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Anti-Transcription factor SOX-2 SOX2 Antibody Picoband®

Catalog Number: PA2284

About SOX2

SRY (sex determining region Y)-box 2, also known as SOX2, is a transcription factor that is essential for maintaining self-renewal, or pluripotency of undifferentiated embryonic stem cells. Sox2 is a member of the Sox family of transcription factors, which have been shown to play key roles in many stages of mammalian development. This gene is mapped to 3q26.33. It is found that SOX2 can regulate OCT3/4 expression and maintains ES pluripotency through upstream transcription factors. SOX2 is identified as a lineage-survival oncogene in lung and esophageal squamous cell carcinoma. In addition to those, SOX2 has a critical role in maintenance of embryonic and neural stem cells and holds great promise in research involving induced pluripotency, an emerging and very promising field of regenerative medicine.

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Overview

Product Name	Anti-Transcription factor SOX-2 SOX2 Antibody Picoband®
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Transcription factor SOX-2 SOX2 Antibody catalog # PA2284. Tested in IHC, WB applications. This antibody reacts with Human, Mouse, Rat. The brand Picoband indicates this is a premium antibody that guarantees superior quality, high affinity, and strong signals with minimal background in Western blot applications. Only our best-performing antibodies are designated as Picoband, ensuring unmatched performance.
Application	IHC, WB
Clonality	Polyclonal
Formulation	Each vial contains 4 mg Trehalose, 0.9 mg NaCl and 0.2 mg Na2HPO4.
Storage Instructions	Store at -20˚C for one year from date of receipt. After reconstitution, at 4˚C for one month. It can also be aliquotted and stored frozen at -20˚C for six months. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P48431

Technical Details

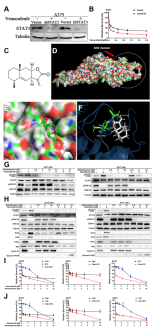
Immunogen	A synthetic peptide corresponding to a sequence in the middle region of human SOX2, different from the related mouse and rat sequence by one amino acid.

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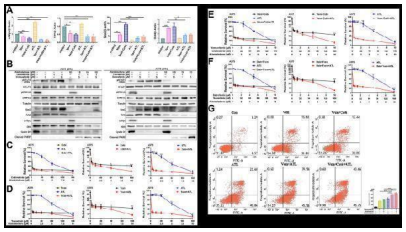
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot, and HRP Conjugated anti-Rabbit IgG Super Vision Assay Kit (SV0002-1) for IHC(P).
Cross Reactivity	No cross-reactivity with other proteins
Isotype	Rabbit IgG
Form	Lyophilized
Concentration	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml.
Purification	Immunogen affinity purified.
Suggested Dilutions	Immunohistochemistry (Paraffin-embedded Section), 2-5ug/ml, Human, Mouse, Rat Western blot, 0.1-0.5ug/ml, Human, Mouse, Rat

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Anti-Transcription factor SOX-2 SOX2 Antibody Picoband® (PA2284) Images



