HERP (human) polyclonal antibody

Homocysteine-responsive endoplasmic reticulum-resident ubiquitin-like domain member 1 protein (HERP) is a membrane-associated ER protein strongly up-regulated by inducers of ER stress.

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BML-PW9705-0100	100µl

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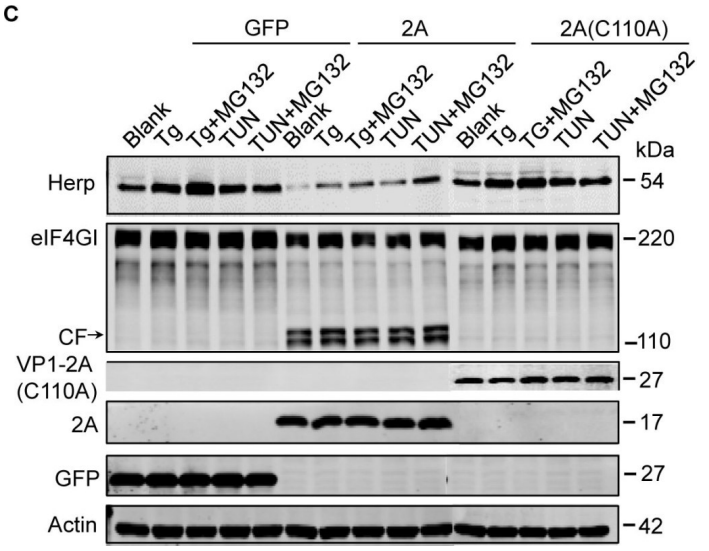
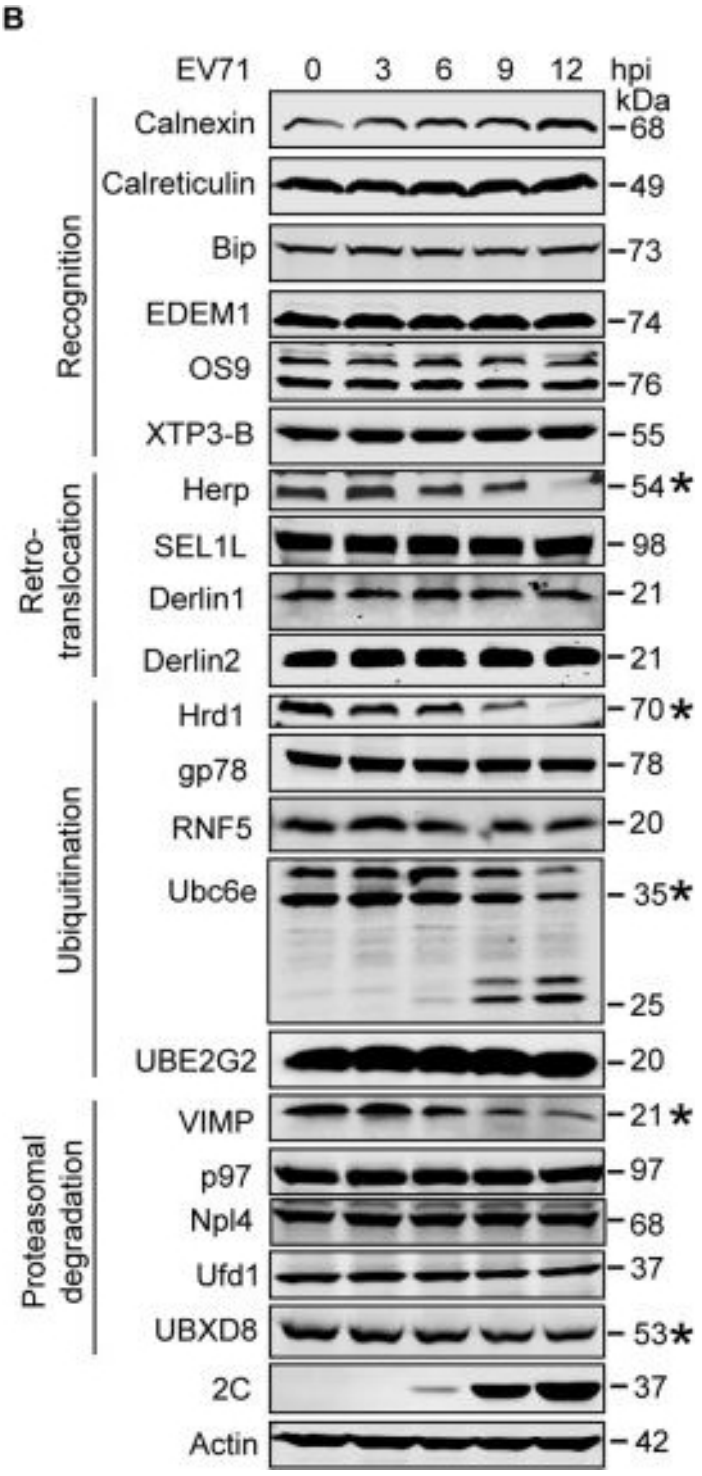
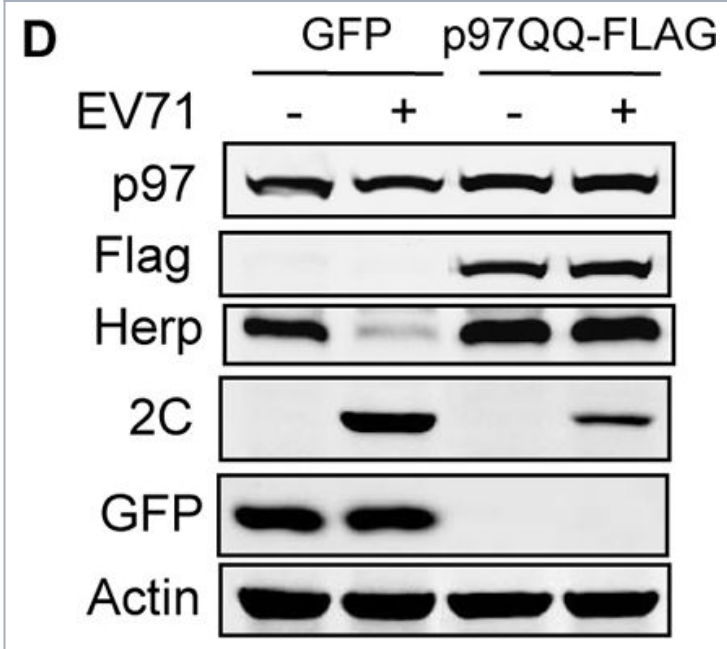


