

Anti-AKR1C1/C2 Antibody Picoband®

Catalog Number: PB10036

About AKR1C2

This gene encodes a member of the aldo/keto reductase superfamily, which consists of more than 40 known enzymes and proteins. These enzymes catalyze the conversion of aldehydes and ketones to their corresponding alcohols using NADH and/or NADPH as cofactors. The enzymes display overlapping but distinct substrate specificity. This enzyme binds bile acid with high affinity, and shows minimal 3-alpha-hydroxysteroid dehydrogenase activity. And this gene shares high sequence identity with three other gene members and is clustered with those three genes at chromosome 10p15-p14. Three transcript variants encoding two different isoforms have been found for this gene.

Overview

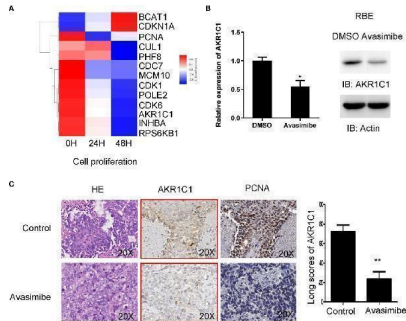
Product Name	Anti-AKR1C1/C2 Antibody Picoband®
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-AKR1C1/C2 Antibody Picoband® catalog # PB10036. Tested in Flow Cytometry, IF, ICC, WB applications. This antibody reacts with Human, Mouse, Rat. The brand Picoband indicates this is a premium 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	Flow Cytometry, IF, ICC, WB
Clonality	Polyclonal
Formulation	Each vial contains 4mg Trehalose, 0.9mg NaCl and 0.2mg Na ₂ HPO ₄ .
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	P52895

Technical Details

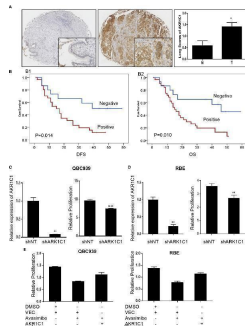
Immunogen	E. coli-derived human AKR1C1/C2 recombinant protein (Position: M1-K123).
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot, and HRP Conjugated anti-Rabbit IgG Super Vision Assay Kit (SV0002-1) for ICC.
Cross Reactivity	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	Western blot, 0.1-0.5ug/ml, Human, Mouse, Rat Immunocytochemistry/Immunofluorescence, 5 ug/ml, Human Flow Cytometry (Fixed), 1-3 ug/1x10 ⁶ cells, Human

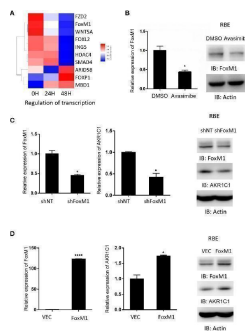
Anti-AKR1C1/C2 Antibody Picoband® (PB10036) Images



cDNA array analysis identified AKR1C1 as a potential target of avasimibe. (A) RBE cells were treated with avasimibe at the concentration of 20 μM for 24 and 48 hours and subjected to cDNA array analysis. Cluster of changed genes in cell proliferation was presented. (B) The level of AKR1C1 mRNA and protein was detected by RT-PCR and western blotting in RBE cells when treated with avasimibe (20 μM) for 48 hours. (C) The expression of AKR1C1 and PCNA was detected by IHC on the resected xenografts. IHC, ×200. *P < 0.05, **P < 0.01. 'long scores of AKR1C1' means AKR1C1 staining score. IHC, immunohistochemistry. Index in PubMed under a CC BY license. PMID: 34127944

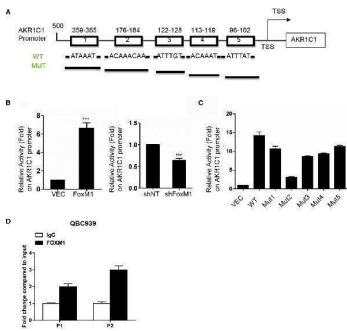


The oncogenic role of AKR1C1 in cholangiocarcinoma. (A) Representative images of negative staining of AKR1C1 in noncancerous tissues (N) and high expression of AKR1C1 in CCA (T). IHC, ×200 for small pictures and ×40 for large pictures. *P < 0.05. (B) Patients with AKR1C1 expression had a shorter time to recurrence (B1) and a worse overall survival (B2) than those without AKR1C1 expression. (C, D) QBC939 (C) and RBE (D) cells were transfected with AKR1C1-shRNA for 48 hours and the level of AKR1C1 mRNA was detected by RT-PCR. CCK8 assay was used to measure the cell viability of both cell lines. **P < 0.01, ****P < 0.0001. (E) QBC939 and RBE cells were treated with avasimibe with or without exogenous AKR1C1 plasmid for 48 hours and CCK8 was used to detect the changes of cell viability. IHC, immunohistochemistry; CCK8, Cell Counting Kit-8; Vec, vector. Index in PubMed under a CC BY license. PMID: 34127944



AKR1C1 is regulated by FoxM1 in cholangiocarcinoma. (A) RBE cells were treated with avasimibe (20 μM) for 24 and 48 hours and subjected to cDNA array analysis. Cluster of changed genes in regulation of transcription was presented. (B) The level of FoxM1 mRNA and protein was detected by RT-PCR and western blotting in RBE cells when treated with avasimibe (20 μM) for 48 hours. (C) RBE cells were transfected with FoxM1-shRNA for 48 hours and the levels of FoxM1 and AKR1C1 mRNA and proteins were detected by RT-PCR and western blotting. (D) RBE cells were transfected with FoxM1 plasmid for 48 hours and the levels of FoxM1 and AKR1C1 mRNA and proteins were detected by RT-PCR and western blotting. *P < 0.05, ****P < 0.0001. Index in PubMed under a CC BY license. PMID: 34127944

