HSP90 monoclonal antibody (16F1)

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: 58 View Online »

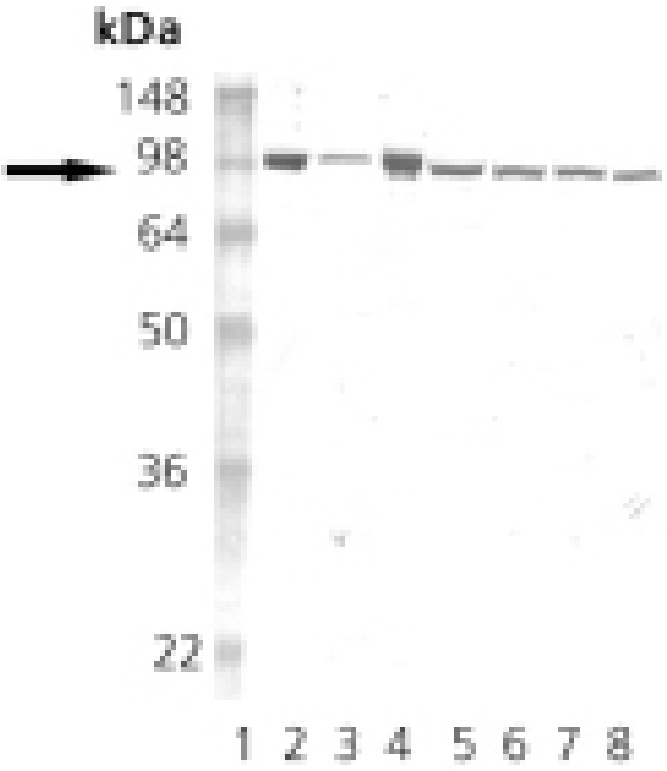
Ordering Information

Order Online »

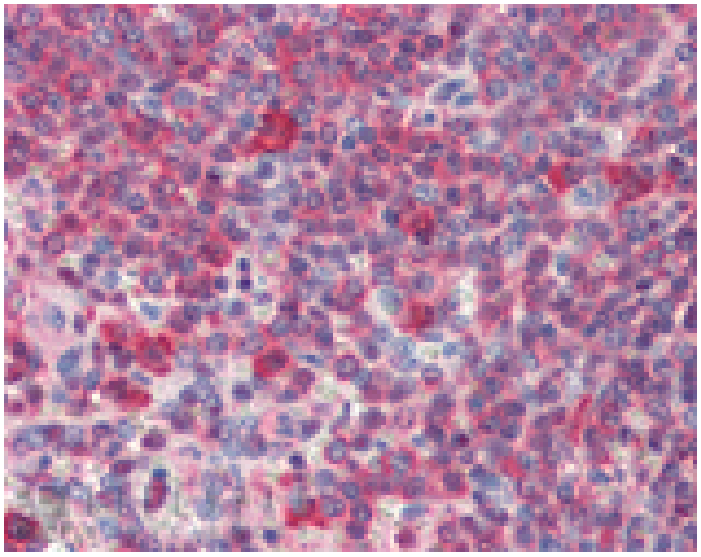
ADI-SPA-835-D	50µg
ADI-SPA-835-F	200µg

Manuals, SDS & CofA

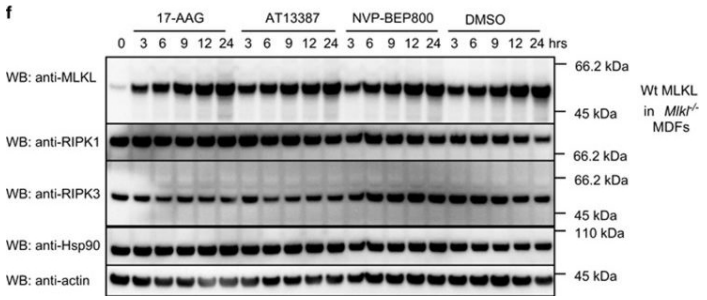
View Online »



Western blot analysis of HSP90: Lane 1: MW marker, Lane 2: HSP90 native protein (ADI-SPP-770), Lane 3: HSP90b recombinant protein (Prod. No. ADI-SPP-772), Lane 4: HSP90alpha recombinant protein (Prod. No. ADI-SPP-776), Lane 5: HeLa (HS), Lane 6: L-929 (HS), Lane 7: Rat-2 (HS), Lane 8:RK-13 (HS).

