BOSTER BIOLOGICAL TECHNOLOGY
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Anti-TIM3/HAVCR2 Antibody

Catalog Number: A00657-5

About HAVCR2

The protein encoded by this gene belongs to the immunoglobulin superfamily, and TIM family of proteins. CD4-positive T helper lymphocytes can be divided into types 1 (Th1) and 2 (Th2) on the basis of their cytokine secretion patterns. Th1 cells are involved in cell-mediated immunity to intracellular pathogens and delayed-type hypersensitivity reactions, whereas, Th2 cells are involved in the control of extracellular helminthic infections and the promotion of atopic and allergic diseases. This protein is a Th1-specific cell surface protein that regulates macrophage activation, and inhibits Th1-mediated auto- and alloimmune responses, and promotes immunological tolerance.

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Overview

Product Name	Anti-TIM3/HAVCR2 Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-TIM3/HAVCR2 Antibody catalog # A00657-5. Tested in WB, IHC, IF applications. This antibody reacts with Human, Mouse, Rat.
Application	IF, IHC, WB
Clonality	Polyclonal
Formulation	500 ug/ml antibody with PBS, 0.02% NaN3, 1 mg stabilizing protein and 50% glycerol *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	12 months from date of receipt□-20°C as supplied. 6 months 2 to 8°C after reconstitution. Avoid repeated freezing and thawing.
Host	Rabbit
Uniprot ID	Q8TDQ0

Technical Details

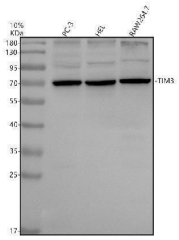
Immunogen	E.coli-derived human TIM3/HAVCR2 recombinant protein (Position: M1-L140).

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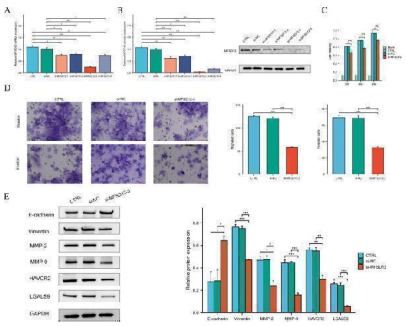
Form	Liquid
Concentration	500 ug/ml
Purification	Immunogen affinity purified.
Suggested Dilutions	Western blot, 1:500-2000 Immunohistochemistry, 1:50-400 Immunofluorescence, 1:50-400

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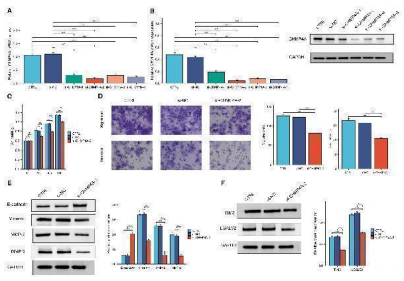
Anti-TIM3/HAVCR2 Antibody (A00657-5) Images



Western blot analysis of TIM3/HAVCR2 using anti-TIM3/HAVCR2 antibody (A00657-5). Electrophoresis was performed on a 10% SDS-PAGE gel at 80V (Stacking gel) / 120V (Resolving gel) for 2 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: human PC-3 whole cell lysates, Lane 2: human HEL whole cell lysates, Lane 3: mouse RAW264.7 whole cell lysates.After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-TIM3/HAVCR2 antigen affinity purified polyclonal antibody (A00657-5) at 1:1000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:5000 for 1.5 hour at RT. The signal is developed using an ECL Plus Western Blotting Substrate (Catalog # AR1196-200) with Tanon 5200 system. A specific band was detected for TIM3/HAVCR2 at approximately 70 kDa. The expected band size for TIM3/HAVCR2 is at 33 kDa.



The knockdown of MFSD12 inhibited the proliferation, migration, and invasion of LIHC cells, as well as the TIM-3/Galectin-9 signaling pathway. (A, B) RT-qPCR and Western blot validation of MFSD12 silencing efficiency using siRNAs (si-MFSD12–1 to −4) with GAPDH as loading control. (C) CCK-8 cell viability assay showing reduced HEP 3B2.1–7 cells proliferation after MFSD12 knockdown (si-MFSD12-3). (D) Transwell assay revealed a reduction in the migratory and invasive capabilities of HEP 3B2.1–7 cells following the knockdown of MFSD12. (E) Immunoblot analysis of EMT markers and TIM-3 axis components showing up-regulation of E-cadherin and down-regulation of Vimentin, MMP-2, MMP-9, HAVCR2 (TIM-3) and LGALS9 in si-MFSD12-treated cells. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. CTRL, control untreated; si-NC, negative control siRNA; si-MFSD12, MFSD12-targeting siRNA; E-cadherin, epithelial cadherin; MMP-2/9, matrix metalloproteinase-2/9; HAVCR2, hepatitis A virus cellular receptor 2 (TIM-3); LGALS9, lectin galactoside-binding soluble 9 (Galectin-9).Index in PubMed under a CC BY license. PMID: 41194934



The knockdown of CHMP4A inhibited the proliferation, migration, and invasion of LIHC cells, as well as the TIM-3/Galectin-9 signaling pathway. (A, B) RT-qPCR and Western blot validation of CHMP4A silencing efficiency using siRNAs (si-CHMP4A-1 to -4) with GAPDH as a loading control. (C) CCK-8 cell viability assay showing reduced proliferation of HEP 3B2.1–7 cells after CHMP4A knockdown (si-CHMP4A-2). (D) Transwell assay revealing a reduction in the migratory and invasive capabilities of HEP 3B2.1–7 cells following the knockdown of CHMP4A. (E) Immunoblot

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analysis of EMT markers and TIM-3 axis components showing upregulation of E-cadherin and downregulation of Vimentin, MMP-2, and MMP-9 in si-CHMP4A-treated cells. (F) Immunoblot analysis of TIM-3 axis components showing downregulation of HAVCR2 (TIM-3) and LGALS9 in si-CHMP4A-treated cells. ** P <0.01, *** P <0.001. CTRL, control untreated; si-NC, negative control siRNA; si-CHMP4A, CHMP4A-targeting siRNA; E-cadherin, epithelial cadherin; MMP-2/9, matrix metalloproteinase-2/9; HAVCR2, hepatitis A virus cellular receptor 2 (TIM-3); LGALS9, lectin galactoside-binding soluble 9 (Galectin-9).Index in PubMed under a CC BY license. PMID: 41112257

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