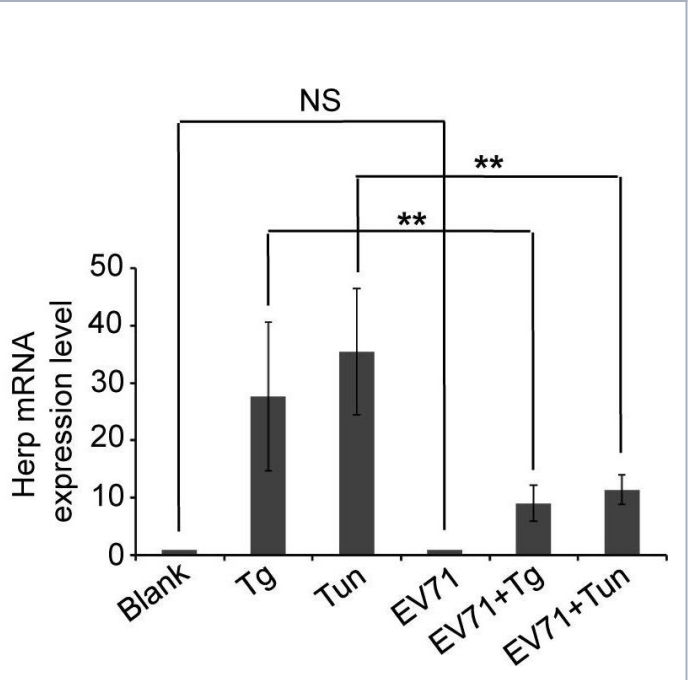
EV71 2Apro inhibits the biosynthesis of Herp and VIMP.(A) RD cells were mock-infected (−) or infected (+) with EV71 (MOI = 10) for 9 h and then treated with MG132 (50 μM), Tg (300 nM), Tg plus MG132, Tun (10 μg/ml), Tun plus MG132 for an additional 6 h. Then, the cells were harvested and analyzed by western blotting with antibodies against Herp, EV71 VP1, and actin. The lower panel graph shows the quantification of Herp. The data are presented as means ± SD of two independent experiments. (B) RD cells were mock-infected (−) or infected (+) with EV71 (MOI = 10) for 9 h and then treated with Tg (300 nM) or Tun (10 μg/ml) for 6 h to induce Herp expression. Then, the mRNA expression levels of Herp were evaluated by quantitative real-time PCR. The data are presented as means ± SD of two independent experiments. NS, non-significant, P≥0.05; ** P<0.01. (C) BSRT7 cells were transfected with pcDNA3.1-EGFP, pcDNA3.1-IRES-2A, or pcDNA3.1-IRES-2A(C110A). At 36 h post-transfection, cells were treated with Tg (300 nM), Tg plus MG132 (50 μM), Tun (10 μg/ml), or Tun plus MG132 for 6 h. Then, cell lysates were analyzed by western blotting with antibodies against Herp, eIF4GI, V5, GFP, and actin. eIF4GI was included as a positive control of protease activity of 2Apro, and arrows indicate the cleavage fragments (CF) of eIF4GI. (D) BSRT7 cells were transfected with increasing doses of pcDNA3.1-EGFP plasmid or pcDNA3.1-IRES-2A plasmid (1–3 μg). At 36 h post-transfection, the cells were harvested, RNA was extracted, and quantitative real-time PCR was used to analyze VIMP mRNA expression. The data are presented as means ± SD of three independent experiments. NS, non-significant, P≥0.05. (E) BSRT7 cells were transfected with increasing doses of pcDNA3.1-EGFP, pcDNA3.1-IRES-2A, or pcDNA3.1-IRES-2A(C110A) plasmids (1–3 μg). At 36 h post-transfection, the cells were harvested and cell lysates were analyzed by western blotting with antibodies



