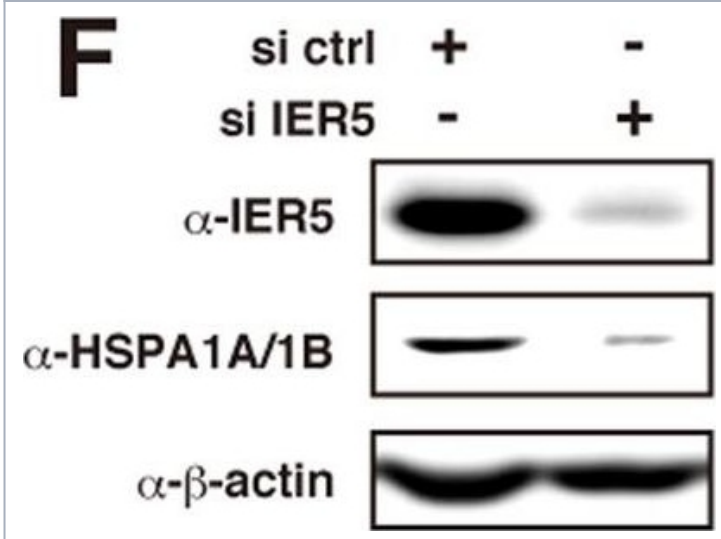
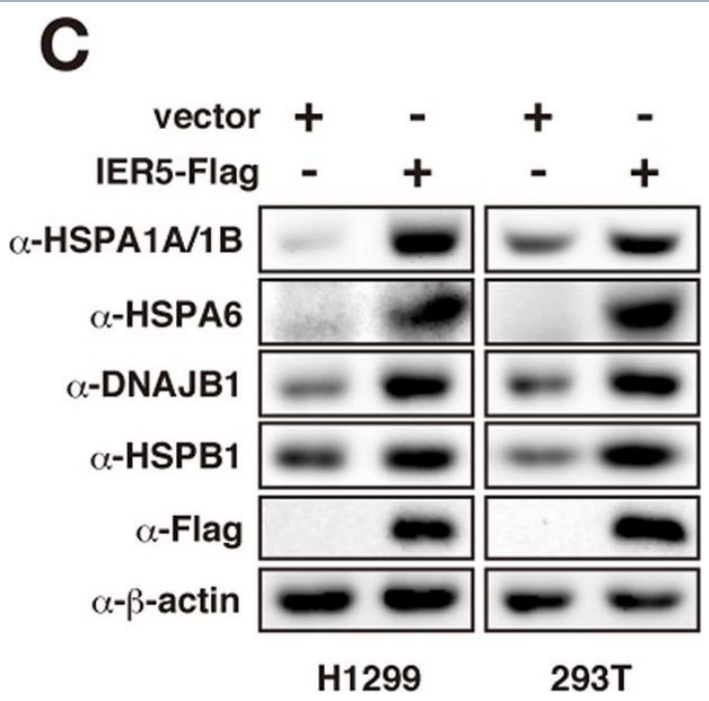


HSP family genes are induced by IER5.(A) H1299 or 293T cells were transfected with control vector or an IER5 expression vector. Cells were harvested 21 hrs or 27 hrs post-transfection and microarray expression analysis was performed. The table shows the HSP family genes, among the genes induced by IER5. (B) H1299 cells were transfected with control, IER5-Flag or mutant IER5-Flag expression vectors (representative image of mut 1 is shown in Fig. S1). Cells were harvested 27 hrs post-transfection, and mRNA expressions of the HSP family genes were analyzed by Northern blotting. (C) H1299 and 293T cells were transfected with control vector or IER5-Flag expression vector, and cells were harvested 24 hrs post-transfection. Expressions of the HSP family proteins were analyzed by Western blotting. (D–F) Control or IER5-targeting siRNAs were introduced into OE33 cells. Cells were harvested 52 hrs post-transfection. Expression of IER5 (D,F) and HSPA1A (E,F) were analyzed by quantitative RT-PCR (D,E) and Western blotting (F). (**p < 0.01). (G) The promoter regions of HSPA1A, HSPA1B and HSPA6 were inserted into the luciferase reporter plasmid containing a minimal promoter, and assayed 24 hrs post-transfection. Experiments were run in triplicate, and data are represented as the mean-fold activation ±SD. (H) Serially deleted regions of the HSPA1A promoter were analyzed as in (G). Numbers indicate the position of the 5′ most nucleotide relative to the transcription initiation site. A heat shock element (HSE), to which HSF1 binds, was found between positions −132 and −109.



