

Product Datasheet

IRF3 Antibody - BSA Free NBP1-78769

Unit Size: 100 ul

Store at 4C. Do not freeze.

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NBP1-78769

IRF3 Antibody - BSA Free

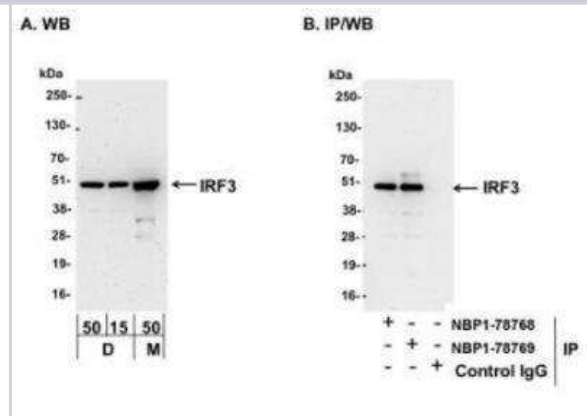
Product Information	
Unit Size	100 ul
Concentration	1.0 mg/ml
Storage	Store at 4C. Do not freeze.
Clonality	Polyclonal
Preservative	0.09% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	Tris-Citrate/Phosphate (pH 7.0 - 8.0)
Target Molecular Weight	47 kDa

Product Description	
Description	Novus Biologicals Rabbit IRF3 Antibody - BSA Free (NBP1-78769) is a polyclonal antibody validated for use in WB and IP. Anti-IRF3 Antibody: Cited in 3 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	3661
Gene Symbol	IRF3
Species	Mouse
Immunogen	The immunogen for this product maps to a region between residue 375 and 419 of mouse Interferon Regulatory Factor 3 using the numbering given in entry NP_058545.1 (GeneID 54131).

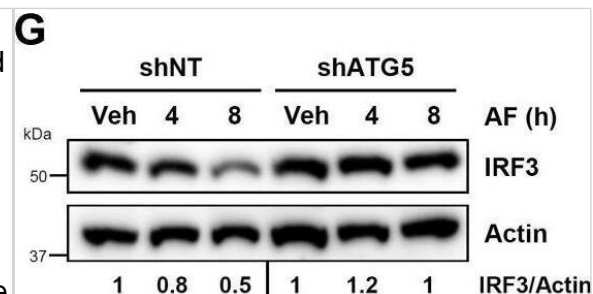
Product Application Details	
Applications	Western Blot, Immunoprecipitation
Recommended Dilutions	Western Blot 1:1000-1:2500, Immunoprecipitation 2-10 ug/mg lysate
Application Notes	Western blot of lysates performed using standard western blot reagents and 4-20% SDS-PAGE.

Images

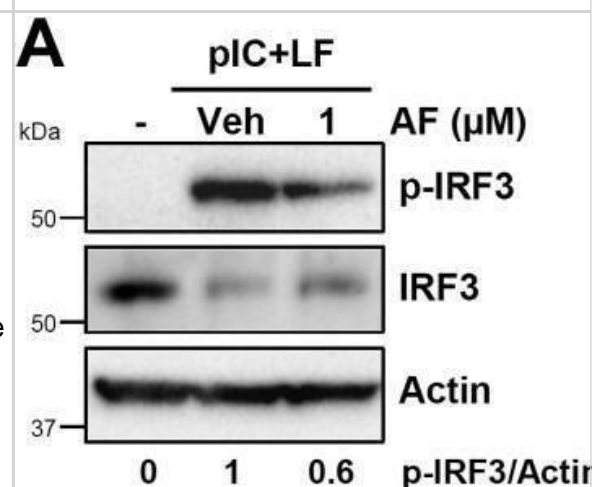
Western Blot: IRF3 Antibody [NBP1-78769] - Whole cell lysate from mouse bone marrow dendritic (D; 50 and 15 mcg for WB; 1 mg for IP, 20% of IP loaded), and NIH3T3 (M; 50 mcg) cells. Affinity purified rabbit anti-IRF3 antibody used for WB at 0.4 mcg/ml (A) and 1 mcg/ml (B) and used for IP at 6 mcg/mg lysate. IRF3 was also immunoprecipitated by rabbit anti-IRF3 antibody NBP1-78768 which recognizes a disparate epitope.



