

HSP90 polyclonal antibody

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: 22

[View Online »](#)

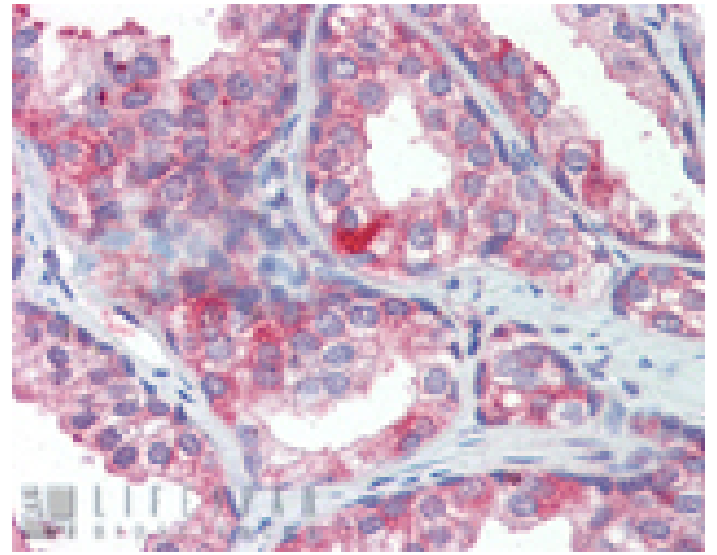
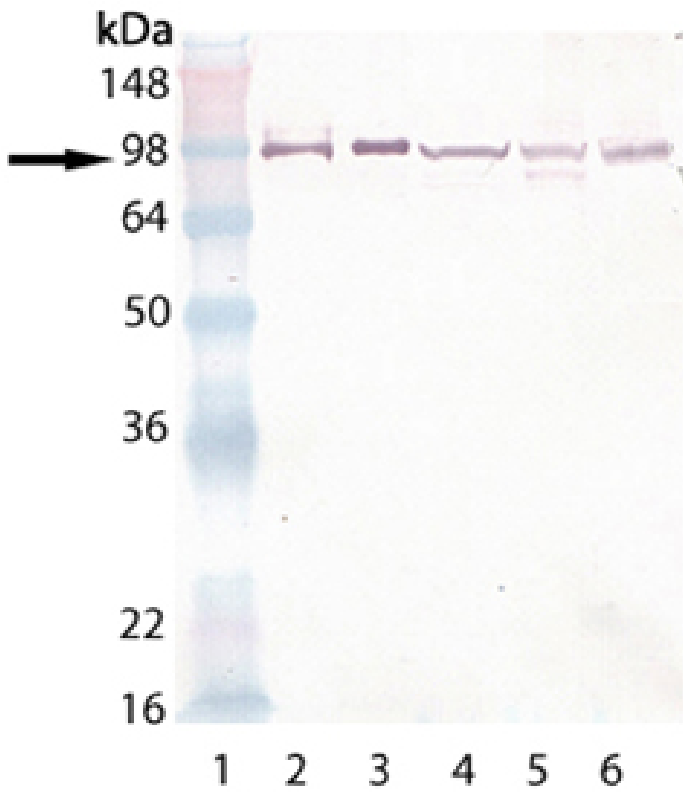
Ordering Information

[Order Online »](#)

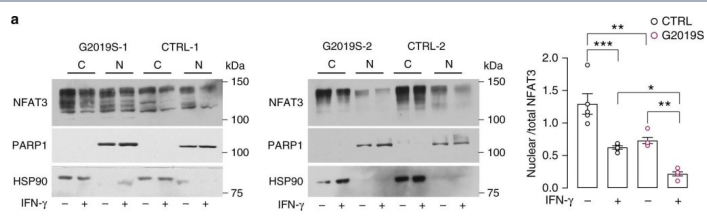
ADI-SPA-836-F	200µg
---------------	-------

Manuals, SDS & CofA

[View Online »](#)

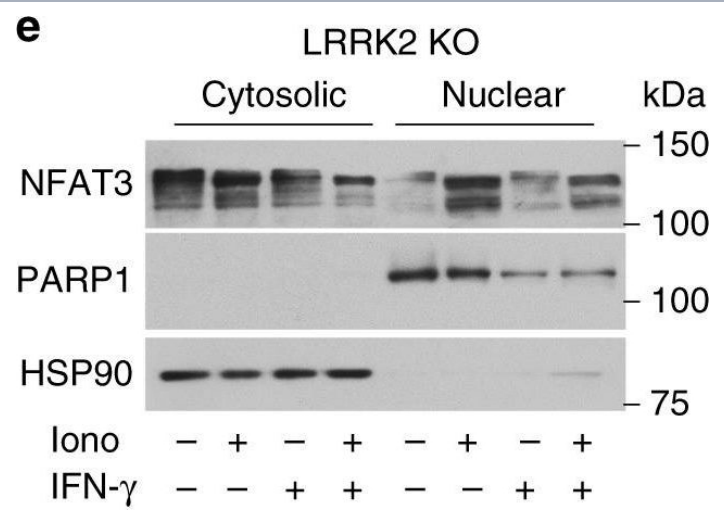


Immunohistochemistry analysis of human prostate tissue stained with HSP90, pAb at 10 μ g/ml.

