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Anti-Aryl hydrocarbon Receptor/AHR Antibody Picoband®

Catalog Number: PA1782-1

About AHR

AHR (aryl hydrocarbon receptor), also called bHLHe76, is a member of the family of basic helix-loop-helix transcription factors. AhR is a cytosolic transcription factor that is normally inactive, bound to several co-chaperones. The AHR gene is mapped on 7p21.1. Estrogenic actions of AHR agonists were detected in wildtype ovariectomized mouse uteri, but were absent in Ahr -/- or Er-alpha -/- ovariectomized mice. Complex assembly and ubiquitin ligase activity of CUL4B (AHR) in vitro and in vivo are dependent on the AHR ligand. In the CUL4B (AHR) complex, ligand-activated AHR acts as a substrate-specific adaptor component that targets sex steroid receptors for degradation. Cd4-positive cells from mice lacking Ahr developed Th17 responses but failed to produce Il22 and did not show enhanced Th17 development. Activation of Ahr during induction of EAE accelerated disease onset and increased pathology in wildtype mice, but not in Ahr -/- mice. The TDO-AHR pathway is active in human brain tumors and is associated with malignant progression and poor survival. Ahr activity within ROR-gamma-t-positive ILC could be induced by dietary ligands such as those contained in vegetables of the family Brassicaceae.

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Overview

Product Name	Anti-Aryl hydrocarbon Receptor/AHR Antibody Picoband®
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Aryl hydrocarbon Receptor/AHR Antibody catalog # PA1782-1. Tested in Flow Cytometry, IF, IHC, 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, IHC, ICC, WB
Clonality	Polyclonal
Formulation	Each vial contains 4mg Trehalose, 0.9mg NaCl and 0.2mg 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	P35869

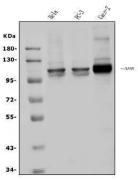
Technical Details

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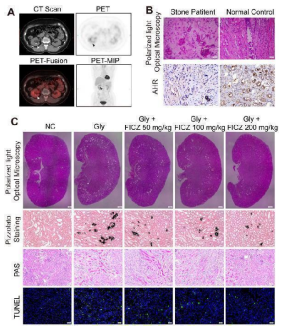
Immunogen	A synthetic peptide corresponding to a sequence at the C-terminus of human AHR.
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot, and HRP Conjugated anti-Rabbit IgG Super Vision Assay Kit (SV0002-1) for IHC(P) and ICC.
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, 0.25-0.5ug/ml, Human Immunohistochemistry (Paraffin-embedded Section), 2-5ug/ml, Human, Mouse, Rat Immunocytochemistry/Immunofluorescence, 5ug/ml, Human Flow Cytometry (Fixed), 1-3ug/1x10^6 cells, Human Note: For flow cytometry (FCM), suitable for fixed cells only. Not suitable for FACS or surface flow cytometry using live cells.

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Anti-Aryl hydrocarbon Receptor/AHR Antibody Picoband® (PA1782-1) Images



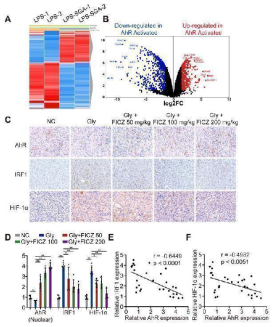
Western blot analysis of AHR using anti-AHR antibody (PA1782-1). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 50ug of sample under reducing conditions. Lane 1: human HELA whole cell lysates, Lane 2: human PC-3 whole cell lysates, Lane 3: human CACO-2 whole cell lysates. After Electrophoresis, proteins were transferred to a Nitrocellulose membrane at 150mA 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-AHR antigen affinity purified polyclonal antibody (Catalog # PA1782-1) 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 Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for AHR at approximately 100-110KD. The expected band size for AHR is at 100-110KD.



