BOSTER
antibody and ELISA experts

BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com

Anti-CD203c ENPP3 Antibody

Catalog Number: A05615

Overview

Product Name	Anti-CD203c ENPP3 Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-CD203c ENPP3 Antibody catalog # A05615. Tested in WB, IHC, IF, ELISA applications. This antibody reacts with Human, Mouse, Rat.
Application	ELISA, IF, IHC, WB
Clonality	Polyclonal
Formulation	Liquid in PBS containing 50% glycerol, 0.5% stabilizing protein and 0.02% sodium azide. *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	Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	O14638

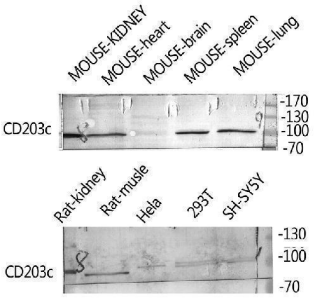
Technical Details

Immunogen	The antiserum was produced against synthesized peptide derived from the Internal region of human ENPP3. AA range:281-330
Cross Reactivity	No cross reactivity with other proteins.
Isotype	IgG
Form	Liquid
Concentration	1 mg/ml
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using a epitope-specific immunogen.
Suggested Dilutions	WB 1:500-1:2000 IHC: 1:100-300 ELISA 1:20000 IF 1:50-200

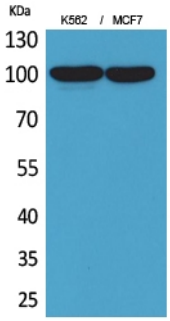
BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com

Anti-CD203c ENPP3 Antibody (A05615) Images

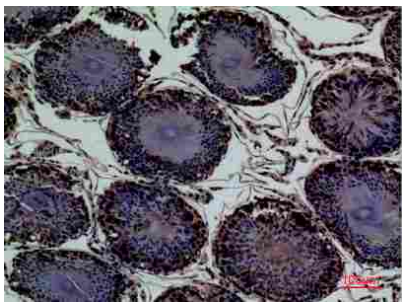
Western Blot (WB) analysis of Mouse Kidney lysis using CD203c antibody.



Western Blot (WB) analysis of Rat Kidney HeLa 293T SH-SY5Y Mouse Kidney, Mouse Spleen, Mouse Lung, using ENPP3 Polyclonal antibody.



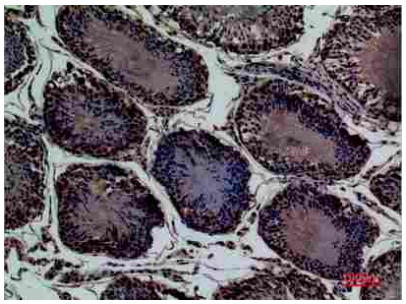
Western Blot (WB) analysis of K562, MCF7 cells using CD203c Polyclonal antibody.

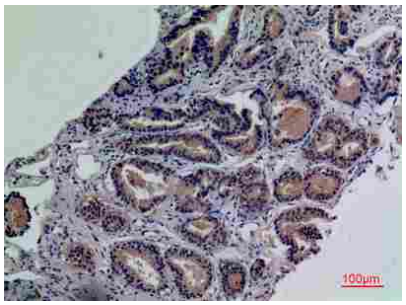


Immunohistochemistry (IHC) analysis of paraffin-embedded Rat Testis, antibody was diluted at 1:100.

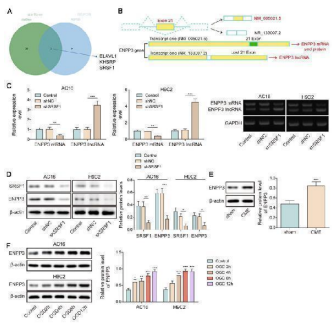
Immunohistochemistry (IHC) analysis of paraffin-embedded Rat Testis, antibody was diluted at 1:100.

BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com



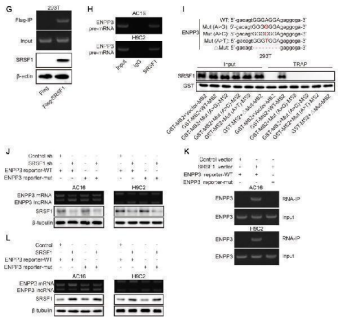


Immunohistochemistry (IHC) analysis of paraffin-embedded Human Prostate Cancer, antibody was diluted at 1:100.