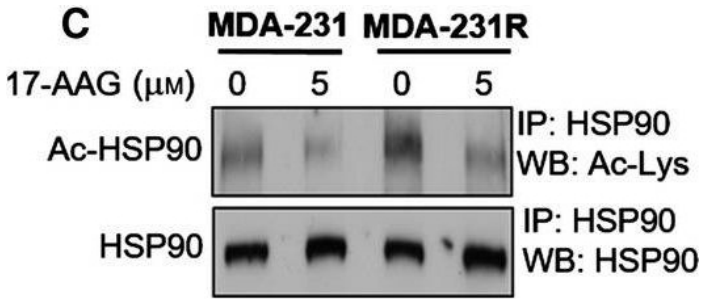


Immunohistochemistry analysis of human spleen tissue stained with HSP90, mAb (16F1) at 10µg/ml.



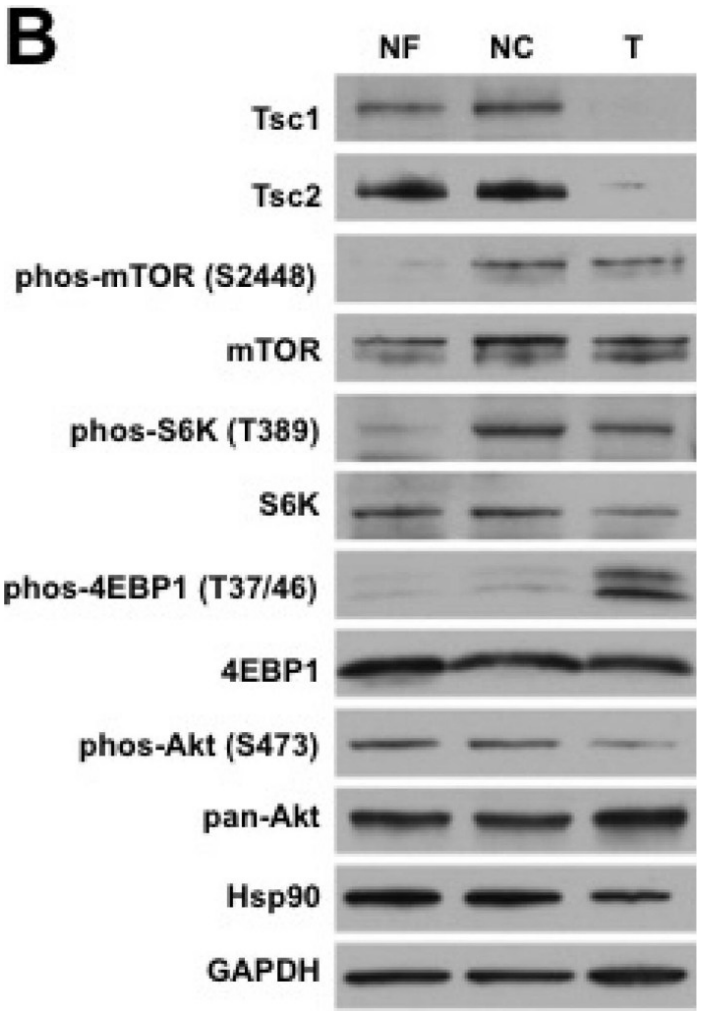
Hsp90 inhibition affects MLKL activity. (a–c) Wild-type (a), Mlkl−/− (b) or Ripk3−/− (c) MDFs were pretreated for 1 h with AT13387 (1 μM), NVP-BEP800 (125 nM), 17-AAG (250 nM) or DMSO, then expression of MLKL(1–180) was induced using 50 ng/ml doxycycline. After 24 h, PI uptake was measured using flow cytometry. Two experiments were performed using three independent cell lines (n=6). (d) Mlkl−/−Ripk3−/− MDFs were pretreated with HSP90 inhibitors as described above, then expression of MLKL S345D was induced using 50 ng/ml doxycycline. After 24 h, PI uptake was measured using flow cytometry. Two experiments were performed using three independent cell lines (n=6). (e and f) Cells were pretreated for 1 h with AT13387 (1 μM), NVP-BEP800 (125 nM), 17-AAG (250 nM) or DMSO, then expression of MLKL S345D in Mlkl−/−Ripk3−/− double knockout MDFs (e) or endogenous MLKL in Mlkl−/− MDFs (f) was induced using 50 ng/ml doxycycline. Treatment was performed over 24 h at the times shown, then cell lysates analysed with western blotting using the indicated antibodies. Data are representative of three independent experiments

Image collected and cropped by CiteAb under a CC-BY license from the following publication: HSP90 activity is required for MLKL oligomerisation and membrane translocation and the induction of necroptotic cell death. *Cell Death Dis* (2016)