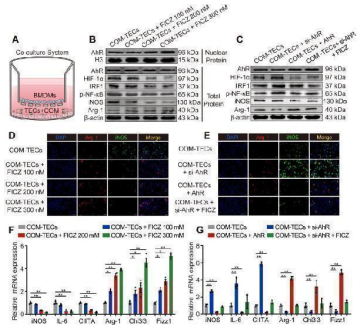
High inflammation status and AhR dysregulation in stone patient kidneys while reducing renal inflammation and injury found in AhR-activated CaOx nephrocalcinosis mice. (A) PET-CT imaging studies assessing renal inflammatory responsiveness. Axial CT, axial PET, axial fused PET-CT and coronal PET maximum intensity projection (MIP) images suggesting enhanced renal uptake of 18 F-FDG. Arrows are used to mark focal 18 F-FDG accumulation in the form of a ring surrounding the stone. (B) crystal deposition in Randall's plaques (n = 10) was analysed via polarized light optical microscopy (100×; scale bar: 20 μm) and IHC staining for AhR in Randall's plaques (200×; scale bar: 20 μm). (C) Deposition of renal CaOx crystal in the corticomedullary junction of mice (n = 6) treated with increasing concentrations of FICZ was analysed via polarized light optical microscopy (20×, scale bar: 500 μm). Crystal deposition within corticomedullary junction regions was further confirmed by Pizzolato staining (200×; scale bar: 20 μm). Kidney injury and necrosis were evaluated by PAS staining (200×; scale bar: 20 μm) and TUNEL staining (200×; scale bar: 50 μm) in kidney tissues, respectively.Index in PubMed under a CC BY license. PMID: 33204326

AhR significantly suppressed IRF1 and HIF-1alpha expression in a murine CaOx nephrocalcinosis model. (A) RNA-seq heatmap showing significantly altered mRNAs in SGA-treated BMDMs. (B) Volcano plots showing mRNA transcripts that were differentially expressed between LPS-treated and SGA-treated BMDMs. Significantly downregulated and upregulated mRNAs are shown in green and red, respectively, whereas genes that were not significantly

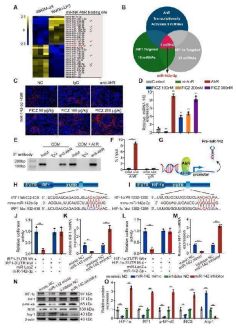
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changed are shown in black. (C) IHC staining for AhR, IRF1, and HIF-1alpha in the kidneys of FICZ-treated mice with CaOx nephrocalcinosis (200×; scale bar: 20 μm). (D) qRT-PCR was used to assess AhR, IRF1, and HIF-1alpha expression in kidney samples from FICZ-treated mice (n = 6) with CaOx nephrocalcinosis compared to kidney samples from model mice. (E, F) Pearson's correlation coefficient analysis (n = 30) of the expression levels of AhR and IRF1 (E) or HIF-1alpha (F). Each dot represents an individual animal. *P < 0.05; **P < 0.01, as assessed via one-way ANOVA (D).Index in PubMed under a CC BY license. PMID: 33204326



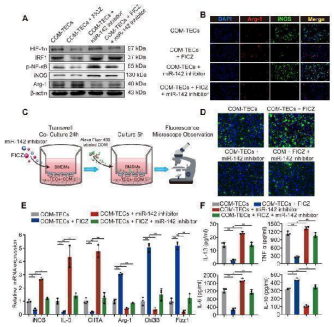
AhR suppressed IRF1 and HIF-1alpha to attenuate CaOx crystal-stimulated M1 macrophage polarization in vitro . (A) BMDMs and COM-treated TECs co-culture model. (B, C) Western blotting analysis was used to detect AhR, HIF-1alpha, IRF1, NF-kappaB p65, iNOS, and Arg-1 expression after FICZ treatment and the upregulation or downregulation of AhR in BMDMs. beta-actin served as a normalization control. (D, E) iNOS (M1 macrophage marker, green) and Arg-1 (M2 macrophage marker, red) distribution in BMDMs were detected by immunofluorescence (200×; scale bar: 20 μm). (F, G) qRT-PCR analysis of iNOS, IL-6, CIITA, Arg-1, Chi3l3 and Fizz1 expression to further determine polarization state of BMDM. The data are shown as the means ± SD of triplicate experiments. *P < 0.05; **P < 0.01, as assessed via one-way ANOVA (F, G).Index in PubMed under a CC BY license. PMID: 33204326