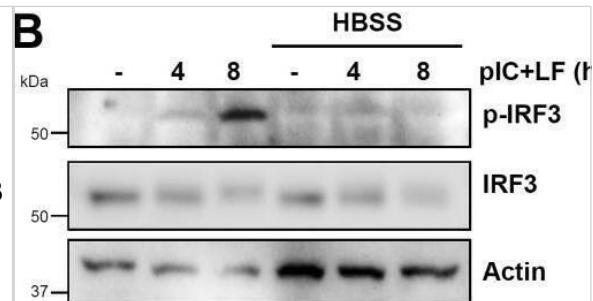
Auranofin activates the cellular autophagy pathway to degrade IRF3 protein. A, HT1080 cells, pretreated with auranofin (AF), were transfected with poly(I:C) (pIC+LF) for 24 h, when LC3 and IRF3 were analyzed by immunoblot. B and C, MDA-MB-453 cells were treated with AF for the indicated times when the p62 levels were analyzed by immunoblot (B), and the immunoblots from the biological replicates were quantified using ImageJ (C). D, RAW264.7 cells were treated with AF for the indicated times when the p62 levels were analyzed by immunoblot. E–G, HT1080 cells, stably expressing ATG5-specific or nontargeting (NT) shRNA, were treated with AF for the indicated times, when the levels of p62 (E and F) or IRF3 (G) were analyzed by immunoblot. The quantification in F is from the biological replicates. ATG5 knockdown levels were analyzed by immunoblot (E, lower panel). H, HT1080 cells, stably expressing ATG5-specific or nontargeting (NT) shRNA, were treated with AF for the indicated times, when the levels of RIG-I, MAVS, TRAF2, TBK1, BAX, and ATG5 were analyzed by immunoblot. Veh, Vehicle (DMSO). Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



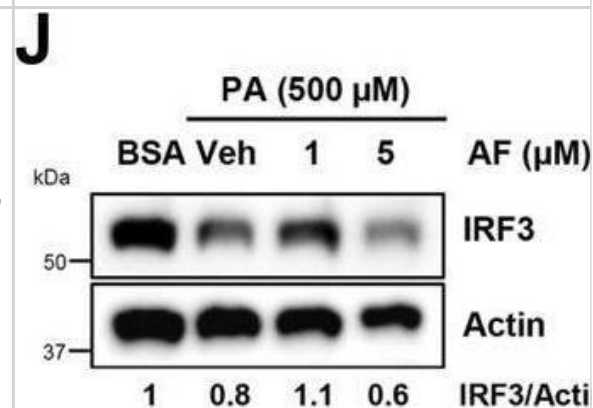
Auranofin promotes the degradation of IRF3 protein. A, HT1080 cells, pretreated with auranofin (AF), were transfected with poly(I:C) (pIC+LF) and the phosphorylated (Ser396) and total IRF3 were analyzed by immunoblot after 8 h. B, MDA-MB-453 cells, pretreated with AF, were transfected with poly(I:C) (pIC+LF) for the indicated times when the IRF3 levels were analyzed by immunoblot. C and D, MDA-MB-453 cells were treated with AF for the indicated times when the IRF3 levels were analyzed by immunoblot (C), and the immunoblots from the biological replicates were quantified using ImageJ (D). E, HT1080 cells were treated with AF for the indicated times when the IRF3 protein levels were analyzed by immunoblot. F, RAW264.7 cells were treated with AF at the indicated concentrations for 16 h, when the levels of Irf3 were analyzed by immunoblot. G, RAW264.7 cells were treated with AF (5 μ M) for the indicated times when the levels of Irf3 were analyzed by immunoblot. H, HT1080 cells were treated with AF for the indicated times, and the BAX protein levels were analyzed by immunoblot. I, HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF and analyzed for phosphorylated (on Ser536) and total p65 by immunoblot after 4 h. J, HT1080 cells were treated with interferon ($\text{hIFN}\beta$, 1000 U/ml) in the absence or the presence of AF and analyzed for phosphorylated (on Ser727) and total STAT1 by immunoblot after 2 h. K, HT1080 cells were treated with AF at the indicated concentrations for 8 h when cell viability was assessed by trypan blue exclusion assay. Veh, Vehicle (DMSO). Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



