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# Anti-Apoptosis regulator BAX Bax Antibody Picoband®

**Catalog Number:** PA1013-1

## About BAX

Apoptosis regulator BAX, also known as bcl-2-like protein 4, is a protein that in humans is encoded by the BAX gene. The protein encoded by this gene belongs to the BCL2 protein family. BCL2 family members form hetero- or homodimers and act as anti- or pro-apoptotic regulators that are involved in a wide variety of cellular activities. This protein forms a heterodimer with BCL2, and functions as an apoptotic activator. Additionally, this protein is reported to interact with, and increase the opening of, the mitochondrial voltage-dependent anion channel (VDAC), which leads to the loss in membrane potential and the release of cytochrome c. The expression of this gene is regulated by the tumor suppressor P53 and has been shown to be involved in P53-mediated apoptosis. Multiple alternatively spliced transcript variants, which encode different isoforms, have been reported for this gene.

## Overview

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| Product Name | Anti-Apoptosis regulator BAX Bax Antibody Picoband® |
| Reactive Species | Human, Mouse, Rat |
| Description | Boster Bio Anti-Apoptosis regulator BAX Bax Antibody catalog # PA1013-1. Tested in Flow Cytometry, IP, ICC/IF, 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 | Flow Cytometry, IP, IF, IHC, ICC, 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 | Q07812 |

## Technical Details

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| Immunogen | A synthetic peptide corresponding to a sequence at the N-terminus of human Bax, different from the related mouse and rat sequences by one amino acid. |
| 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 |

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| 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<br>Immunohistochemistry (Paraffin-embedded Section), 2-5ug/ml, Human<br>Immunocytochemistry/Immunofluorescence, 5 ug/ml, Human<br>Immunoprecipitation, 0.5-2 ug/ml, Human<br>Flow Cytometry (Fixed), 1-3ug/1x$10^6$ cells, Human |

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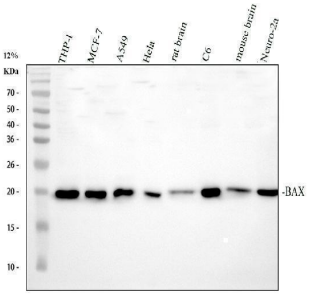
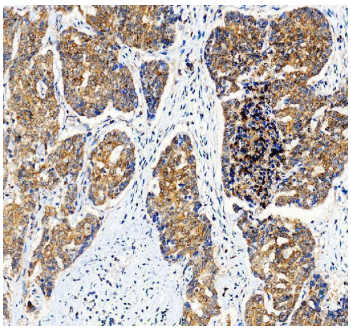
# Anti-Apoptosis regulator BAX Bax Antibody Picoband® (PA1013-1) Images




Western blot analysis of BAX using anti-BAX antibody (PA1013-1). Electrophoresis was performed on a 12% 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 THP-1 whole cell lysates, Lane 2: human MCF-7 whole cell lysates, Lane 3: human A549 whole cell lysates, Lane 4: human Hela whole cell lysates, Lane 5: rat brain tissue lysates, Lane 6: rat C6 whole cell lysates, Lane 7: mouse brain tissue lysates, Lane 8: mouse Neuro-2a 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-BAX antigen affinity purified polyclonal antibody (PA1013-1) 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 ECL Plus Western Blotting Substrate (Catalog # AR1196-200) with Tanon 5200 system. A specific band was detected for BAX at approximately 21 kDa. The expected band size for BAX is at 21 kDa.



IHC analysis of BAX using anti-BAX antibody (PA1013-1). BAX was detected in a paraffin-embedded section of human liver cancer 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-BAX Antibody (PA1013-1) 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 BAX using anti-BAX antibody (PA1013-1). BAX was detected in a paraffin-embedded section of human tonsil 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-BAX Antibody (PA1013-1) 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.