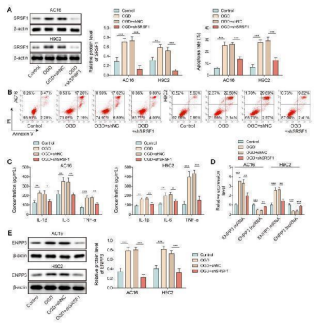


SRSF1 regulated alternative splicing of ENPP3 pre-mRNA. (A) StarBase and RBPDB databases were used to predict the potential RNA-binding protein (RBP) of ENPP3. (B) Illustration of two transcripts of ENPP3 gene and the potential binding sites of SRSF1 to the exon 21 of ENPP3 gene. H9C2 and AC-16 cells were transfected with shSRSF1. (C) RT-qPCR analysis of ENPP3 mRNA and lncRNA ENPP3 expression in cardiomyocytes. (D) The protein level of SRSF1 and ENPP3 was assessed by Western blotting. (E)&(F) The protein level of ENPP3 in the in vivo and in vitro models of CME was measured by Western blotting. (G)&(H) RIP assay validated the endogenous and exogenous binding of SRSF1 to ENPP3 pre-mRNA. (I) The wild type (WT) and mutant (MUT) ENPP3 splicing reporters containing SRSF1 binding sites were transfected into 293T cells. TRAP assay determined the interplay between SRSF1 and ENPP3 pre-mRNA. (J) H9C2 cells were transfected with shSRSF1 together with ENPP3 reporter-WT or ENPP3 reporter-MUT, and expression of ENPP3 mRNA/lncRNA and SRSF1 was assessed by RT-qPCR and Western blotting, respectively. H9C2 cells were transfected with SRSF1 overexpression plasmid together with ENPP3 reporter-WT or ENPP3 reporter-MUT. (K) The interaction between SRSF1 and ENPP3 mRNA was detected by RIP. (L) RT-qPCR and Western blotting analysis of ENPP3 mRNA/lncRNA and SRSF1 levels, respectively. For C, D, F-K, n=3. For E, n=6. Student's t test (for E, G) and one-way ANOVA (for C-F) were performed to analyze data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.Index in PubMed under a CC BY license. PMID: 40585977

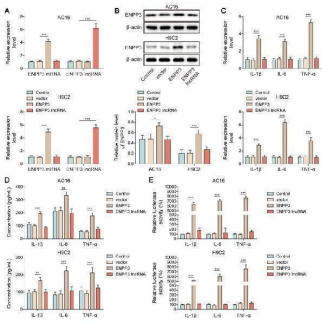
SRSF1 regulated alternative splicing of ENPP3 pre-mRNA. (A) StarBase and RBPDB databases were used to predict the potential RNA-binding protein (RBP) of ENPP3. (B) Illustration of two transcripts of ENPP3 gene and the potential binding sites of SRSF1 to the exon 21 of ENPP3 gene. H9C2 and AC-16 cells were transfected with shSRSF1. (C) RT-qPCR



analysis of ENPP3 mRNA and lncRNA ENPP3 expression in cardiomyocytes. (D) The protein level of SRSF1 and ENPP3 was assessed by Western blotting. (E)&(F) The protein level of ENPP3 in the in vivo and in vitro models of CME was measured by Western blotting. (G)&(H) RIP assay validated the endogenous and exogenous binding of SRSF1 to ENPP3 pre-mRNA. (I) The wild type (WT) and mutant (MUT) ENPP3 splicing reporters containing SRSF1 binding sites were transfected into 293T cells. TRAP assay determined the interplay between SRSF1 and ENPP3 pre-mRNA. (J) H9C2 cells were transfected with shSRSF1 together with ENPP3 reporter-WT or ENPP3 reporter-MUT, and expression of ENPP3 mRNA/lncRNA and SRSF1 was assessed by RT-qPCR and Western blotting, respectively. H9C2 cells were transfected with SRSF1 overexpression plasmid together with ENPP3 reporter-WT or ENPP3 reporter-MUT. (K) The interaction between SRSF1 and ENPP3 mRNA was detected by RIP. (L) RT-qPCR and Western blotting analysis of ENPP3 mRNA/lncRNA and SRSF1 levels, respectively. For C, D, F-K, n=3. For E, n=6. Student's t test (for E, G) and one-way ANOVA (for C-F) were performed to analyze data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. PMID: 40585977