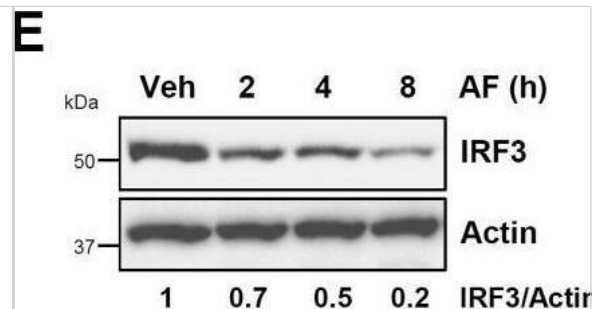
Autophagic degradation inhibits the transcriptional activity of IRF3. A, MDA-MB-453 cells were treated with Hank's balanced salt solution (HBSS) for the indicated times when the protein levels of IRF3 were analyzed by immunoblot. B and C, MDA-MB-453 cells were transfected with polyI:C (pIC+LF) in the absence or the presence of HBSS for the indicated times, when the protein levels of pIRF3 and IRF3 (B) and IFIT3 (C) were analyzed by immunoblot. D–F, MDA-MB-453 (D, E) or RAW264.7 (F) cells were transfected with polyI:C (pIC+LF) in the absence or the presence of HBSS for 8 h, when IFIT3 (D), IFNB1 (E), and Ifit1 (F) mRNA levels were analyzed by qRT-PCR. G, HT1080 cells were transfected with polyI:C (pIC+LF) in the absence or the presence of HBSS and phosphorylated (on Ser172), and total TBK1 was analyzed by immunoblot after 4 h. H, HT1080 cells were transfected with polyI:C (pIC+LF) in the absence or the presence of HBSS and phosphorylated (on Thr180/Tyr182) and total p38 MAPK was analyzed by immunoblot after 4 h. I, U4C (Wt) and IRF3-overexpressing U4C (IRF3hi) cells were transfected with polyI:C (pIC+LF) in the absence or the presence of HBSS, and IFIT3 and IRF3 were analyzed by immunoblot at the indicated times posttransfection. □ indicates $p < 0.05$. Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



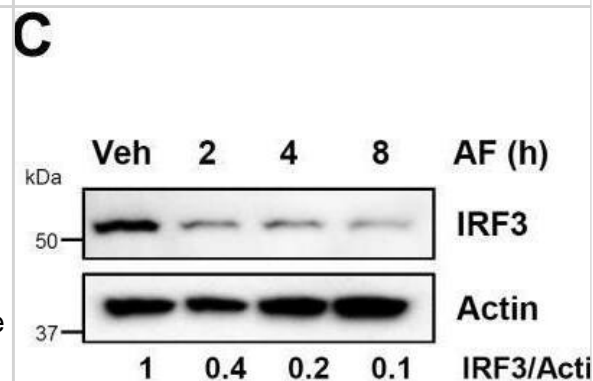
Autophagic degradation of IRF3 by auranofin inhibits palmitic-acid-induced apoptotic cell death in human hepatocytes. A, Huh7 cells were treated with palmitic acid (PA) or vehicle (bovine serum albumin, BSA) for 24 h, when the caspase-3 activity was measured. B and C, Huh7 cells were treated with PA, and apoptotic cell death was analyzed by immunoblot of cleaved PARP (B) or bright field microscopy (C). D and E, Huh7 cells were treated with PA, and the caspase activity (D), and cleaved PARP (E) were analyzed at the indicated times post-treatment. F and G, Huh7 cells were pretreated with auranofin (AF) at the indicated concentrations and treated with PA. Caspase activity (F) and cleaved PARP (G) were measured 16 h posttreatment. H, Huh7 cells were treated with AF at the indicated concentrations for 8 h when cell viability was assessed by trypan blue exclusion assay. I, Huh7 cells were transfected with nontargeting (NT) or IRF3-specific siRNA, and the cells were analyzed for caspase-3 activity upon PA treatment for 16 h. The lower panel indicates the knockdown levels of IRF3 protein. J and K, Huh7 cells were pretreated with AF at the indicated concentrations and treated with PA. IRF3 (J) and p62 (K) were analyzed 16 h posttreatment. L, Huh7 cells were treated with AF, as indicated, and the LC3 levels were analyzed by immunoblot. Veh, Vehicle (DMSO), □ indicates $p < 0.05$, NS, nonsignificant, scale bar, 100 μm . Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



