HSF1 polyclonal antibody

HSFs (Heat Shock family of transcription factors), which consists of HSF 1-4, bind to highly conserved Heat shock elements (HSEs) in the promoter regions of heat shock genes, ultimately regulating the expression of Heat shock proteins (Hsps). On exposure to heat shock and other stresses, HSF1 localizes within seconds to discrete nuclear granules and on recovery from stress, HSF1 rapidly dissipates from the stress granules to a diffuse nucleoplasmic distribution.

This antibody is covered by our Worry-Free Guarantee.

Citations: 82 View Online »

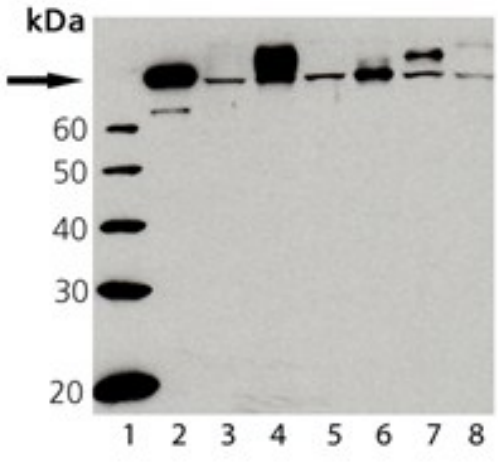
Ordering Information

Order Online »

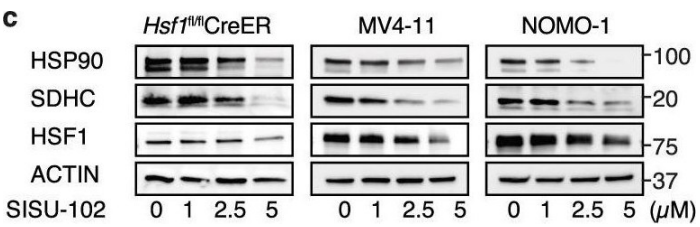
ADI-SPA-901-J	1mg
ADI-SPA-901-D	50µg
ADI-SPA-901-F	200µg

Manuals, SDS & CofA

View Online »

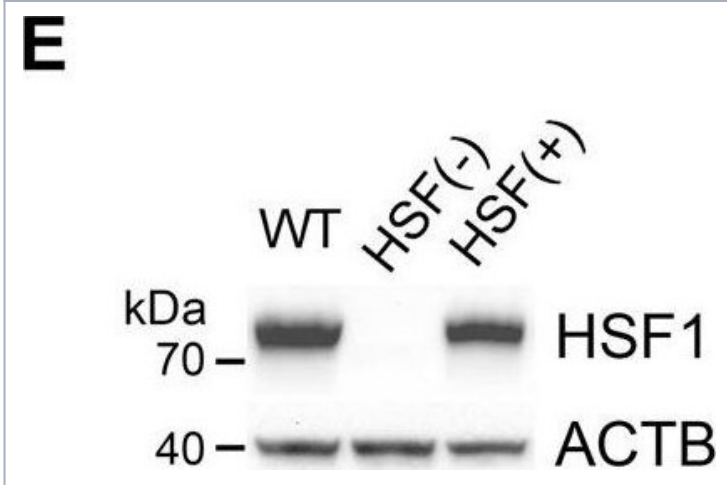


Western blot analysis of HSF1: Lane 1: Molecular Weight Marker, Lane 2: HSF1 (human), (recombinant) (Prod. No. ADI-SPP-900), Lane 3: HeLa Cell Lysate (Prod. No. ADI-LYC-HL100), Lane 4: HeLa Cell Lysate (Heat Shocked) (Prod. No. ADI-LYC-HL101), Lane 5: 3T3 Cell Lysate (Prod. No. ADI-LYC-3T100), Lane 6: PC-12 Cell Lysate (Prod. No. ADI-LYC-PC100), Lane 7: RK-13 Cell Lysate, Lane 8: Vero Cell Lysate