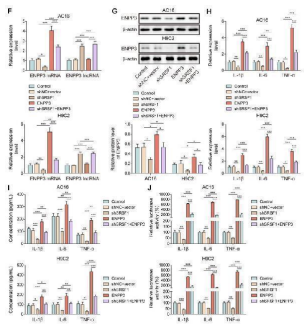


SRSF1 silencing suppressed cardiac inflammation in CME via modulation of ENPP3 splicing. H9C2 and AC-16 cells were transfected with shSRSF1, followed by stimulation with OGD. (A) Western blotting detected SRSF1 protein level. (B) Apoptosis of cardiomyocytes was detected by flow cytometry. (C) The production of TNF-alpha, IL-1beta, and IL-6 was measured by ELISA. (D) RT-qPCR analysis of ENPP3 mRNA and lncRNA ENPP3 levels in each group. (E) The protein level of ENPP3 was determined by Western blotting. n=3 for A-E. One-way ANOVA was performed to analyze data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. PMID: 40585977

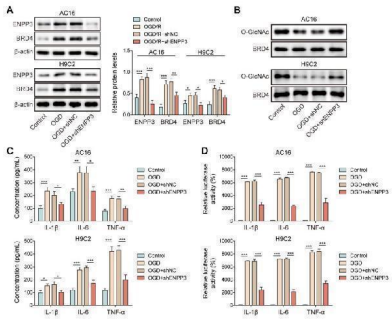


SRSF1 enhanced ENPP3 mRNA expression to trigger NF-kappaB p65-mediated transcription of pro-inflammatory cytokines. H9C2 and AC-16 cells were transfected with overexpression plasmid for ENPP3 or lncRNA ENPP3. (A) ENPP3 mRNA and lncRNA ENPP3 levels were evaluated by RT-qPCR. (B) Western blotting measured ENPP3 protein level in each group. (C) The mRNA levels of TNF-alpha, IL-1beta, and IL-6 were assessed by RT-qPCR. (D) ELISA detected the release of TNF-alpha, IL-1beta, and IL-6 from cardiomyocytes. (E) The binding of NF-kappaB p65 to the promoters of TNF-alpha, IL-1beta, and IL-6 was determined by dual-luciferase reporter assay. H9C2 and AC-16 cells were transfected with shSRSF1, overexpression plasmid for ENPP3, or a combination of them. (F) ENPP3 mRNA and lncRNA ENPP3 levels were measured by RT-qPCR. (G) Western blotting analysis of ENPP3 protein level in cardiomyocytes. (H)&(I) The levels of TNF-alpha, IL-1beta, and IL-6 in cardiomyocytes were evaluated by RT-qPCR and ELISA. (J) Dual-luciferase reporter assay analyzed the interaction between NF-kappaB p65 and the promoters of TNF-alpha, IL-1beta, and IL-6. n=3 for A-J. One-way ANOVA

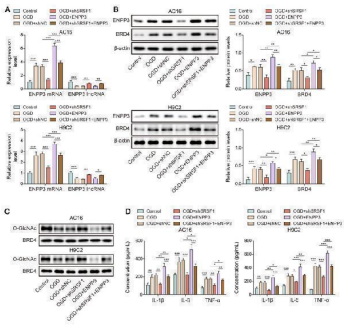
was performed to analyze data. * p < 0.05, ** p < 0.01, *** p < 0.001.Index in PubMed under a CC BY license. PMID: 40585977