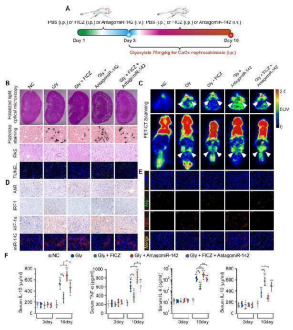
AhR transcriptionally activates miR-142a to inhibit IRF1 and HIF-1alpha expression. (A) The top 30 miRNAs in BMDMs that are regulated by LPS are arranged in a miRNA array heatmap. In addition, miRNAs predicted to be under the transcriptional control of AhR (according to analysis with the JASPAR database) are noted. (B) Venn diagram analyses were performed to identify miRNAs that can both target IRF1 and HIF-1alpha and that are under the transcriptional control of AhR. (C) Renal expression of mmu-miR-142a-3p in mice (n = 6) with CaOx nephrocalcinosis following treatment with an AhR neutralizing antibody or FICZ treatment was assessed via FISH (200×; scale bar: 20 μm). (D) qRT-PCR was performed to measure mmu-miR-142a-3p expression in BMDMs using U6 RNA as a normalization control. (E, F) ChIP assays and ChIP qPCR analysis showed that AhR bound to the miR-142a promoter in BMDMs treated with the AhR overexpression plasmid. (G) A schematic model showed that AhR directly binds to the miR-142a promoter and activates its transcription. (H, I) WT and mutated miR-142a targeting sequences in the IRF1 and HIF-1alpha 3'-UTR regions that were used to construct luciferase reporters, with reporters bearing these IRF1 (J) or HIF-1alpha (L) 3'-UTR sequences co-transfected along with miR-142a mimic (100 nM). IRF1 (K) and HIF-1alpha (M) mRNA levels were detected via qRT-PCR in BMDMs following miR-142a mimic or inhibitor transfection. Western blotting (N, O) analysis enabled the detection of IRF1 and HIF-1alpha expression while also assessing the levels of iNOS and Arg-1 to monitor the polarization state of

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BMDMs following miR-142a mimic or inhibitor transfection. beta-actin was employed as a normalization control. The data are shown as the means ± SD of triplicate experiments. *P < 0.05; **P < 0.01, as assessed via Student's t test (D, F) or one-way ANOVA (J-M, O).Index in PubMed under a CC BY license. PMID: 33204326



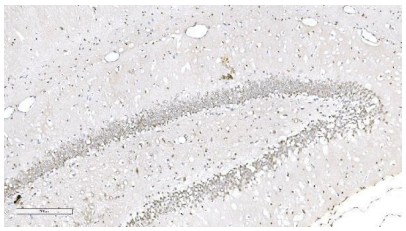
AhR activation in vitro decrease M1 macrophage polarization to inhibit kidney inflammation and injury through the AhR-miR-142a-IRF1/HIF-1alpha axis in vitro . (A) Western blotting analysis enabled the detection of AhR, HIF-1alpha, IRF1, NF-kappaB p65, iNOS, and Arg-1 expression in BMDMs. beta-actin was detected as an internal control. (B) iNOS (M1 macrophage marker, green) and Arg-1 (M2 macrophage marker, red) distributions in BMDMs were detected by immunofluorescence (200×; scale bar: 20 μm). (C) Schematic diagram of BMDMs phagocytic capacity testing. (D) Fluorescence microscopy was performed to analyse the phagocytic ability of BMDMs (200×; scale bar: 20 μm). (E) qRT-PCR analysis of iNOS, IL-6, CIITA, Arg-1, Chi3l3 and Fizz1 expression to further determine polarization state of BMDM. (F) ELISA was used to quantify cytokine levels in the co-culture media. The data are shown as the means ± SD of triplicate experiments. *P < 0.05; **P < 0.01, as assessed via one-way ANOVA (E, F).Index in PubMed under a CC BY license. PMID: 33204326



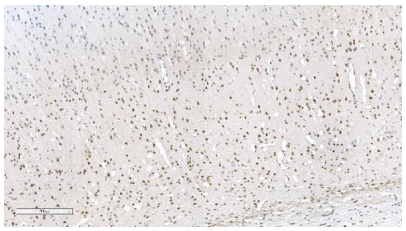
AhR activation suppressed the deposition of CaOx crystal and CaOx nephrocalcinosis-mediated kidney inflammation and injury through the AhR-miR-142a-IRF1/HIF-1alpha axis in vivo . (A) Experimental overview. (B) The deposition of renal CaOx crystal in FICZ- and/or antagomiR-142a-treated mice was assessed via polarized light optical microscopy (20×; scale bar: 500 μm). Pizzolato staining was employed as a means of detecting these CaOx crystal in corticomedullary tissue, while PAS was utilized to evaluate injury to TECs (200×; scale bar: 20 μm), and TUNEL staining was employed to assess renal TECs death (200×; scale bar: 50 μm). (C) PET-CT scanning was employed as a means of assessing renal inflammation state in CaOx nephrocalcinosis mice. (D) IHC was used to analyse AhR, IRF1, and HIF-1alpha expression, and FISH was used to detect miR-142a expression in renal tissue (200×; scale bar: 20 μm). (E) iNOS (M1 macrophage marker, red) and Arg-1 (M2 macrophage marker, green) distributions in renal tissues were detected by immunofluorescence (200×; scale bar: 50 μm). (F) On days 3 and 10, the serum pro-inflammatory IL-1beta, TNF-alpha, and IL-6 levels and the anti-inflammatory IL-10 levels were measured by ELISA. n = 6 per group. *P < 0.05; **P < 0.01, as assessed via one-way ANOVA (F).Index in PubMed under a CC BY license. PMID: 33204326