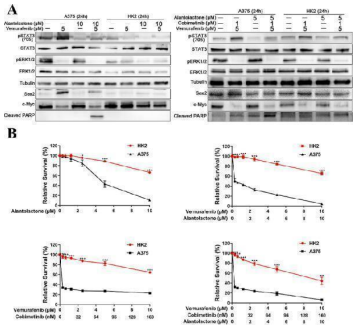
Alantolactone suppressed STAT3 feedback activation induced by BRAFi, downregulating protein expression of Oct4 and Sox2 in A375 cells. (A) A375 cells were transfected with shSTAT3 plasmid, and the expression of STAT3 protein was detected 48 h later. (B) A375 cells were transfected with shSTAT3 plasmid for 24 h and treated with different concentrations of vemurafenib. After another 72 h, cell viability was detected by CCK8 assays. (C) Chemical structure of alantolactone. (D) Predicted model of alantolactone binding to STAT3beta SH2, as shown by computational modeling. Protein structure information was obtained from Protein Data Bank (PDB) entry 6NJS. (E) Binding model of alantolactone to the SH2 domain. The molecular surface of the STAT3beta SH2 domain is electrostatically colored with blue and red representing potentially positively- and negatively-charged regions, respectively. (F) Predicted interactions between the amino acid residues of the SH2 domain and alantolactone. Oxygen atoms of alantolactone are shown in red. Alantolactone forms carbon hydrogen bond with MET554, and there is a alkyl bond between alantolactone and ALA555. (G and H) A375 cells were treated with alantolactone and BRAFi single or combination for 6 (G) or 24 (H) hours, phospho-STAT3 (705), STAT3, phospho-ERK1/2, ERK1/2, SOX2, Oct4, c-Myc, Klf4, cyclin D1 and cleaved PARP levels were analyzed by western blotting, and tubulin served as a loading control. (I and J) A375 cells were treated with different concentrations of alantolactone and vemurafenib (I) or alantolactone and dabrafenib (J) for 3 days. Cell viability was determined by CCK8 assays. The two images on the left in Figures G and H are split from the image on the right. Data are mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t -test Index in PubMed under a CC BY license. PMID: 38822350



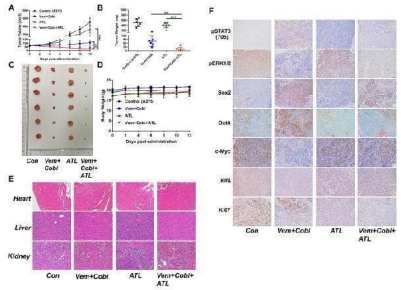
The combination of alantolactone and MAPKi simultaneously inhibited the STAT3 and BRAF/MEK/ERK pathways, regulating the expression level of downstream effectors in A375 cells. (A) A375 cells were treated with vemurafenib, cobimetinib and alantolactone alone or in combination for 6 h. The gene expression levels of c-Myc, Klf4, Sox2 and Oct4 were detected by real-time fluorescent quantitative PCR, and beta-actin served as a loading control. (B) A375 cells were treated with vemurafenib, cobimetinib and alantolactone for 24 h. Phospho-STAT3 (705), STAT3, phospho-ERK1/2, ERK1/2, Sox2, Oct4, c-Myc, Klf4, cyclin D1 and cleaved PARP levels were analyzed by western blotting, and tubulin served as a loading control. (C and D) A375 cells were treated with different concentrations of cobimetinib and alantolactone (C) or trametinib and alantolactone (D) for 72 h. Cell viability was determined by CCK8 assays. The two images on the left in Figures C and D are split from the image on the right. (E and F) A375 cells were treated with different concentrations of vemurafenib, cobimetinib, and alantolactone (E) or dabrafenib, trametinib and

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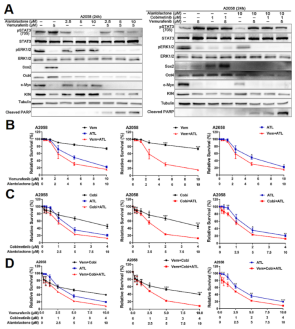
alantolactone (F) for 72 h. Cell viability was determined by CCK8 assays. The two images on the left in Figures C and D are split from the image on the right. (G) A375 cells were treated with vemurafenib (5 μM), cobimetinib (1 μM), and alantolactone (6 μM) alone or in combination for 24 h. Flow cytometry analysis of cell death (Annexin V/PI labelling) in A375 cells. The histogram on the right shows the proportion of dead cells. Data, means ± SDs. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350