IF analysis of BAX using anti-BAX antibody (PA1013-1). BAX was detected in an immunocytochemical section of A549

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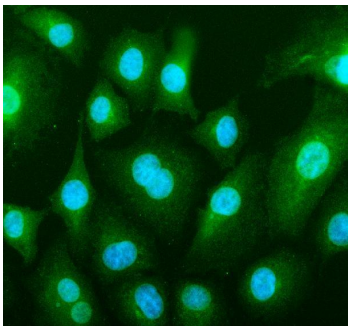
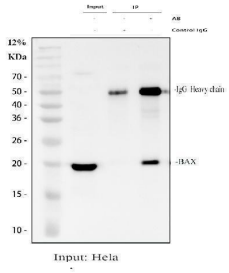
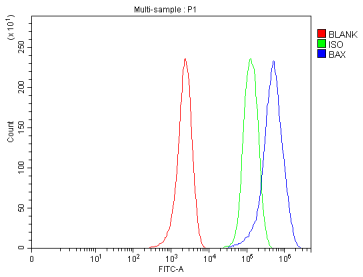


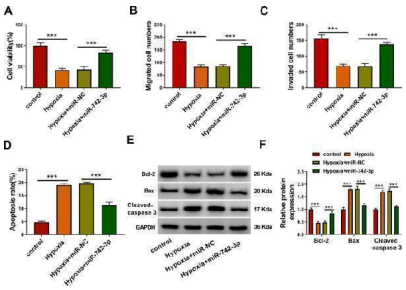
cells. Enzyme antigen retrieval was performed using IHC enzyme antigen retrieval reagent (AR0022) for 15 mins. The cells were blocked with 10% goat serum. And then incubated with 5 ug/mL rabbit anti-BAX Antibody (PA1013-1) overnight at 4°C. Fluoro488 Conjugated Goat Anti-Rabbit IgG (BA1127) was used as secondary antibody at 1:500 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.




Immunoprecipitating (IP) BAX in Hela whole cell lysate. Western blot analysis of BAX using anti-BAX antibody (PA1013-1); Lane 1: Hela whole cell lysates (30ug); Lane 2: Rabbit control IgG instead of anti-BAX antibody in Hela whole cell lysate; Lane 3: anti-BAX antibody (2ug) + Hela whole cell lysate (500ug). After electrophoresis, proteins were transferred to a membrane. Then the membrane was incubated with rabbit anti-BAX antigen affinity purified polyclonal antibody (PA1013-1) at a dilution of 0.5 ug/mL and probed with a goat anti-rabbit IgG-HRP secondary antibody (Catalog # BA1054). The signal is developed using ECL Plus Western Blotting Substrate (Catalog # AR1196-200). A specific band was detected for BAX at approximately 21 kDa. The expected band size for BAX is at 21 kDa.




Flow Cytometry analysis of MCF-7 cells using anti-BAX antibody (PA1013-1). Overlay histogram showing MCF-7 cells stained with PA1013-1 (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-BAX Antibody (PA1013-1, 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.




MiR-742-3p relieved hypoxia-induced H9c2 cell injury. H9c2 cells were transfected with or without miR-NC (50 nM) or miR-742-3p mimic (50 nM), and then treated with hypoxia. Untreated H9c2 cells were used as control. Cell viability, migrated and invaded cell numbers, and cell apoptosis rate were measured using CCK8 assay ( A ), transwell assay ( B , C ) and flow cytometry ( D ). E , F The protein levels of Bcl-2, Bax and Cleaved-caspase 3 were determined using WB analysis. All experiments were repeated three times. *** P

Effects of circRbms1 silencing and miR-742-3p inhibitor on hypoxia-induced H9c2 cell injury. A After transfecting with anti-NC (50 nM) or anti- miR-742-3p (50 nM) into H9c2 cells,

