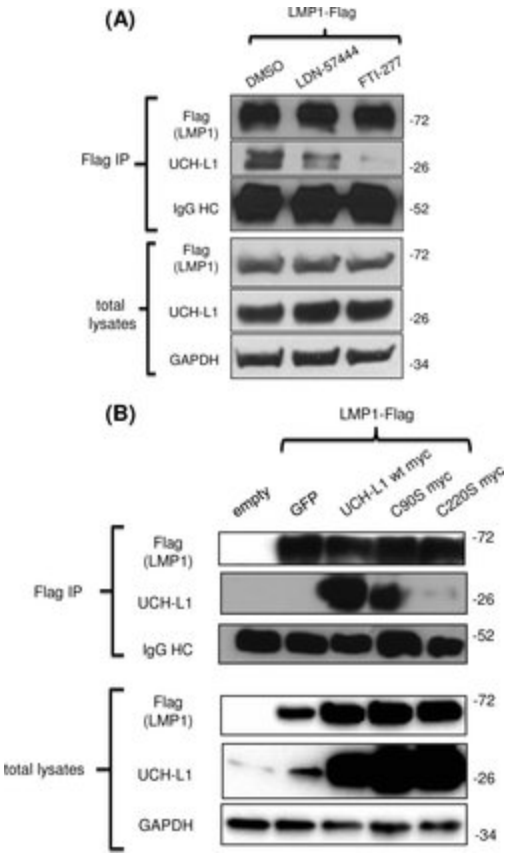
PGP9.5 Polyclonal Antibody

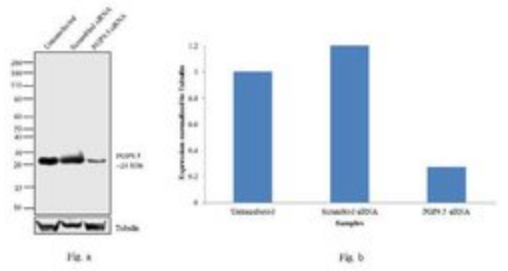
Product Details	
Size	100 µg
Species Reactivity	Human, Mouse, Rat
Published Species	Rat, Human, Mouse, Chicken
Host/Isotype	Rabbit / IgG
Class	Polyclonal
Type	Antibody
Conjugate	Unconjugated
Immunogen	Synthetic peptide derived from an internal region of the human USF-1 protein, isoforms 1 and 2, which are identical to mouse, rat, dog, and bovine isoform 1 and to mouse and dog isoform 2
Form	Liquid
Concentration	0.25 mg/mL
Purification	Antigen affinity chromatography
Storage buffer	PBS, pH 7.4
Contains	0.1% sodium azide
Storage conditions	-20°C
RRID	AB_2533355

Applications	Tested Dilution	Publications
Western Blot (WB)	1-3 µg/mL	2 Publications
Immunohistochemistry (IHC)	Assay-dependent	6 Publications
ELISA (ELISA)	Assay-dependent	-

