

Cardif (human) polyclonal antibody (AT107)

RIG-I (retinoic acid-inducible gene I; Ddx58) and Mda5 (melanoma differentiation-associated gene 5, also known as Ifih1 or Helicard) are proteins that sense viral replication intermediates, such as double-stranded RNA and triggers the host antiviral programs. These molecules signal the downstream activation of NF- κ B and IFN regulatory factor (IRF) -3, which coordinately regulate the expression of type-I interferons. Cardif (also called VISA/IPS-1/MAVS) is a CARD (caspase activation and recruitment domain)-containing adaptor protein that interacts with the CARD domain of RIG-I and Mda5, leading to the activation of NF- κ B and IRF3. Cardif is located to the mitochondrial outer membrane. Removal of the mitochondrial-targeting domain of cardif abolishes its ability to induce IFNs. Cardif is cleaved and inactivated by NS3-4A, a serine protease from hepatitis C virus known to block interferon- β production.

This antibody is covered by our [Worry-Free Guarantee](#).

Citations: 39

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Ordering Information

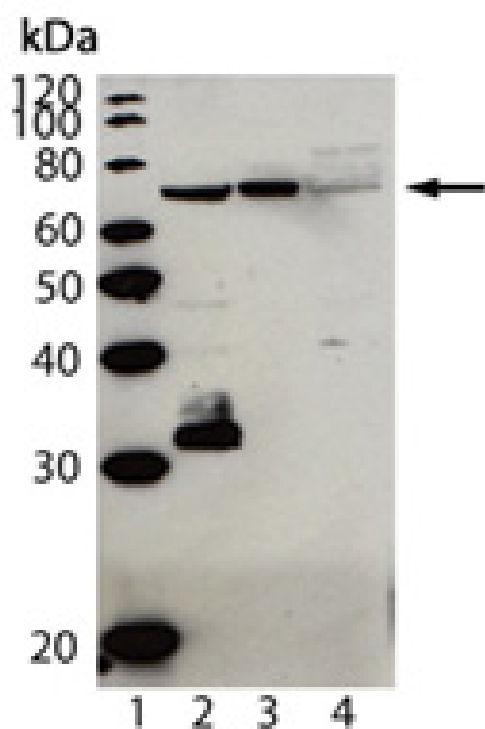
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ALX-210-929-C100

100 μ g

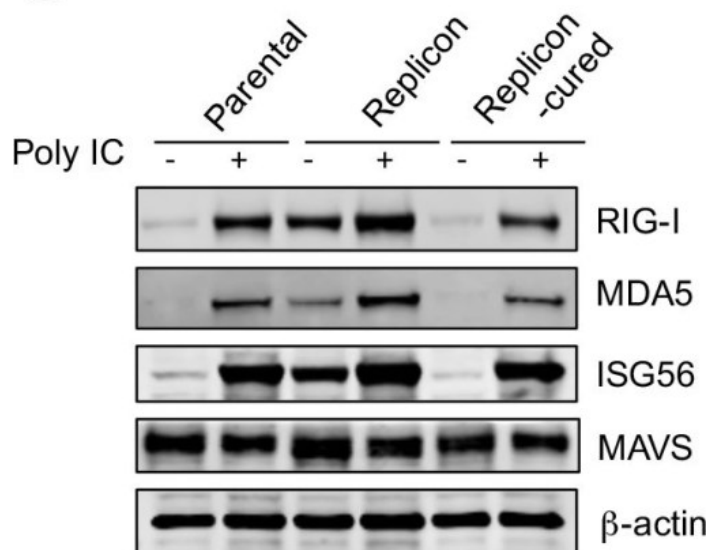
Manuals, SDS & CofA

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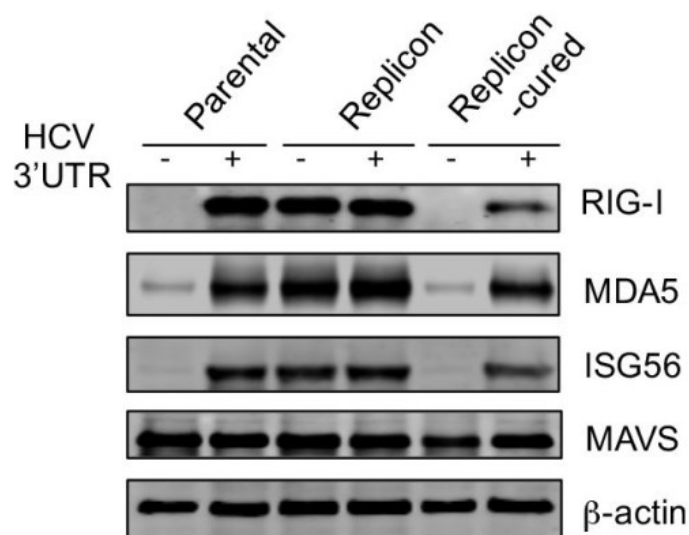


Western blot analysis of Cardif (human), pAb (AT107) (Prod. No. ALX-210-929): Lane 1: MW marker; Lane 2: HepG2; Lane 3: PALA; and Lane 4: HeLa. Additional bands observed probably represent isoforms or cleaved products of Cardif.