IHC analysis of AHR using anti-AHR antibody (PA1782-1). AHR was detected in paraffin-embedded section of rat brain tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue

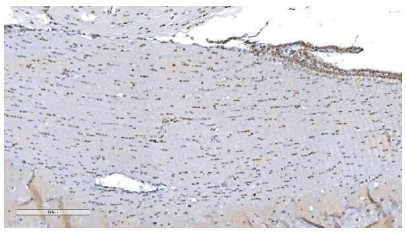
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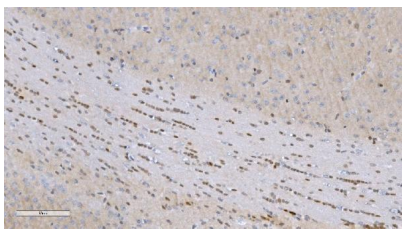
section was then incubated with 2ug/ml rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. Biotinylated goat anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using Strepavidin-Biotin-Complex (SABC) (Catalog # SA1022) with DAB as the chromogen.



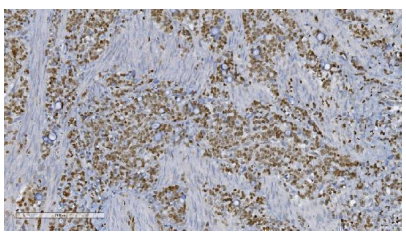
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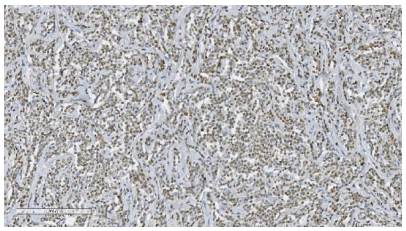


IHC analysis of AHR using anti-AHR antibody (PA1782-1). AHR was detected in paraffin-embedded section of mouse brain tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2ug/ml rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. Biotinylated goat anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using Strepavidin-Biotin-Complex (SABC) (Catalog # SA1022) with DAB as the chromogen.

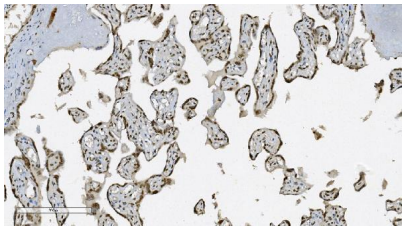


IHC analysis of AHR using anti-AHR antibody (PA1782-1). AHR was detected in paraffin-embedded section of human gastric cancer tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2ug/ml rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. Biotinylated goat anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using Strepavidin-Biotin-Complex (SABC) (Catalog # SA1022) with DAB as the chromogen.

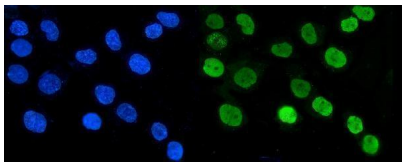
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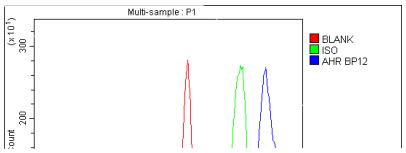
IHC analysis of AHR using anti-AHR antibody (PA1782-1). AHR was detected in paraffin-embedded section of human lymphadenoma tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2ug/ml rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. Biotinylated goat anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using Strepavidin-Biotin-Complex (SABC) (Catalog # SA1022) with DAB as the chromogen.



IHC analysis of AHR using anti-AHR antibody (PA1782-1). AHR was detected in paraffin-embedded section of human placenta tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2ug/ml rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. Biotinylated goat anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using Strepavidin-Biotin-Complex (SABC) (Catalog # SA1022) with DAB as the chromogen.



IF analysis of AHR using anti-AHR antibody PA1782-1). AHR was detected in immunocytochemical section of A431 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 5ug/mL rabbit anti-AHR Antibody (PA1782-1) overnight at 4°C. DyLight®488 Conjugated Goat Anti-Rabbit IgG (BA1127) 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 A431 cells using anti-AHR antibody (PA1782-1). Overlay histogram showing A431 cells stained with PA1782-1 (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-AHR Antibody (PA1782-1, 1ug/1x106 cells) for 30 min at 20°C. DyLight®488 conjugated goat anti-rabbit IgG (BA1127, 5-10ug/1x106 cells) was used as secondary antibody for 30 minutes at 20°C. Isotype control antibody (Green line) was rabbit IgG (1ug/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-Aryl hydrocarbon Receptor/AHR Antibody

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