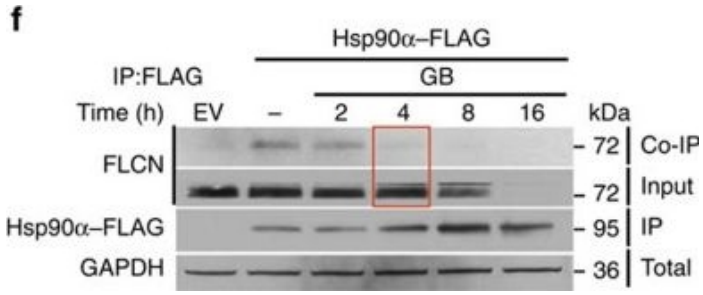


Altered NQO1 levels, HDAC family member expression and altered acetylation status in 17□AAG□resistant cell lines. Analysis of parental and resistant cell lines demonstrated altered expression levels of a number of molecules. Semiquantitative PCR demonstrated that the expression levels of NQO1 in resistant MDA□435 cells were decreased when compared with parental cells, while no alteration was noted between MDA□231 and MDA□231R cell lines (A). Western blot analysis of parental and resistant MDA□231 total cell lysates examining levels of HDAC family members in the presence and absence of 17□AAG for a period of 24 h (B). Analysis of acetylated HSP90 by immunoprecipitation of HSP90 and western blot analysis with antiacetylated lysine antibody of total cell lysates of parental and resistant MDA□231 cells treated with and without 17□AAG demonstrated increased acetylated HSP90 (C). Analysis of acetylation of Grp94 (D) and Trap1 (E) by immunoprecipitation and western blot analysis of MDA□231 and MDA□231R total cell lysates demonstrated no alteration in acetylation status. Acetylated lysine residue was detected by western blotting. Western blot analysis of acetylated histone 3 in parental and resistant MDA□231 cells treated with and without 17□AAG demonstrated decreased nuclear acetylation (F).

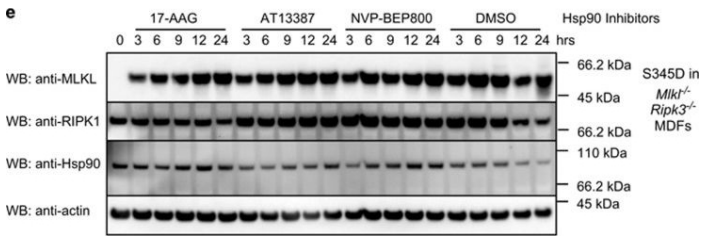
Image collected and cropped by CiteAb under a CC-BY license from the following publication: Histone deacetylase activity mediates acquired resistance towards structurally diverse HSP90 inhibitors. *Mol Oncol* (2017)



Sporadic renal AML demonstrates somatic loss of Tsc1/2 expression(A) Protein was extracted from adjacent normal far (NF) and close (NC) kidney and tumor (T). Expression of FLCN was examined by immunoblotting. GAPDH was used as a loading control. Short (SE) and long (LE) exposure of the radiographic film. (B) Protein was extracted from adjacent normal far (NF) and close (NC) kidney and tumor (T). Expression of Tsc1/2 and mTOR pathway components was examined by immunoblotting. GAPDH was used as a loading control.



Folliculin is a new client of Hsp90.(a) FLAG–FLCN was expressed and isolated from HEK293 cells. Profile of interacting proteins determined by MALDI–time of flight. Red nodes represent chaperones and co-chaperones, blue nodes are chaperonins and green nodes are splicing factors and ribosomal proteins. (b) FLCN was isolated from HEK293 cell lysates using anti-FLCN or IgG (control) and immunoblotted with indicated antibodies to confirm protein interactions. (c) HEK293 cells were transiently transfected with FLAG–FLCN or empty vector control (EV), immunoprecipitated and immunoblotted with indicated antibodies to confirm interacting proteins. (d) HEK293 cells were treated with 10 μM of the Hsp70 inhibitor JG-98 at the indicated time points. FLCN protein stability in soluble and insoluble fraction was assessed by immunoblotting. (e) HEK293 cells were treated with 1 μM GB at the indicated time points. FLCN protein stability was assessed by immunoblotting. Akt and Phospho-S473-Akt were used as positive controls. (f) Hsp90α–FLAG was transiently expressed in HEK293 cells. Cells were treated with 1 μM GB for the indicated times. Hsp90α–FLAG was immunoprecipitated and co-IP of FLCN was examined by immunoblotting. (g) HEK293 cells were treated with 50 nM of the proteasome inhibitor bortezomib (BZ) for the indicated times. FLCN protein levels were evaluated at the indicated time points by immunoblotting (upper blots). HEK293 cells were also treated with 1 μM GB for 1 h before addition of 50 nM BZ. Immunoblotting was used to evaluate the FLCN level for the indicated time points (lower blots). (h) Empty vector (EV) or FLAG–FLCN was used to transiently transfect HEK293 cells for 24 h then treated for 4 h with either 50 nm BZ or 1 μM GB. FLAG–FLCN was immunoprecipitated and ubiquitination was examined by immunoblotting with a pan-anti-ubiquitin antibody.



