

AHR
Purified Mouse Monoclonal Antibody
Catalog # AO2632a**Specification**

AHR - Product Information

Application	WB, IHC, ICC, E
Primary Accession	P35869
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	Mouse IgG1
Calculated MW	96.1kDa KDa
Immunogen	

Purified recombinant fragment of human AHR (AA: 721-820) expressed in E. Coli.

Formulation

Purified antibody in PBS with 0.05% sodium azide

AHR - Additional Information

Gene ID 196

Other Names

bHLHe76

Dilution

WB~~ 1/500 - 1/2000

IHC~~1:100~500

ICC~~N/A

E~~ 1/10000

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

AHR is for research use only and not for use in diagnostic or therapeutic procedures.

AHR - Protein Information

Name AHR {ECO:0000303|PubMed:8393992, ECO:0000312|HGNC:HGNC:348}

Function

Ligand-activated transcription factor that enables cells to adapt to changing conditions by sensing compounds from the environment, diet, microbiome and cellular metabolism, and which plays important roles in development, immunity and cancer (PubMed:<a

[>23275542, PubMed:30373764, PubMed:32818467, PubMed:7961644\). Upon ligand binding, translocates into the nucleus, where it heterodimerizes with ARNT and induces transcription by binding to xenobiotic response elements \(XRE\) \(PubMed:23275542, PubMed:30373764, PubMed:7961644\). Regulates a variety of biological processes, including angiogenesis, hematopoiesis, drug and lipid metabolism, cell motility and immune modulation \(PubMed:12213388\). Xenobiotics can act as ligands: upon xenobiotic- binding, activates the expression of multiple phase I and II xenobiotic chemical metabolizing enzyme genes \(such as the CYP1A1 gene\) \(PubMed:7961644, PubMed:33193710\). Mediates biochemical and toxic effects of halogenated aromatic hydrocarbons \(PubMed:34521881, PubMed:7961644\). Next to xenobiotics, natural ligands derived from plants, microbiota, and endogenous metabolism are potent AHR agonists \(PubMed:18076143\). Tryptophan \(Trp\) derivatives constitute an important class of endogenous AHR ligands \(PubMed:32818467, PubMed:32866000\). Acts as a negative regulator of anti-tumor immunity: indoles and kynurenic acid generated by Trp catabolism act as ligand and activate AHR, thereby promoting AHR-driven cancer cell motility and suppressing adaptive immunity \(PubMed:32818467\). Regulates the circadian clock by inhibiting the basal and circadian expression of the core circadian component PER1 \(PubMed:28602820\). Inhibits PER1 by repressing the CLOCK-BMAL1 heterodimer mediated transcriptional activation of PER1 \(PubMed:28602820\). The heterodimer ARNT:AHR binds to core DNA sequence 5'-TGCGTG-3' within the dioxin response element \(DRE\) of target gene promoters and activates their transcription \(PubMed:28602820\).](http://www.uniprot.org/citations/23275542)

Cellular Location

Cytoplasm. Nucleus. Note=Initially cytoplasmic; upon binding with ligand and interaction with a HSP90, it translocates to the nucleus.

Tissue Location

Expressed in all tissues tested including blood, brain, heart, kidney, liver, lung, pancreas and skeletal muscle Expressed in retinal photoreceptors (PubMed:29726989)

AHR - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)

- [Flow Cytometry](#)
- [Cell Culture](#)

AHR - Images

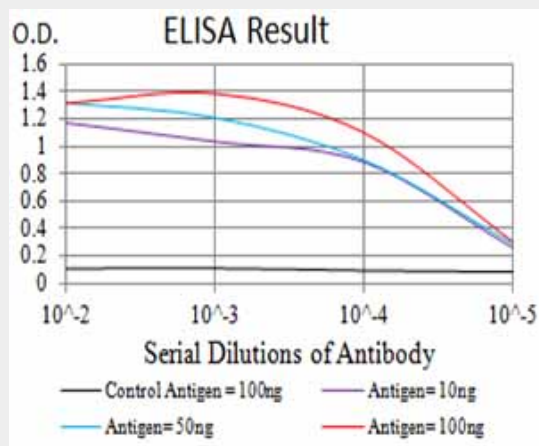


Figure 1: Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line: Antigen (100 ng)

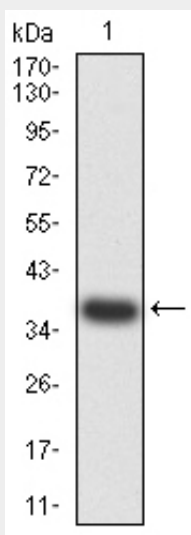


Figure 2: Western blot analysis using AHR mAb against human AHR (AA: 721-820) recombinant protein. (Expected MW is 37 kDa)

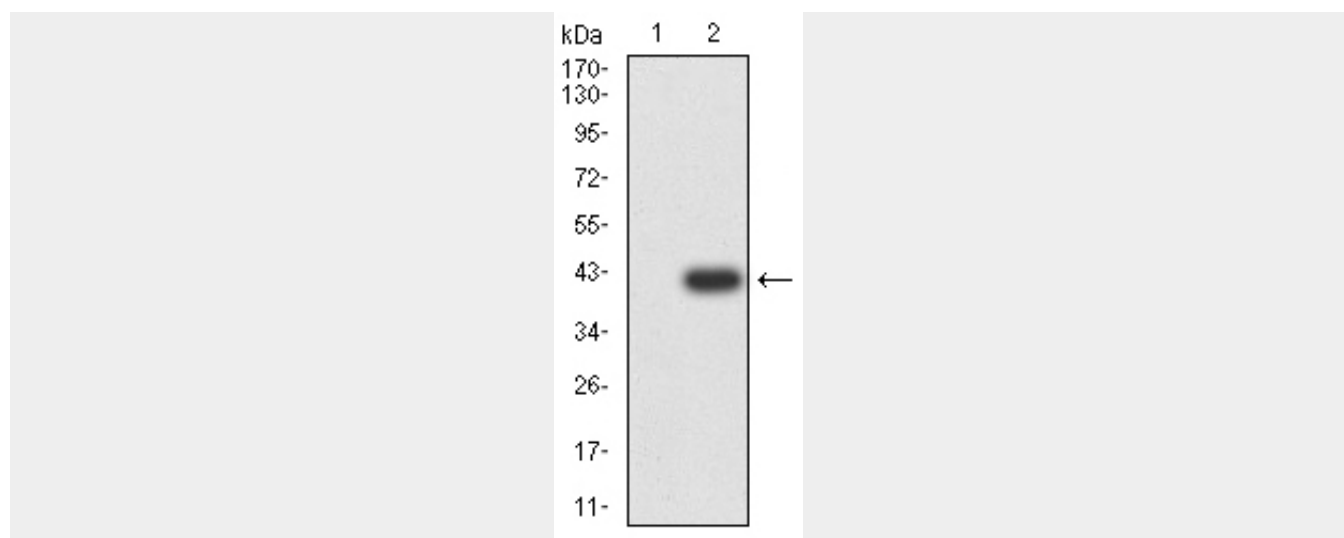


Figure 3: Western blot analysis using AHR mAb against HEK293 (1) and AHR (AA: 721-820)-hIgGFc transfected HEK293 (2) cell lysate.

AHR - References

1. Pharmacol Rev. 2015;67(2):259-79. 2. Int J Cancer. 2015 Jul 15;137(2):299-310.