EV71 targets ERAD at multiple points.(A) Diagram of the key molecules involved in ERAD. (B) RD cells were infected with EV71 (MOI = 10) for the indicated times (hpi: hours post-infection). The cells were then harvested and western blotting was performed using the indicated antibodies to detect the indicated ERAD components, EV71 2C, and actin. The ERAD molecules assessed in this study were separated into four categories: substrate recognition, retrotranslocation, ubiquitination, and proteasomal degradation. Asterisks indicate the molecules that were obviously downregulated. (C) Full-size western blots for Herp, Hrd1, VIMP, and UBXD8 described in (B). (D) Quantification of Ubc6e, Herp, Hrd1, VIMP, and UBXD8 in (B). The data are presented as means ± SD of three independent experiments.



p97 and its ATPase activity are essential for EV71 replication, and it co-localizes with EV71 2C.(A) RD cells were transfected with control siRNA or siRNA targeting Ubc6e or Herp. At 36 h post-transfection, cells were infected with EV71 (MOI = 10) for 12 h. Then real-time PCR was used to qualified viral RNA replication. Data are expressed as fold-change of the EV71 RNA level relative to cells transfected with siRNA control. The data were examined in at least two independent experiments; NS, non-significant, P≥0.05. (B) RD cells were transfected with control siRNA and siRNA targeting different ERAD components including EDEM1, OS9, SEL1L, Derl1, Derl2, Hrd1, gp78, RNF5, UBE2G2, VIMP, UBXD8, p97, Npl4, and Ufd1. At 36 h post-transfection, cells were mock infected (−) or infected (+) with EV71 (MOI = 10) for 12 h. Then, western blotting was carried out to detect the knockdown efficiency of different molecules, EV71 2C, and actin. (C) RD cells were pre-treated with increasing doses of DBeQ (0–10 μM) for 3 h and then mock-infected (−) or infected (+) with EV71 (MOI = 10) for 12 h. The cell lysates were then analyzed by western blotting with the indicated antibodies. Herp expression was used as an indicator of DBeQ efficiency, and actin was used as the loading control. (D) RD cells were transfected with plasmids encoding GFP or p97QQ-FLAG (dominant negative p97 mutant). At 36 h after transfection, the cells were mock-infected (−) or infected (+) with EV71 (MOI = 10) for 12 h, and the cell lysates were analyzed by western blotting with the indicated antibodies. (E) RD cells were mock-infected or infected with EV71 (MOI = 10) for 12 h, and immunostaining was then performed to detect the intracellular distribution of p97 and EV71 2C (p97, green; 2C, red; nuclei, blue). Insets show magnified views of the merged channels in the boxed region. (F) RD cells were mock infected (−) or infected (+) with



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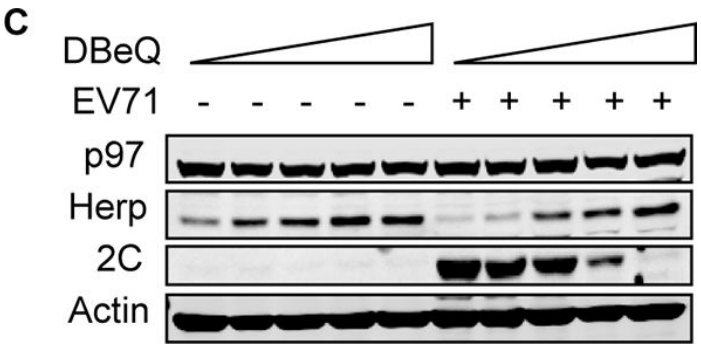