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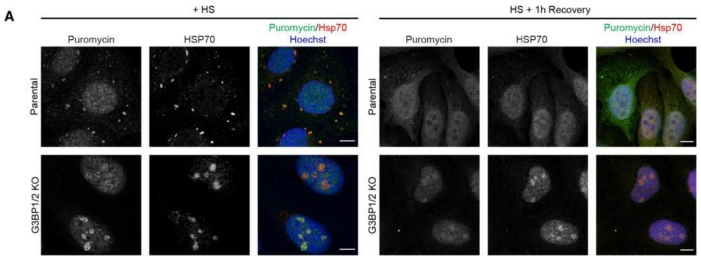

Altered heat shock response in stress granule‐deficient cellsRepresentative confocal images of immunofluorescent staining of and puromycin‐labeled proteins and Hsp70 in parental and G3BP1/2 knockout U2OS cells exposed to a heat shock at 43°C for 30 min (+ HS; left panel), and after 1 h recovery (HS + 1 h Recovery; right panel). Scale bar, 10 μm.Representative confocal images of immunofluorescent staining of HSF1 in parental and G3BP1/2 knockout U2OS cells directly after heat shock (+ HS) and 2 h after 2 h recovery (HS + 2 h Recovery). Scale bar, 10 μm.Quantification of the number of HSF1 foci per cell in images from (B), shown as a box plot with median and 5–95 percentiles (n = 3 independent experiments, > 300 cells analyzed per condition, Kruskal‐Wallis test, **P < 0.01).Western blot analysis of parental and G3BP1/2 knockout U2OS cells that were left untreated (− HS), heat‐shocked (HS), and followed for 1, 2, and 4 h after heat shock (Rec). The blots were probed with antibodies against HSF1 and GAPDH (* indicated phosphorylated HSF1, and ** indicates unphosphorylated HSF1).Analysis of HSP70 mRNA levels in parental and G3BP1/2 knockout U2OS cells that were left untreated (− HS), heat‐shocked (+ HS), and followed for 1, 2, and 4 h after heat shock (Rec.). mRNA levels were normalized to untreated samples. Data represent the geometric mean with 95% confidence interval of triplicate samples (Student's unpaired t‐test, **P < 0.01).