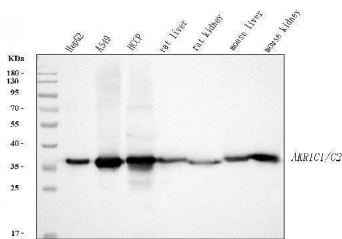
AKR1C1 is a direct transcriptional target of FoxM1. (A) Diagram shows the sequence and position of five putative FoxM1-binding elements in the AKR1C1 promoter. TSS, transcriptional start site; WT, wild type; Mut, mutant type. (B) Left panel, RBE cells were cotransfected with the AKR1C1 promoter reporter, pRL-TK, and pcDNA3.1-FoxM1 or pcDNA



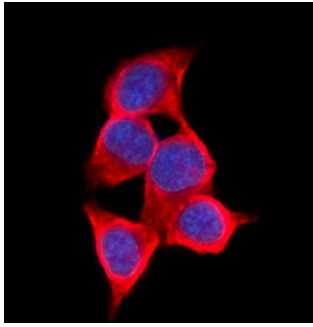
3.1; right panel, RBE cells were cotransfected with the AKR1C1 promoter reporter, pRL-TK, and FoxM1-shRNA or shcontrol (50 nM). 36 hours after transfection, the cells were collected, and the relative AKR1C1 promoter activities were measured. The assay was repeated three times independently. ***P < 0.001. (C) Reporter plasmids harboring the wild-type AKR1C1 promoter or the corresponding mutant promoter in the FoxM1-binding sites were transfected into RBE cells, and the relative promoter activities were measured as above. (D) The chromatin immunoprecipitation (ChIP) assay results show the in vivo binding of FoxM1 to the AKR1C1 promoter. QBC939 cell lysis was immunoprecipitated using an anti-FoxM1 antibody or immunoglobulin G. The resulting samples were subjected to RT-PCR using the site-specific primers. Index in PubMed under a CC BY license. PMID: 34127944



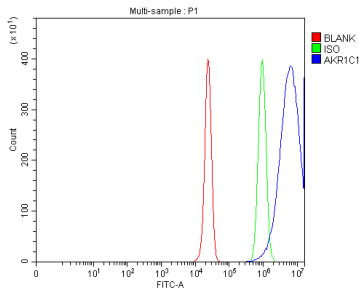
Avasimibe inhibits cholangiocarcinoma cells proliferation via targeting AKR1C1 and FoxM1. (A, B) RBE cells (A) and QBC939 cells (B) were treated with avasimibe or DMSO, then transfected with FoxM1 or control vector, along with the transfection with AKR1C1 shRNA or shNT. 48 h after transfection, cell viability was analyzed by CCK8 assay. Data are from three independent assays. *P < 0.05, **P < 0.01, ***P < 0.001. (C) Left panel, the expression of FoxM1 and AKR1C1 was detected by IHC on the resected xenografts. IHC, $\times 400$. Right panel, diagram showing the different expression of FoxM1 and AKR1C1 in these samples when treated with avasimibe. (D) The representative images of FoxM1 and AKR1C1 expressions and their correlations determined by Spearman's correlation test. r, Spearman correlation coefficient; IHC, $\times 40$ or $\times 200$. Index in PubMed under a CC BY license. PMID: 34127944



Western blot analysis of AKR1C1/C2 using anti-AKR1C1/C2 antibody (PB10036). 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 μ g of sample under reducing conditions. Lane 1: human HepG2 whole cell lysates, Lane 2: human A549 whole cell lysates, Lane 3: human HCCP tissue lysates, Lane 4: rat liver tissue lysates, Lane 5: rat kidney tissue lysates, Lane 6: mouse liver tissue lysates, Lane 7: mouse kidney tissue 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-AKR1C1/C2 antigen affinity purified polyclonal antibody (Catalog # PB10036) at 0.5 μ g/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 AKR1C1/C2 at approximately 37 kDa. The expected band size for AKR1C1/C2 is at 37 kDa.



IF analysis of AKR1C1/C2 using anti-AKR1C1/C2 antibody (PB10036). AKR1C1/C2 was detected in an immunocytochemical section of HepG2 cells. Enzyme antigen retrieval was performed using IHC enzyme antigen retrieval reagent (AR0022) for 15 mins. The cells were blocked with 10% goat serum. And then incubated with 5 ug/mL rabbit anti-AKR1C1/C2 Antibody (PB10036) overnight at 4°C. DyLight®594 Conjugated Goat Anti-Rabbit IgG (BA1142) was used as secondary antibody at 1:100 dilution and incubated for 30 minutes at 37°C. The section was counterstained with DAPI. Visualize using a fluorescence microscope and filter sets appropriate for the label used.



Flow Cytometry analysis of HepG2 cells using anti-AKR1C1/C2 antibody (PB10036). Overlay histogram showing HepG2 cells stained with PB10036 (Blue line). To facilitate intracellular staining, cells were fixed with 4% paraformaldehyde and permeabilized with permeabilization buffer. The cells were blocked with 10% normal goat serum. And then incubated with rabbit anti-AKR1C1/C2 Antibody (PB10036, 1 ug/1x10⁶ cells) for 30 min at 20°C. DyLight®488 conjugated goat anti-rabbit IgG (BA1127, 5-10 ug/1x10⁶ cells) was used as secondary antibody for 30 minutes at 20°C. Isotype control antibody (Green line) was rabbit IgG (1 ug/1x10⁶) used under the same conditions. Unlabelled sample without incubation with primary antibody and secondary antibody (Red line) was used as a blank control.

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Anti-AKR1C1/C2 Antibody

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