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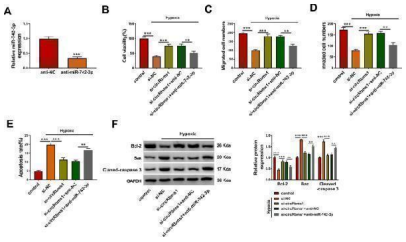
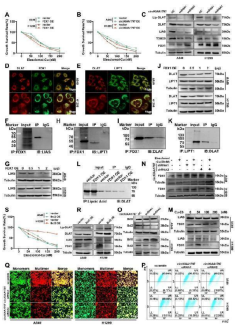


the expression of miR-742-3p was assessed by qRT-PCR. B–F H9c2 cells were transfected with si-NC (50 nM), si-circRbms1 (50 nM), si- circRbms1 (50 nM) + anti-NC (50 nM), or si- circRbms1 (50 nM) + anti- miR-742-3p (50 nM), and then treated with hypoxia. Untreated H9c2 cells were used as control. CCK8 assay ( B ), transwell assay ( C , D ), and flow cytometry ( E ) were employed to examine cell viability, migrated and invaded cell numbers, and cell apoptosis rate, respectively. F WB analysis was used to test the protein levels of Bcl-2, Bax, and Cleaved-caspase 3. All experiments were repeated three times. ** P



Exploration of the mechanism of cuproptosis induction. ( A ) After the transient overexpression of FDX1, a CCK-8 assay was used to detect changes in cellular resistance to cuproptosis. ( B ) After the transient overexpression of circKIAA1797, a CCK-8 assay was performed to detect changes in cellular resistance to cuproptosis. ( C ) Western blot analysis of Lip-DLAT, DLAT, LIAS, TOM20, and FDX1 protein expression after the stable silencing of circKIAA1797. ( D ) Immunofluorescence staining showed the colocalisation of the DLAT and FDX1 proteins. ( E ) Immunofluorescence staining was performed to detect the colocalisation of DLAT with the LIPT1 protein. ( F ) Western blot results of the Co-IP assay used to detect FDX1 binding to LIAS. ( G ) Western blot analysis of LIAS protein expression levels after the gradient overexpression of FDX1. ( H ) Western blot results of the Co-IP assay for detecting FDX1 binding to LIPT1. ( I ) Western blot results of Co-IP experiments for detecting FDX1 binding to DLAT. ( J ) Gradient overexpression of FDX1 followed by Western blot detection of DLAT and LIPT1 protein expression levels. ( K ) Western blot results of the Co-IP assay used to detect LIPT1 binding to DLAT. ( L ) After the overexpression of FDX1, Co-IP experiments were performed to detect changes in the ability of lipoic acid to bind DLAT. ( M ) Cells were treated with 0 nM, 50 nM, 100 nM and 200 nM elesclomol-Cu, and Western blot was performed to examine the protein expression of LIAS and FDX1. ( N ) circKIAA1797 stably transfected cells were treated with 50 nM elesclomol-Cu, and FDX1 protein expression was examined by Western blot. ( O ) Western blot analysis of BAX and Bcl2 protein expression in cell lines with stable circKIAA1797 silencing. ( P ) Flow cytometry detection of changes in the mitochondrial membrane potential after the transient silencing of circKIAA1797. ( Q ) Fluorescence microscopy comparing the changes in JC monomer and multimer levels after the transient silencing of circKIAA1797. ( R ) After the overexpression of Bcl2, Western blot assays were performed to detect Lip-DLAT, DLAT, LIAS, and FDX1 protein expression. ( S ) After the overexpression of Bcl2, a CCK-8 assay was performed to detect changes in cellular resistance to cuproptosis Index in PubMed under a CC BY license. PMID: 40176113

Knockdown of circRbms1 alleviated hypoxia-induced H9c2 cell injury. A , B qRT-PCR was used to assess circRbms1 expression to evaluate the transfection efficiency of si-circRbms1 (50 nM) or circRbms1 overexpression vector (4.0

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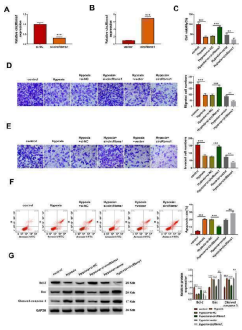
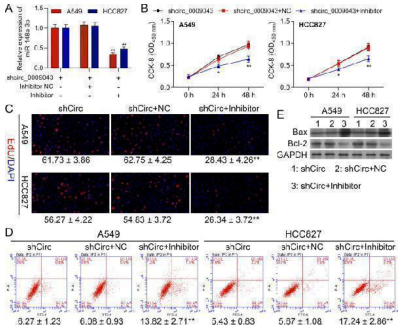
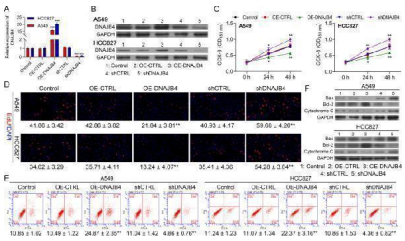