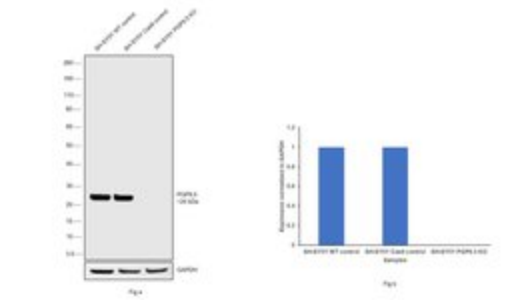
Advanced Verification Data



PGP9.5 Antibody (38-1000)
FIG 2 C-terminal farnesylation of UCH-L1 is required for its association with the EBV primary oncogene LMP1. (A) Inhibition of cellular farnesylation reduces LMP1 association with UCH-L1. 293 cells were transfected with empty vector, GFP (green fluorescent protein), LMP1-Flag and UCH-L1 wild-type expression vectors and treated with DMSO and either UCH-L1 DUB activity inhibitor LDN-57444 or farnesyltransferase inhibitor FTI-277 (5 muM each). At 48 h after transfection, LMP1 was immunoprecipitated with anti-Flag-agarose beads. Band intensity was quantified by the use of ImageJ (http://rsbweb.nih.gov/ij/) software. The result shows less UCH-L1 in the LMP1 complexes under conditions of treatment with FTI-277 than was seen with the DMSO control or LDN-57444 treatment. (B) Inhibition of UCH-L1-specific farnesylation inhibits LMP1/UCH-L1 complex formation. 293 cells were transfected with LMP1-Flag and the UCH-L1 wild type or one of two UCH-L1 mutants: an enzymatically inactive mutant (C90S mutant) or UCH-L1 with a mutated farnesylation site (C220S mutant). Cells were harvested 48 h posttransfection for LMP1 complex formation analysis. After IP with anti-Flag-agarose beads, Western blot analysis was performed with the indicated antibodies. The results revealed less UCH-L1 C220S mutant than wild type or C90S mutant in complex with LMP1. Cell treatment validation info.



PGP9.5 Antibody (38-1000)
Antibody specificity was demonstrated by siRNA mediated knockdown of target protein. HeLa cells were transfected with PGP9.5 siRNA and loss of signal was observed in Western Blot using Anti-PGP9.5 Antibody (Product # 38-1000). Knockdown validation info.



PGP9.5 Antibody (38-1000)
Antibody specificity was demonstrated by CRISPR-Cas9 mediated knockout of target protein. A loss of signal was observed for target protein in PGP9.5/UCHL1 (KO) cell line compared to control cell line using Anti-PGP9.5/UCHL1 Polyclonal Antibody (Product # 38-1000). Knockout validation info.

View more figures on thermofisher.com

8 References

Western Blot (2)

mSphere

C-Terminal Farnesylation of UCH-L1 Plays a Role in Transport of Epstein-Barr Virus Primary Oncoprotein LMP1 to Exosomes.

"Published figure using PGP9.5 polyclonal antibody (Product # 38-1000) in Western Blot"

Authors: Kobayashi E,Aga M,Kondo S,Whitehurst C,Yoshizaki T,Pagano JS,Shackelford J

Species
Human

Dilution
Not Cited

Year
2019

International journal of molecular sciences

Inhibition of UCH-L1 Deubiquitinating Activity with Two Forms of LDN-57444 Has Anti-Invasive Effects in Metastatic Carcinoma Cells.

"Published figure using PGP9.5 polyclonal antibody (Product # 38-1000) in Western Blot"

Authors: Kobayashi E,Hwang D,Bheda-Malge A,Whitehurst CB,Kabanov AV,Kondo S,Aga M,Yoshizaki T,Pagano JS, Sokolsky M,Shakelford J

Species
Not Applicable

Dilution
Not Cited

Year
2019

Immunohistochemistry (6)

Diabetes

NLRP3 Promotes Diabetic Bladder Dysfunction and Changes in Symptom-Specific Bladder Innervation.

"Published figure using PGP9.5 polyclonal antibody (Product # 38-1000) in Immunohistochemistry"

Authors: Hughes FM,Hirshman NA,Inouye BM,Jin H,Stanton EW,Yun CE,Davis LG,Routh JC,Purves JT

Species
Not Applicable

Dilution
Not Cited

Year
2019

American journal of physiology. Renal physiology

Bladder decompensation and reduction in nerve density in a rat model of chronic bladder outlet obstruction are attenuated with the NLRP3 inhibitor glyburide.

"38-1000 was used in Immunohistochemistry to suggest a critical role for NLRP3 in mediating bladder decompensation and nerve density during chronic BOO."

Authors: Hughes FM,Sexton SJ,Ledig PD,Yun CE,Jin H,Purves JT

Species
Rat

Dilution
1:200

Year
2019

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More applications with references on thermofisher.com