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antibody and ELISA experts

BOSTER BIOLOGICAL TECHNOLOGY
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Anti-SYNJ2BP antibody

Catalog Number: A11249

Overview

Product Name	Anti-SYNJ2BP antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-SYNJ2BP antibody catalog # A11249. Tested in WB, IHC, ICC, IF, IP, Flow Cytometry, ELISA applications. This antibody reacts with Human, Mouse, Rat.
Application	ELISA, Flow Cytometry, IP, IF, IHC, ICC, 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	P57105

Technical Details

Immunogen	E.coli-derived human SYNJ2BP recombinant protein (Position: M1-P115)
Form	Liquid
Concentration	500 ug/ml
Purification	Immunogen affinity purified.
Suggested Dilutions	Western blot, 1:500-2000 Immunohistochemistry, 1:50-400 Immunocytochemistry/Immunofluorescence, 1:50-400 Immunoprecipitation, 1:50 Flow Cytometry (Fixed), 1-3ug/1x10^6 cells ELISA, 1:100-1000

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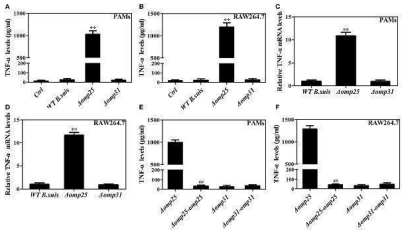
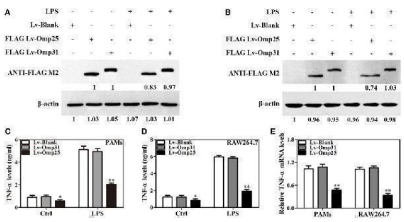
Anti-SYNJ2BP antibody (A11249) Images



Deficiency of omp25 enhances B. suis -induced tumor necrosis factor (TNF)-alpha production in porcine alveolar macrophages (PAMs) and RAW264.7 cells. (A,B) PAMs and RAW264.7 cells were infected with wild-type (WT) B. suis , Omp25-deficient mutant (Δ omp25 B. suis), Omp31-deficient mutant (Δ omp31 B. suis) or were uninfected (ctrl), and TNF-alpha secretion was measured at 24 h post-infection in culture supernatants by enzyme-linked immunosorbent assay (ELISA). (C,D) PAMs and RAW264.7 cells were infected with WT B. suis , Δ omp25 , or Δ omp31 and cultured for 6 h, Q-PCR was used to measure TNF-alpha mRNAs levels. (E,F) PAMs and RAW264.7 cells were infected with Δ omp25 , the complemented Δ omp25 strain of B. suis (Δ omp25 - omp25 B. suis), Δ omp31 , or the complemented Δ omp31 strain of B. suis (Δ omp31 - omp31 B. suis), followed by ELISA detection of TNF-alpha in culture supernatants. The results are mean ± SEM of three independent experiments. ** P < 0.01 versus WT B. suis -infected cells. ## P < 0.01 versus Δ omp25 B. suis -infected cells.Index in PubMed under a CC BY license. PMID: 29387067



Omp25, but not Omp31, inhibits LPS-induced tumor necrosis factor (TNF)-alpha production in porcine alveolar macrophages (PAMs) and RAW264.7 cells. (A,B) Evaluation of the expression of Omp25 and Omp31 in PAMs and RAW264.7 cells infected with LV-Blank, lentivirus expressing Omp25 (LV-Omp25), or LV-Omp31. PAMs and RAW264.7 cells were, respectively, infected with 100 multiplicities of infection (MOIs) of lentivirus for 24 h, and then treated with or without LPS for 24 h. The expression of protein was detected by western blotting. (C,D) Omp25 inhibits LPS-induced TNF-alpha production in PAMs and RAW264.7 cells. Cells were infected and expression of TNF-alpha was detected by enzyme-linked immunosorbent assay in culture supernatants. (E) Omp25 decreases the levels of TNF-alpha mRNA in LPS-treated PAMs and RAW264.7 cells. Cells were, respectively, infected with 100 MOIs of lentivirus for 24 h and stimulated with LPS for 6 h, Q-PCR was used to measure the levels of TNF-alpha mRNA. Values are mean ± SEM of three independent experiments. * P < 0.05, ** P < 0.01 versus LV-Blank-infected cells in the same processing.Index in PubMed under a CC BY license. PMID: 29387067