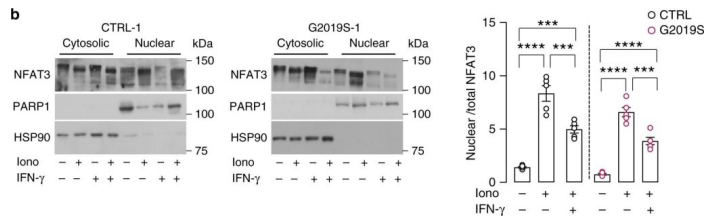


a LRRK2 inhibits nuclear NFAT3 shuttling in human neurons. **a** Representative Western blots of NFAT3 in nuclear (N) and cytosolic (C) fractions of LRRK2 G2019S neurons and isogenic controls (left and middle panel). Treatment with 200 IU/mL IFN- γ is indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, *** P = 0.0003, ** P = 0.0015, 0.0035 in sequence, * P = 0.0226; n = 5 independent experiments). **b** Representative Western blots of NFAT3 in N and C fractions of control (left panel) and isogenic LRRK2 G2019S neurons (middle panel). Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, **** P < 0.0001, *** P = 0.0005, 0.0008, 0.0002 in sequence; n = 5 independent experiments). **c** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in LRRK2 G2019S neurons and isogenic controls. Treatments with 200 IU/mL IFN- γ are indicated (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, *** P = 0.0009, * P = 0.0421; n = 5 independent experiments). **d** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in control iPSC-derived neurons upon treatment with 200 IU/mL IFN- γ for 24 h, with and without treatment with a proteasome inhibitor (MG132, 20 nM for 24 h) (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, * P = 0.0399, 0.0437 in sequence; n = 3 independent experiments). **e** Representative Western blots of NFAT3 in nuclear and cytosolic fractions of LRRK2 KO neurons. Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, **** P < 0.0001; n = 5 independent experiments).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Interferon- γ signaling synergizes with LRRK2 in neurons and microglia derived from human induced pluripotent stem cells. *Nat Commun* (2020)



e LRRK2 inhibits nuclear NFAT3 shuttling in human neurons. **a** Representative Western blots of NFAT3 in nuclear (N) and cytosolic (C) fractions of LRRK2 G2019S neurons and isogenic controls (left and middle panel). Treatment with 200 IU/mL IFN- γ is indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, *** P = 0.0003, ** P = 0.0015, 0.0035 in sequence, * P = 0.0226; n = 5 independent experiments). **b** Representative Western blots of NFAT3 in N and C fractions of control (left panel) and isogenic LRRK2 G2019S neurons (middle panel). Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, **** P < 0.0001, *** P = 0.0005, 0.0008, 0.0002 in sequence; n = 5 independent experiments). **c** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in LRRK2 G2019S neurons and isogenic controls. Treatments with 200 IU/mL IFN- γ are indicated (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, *** P = 0.0009, * P = 0.0421; n = 5 independent experiments). **d** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in control iPSC-derived neurons upon treatment with 200 IU/mL IFN- γ for 24 h, with and without treatment with a proteasome inhibitor (MG132, 20 nM for 24 h) (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, * P = 0.0399, 0.0437 in sequence; n = 3 independent experiments). **e** Representative Western blots of NFAT3 in nuclear and cytosolic fractions of LRRK2 KO neurons. Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, **** P < 0.0001; n = 5 independent experiments).



LRRK2 inhibits nuclear NFAT3 shuttling in human neurons. **a** Representative Western blots of NFAT3 in nuclear (N) and cytosolic (C) fractions of LRRK2 G2019S neurons and isogenic controls (left and middle panel). Treatment with 200 IU/mL IFN- γ is indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, ****P = 0.0003, **P = 0.0015, 0.0035 in sequence, *P = 0.0226; n = 5 independent experiments). **b** Representative Western blots of NFAT3 in N and C fractions of control (left panel) and isogenic LRRK2 G2019S neurons (middle panel). Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 in LRRK2 G2019S iPSC-derived neurons and isogenic controls (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, ****P < 0.0001, ***P = 0.0005, 0.0008, 0.0002 in sequence; n = 5 independent experiments). **c** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in LRRK2 G2019S neurons and isogenic controls. Treatments with 200 IU/mL IFN- γ are indicated (mean \pm SEM, two-way ANOVA, Bonferroni post hoc, ***P = 0.0009, *P = 0.0421; n = 5 independent experiments). **d** Representative Western blot (left) and quantification (right) of NFAT3 from whole-cell extracts in control iPSC-derived neurons upon treatment with 200 IU/mL IFN- γ for 24 h, with and without treatment with a proteasome inhibitor (MG132, 20 nM for 24 h) (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, *P = 0.0399, 0.0437 in sequence; n = 3 independent experiments). **e** Representative Western blots of NFAT3 in nuclear and cytosolic fractions of LRRK2 KO neurons. Treatments with 1 μ M ionomycin for 30 min and 200 IU/mL IFN- γ for 24 h are indicated. On the right, quantification of nuclear to total (N and C fraction) NFAT3 (mean \pm SEM, one-way ANOVA, Bonferroni post hoc, ****P < 0.0001; n = 5 independent experiments).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Interferon- γ signaling synergizes with LRRK2 in neurons and microglia derived from human induced pluripotent stem cells. *Nat Commun* (2020)

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.

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

Host Rabbit

Immunogen Synthetic peptide corresponding to aa 250-325 of human Hsp90.

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 Human, Mouse, Rabbit, Rat

UniProt ID P07900 (HSP90α), P08238 (HSP90β)

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](mailto:info-usa@enzolifesciences.com)

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

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

France
Phone: +33 472 440 655
[info-
fr@enzolifesciences.com](mailto:info-fr@enzolifesciences.com)

Germany
Phone: +49 7621 5500 526
[info-
de@enzolifesciences.com](mailto:info-de@enzolifesciences.com)

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