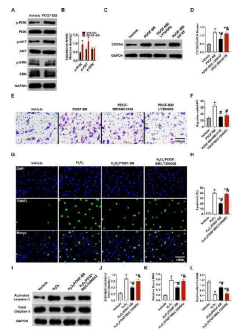
μg) in H9c2 cells. C – G H9c2 cells were transfected with or without si-NC (50 nM), si- circRbms1 (50 nM), vector (4.0 μg) or circRbms1 (4.0 μg), and then treated with hypoxia. Untreated H9c2 cells were used as control. CCK8 assay ( C ), transwell assay ( D , E ) and flow cytometry ( F ) were used to determine cell viability, migrated and invaded cell numbers, and cell apoptosis rate, respectively. G WB analysis was performed to test the protein levels of Bcl-2, Bax, and Cleaved-caspase 3. All experiments were repeated three times. ** P



MiR-148a-3p Reversed the Regulatory Effect of circ_0009043 on A549 and HCC827 cells. A549 and HCC827 cells were transfected with shCirc, shCirc + NC, or shCirc + miR-148a-3p inhibitor. (A) MiR-148a-3p mRNA levels in A549 and HCC827 were determined with RT-qPCR assay. B Viability in A549 and HCC827 cells at 0, 24, and 48 h. C EdU assay to detect A549 and HCC827 cell proliferation. (D) FSC assay to detect A549 and HCC827 cell apoptosis. E Protein levels of Bax and Bcl-2 in A549 and HCC827 cells with the indicated transfection were determined by western blot. GAPDH is a loading control. Data are presented as mean ± standard deviation. ** P



Overexpression of DNAJB4 inhibits the proliferation but promotes apoptosis abilities of NSCLC cells. A DNAJB4 mRNA expression in HCC827 and A549 cells transfected with pcDNA4.0 vector, pcDNA4.0- DNAJB4 vector, CTRL-shRNA, or DNAJB4 -shRNA were determined by RT-qPCR. B DNAJB4 protein expression in HCC827 and A549 cells transfected with indicated vectors, which divided into five groups, including Control, OE-CTRL, OE-DNAJB4, shCTRL and shDNAJB4. C CCK8 assay was used to compare the cell proliferation of CTRL, OE-CTRL, OE- DNAJB4, shCTRL and sh DNAJB4 groups in HCC827 and A549 cells. D Edu assay of Control, OE-CTRL, OE- DNAJB4, shCTRL and sh DNAJB4 groups in HCC827 and A549 cells, respectively. E FSC assay to detect cell apoptosis of Control, OE-CTRL, OE- DNAJB4, shCTRL and sh DNAJB4 groups in HCC827 and A549 cells. F Expression level of Bax, Bcl-2 and Cytochrome C in the HCC827 and A549 cells of the 5 groups (Control, OE-CTRL, OE- DNAJB4, shCTRL and sh DNAJB4 groups) were determined by western blotting. Control negative control, OE over expression. Data represent mean values ± SD from three replicates of each sample; ** P



PDGF-BB promotes MSC migration and protects MSCs against apoptosis via PI3K/Akt signaling. ( A - B ) Western blot analysis of total and phosphorylated PI3K, Akt, and ERK in MSCs with or without PDGF-BB treatment. PDGF-BB, 50 ng/ml. Values are the mean ± SEM. Significant differences were determined by Student's t test. N = 6/group. *P<0.05 vs. vehicle. ( C - D ) Western blot analysis of CXCR4 in MSCs. LY294002, 10 μM, U0126, 10 μM, 2 h before PDGF-BB treatment. Values are the mean ± SEM. Significant differences were determined by using one-way ANOVA. N =