Omp25 inhibits the transcriptional expression of tumor necrosis factor (TNF)-alpha by suppressing NF-kappaB pathway activation. (A–D) Porcine alveolar macrophages (PAMs) and RAW264.7 cells were, respectively, transfected with pCI-neo, pCI-neo-Omp31, or pCI-neo-Omp25 along with TNF-alpha or NF-kappaB luciferase reporter plasmids for 24 h; cells were stimulated with or without LPS for another 24 h, and TNF-alpha promoter activities (A,B) and the relative transcriptional activities of NF-kappaB (C,D) were examined.

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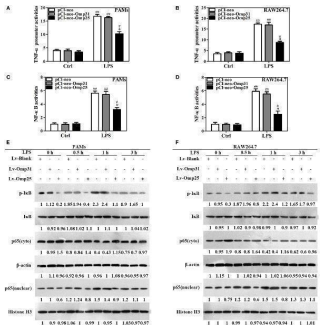
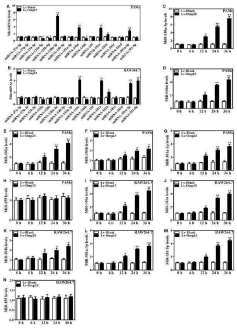
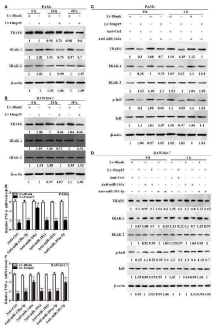


(E,F) PAMs and RAW264.7 cells were infected with 100 multiplicities of infection of LV-Blank, lentivirus expressing Omp25 (LV-Omp25), or LV-Omp31 for 24 h and the expression levels of cytoplasmic p-IkappaB, IkappaB, or p65 and nucleoprotein p65 at 0, 0.5, 1, and 3 h following LPS stimulation were determined by western blotting. The results are mean ± SEM of three independent experiments. * P < 0.05 versus LV-Blank-infected cells; # P < 0.05, ## P < 0.01 versus control (Ctrl) for same transfection.Index in PubMed under a CC BY license. PMID: 29387067

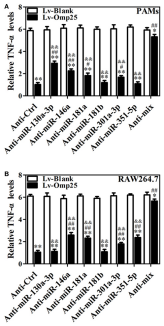


Omp25 upregulates miR-130a-3p, -146a, -181a, -181b, or -301a-3p in porcine alveolar macrophages (PAMs) and miR-146a, -181a, -181b, -301a-3p, or -351-5p in RAW264.7 cells. (A,B) Expression profiling of microRNAs in Omp25-expressing PAMs and RAW264.7 cells. Quantitative polymerase chain reaction (Q-PCR) assay was used to measure the levels of 17 specific miRNAs normalized by RNU6B at 24 h following infection. (C–H) Q-PCR was used to measure the kinetics of miR-130a-3p, miR-146a, miR-181a, miR-181b, miR-301a-3p, and miR-155 expression in PAMs infected with LV-Blank or lentivirus expressing Omp25 (LV-Omp25). (I–N) Q-PCR was used to measure the kinetics of miR-146a, miR-181a, miR-181b, miR-301a-3p, miR-351-5p, and miR-155 expression in RAW264.7 cells infected with LV-Blank or LV-Omp25. Results are mean ± SEM of three independent experiments. * P < 0.05, ** P < 0.01 versus LV-Blank-infected cells for same miRNAs or same time point.Index in PubMed under a CC BY license. PMID: 29387067