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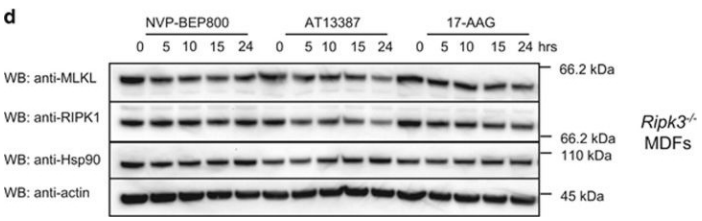

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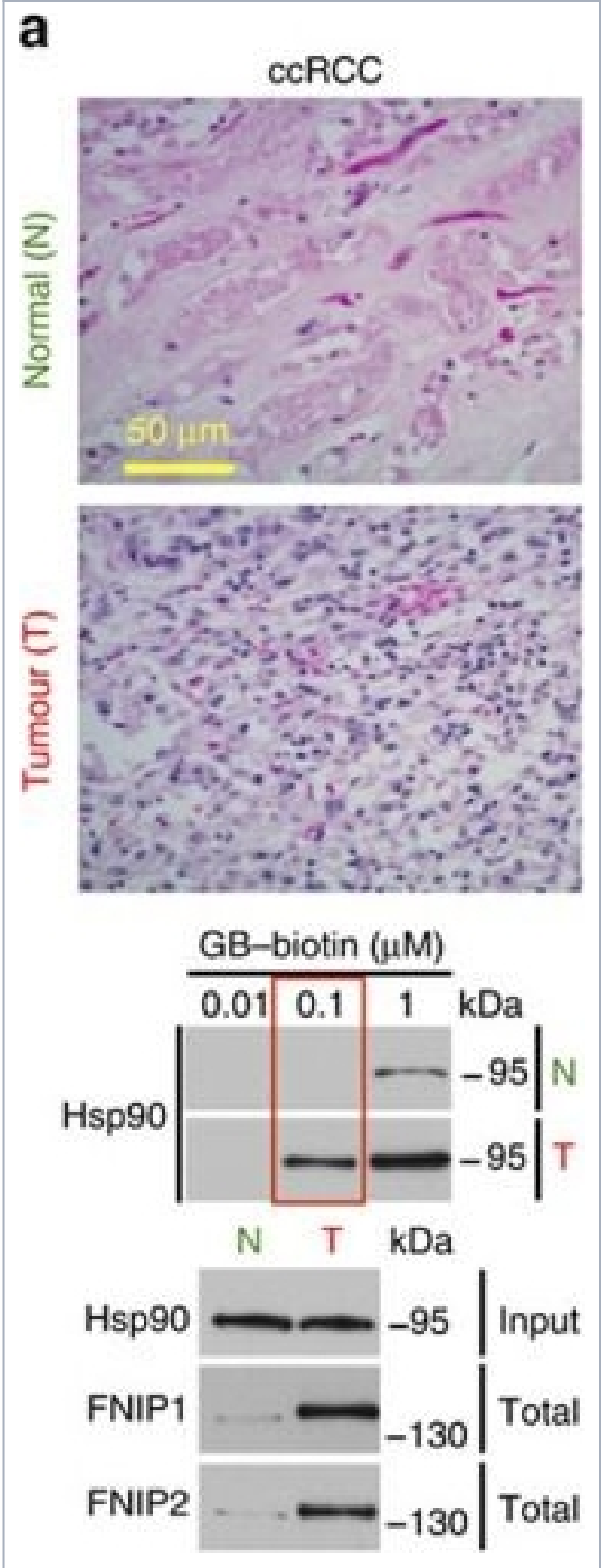
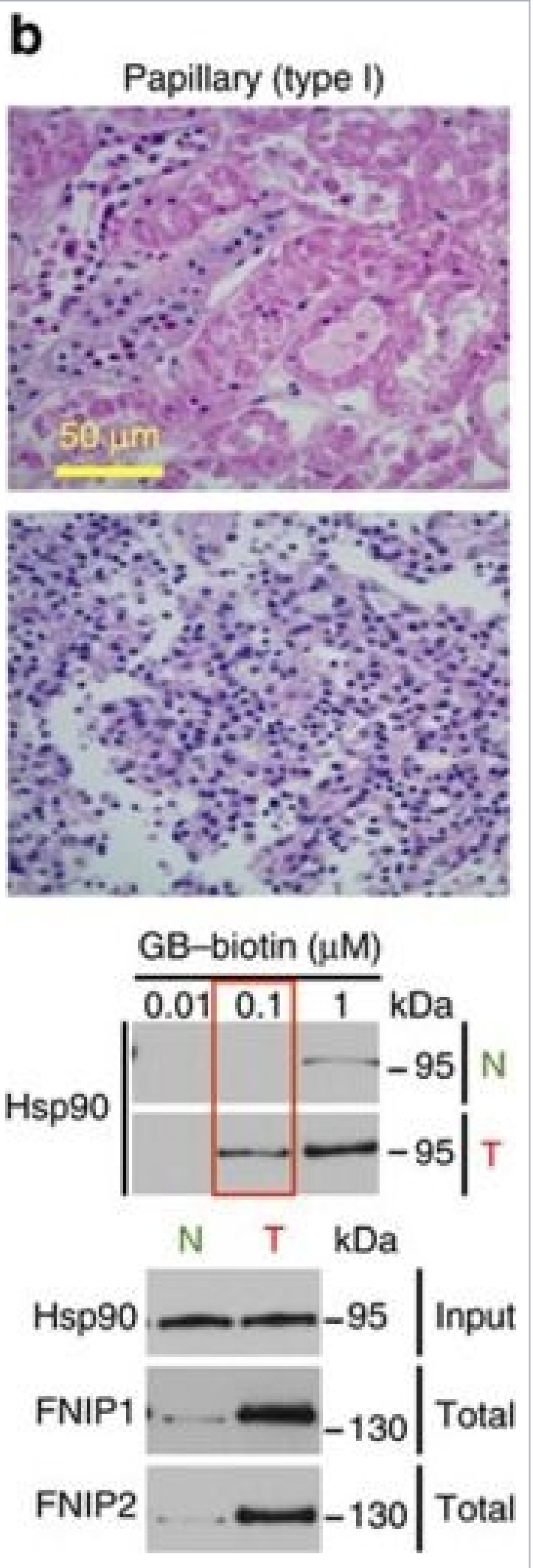
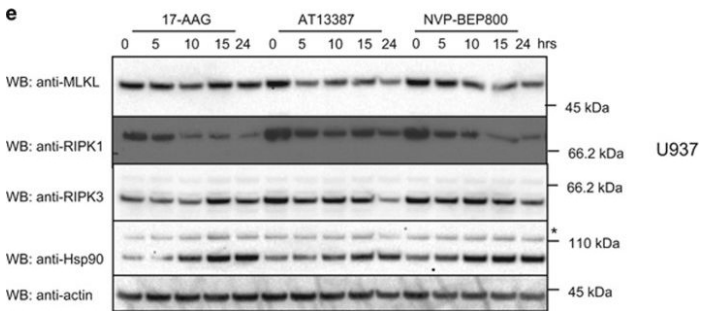


High levels of FNIPs make renal tumours sensitive to Hsp90 inhibitor GB.(a) Clear cell renal cell carcinoma (ccRCC), (b) Papillary type I, (c) Papillary type II, (d) Oncocytoma (Tumours, T) and adjacent normal tissues (Normal, N) were stained with haematoxylin and eosin (H&E). Proteins were also extracted from these tumours and adjacent normal tissues and incubated with



High levels of FNIPs make renal tumours sensitive to Hsp90 inhibitor GB.(a) Clear cell renal cell carcinoma