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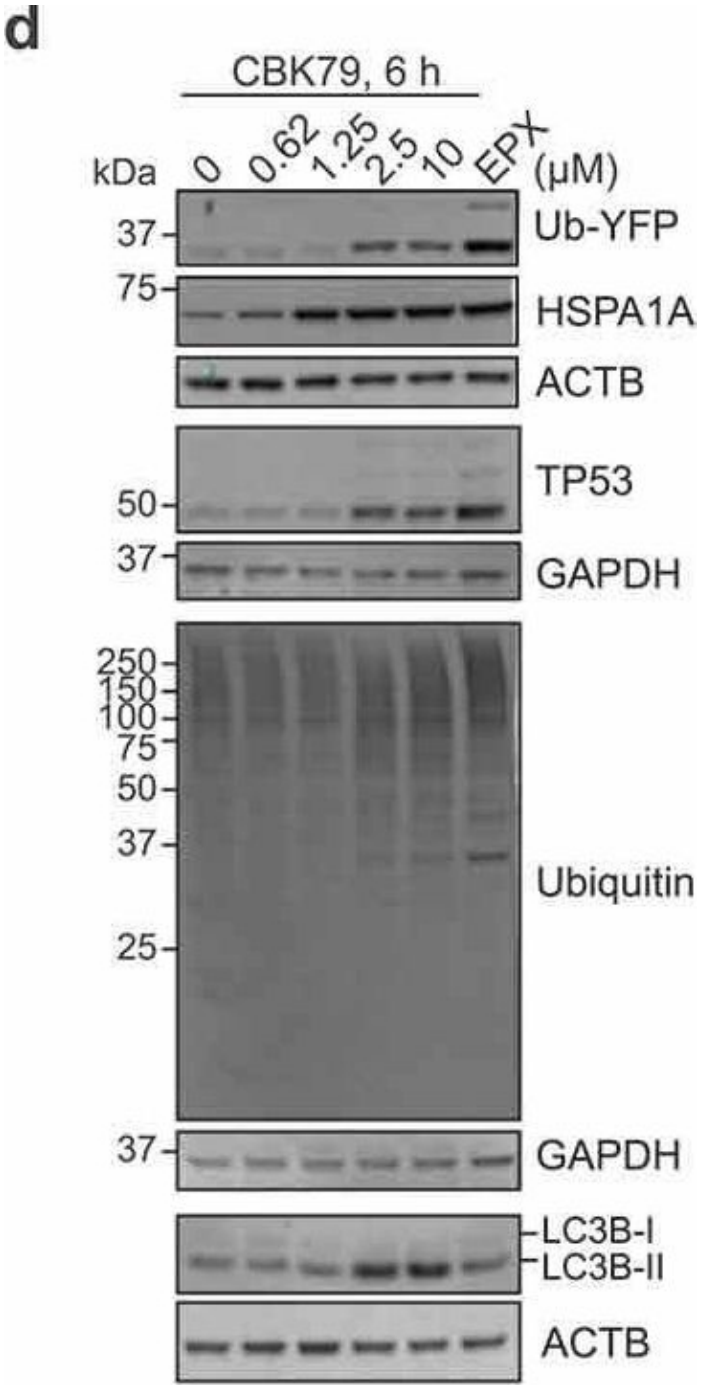
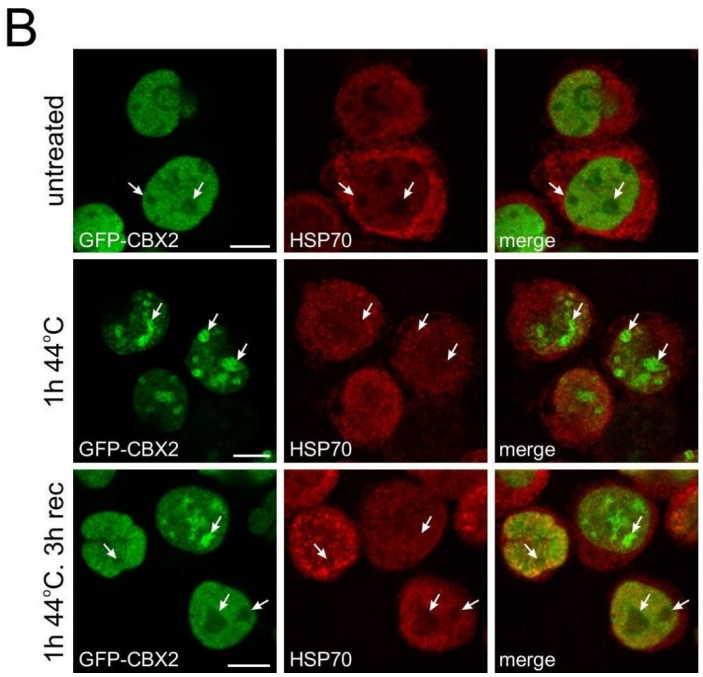
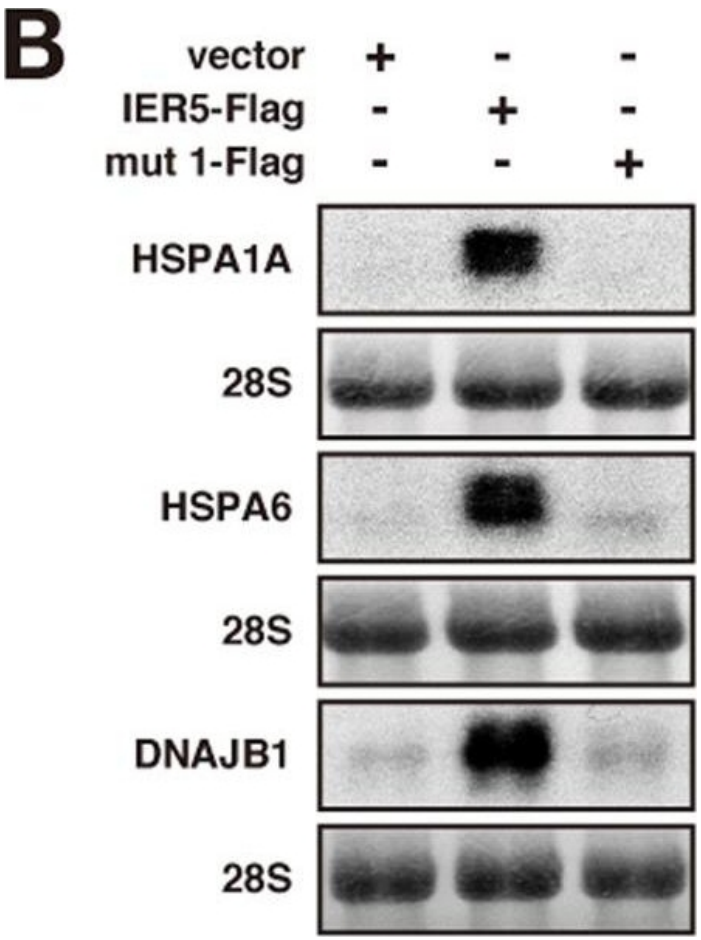