Combined treatment with alantolactone and MAPK pathway inhibitors showed highly selective cytotoxic effects on A375 cells, but had no obvious cytotoxicity on renal tubular epithelial cells. (A) HK2 and A375 cells were treated with vemurafenib and alantolactone or vemurafenib, cobimetinib and alantolactone for 24 h. Phospho-STAT3 (705), STAT3, phospho-ERK1/2, ERK1/2, Sox2, c-Myc and cleaved PARP levels were analyzed by western blotting, and tubulin served as a loading control. (B) HK2 and A375 cells were treated with different concentrations of alantolactone, vemurafenib and cobimetinib for 72 h. Cell viability was determined by CCK8 assays. Data are the mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350



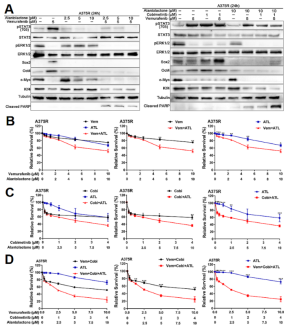
Alantolactone could synergistically enhance the cytotoxic effects with MAPKi in A375 xenografts of nude mice. (A) A375 xenografts were administrated by alantolactone (ATL; 20 mg/kg administered intraperitoneally once daily) and vemurafenib (Vem; 25 mg/kg administered intraperitoneally once daily) + cobimetinib (Cobi; 1 mg/kg administered intraperitoneally once daily) individually or in combination. During the treatment period, measure and record the tumor volume every other day. (B) After 12 days of treatment, tumor grafts were removed and weighed. (C) Photographs of xenograft A375 tumors treated with single or combination drugs. (D) During the drug treatment, the animals were weighed every other day. (E) Haematoxylin-eosin staining of heart, liver and kidney tissue sections (magnification: ×100). (F) Representative images of immunohistochemical staining of p-STAT3(705), p-ERK1/2, c-Myc, Klf4, Sox2, Oct4 and Ki67 in tumor tissues. Data are the mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350



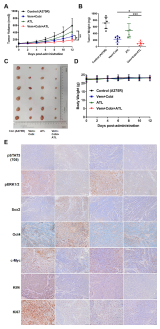
Alantolactone sensitized intrinsic-resistant A2058 cells to MAPKi targeted therapy by inhibiting STAT3 activation. (A) A2058 cells were treated with vemurafenib and alantolactone or vemurafenib, cobimetinib and alantolactone for 24 h. Phospho-STAT3 (705), STAT3, phospho-ERK1/2, ERK1/2, Sox2, Oct4, c-Myc, Klf4, cyclin D1 and cleaved PARP levels were analyzed by western blotting, and tubulin served as a loading control. (B-D) A2058 cells were treated with different concentrations of vemurafenib and alantolactone (B), cobimetinib and alantolactone (C), or vemurafenib, cobimetinib and alantolactone (D) for 72 h. Cell viability

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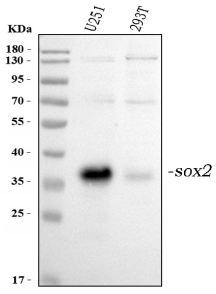
was determined by CCK8 assays. Data are the mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350



The combination of alantolactone and MAPKi could inhibit the proliferation of MAPKi-resistant A375R cells. (A) A375R cells were treated with vemurafenib and alantolactone or vemurafenib, cobimetinib and alantolactone for 24 h. Phospho-STAT3 (705), STAT3, phospho-ERK1/2, ERK1/2, Sox2, Oct4, c-Myc, Klf4, cyclin D1 and cleaved PARP levels were analyzed by western blotting, and tubulin served as a loading control. (B-D) A375R cells were treated with different concentrations of vemurafenib and alantolactone (B), cobimetinib and alantolactone (C), or vemurafenib, cobimetinib and alantolactone (D) for 72 h. Cell viability was determined by CCK8 assays. Data are the mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350