G

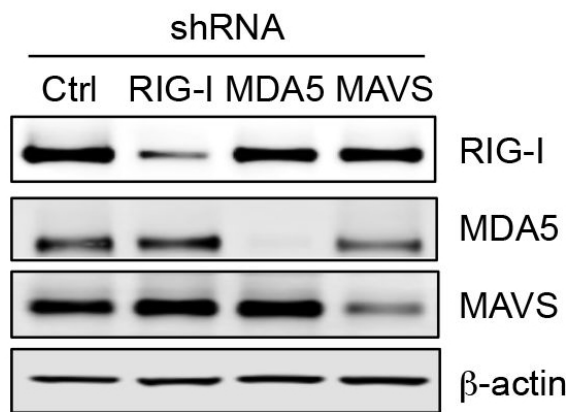


J



HEV does not target MAVS. (A) Confocal images showing MAVS and viral antigens in HepG2 cells infected with either HEV (top) or HAV (bottom). Cells were stained 5 days after infection with a rabbit anti-MAVS, chimpanzee 1313 serum (HEV), or a murine monoclonal antibody K24F2 (HAV). DAPI was used to stain the nuclei. Scale bar: 10 μ m. (B) Confocal images showing the mitochondrial localization of MAVS in HepG2 cells with or without HEV replicon. MAVS was stained with a rabbit antibody against MAVS (green). Mitochondria was visualized with MitoTracker (red). Nuclei were stained with DAPI. Scale bar: 10 μ m. (C) HepG2 cells with or without the HEV replicon were transfected with a MAVS-expressing plasmid along with a HAV 3ABC-expressing plasmid or an empty vector. The endogenous (closed arrowheads) and overexpressed MAVS (open arrowheads) were detected with a rabbit anti-MAVS antibody. Note that co-

A



Signaling pathways involved in HEV-induced IFN response. Immunoblots of RIG-I, MDA5, MAVS and β -actin in HepG2 cells expressing gene-specific shRNA, or GFP (Ctrl). (B-C) IFN- β promoter activity in HepG2 cells with different gene knockdown following Sendai virus (SeV) infection (B) or poly IC transfection (C). Cells were transfected with IFN- β -Luc and TK-RLuc (for normalization of transfection efficiency) 20 h prior to SeV infection or poly IC transfection. Cells were lysed and luciferase activity was determined 20 h after SeV infection or 12 h after poly IC transfection. Data are presented as fold changes relative to non-treated cells. Shown are representative results from two independent experiments each performed in triplicate. (D-F) Effect of RIG-I, MDA5 or MAVS knockdown on HEV replication and host IFN responses. Control and shRNA-expressing HepG2 cells were inoculated with HEV (1000 GE/cell). IFN- λ mRNA expression (D), HEV RNA abundance (E), and HEV-positive foci (F) were determined at 5 days after infection. The results show the mean \pm SEM of 2 independent experiments performed in duplicate each. * $P < 0.05$; ** $P < 0.01$. (G) Immunoblots of IRF-3, IRF-7 and β -actin in HepG2 cells transduced with lentiviruses expressing GFP (Ctrl) or gene-specific shRNA. (H-J) Effect of IRF-3 or IRF-7 knockdown on HEV replication and IFN responses. Control and shRNA-expressing cells were inoculated with HEV (1,000 GE/cell). IFN- λ mRNA expression (H), HEV RNA abundance (I), and HEV-positive foci (J) in different cells were determined after 5 days. The results show the mean \pm SEM of 2 independent experiments each performed in duplicate wells. * $P < 0.05$; ** $P < 0.01$. Scale bar (upper panel in F), 100 μ m.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Hepatitis E virus persists in the presence of a type III interferon response. *PLoS Pathog* (2017)

Handling & Storage

| | |
|--------------------|--|
| Use/Stability | Stable for at least 1 year after receipt when stored at -20°C. |
| Handling | Avoid freeze/thaw cycles. |
| Short Term Storage | +4°C |
| Long Term Storage | -20°C |
| Shipping | Blue Ice |

Regulatory Status

RUO - Research Use Only

Product Details

| | |
|-------------------------------------|---|
| Alternative Name | CARD adapter inducing interferon-β, IPS-1, Interferon-β promoter stimulator protein 1, MAVS, Mitochondrial antiviral signalling protein, VISA, Virus-induced signalling adapter |
| Application | ICC, IP, WB |
| Application Notes | Detects a band of ~65kDa by Western blot. |
| Formulation | Liquid. In PBS containing 0.02% sodium azide. |
| Host | Rabbit |
| Immunogen | Recombinant human Cardif (CARD adapter inducing interferon-β) (aa 160-450). |
| Purity Detail | Protein A-affinity purified. |
| Recommendation Dilutions/Conditions | Immunocytochemistry (1:500)Immunoprecipitation (1:100)Western Blot (1:2,000)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 |

UniProt ID

Q7Z434

Worry-free Guarantee

This antibody is covered by our [Worry-Free Guarantee](#)

Last modified: May 29, 2024



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