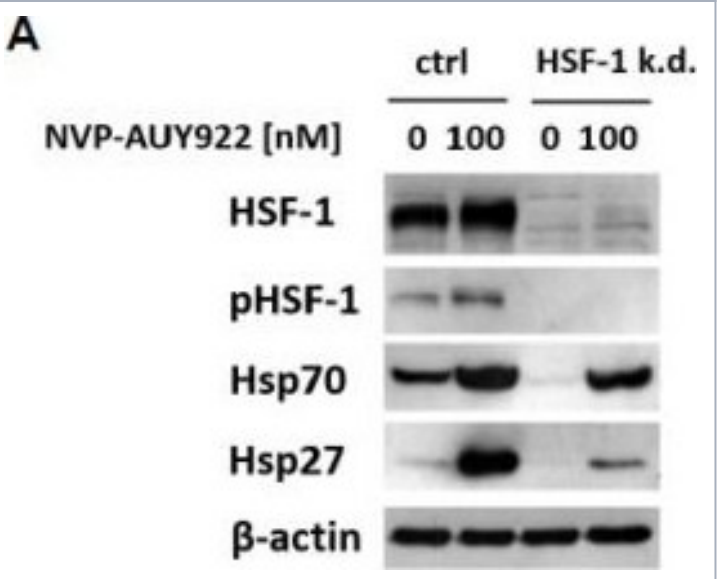
HSF1 is critical for maintenance of LSC self-renewal.a Engraftment of three different primary AML LSCs in the presence or absence of CRISPR-mediated HSF1KD (started 4 mice/AML). *p = 0.0404, **p = 0.0003, ***p = 0.0006. b ATP production in control or HSF1 knockdown AML cells (post-transplantation from a) measured by Seahorse assay (n = 3 independent replicates). AML-a: *p = 0.0046. **p = 0.0349; AML-b: *p = 0.0201. **p = 0.0124; AML-c: *p = 0.0325. **p = 0.0033; c The expression of HSP90, SDHC, and HSF1 proteins in SISU-102 treated mouse MLL-AF9 cells and the human AML cell lines MV4–11 and NOLM-1 (n = 3 independent replicates). ACTIN is used as a load control. d Cell growth of murine MLL-AF9 cells (*p = 1.6 × 10−5, **p = 3.1 × 10−5, ***p = 6.1 × 10−5) and human AML cell line MV4–11 (*p = 4.8 × 10−3, **p = 2.3 × 10−4, ***p = 1.6 × 10−4) and NOLM-1 (*p = 6.3 × 10−4, **p = 3.01 × 10−5, ***p = 6.38 × 10−5) in the presence of absence of SISU-102 (n = 3 independent replicates). e OCR in Hsf1fl/flcreER LSCs (*p = 8.87 × 10−31, **p = 1.09 × 10−7) or human AML cell line MV4–11 (*p = 4.89 × 10−13, **p = 4.97 × 10−12) and NOLM-1 (*p = 1.19 × 10−18, **p = 0.00028) treated with or without SISU-102 (n = 8 independent replicates). f Engraftment of human AML cell line MV4–11 (n = 5 mice/group, *p = 9.8 × 10−5) or three different primary AML cells in the presence or absence of SISU-102. AML-d: *p = 1.8 × 10−5, n = 4 mice/group, and AML-e: *p = 8.98 × 10−6, Con n = 4 mice, SISU-102, n = 5 mice; AML-f: *p = 0.0011, Con n = 5 mice, SISU-102, n = 4 mice. g Engraftment of human BM CD34+ HSPCs treated with (n = 4 mice) or without (n = 3 mice) SISU-102 (5 mg/kg, IP, daily). In (a, b, d–f), two-tailed t test was used, data are presented as mean values ± SEM. Source data are provided as a Source Data file.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: HSF1 is a driver of leukemia stem cell self-renewal in acute myeloid leukemia. *Nat Commun* (2022)