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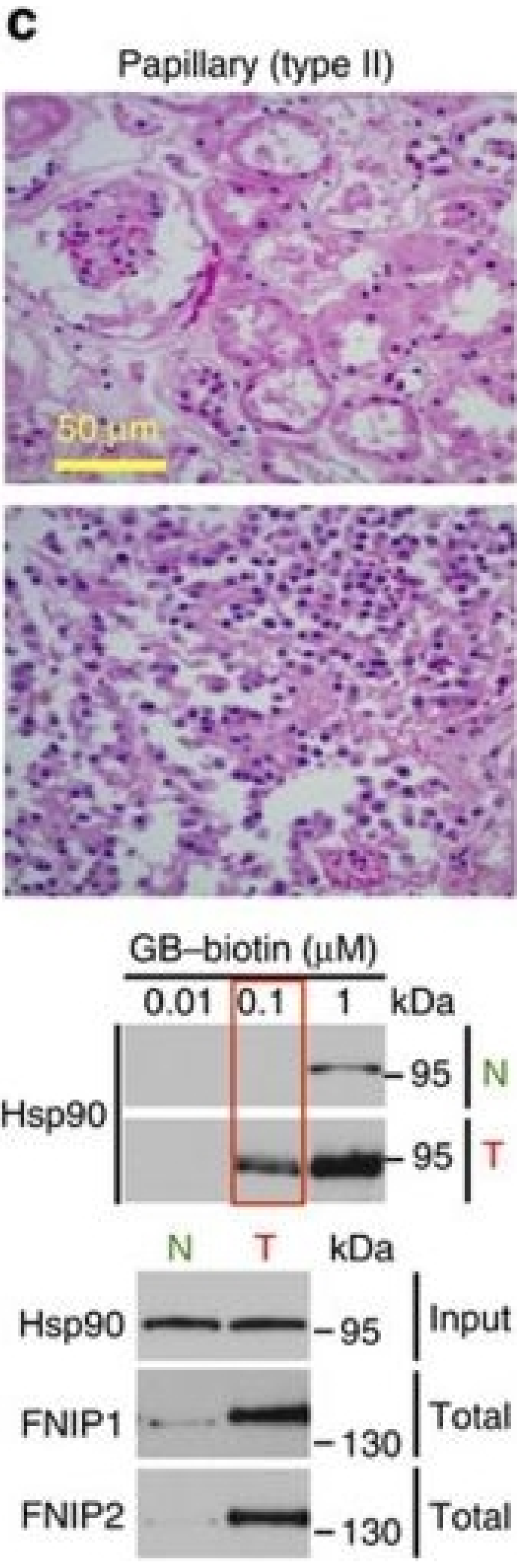

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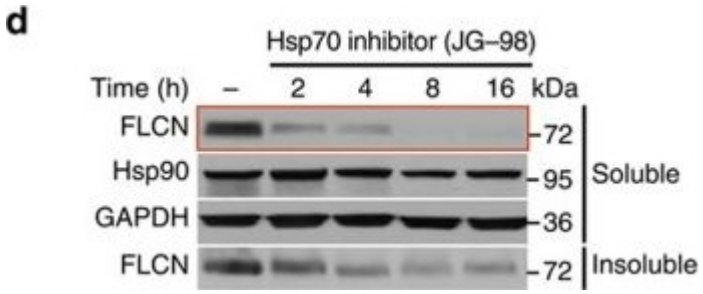

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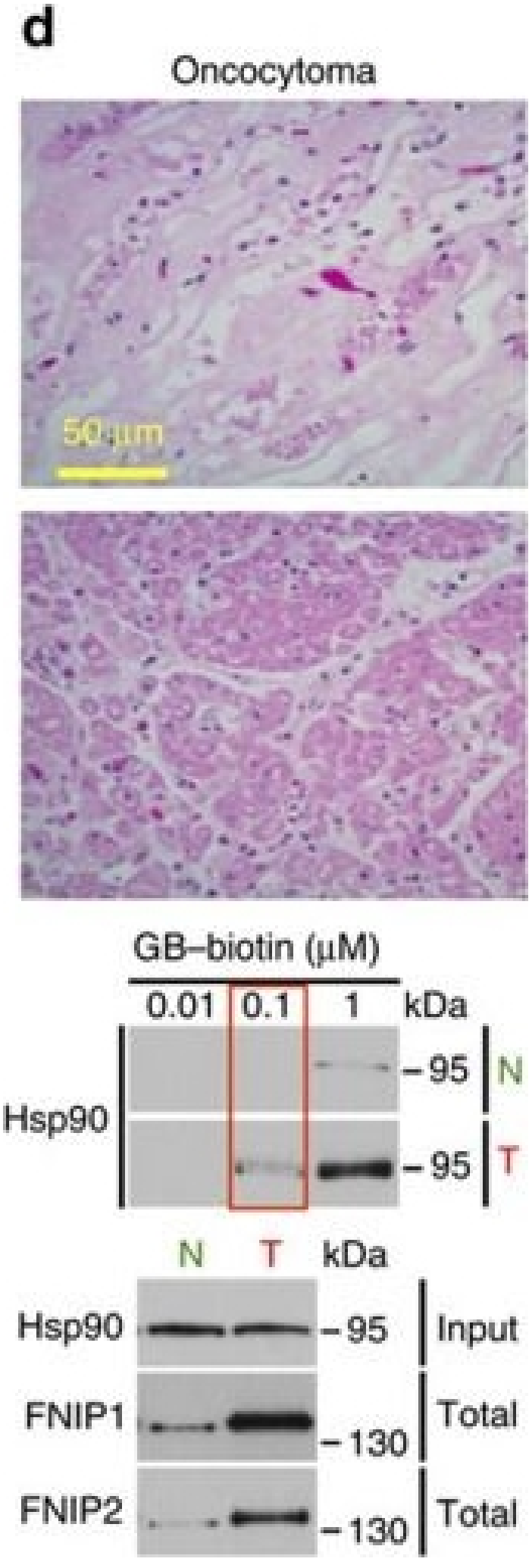