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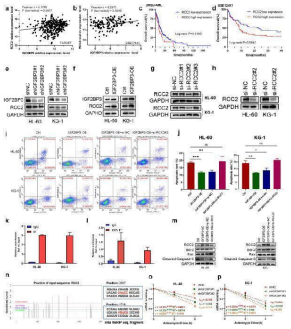
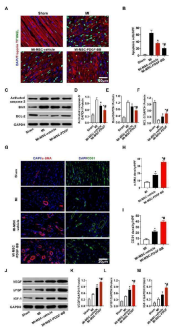
6/group. *p<0.01 vs. vehicle; # p<0.01 vs. PDGF-BB; & p<0.01 vs. PDGF-BB/LY294002. ( E - F ). MSC migratory capacities were evaluated by Transwell assay. LY294002, 50 μM. AMD3100, 44 nM. PDGF-BB, 50 ng/ml. Bar, 100 um. Values are the mean ± SEM. Significant differences were determined by using one-way ANOVA. N = 6/group. *p<0.01 vs. vehicle; # p<0.01 vs. PDGF-BB; & p<0.01 vs. PDGF-BB/AMD3100. ( G - H ). MSC apoptosis was evaluated by TUNEL assay. Bar, 50 um. ( I - J ) Western blot analysis of activated caspase-3 in MSCs. ( K - L ) Bax and BCL-2 mRNA expression levels were determined by RT–PCR in MSCs. H 2 O 2 , 200 μM, 6 h. LY294002, 30 μM. Values are the mean ± SEM. Significant differences were determined by using one-way ANOVA. N = 6/group. *p<0.01 vs. vehicle; # p<0.01 vs. H 2 O 2 ; & p<0.01 vs. H 2 O 2 /PDGF-BB Index in PubMed under a CC BY license. PMID: 38102643



IGF2BP3 regulates RCC2 expression in an m6A-dependent manner. a , b RCC2 expression was positively correlated with IGF2BP3 expression in the GSE37642 and TARGET datasets. c , d Kaplan–Meier survival analysis revealed that high RCC2 expression indicated a poor prognosis in AML patients. e , f Protein expression level of RCC2 following knockdown or overexpression of IGF2BP3 in HL-60 and KG-1 cells. g , h The interference efficiency of the siRNAs was evaluated to confirm the feasibility of the siRNAs, and si-RCC2#2 was found to be effective in reducing RCC2 expression. i , j Apoptosis was detected by flow cytometry. RCC2 deficiency promoted the induction of apoptosis by IGF2BP3 overexpression. k The mRNA of RCC2 was enriched by the anti-IGF2BP3 antibody compared to IgG in the HL-60 and KG-1 cell lines. l The mRNA of RCC2 was enriched by the m6A-specific antibody compared to IgG in the HL-60 and KG-1 cell lines. m Overexpression of IGF2BP3 restored the increases in the levels of proapoptotic proteins (Bax and cleaved Caspase 3) caused by silencing RCC2, and the level of the antiapoptotic protein Bcl-2 was slightly decreased. n The potential m6A sites in RCC2 were predicted by SRAMP. The different colored lines indicate different confidence levels. o , p Loss of IGF2BP3 reduced RCC2 stability in HL-60 and KG-1 cells. Transfected cells were treated with 5 μg/ml actinomycin D for 0 h, 3 h, or 6 hours prior to RNA extraction. * P



Transplantation of PDGF-BB-primed MSCs via UTMD reduces cardiomyocyte apoptosis and improves angiogenesis in rat hearts post-MI. ( A ) Representative images of TUNEL-positive cardiomyocytes in the ischemic area 30 days after MI. Apoptotic nuclei were identified as TUNEL positive (green fluorescence), and total nuclei were identified by DAPI counterstaining (blue fluorescence). Myocardium was stained using a monoclonal antibody against cardiac troponin I (red fluorescent). Bar, 20 um. ( B ). Quantification of TUNEL-positive cardiomyocytes. ( C - F ) Western blotting of activated caspase 3, Bax, and BCL-2 in the ischemic heart. GAPDH was used as a loading control. ( G ) Representative images of CD31 staining and alpha-SMA staining in the ischemic hearts of rats 30 days post-MI. Bar,