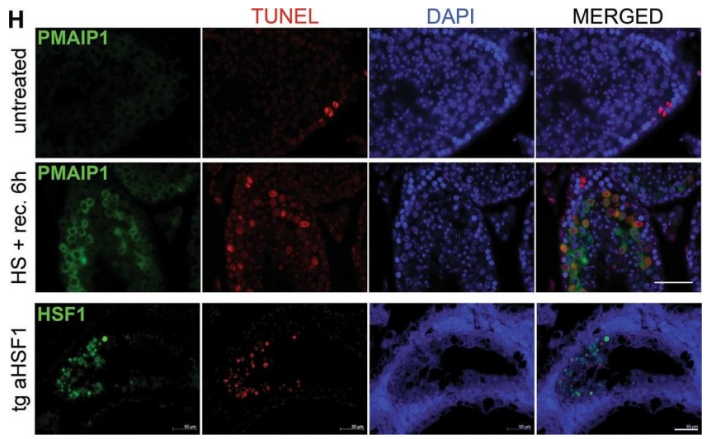
HSF1 and HSEs present in the introns of Pmaip1 gene are essential for its activation after heat shock.a Heat shock-induced HSF1 binding in introns of Pmaip1 analyzed by ChIP-qPCR in wild-type (WT) HECa10 cells and the clone with hemideletion (1/2HSE) of the perfect HSE in the second intron. b RT-qPCR assays of Pmaip1 and Hspa1a genes transcript levels after heat shock (HS) in cells described in panel a. Fold changes in reference to untreated cells are shown. c PMAIP1 level in the same cells analyzed by western blot. CPT treatment served as positive control for PMAIP1 upregulation. ACTB is shown as a control for loading. Lower panel shows the representative results of densitometric analyses of western blots; *p < 0.05. d Relative luciferase activity in the human 1205Lu cells stably expressing constitutively active HSF1 (aHSF1) in relation to control cells with the empty vector (Neo). Cells were transiently transfected with: the pGL3-Promoter vector (a), its derivatives with the part of the second intron of the mouse Pmaip1 gene acting as an enhancer, containing either wild-type (a1) or mutated HSE (a2), and the vector with the HSPA7 promoter (b) used as a positive control. Sequences of wild-type HSE from the second intron of mouse Pmaip1 (nucleotides 93–112 downstream from the exon2/intron2 boundary) and mutated HSE (mutHSE) are shown above the graph. Hats indicate the most essential G and C nucleotides in the HSE sequence. Presented are mean values and ± SD from three independent experiments (with three-five technical repeats each); *p < 0.05. e HSF1 protein levels detected by western blot documenting the complete HSF1 knockout (−) obtained in RKO cells by CRISPR/Cas9 editing. ACTB is shown as control for loading. f RT-qPCR assays of PMAIP1 and HSPA1A transcript levels after heat shock treatment in HSF1(+) (mix of control clones) and HSF1(−) (one of six individual clones; the same result was obtained for all clones) RKO cells. **p < 0.001.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Pro-death



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 ≤ 0.05; ** p ≤ 0.01; *** p ≤ 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 ≤ 0.05; ** p ≤ 0.01; *** p ≤ 0.001.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Radiosensitization of HSF-1 Knockdown Lung Cancer Cells by Low Concentrations of Hsp90 Inhibitor NVP-AUY922. *Cells* (2019)



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

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	HSTF1, Heat shock factor protein 1
Application	ELISA, EMSA, IP, WB
Application Notes	Detects bands ~80 to ~95kDa (depending on phosphorylation status) by Western blot analysis.
Formulation	Liquid. In PBS containing 50% glycerol and 0.09% sodium azide.
GenBank ID	M64673
Host	Rabbit
Immunogen	Recombinant human HSF1.
Purity Detail	Protein A affinity purified.
Recommendation Dilutions/Conditions	Western Blot (1:1,000, colorimetric)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	Gerbil, Human, Monkey, Mouse, Rabbit, Rat
Technical Info / Product Notes	ADI-SPA-901 is tested against HeLa and HeLa Heat-Shocked lysates. Upon heat shock, HSF1 is hyperphosphorylated. ADI-SPA-901 recognizes both phosphorylated and non-phosporylated HSF1 in Western Blot.

UniProt ID Q00613

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

Last modified: May 29, 2024

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

For local distributors and detailed product information visit us online: enzo.com All product names, logos, and brands are the property of their respective owners.