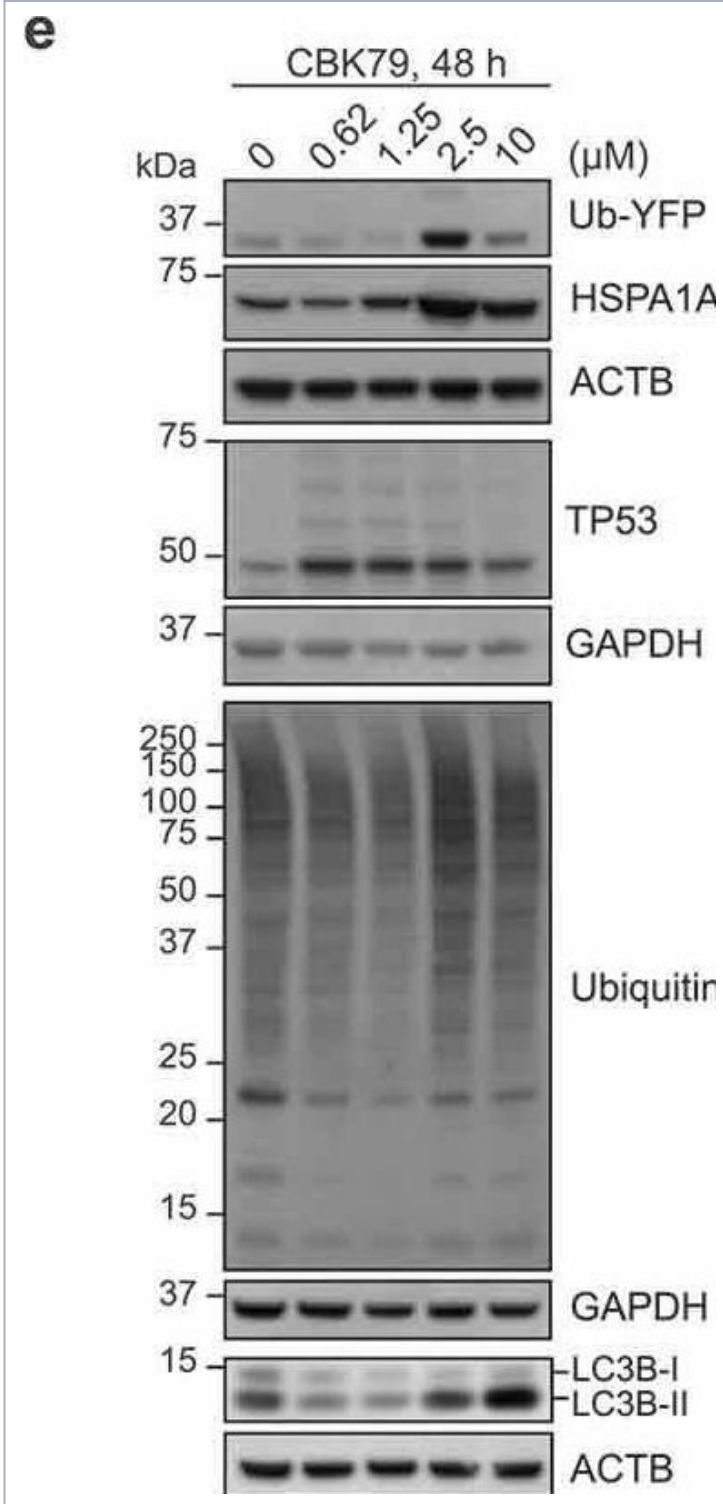
CBK79 induces the heat shock response. (A) HOS GFP-LC3B cells were treated with DMSO 0.1% (8 h), CBK79 10 μM (0 to 8 h; a representative image for the 2 h timepoint is shown or epoxomicin (EPX, 100 nM) for 8 h. Cells were fixed and immunostained with an HSF1 antibody. HSF1 nuclear foci are marked with white arrows. Scale bar: 20 μm. (B) The percentage of cells with HSF1 foci from one of two independent experiments are shown. (C) The number of HSF1 foci per cell were quantified using CellProfiler. Data from one of two independent experiments (n > 200 cells/condition) are shown as box plots with median and 5–95 percentiles. (D) MelJuSo Ub-YFP cells were treated with DMSO 0.1% (0) or the indicated concentrations of CBK79 for 6 h. Cell lysates were analyzed by immunoblotting with the indicated antibodies. Beta-actin (ACTB) is shown as loading control. Representative blots from one of three independent experiments are shown. (E) MelJuSo Ub-