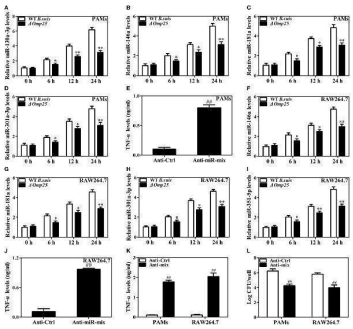


Omp25-induced miR-146a and miR-351-5p inhibit the transcriptional expression of tumor necrosis factor (TNF)-alpha by targeting to TRAF6 and IRAK1. (A,B) Porcine alveolar macrophages (PAMs) and RAW264.7 cells were infected with LV-Blank and lentivirus expressing Omp25 (LV-Omp25), and western blotting was used to determine the expressions of TRAF6, IRAK1, and IRAK2 at 0, 24, and 48 h. (C,D) PAMs and RAW264.7 cells were transfected with anti-miRNA control or indicated anti-miRNAs; then, cells were infected with LV-Blank or LV-Omp25 for 24 h, following LPS stimulation for another 1 h, and cells were lysed and examined for TRAF6, IRAK1, IRAK2, p-IkappaB, and IkappaB by western blotting. (E,F) Cells were transfected with anti-miRNA control or indicated anti-miRNAs and then infected with LV-Blank or LV-Omp25 for 24 h; following LPS treatment for 6 h, quantitative polymerase chain reaction was used to measure the level of TNF-alpha mRNA. The results are mean ± SEM of three independent experiments. * P < 0.05, ** P < 0.01 versus LV-Blank-infected cells; ## P < 0.01 versus LV-Omp25-infected cells with anti-control (Anti-Ctrl).Index in PubMed under a CC BY license. PMID: 29387067

miR-146a, miR-181a, and miR-301a-3p participate in the regulation of tumor necrosis factor (TNF)-alpha in both porcine alveolar macrophages (PAMs) and RAW264.7 cells,



whereas miR-130a-3p and miR-351-5p differentially regulate TNF-alpha expression in porcine and murine cells. (A,B) PAMs and RAW264.7 cells were transfected with anti-control, or indicated anti-miRNA, or anti-miRNAs mix (4 miRNA inhibitors); then, cells were infected with LV-Blank or lentivirus expressing Omp25 (LV-Omp25) for 24 h, and the levels of TNF-alpha were measured by enzyme-linked immunosorbent assay. The results are mean ± SEM of three independent experiments. * P < 0.05, ** P < 0.01 versus LV-Blank-infected cells; # P < 0.05, ## P < 0.01 versus LV-Omp25-infected cells with anti-control (Anti-Ctrl); && P < 0.01 versus LV-Omp25-infected cells with anti-mix (Anti-mix).Index in PubMed under a CC BY license. PMID: 29387067



Deficiency of Omp25 decreases B. suis -induced miR-130a-3p, miR-146a, miR-181a, miR-301a-3p, or miR-351-5p whereas inhibition of these miRNAs upregulates tumor necrosis factor (TNF)-alpha and promotes the intracellular clearance of wild-type (WT). B. suis . (A–D) Porcine alveolar macrophages (PAMs) were infected with WT B. suis or Δ omp25 B. suis for 0, 6, 12, and 24 h, and quantitative polymerase chain reaction (Q-PCR) was used to analyze the levels of indicated miRNAs. (E) PAMs were transfected anti-control or anti-miRNAs mix; cells were infected with WT B. suis for 24 h, and TNF-alpha production was measured by enzyme-linked immunosorbent assay (ELISA). (F–I) RAW264.7 cells were infected with WT B. suis or Δ omp25 B. suis for 24 h, and cells were harvested to examine the expression of indicated miRNAs by Q-PCR at 0, 6, 12, and 24 h. (J) RAW264.7 cells were treated as in (E) and followed by ELISA measurement of TNF-alpha in culture supernatants. (K) Cells were transfected with anti-miRNA control or anti-miRNAs mix and infected with WT B. suis for 24 h; following stimulation with LPS for another 24 h, TNF-alpha production was measured by ELISA. (L) Cells were transfected anti-miRNA control or anti-miRNAs mix; cells were infected with WT B. suis for 48 h and the numbers of viable intracellular bacteria were determined as described in Section “Materials and Methods.” The results are mean ± SEM of three independent experiments. * P < 0.05, ** P < 0.01 versus WT B. suis -infected cells at same time point; ## P < 0.01 versus Anti-Ctrl.Index in PubMed under a CC BY license. PMID: 29387067

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