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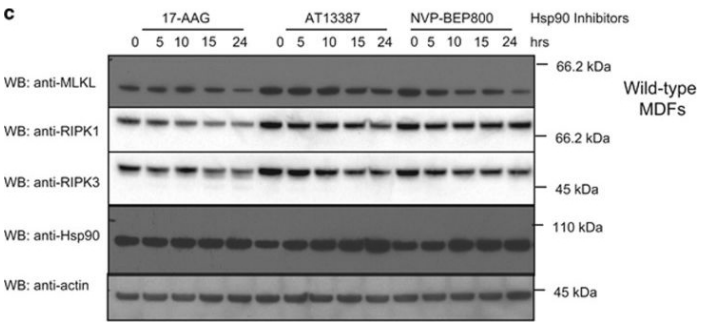

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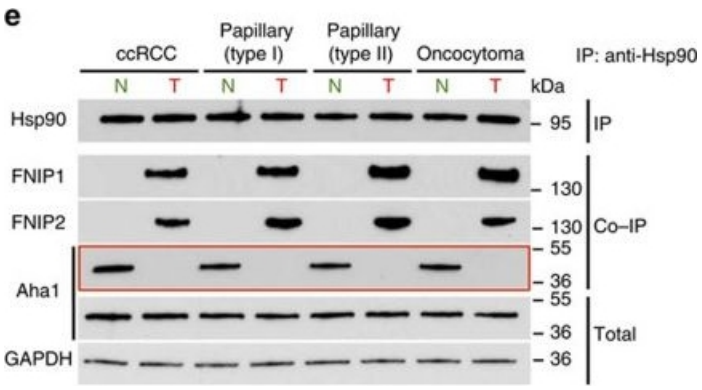

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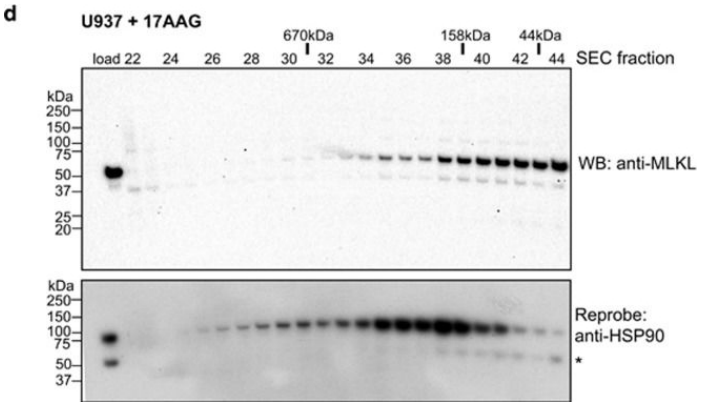
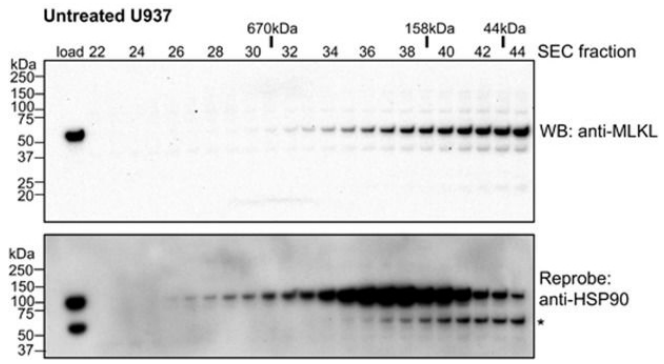