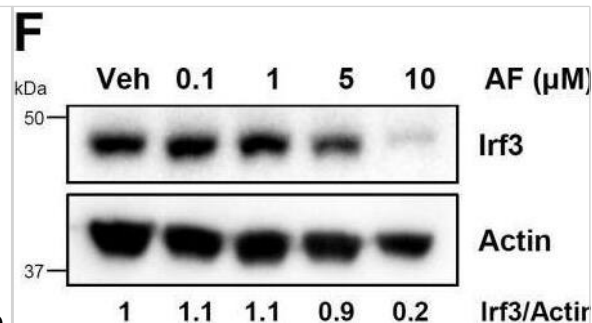
Auranofin promotes the degradation of IRF3 protein. A, HT1080 cells, pretreated with auranofin (AF), were transfected with poly(I:C) (pIC+LF) and the phosphorylated (Ser396) and total IRF3 were analyzed by immunoblot after 8 h. B, MDA-MB-453 cells, pretreated with AF, were transfected with poly(I:C) (pIC+LF) for the indicated times when the IRF3 levels were analyzed by immunoblot. C and D, MDA-MB-453 cells were treated with AF for the indicated times when the IRF3 levels were analyzed by immunoblot (C), and the immunoblots from the biological replicates were quantified using ImageJ (D). E, HT1080 cells were treated with AF for the indicated times when the IRF3 protein levels were analyzed by immunoblot. F, RAW264.7 cells were treated with AF at the indicated concentrations for 16 h, when the levels of Irf3 were analyzed by immunoblot. G, RAW264.7 cells were treated with AF (5 μ M) for the indicated times when the levels of Irf3 were analyzed by immunoblot. H, HT1080 cells were treated with AF for the indicated times, and the BAX protein levels were analyzed by immunoblot. I, HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF and analyzed for phosphorylated (on Ser536) and total p65 by immunoblot after 4 h. J, HT1080 cells were treated with interferon ($\text{hIFN}\beta$, 1000 U/ml) in the absence or the presence of AF and analyzed for phosphorylated (on Ser727) and total STAT1 by immunoblot after 2 h. K, HT1080 cells were treated with AF at the indicated concentrations for 8 h when cell viability was assessed by trypan blue exclusion assay. Veh, Vehicle (DMSO). Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