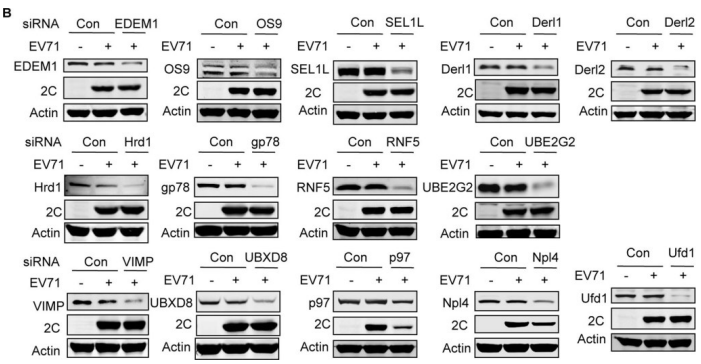

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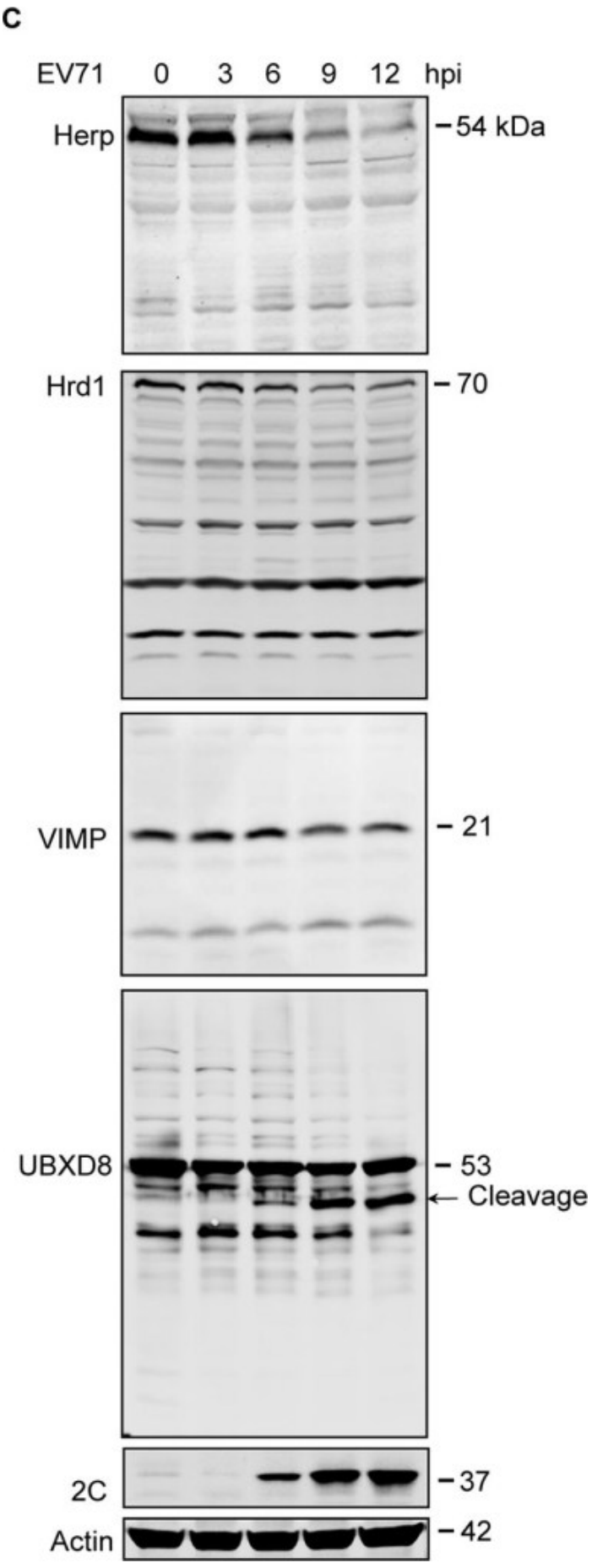

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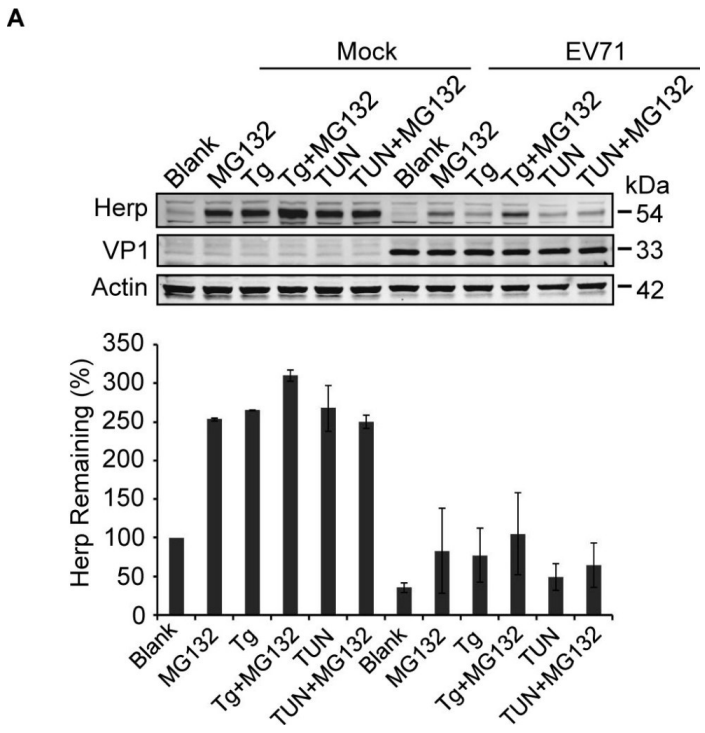

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Handling & Storage

Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	Homocystein-responsive endoplasmic reticulum-resident ubiquitin-like domain member 1 protein
Application	IP, WB
Formulation	Liquid. In PBS containing 10mM sodium azide.
Host	Rabbit
Immunogen	Recombinant N-terminal cytoplasmic region of human HERP (aa 1-240).
Species Reactivity	Human
UniProt ID	Q15011
Worry-free Guarantee	**This antibody is covered by our Worry-Free Guarantee .**

Last modified: May 29, 2024

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