MLKL transiently interacts with HSP90 via the Cdc37 co-chaperone. (a and b) Mlkl−/−Ripk3−/− MDFs stably transduced with a lentiviral construct encoding S345D MLKL were untreated, treated with transfection reagent only, or transfected with scrambled or Cdc37 siRNA pools. Cdc37 knockdown was observed in Cdc37 siRNA-treated cells relative to untreated, transfection reagent and scrambled siRNA-treated controls by western blot (a). Only Cdc37 siRNA knockdown conferred protection from S345D MLKL-mediated death (b). (c and d) Lysates of U937 cells incubated with DMSO (c) or 17-AAG (500 nM, d) were resolved by Superose-6 10/300 size-exclusion chromatography (SEC). Fractions were subjected to SDS-PAGE and western blotted for MLKL (upper panels) before reprobing for HSP90 (lower; * corresponds to residual signal from MLKL blots). The SEC fraction number is shown above the blots along with the elution position of molecular weight standards



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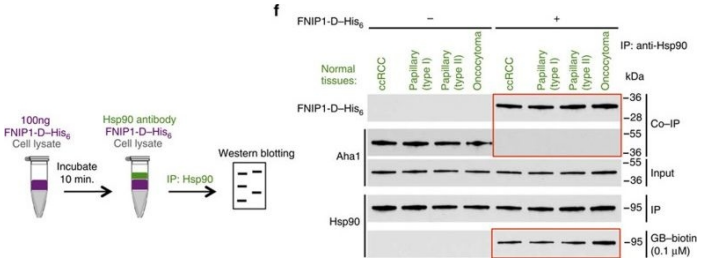

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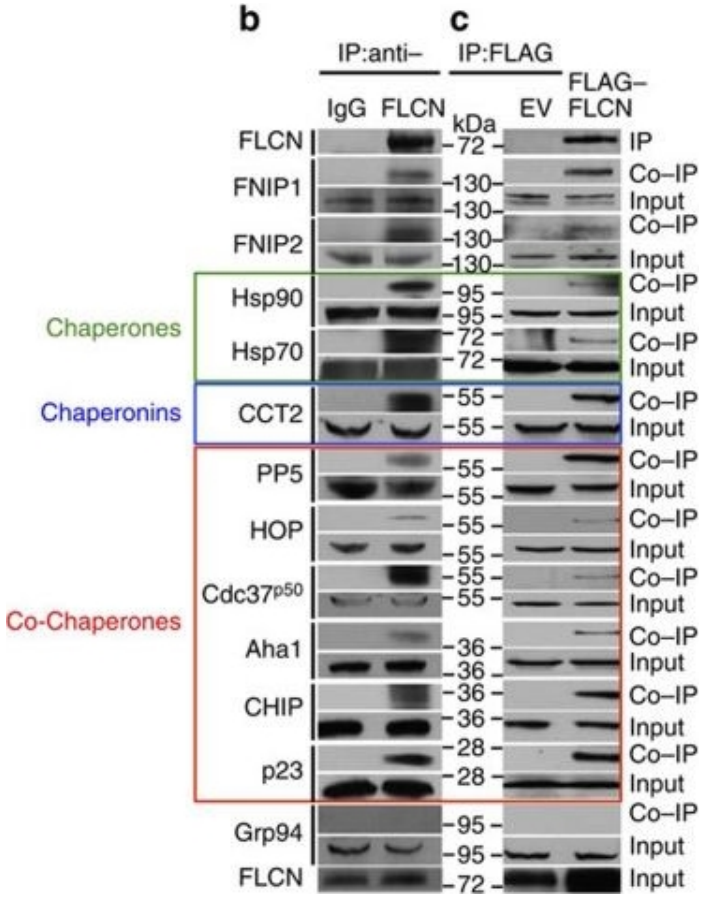

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Handling & Storage

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

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	Heat shock protein 90
Application	IHC (PS), IP, WB
Application Notes	Detects a band of ~90kDa by Western blot.
Clone	16F1
Formulation	Liquid. In PBS, pH 7.2, containing 50% glycerol and 0.09% sodium azide.
Host	Rat
Immunogen	Native human Hsp90.
Isotype	IgG2a
Purity Detail	Protein G 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 ascites.
Species Reactivity	Beluga, Bovine, Chicken, Dog, Drosophila, Fish, Guinea pig, Hamster, Human, Monkey, Mouse, Mussel, Opossum, Plant, Porcine, Rabbit, Rat, Scallop, Sheep, Xenopus
UniProt ID	P07900 (HSP90alpha), P08238 (HSP90beta)

Worry-free Guarantee

This antibody is covered by our Worry-Free Guarantee .

Last modified: May 29, 2024

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info-usa@enzolifesciences.com

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