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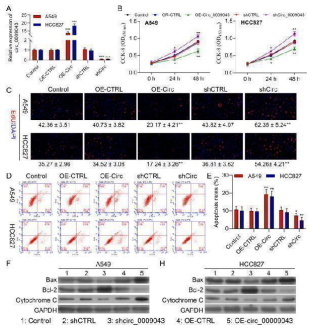
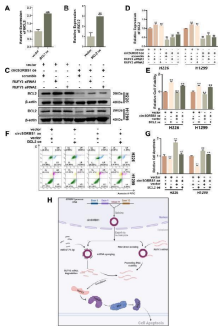
20 um. ( H ) Quantitative analysis of the capillary density in the ischemic heart. (I) Quantitative analysis of the arteriole density in the ischemic heart. ( J - M ) Protein expression of VEGF, bFGF and IGF-1 determined by Western blotting in ischemic myocardium, with GAPDH as the internal control. Values are the mean ± SEM. Significant differences were determined by using one-way ANOVA. N = 5/group. *p<0.01 vs. MI; # p<0.01 vs. MI-MSC-vehicle Index in PubMed under a CC BY license. PMID: 38102643



Overexpression of circ_0009043 inhibits the proliferation, while accelerates apoptosis of NSCLC cells. A circ_0009043 mRNA expression in HCC827 and A549 cells transfected with pcDNA4.0 vector (OE-CTRL), pcDNA4.0- circ_0009043 vector (OE-Circ), CTRL-shRNA (shCTRL), or circ_0009043-shRNA (shCirc) were determined by RT-qPCR. B CCK8 assay was used to compare the cell proliferation of Control, OE-CTRL, OE-Circ, shCTRL, and shCirc groups in HCC827 and A549 cells. C Edu assay of Control, OE-CTRL, OE-Circ, shCTRL, and shCirc groups in HCC827 and A549 cells. D-E The ratio of apoptosis in the HCC827 and A549 cells transfected with indicated vectors, which consisting of the OE-CTRL, OE- Circ, shCTRL and shCirc groups were detected by flow cytometry. Comparison of the ratio of apoptosis in the afore mentioned 5 groups. Each bar indicates the mean apoptosis rate ± standard deviation per group. F-G Expression level of Bax, Bcl-2 and Cytochrome C in the HCC827 and A549 cells of the 5 groups (CTRL, OE-CTRL, OE- Circ, shCTRL and shCirc groups) were determined by western blotting. Data was normalized to GAPDH. All results were representative of three separate experiments. Data represent mean values ± SD from three replicates of each sample; * P



circSORBS1 inhibits lung cancer development through the RUFY3/YWHAE/BAD/BCL2 pathway. A , B qPCR detection of the transient overexpression efficiency of BCL2 mRNA in H226 versus H1299 cells. C , D . Western blot analysis of YWHAE protein expression after transient silencing and overexpression of RUFY3 mRNA and grey value analysis. E A CCK-8 assay was used to detect the viability of H1299 and H226 cells after transfection with BCL2. F , G Flow cytometry was used to detect the apoptotic capacity after BCL2 backfilling. H circSORBS1 acts as a miR-6779-5p sponge and indirectly inhibits RUFY3 mRNA degradation, directly binds to RUFY3 mRNA and enhances its stability, which in turn increases RUFY3 protein expression, activates the YWHAE/BAD/BCL2 apoptotic signalling pathway, and inhibits lung cancer progression Index in PubMed under a CC BY license. PMID: 38915053

circSORBS1 regulates apoptosis via the RUFY3/YWHAE pathway. A KEGG pathway analysis of genes downstream of RUFY3. B–E Correlation analysis of circSORBS1 with RUFY3 and key proteins of the apoptotic pathways YWHAE, BAD, and BCL2 in the UCSC database. F Results of the Western blot analysis for Co-IP experiments. G IF was used to detect the colocalization of the BCL2 protein with the BAD protein.