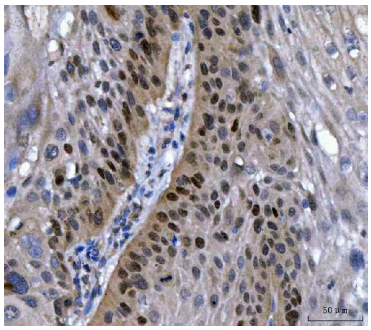


Alantolactone could synergistically enhance the cytotoxic effects with MAPKi in A375 xenografts of nude mice. (A) Tumor growth curves of A375R xenograft models treated with alantolactone (ATL; 20 mg/kg administered intraperitoneally once daily) and vemurafenib (Vem; 25 mg/kg administered intraperitoneally once daily) + cobimetinib (Cobi; 1 mg/kg administered intraperitoneally once daily) alone or in combination. (B) After 12 days of treatment, tumor grafts were removed and weighed. (C) Photographs of xenograft A375R tumors treated with single or combination drugs. (D) During the drug treatment, the animals were weighed every other day. (E) Representative images of immunohistochemical staining of p-STAT3(705), p-ERK1/2, c-Myc, Klf4, Sox2, Oct4 and Ki67 in tumor tissues. Data are the mean ± SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Student's t test Index in PubMed under a CC BY license. PMID: 38822350

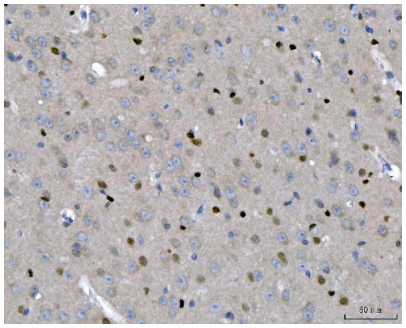


Western blot analysis of SOX2 using anti-SOX2 antibody (PA2284). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: human U251 whole cell lysates, Lane 2: human 293T 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-SOX2 antigen affinity purified polyclonal antibody (Catalog # PA2284) at 0.5 ug/mL 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 Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for SOX2 at approximately 36 kDa. The expected band size for SOX2 is at 34 kDa.

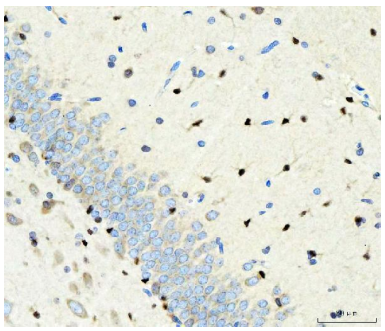
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IHC analysis of SOX2 using anti-SOX2 antibody (PA2284). SOX2 was detected in a paraffin-embedded section of human laryngeal squamous cell carcinoma tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-SOX2 Antibody (PA2284) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.



IHC analysis of SOX2 using anti-SOX2 antibody (PA2284). SOX2 was detected in a paraffin-embedded section of mouse brain tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-SOX2 Antibody (PA2284) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.



IHC analysis of SOX2 using anti-SOX2 antibody (PA2284). SOX2 was detected in a paraffin-embedded section of rat brain tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-SOX2 Antibody (PA2284) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.

2 Publications Citing This Product

1. PubMed ID: 25888093, Cheng Z, Wang Hz, Li X, Wu Z, Han Y, Li Y, Chen G, Xie X, Huang Y, Du Z, Zhou Y. J Exp Clin Cancer Res. 2015 Mar 26;34:27. Doi: 10.1186/S13046-015-0142-9. Microrna-184 Inhibits Cell Proliferation And Invasion, And Specifically Targets Tnfaip2 In G...

2. PubMed ID: 29531534, Yi H, Xie B, Liu B, Wang X, Xu L, Liu J, Li M, Zhong X, Peng F. Stem Cells Int. 2018 Jan 24;2018:3628578. doi: 10.1155/2018/3628578. eCollection 2018. Derivation and Identification of Motor Neurons from Human Urine-Derived Induced Pluripotent Stem...

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