GFP-CBX2, DNAJB1 and HSP70 show comparable HS-induced relocalization kinetics and post-HS GFP-CBX2 recovery is delayed upon HSP70 knockdown.(A) Confocal images of K562 GFP-CBX2 cells (untreated, directly after HS [1 hr, 44°C], or 3 hr after HS) stained with anti-DNAJB1. White arrows indicate the nucleoli. Scale bar represents 10 μm. (B) Confocal images of K562 GFP-CBX2 cells (untreated, directly after HS [1 hr, 44°C], or 3 hr after HS) stained with anti-HSP70. White arrows indicate the nucleoli. Scale bar represents 10 μm. (C) Fluorescent images of HEK293T GFP-CBX2 cells, either untreated, directly after HS (30', 44°C), 1 hr after HS, or 3 hr after HS. Scale bar represents 25 μm. (D) Quantification of post-HS (30', 44°C) GFP-CBX2 recovery in control and HSP70 siRNA transfected HEK293T GFP-CBX2 cells. Error bars indicate the mean ± SD calculated from independent microscopical images (n = 5; total cell number 175–460). Statistical analysis was performed using Student's t-test, *p<0.05, **p<0.01. (E) Western analysis of HEK293T GFP-CBX2 cells, transfected with control or HSP70 siRNAs, both untreated and 3 hr after HS (30', 44°C), using HSPA1A/HSP70 and GAPDH antibodies.



HSP family genes are induced by IER5.(A) H1299 or 293T cells were transfected with control vector or an IER5 expression vector. Cells were harvested 21 hrs or 27 hrs post-transfection and microarray expression analysis was performed. The table shows the HSP family genes, among the genes induced by IER5. (B) H1299 cells were transfected with control, IER5-Flag or mutant IER5-Flag expression vectors (representative image of mut 1 is shown in Fig. S1). Cells were harvested 27 hrs post-transfection, and mRNA expressions of the HSP family genes were analyzed by Northern blotting. (C) H1299 and 293T cells were transfected with control vector or IER5-Flag expression vector, and cells were harvested 24 hrs post-transfection. Expressions of the HSP family proteins were analyzed by Western blotting. (D–F) Control or IER5-targeting siRNAs were introduced into OE33 cells. Cells were harvested 52 hrs post-transfection. Expression of IER5 (D,F) and HSPA1A (E,F) were analyzed by quantitative RT-PCR (D,E) and Western blotting (F). (**p < 0.01). (G) The promoter regions of HSPA1A, HSPA1B and HSPA6 were inserted into the luciferase reporter plasmid containing a minimal promoter, and assayed 24 hrs post-transfection. Experiments were run in triplicate, and data are represented as the mean-fold activation ±SD. (H) Serially deleted regions of the HSPA1A promoter were analyzed as in (G). Numbers indicate the position of the 5′ most nucleotide relative to the transcription initiation



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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	Electron microscopy, ELISA, Flow Cytometry, ICC, IF, IHC, IP, WB
Application Notes	Detects a band of ~70kDa by Western blot.
Clone	C92F3A-5
Formulation	Liquid. In PBS 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	Mouse
Immunogen	Native human Hsp70 protein.
Isotype	IgG1
Purity Detail	Protein G affinity purified.
Recommendation Dilutions/Conditions	Immunohistochemistry (1:50)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 ascites.

Species Reactivity	Beluga, Bovine, C. elegans, Chicken, Dog, Drosophila, Fish, Gerbil, Guinea pig, Hamster, Human, Monkey, Mouse, Porcine, Rabbit, Rat, Sheep, Teal, Xenopus
Technical Info / Product Notes	**Cited samples:** *For an overview on cited samples please click here.*
UniProt ID	P0DMV8 (HSPA1A), P0DMV9 (HSPA1B)
Worry-free Guarantee	**This antibody is covered by our Worry-Free Guarantee .**

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ENZO LIFE SCIENCES, INC.
Phone: 800.942.0430
info-usa@enzolifesciences.com

European Sales Office
ENZO LIFE SCIENCES (ELS) AG
Phone: +41 61 926 8989
info-eu@enzolifesciences.com

Belgium, The Netherlands & Luxembourg
Phone: +32 3 466 0420
info-be@enzolifesciences.com

France
Phone: +33 472 440 655
info-fr@enzolifesciences.com

Germany
Phone: +49 7621 5500 526
info-de@enzolifesciences.com

UK & Ireland
Phone (UK customers): 0845 601 1488
Phone: +44 1392 825900
info-uk@enzolifesciences.com