SRSF1 enhanced ENPP3 mRNA expression to trigger NF-kappaB p65-mediated transcription of pro-inflammatory cytokines. H9C2 and AC-16 cells were transfected with overexpression plasmid for ENPP3 or lncRNA ENPP3. (A) ENPP3 mRNA and lncRNA ENPP3 levels were evaluated by RT-qPCR. (B) Western blotting measured ENPP3 protein level in each group. (C) The mRNA levels of TNF-alpha, IL-1beta, and IL-6 were assessed by RT-qPCR. (D) ELISA detected the release of TNF-alpha, IL-1beta, and IL-6 from cardiomyocytes. (E) The binding of NF-kappaB p65 to the promoters of TNF-alpha, IL-1beta, and IL-6 was determined by dual-luciferase reporter assay. H9C2 and AC-16 cells were transfected with shSRSF1, overexpression plasmid for ENPP3, or a combination of them. (F) ENPP3 mRNA and lncRNA ENPP3 levels were measured by RT-qPCR. (G) Western blotting analysis of ENPP3 protein level in cardiomyocytes. (H)&(I) The levels of TNF-alpha, IL-1beta, and IL-6 in cardiomyocytes were evaluated by RT-qPCR and ELISA. (J) Dual-luciferase reporter assay analyzed the interaction between NF-kappaB p65 and the promoters of TNF-alpha, IL-1beta, and IL-6. n=3 for A-J. One-way ANOVA was performed to analyze data. * p < 0.05, ** p < 0.01, *** p < 0.001.Index in PubMed under a CC BY license. PMID: 40585977



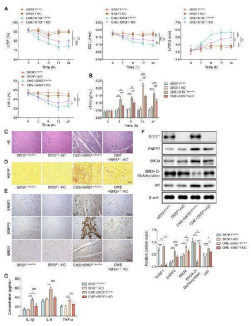
ENPP3 contributed to inflammation by inhibiting O-GlcNAcylation of BRD4. H9C2 and AC-16 cells were transfected with shENPP3, followed by exposure to OGD. (A) ENPP3 and BRD4 protein levels were measured by Western blotting. (B) The O-GlcNAc level of BRD4 protein was assessed. (C) The production of TNF-alpha, IL-1beta, and IL-6 was determined by ELISA. (D) Dual-luciferase reporter assay evaluated the binding of NF-kappaB p65 to TNF-alpha, IL-1beta, and IL-6 promoters. n=3 for A-D. One-way ANOVA was performed to analyze data. * p < 0.05, ** p < 0.01, *** p < 0.001.Index in PubMed under a CC BY license. PMID: 40585977



SRSF1/ENPP3 axis suppressed BRD4 O-GlcNAcylation to promote inflammation in CME. The OGD-stimulated cardiomyocytes were transfected with shSRSF1, ENPP3 overexpression plasmid, or a combination of them. (A) ENPP3 mRNA and lncRNA ENPP3 expression levels were detected by RT-qPCR. (B) The protein abundance of ENPP3 and BRD4 was assessed by Western blotting. (C) The O-GlcNAc level of BRD4 was determined. (D) ELISA was carried out to measure TNF-alpha, IL-1beta, and IL-6 concentrations. n=3 for A-D. One-way ANOVA was performed to analyze data. * p < 0.05, ** p < 0.01, *** p < 0.001.Index in PubMed under a CC BY license. PMID: 40585977

Myocardium-specific SRSF1 knockout alleviated CME-

BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com



induced inflammation via inactivation of the ENPP3/BRD4/NF-kappaB pathway. SRSF1 flox/flox and SRSF1-KO rats were injected with microspheres into the left ventricle to induce CME. (A) LVEF, LVFS, LVEDd, and CO were detected to evaluate cardiac function. (B) The serum cTnI level in different groups was measured by ELISA. (C) Pathological alterations in myocardial tissues were observed by HE staining (scale bar = 100 um). (D) Myocardial infarct size was measured by HBFP staining (scale bar = 100 um). (E) SRSF1, ENPP3, and BRD4 expression in myocardial tissues was evaluated by immunohistochemical staining (scale bar = 100 um). (F) The protein abundance of SRSF1, ENPP3, BRD4, p65, and O-GlcNAcylation of BRD4 was detected by Western blotting or Co-IP, respectively. (G) ELISA was carried out to measure TNF-alpha, IL-1beta, and IL-6 concentrations. n=6 for A-G. ANOVA for repeated measurement (for A, B), and one-way ANOVA (for F, G) was performed to analyze data. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.Index in PubMed under a CC BY license. PMID: 40585977

1 Publications Citing This Product

1. PubMed ID: 10.1002/pmic.201700443, Impacts of Dietary Pleurotus eryngii Polysaccharide on Nutrient Digestion, Metabolism, and Immune Response of the Small Intestine and Colon—An iTRAQ®Based Proteomic Analysis

Visit bosterbio.com/anti-cd203c-antibody-a05615-boster.html to see all 1 publications.

Anti-CD203c ENPP3 Antibody

For Research Use Only. Not for use in diagnostic procedures.