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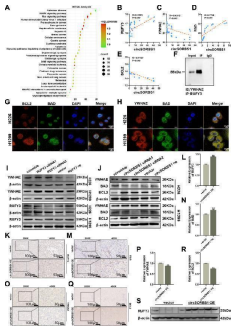
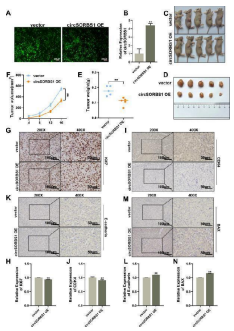
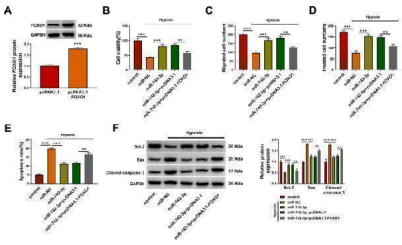


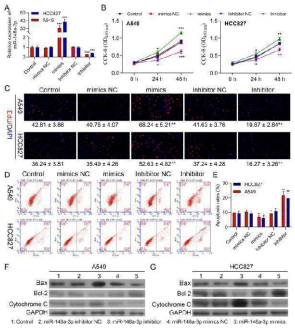
H IF was used to detect the colocalization of YWHAE with the BAD protein. I Western blot analysis of YWHAE and RUFY3 protein expression after transient RUFY3 silencing and overexpression. J Western blot analysis of YWHAE and RUFY3 protein expression after transient circSORBS1 silencing and overexpression. K–R Immunohistochemistry was performed to detect the protein expression levels of RUFY3, YWHAE, BAD, and BCL2 in nude mouse tumours. S Western blot analysis of RUFY3 protein expression in the tumour tissue of nude mice Index in PubMed under a CC BY license. PMID: 38915053



circSORBS1 inhibits lung cancer development in vivo. A Construction of a circSORBS1 stable overexpression cell line by lentiviral transfection of H226 cells. B qPCR detection of circSORBS1 overexpression efficiency in H226 cells stably overexpressing circSORBS1. C , D Nude tumour formation experiments and tumour size determination. E Statistical analysis of tumour weight in nude mice. F Growth of the nude mice. G – N Immunohistochemical analysis of the protein expression of Ki67, CDK4, BAX, and E-cadherin in nude tumours Index in PubMed under a CC BY license. PMID: 38915053



Effects of miR-742-3p and FOXO1 on hypoxia-induced H9c2 cell injury. A H9c2 cells were transfected with pcDNA3.1 and pcDNA3.1- FOXO1 , and the protein expression of FOXO1 was detected by WB analysis. B – F H9c2 cells were transfected with miR-NC (50 nM), miR-742-3p (50 nM), miR-742-3p (50 nM) + pcDNA3.1 (4.0 μg), or miR-742-3p (50 nM) + pcDNA3.1- FOXO1 (4.0 μg), and then treated with hypoxia. Untreated H9c2 cells were used as control. Cell viability, migrated and invaded cell numbers, and cell apoptosis rate were determined by CCK8 assay ( B ), transwell assay ( C , D ), and flow cytometry ( E ). F WB analysis was employed to examine the protein levels of Bcl-2, Bax and Cleaved-caspase 3. All experiments were repeated three times. ** P



MiR-148a-3p promotes proliferation, while inhibits apoptosis in A549 and HCC827 cells. A qPCR assay confirming the transfection efficiency of the miR-148a-3p mimic and miR-148a-3p inhibitor in A549 and HCC827 cells. B Viability in A549 and HCC827 cells transfected with miR-NC, miR-148a-3p mimic, miR-148a-3p inhibitor NC, or miR-148a-3p inhibitor. C Edu assay of cell proliferation in A549 and HCC827 cells transfected with miR-NC, miR-148a-3p mimic, miR-148a-3p inhibitor NC, or miR-148a-3p inhibitor, respectively. D-E Cell apoptosis rates of A549 and HCC827 cells with the indicated transfection were determined with FSC assay. Data are presented as mean ± standard deviation. The experiments were repeated three times. F-G Protein levels of Bax, Bcl-2 and Cytochrome in A549 and HCC827 cells with the indicated transfection were determined by western blot. Data are presented as mean ± standard deviation. The experiments were repeated three times. * P