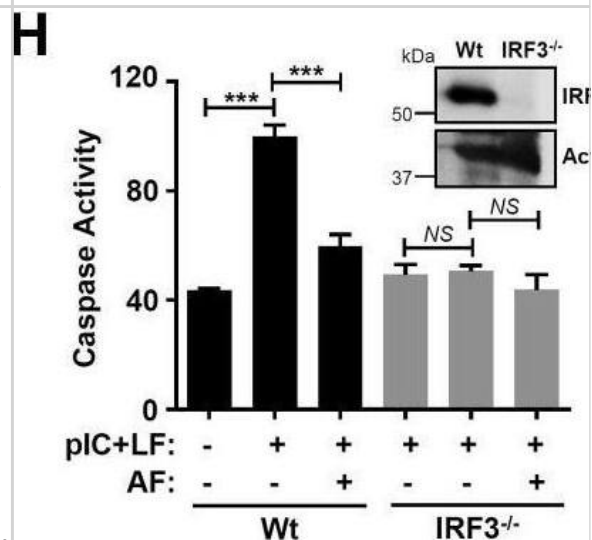
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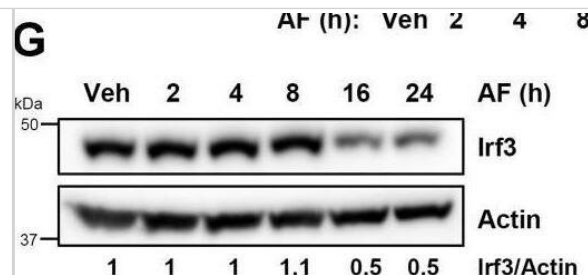
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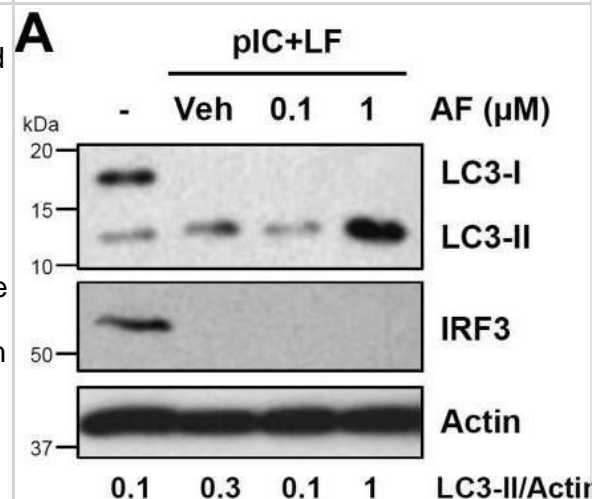
Auranofin inhibits the proapoptotic activity of IRF3. A, HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of auranofin (AF), and the culture fields were analyzed by bright field microscopy 8 h post-pIC stimulation. B, HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF, at the indicated concentrations, for 16 h, when cleaved PARP was analyzed by immunoblot. C, HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF for the indicated times when the caspase-3 activity was measured. D, MDA-MB-453 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF (at the indicated concentrations) for 16 h, when cleaved PARP was analyzed by immunoblot. E, MDA-MB-453 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF for the indicated times when the caspase-3 activity was measured. F, HT1080 cells were transfected with poly(dA:dT) in the absence or the presence of AF for 16 h, when the cleaved PARP was analyzed by immunoblot. G, HT1080 cells were transfected with poly(dA:dT) in the absence or the presence of AF for 16 h, when the caspase-3 activity was measured. H, Wt and IRF3^{-/-} HT1080 cells were transfected with poly(I:C) (pIC+LF) in the absence or the presence of AF for 8 h when the caspase-3 activity was measured. IRF3 protein expression is shown by immunoblot in the inset. Veh, Vehicle (DMSO), \square indicates $p < 0.05$, NS, nonsignificant, scale bar, 100 μ m. Image collected and cropped by CiteAb from the following open publication (<https://linkinghub.elsevier.com/retrieve/pii/S0021925821010772>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



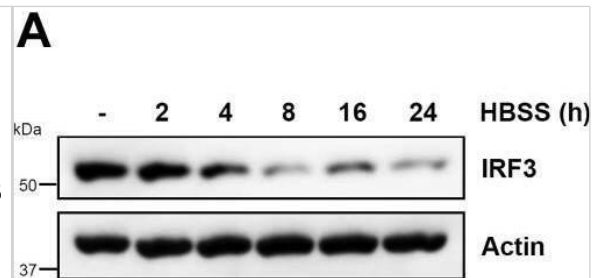
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Publications

N Kerur, S Fukuda, D Banerjee, Y Kim, D Fu, I Apicella, A Varshney, R Yasuma, BJ Fowler, E Baghdasary, KM Marion, X Huang, T Yasuma, Y Hirano, V Serbulea, M Ambati, VL Ambati, Y Kajiwara, K Ambati, S Hirahara, A Bastos-Car, Y Ogura, H Terasaki, T Oshika, KB Kim, DR Hinton, N Leitinger, JC Cambier, JD Buxbaum, MC Kenney, SM Jazwinski, H Nagai, I Hara, AP West, KA Fitzgerald, SR Sadda, BD Gelfand, J Ambati cGAS drives noncanonical-inflammasome activation in age-related macular degeneration *Nat. Med.*, 2017-11-27;0(0):. 2017-11-27 [PMID: 29176737]

Glanz A, Chakravarty S, Fan S Et al. Autophagic degradation of IRF3 induced by the small-molecule auranofin inhibits its transcriptional and proapoptotic activities *The Journal of biological chemistry* 2021-10-05 [PMID: 34619149]

Banerjee D, Langberg K, Abbas S Et al. A non-canonical, interferon-independent signaling activity of cGAMP triggers DNA damage response signaling *Nature communications* 2021-10-27 [PMID: 34707113] (WB, Mouse)



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NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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