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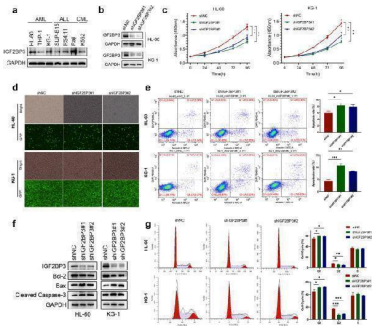
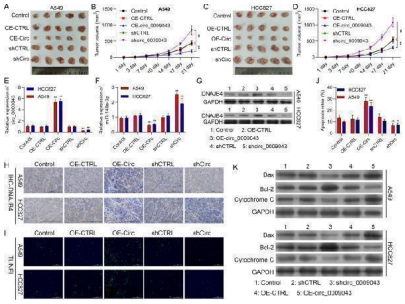


Knockdown of IGF2BP3 significantly inhibits AML progression in vitro. a The protein expression level of IGF2BP3 in various hematologic tumor cell lines was measured by western blotting. b The knockdown efficiency of IGF2BP3 shRNAs (shIGF2BP3#1 and shIGF2BP3#2) delivered via lentiviral vectors in HL-60 and KG-1 cell lines was confirmed by western blotting. GAPDH was used as the internal reference. c Cell proliferation was measured by a CCK-8 assay at different time points (0, 24, 48, 72, and 96 h) in HL-60 and KG-1 cells after shRNA transduction. d The transduction efficiency after puromycin selection was evaluated by GFP fluorescence imaging in both cell lines. e Flow cytometry (representative images are presented) was used to confirm the induction of apoptosis by IGF2BP3 knockdown. f Western blotting was used to explore apoptosis-related protein levels. The levels of cleaved caspase-3 and Bax were increased but the level of Bcl-2 was decreased under shIGF2BP3 treatment compared with control treatment. g Flow cytometry (representative images are presented) was used to analyze the cell cycle distribution. * P



Circ_0009043 inhibits tumor growth via targeting the miR-148a-3p / DNAJB4 pathway in vivo. A, C Tumors formed 6 weeks post-injection in BALB/C nude mice. Tumors in the CTRL, OE-CTRL, OE-Circ, shCTRL and shCirc groups were isolated from mice at the endpoint of experiments. B, D Tumor growth was assessed by tumor volume measurement over time in the 5 afore mentioned groups (mean ± SD; n = 5). ** P

## 128 Publications Citing This Product

1. PubMed ID: 31424657, Meng K,Yuan G,Bao H,Wang L,Ma R,Yu B,Zhao S.Interaction of HCCR-1 and Bax in breast cancer.J BUON.2019 May-Jun;24(3):1027-1037.PMID:31424657.

2. PubMed ID: -, Su-Su Tang,Yi Ren,Xiao-Qian Ren,Jing-Ran Cao,Hao Hong,Hui Ji,Qing-Hua Hu,ERalpha and/or ERbeta activation ameliorates cognitive impairment, neurogenesis and apoptosis in type 2 diabetes mellitus mice,Experimental Neurology,Volume 311,2019,Pages 33-43,ISSN 0014-4886,https://doi.org/10.1016/j.expneurol.2018.09.002.

3. PubMed ID: 31920915, Ren Q,Hu Z,Jiang Y,Tan X,Botchway BOA,Amin N,Lin G,Geng Y,Fang M.SIRT1 Protects Against Apoptosis by Promoting Autophagy in the Oxygen Glucose Deprivation/Reperfusion-Induced Injury.Front Neurol.2019 Dec 5;10:1289.doi:10.3389/fneur.2019.01289.PMID:31920915;PMCID:PMC6915092.

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