SANTA CRUZ BIOTECHNOLOGY, INC.

Ah Receptor (L-15): sc-101104

BACKGROUND

2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is the prototype for a family of toxic halogenated aromatic compounds that are thought to cause adverse reproductive, immunologic and metabolic effects. Many biological responses to TCDD are mediated through ligand binding to the aromatic hydrocarbon (Ah) receptor, also known as AhR. Ah Receptor is a ligand dependent transcription factor that interacts with specific DNA sequences, termed xenobiotic responsive elements (XREs), and that lies upstream of TCDD-inducible genes. Upon binding to the ligand, Ah Receptor binds to the Ah Receptor nuclear translocator (Arnt), and the complex is translocated from the cytoplasm to the nucleus. Arnt is required for Ah Receptor to bind to XRE. Ah Receptor and Arnt are members of a family of transcription factors that contain a basic helix-loop-helix motif and a common "PAS" motif.

CHROMOSOMAL LOCATION

Genetic locus: AHR (human) mapping to 7p21.1; Ahr (mouse) mapping to 12 A3.

SOURCE

Ah Receptor (L-15) is a mouse monoclonal antibody raised against recombinant Ah Receptor of human origin.

PRODUCT

Each vial contains 100 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

Ah Receptor (L-15) is recommended for detection of Ah Receptor of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Ah Receptor siRNA (h): sc-29654, Ah Receptor siRNA (m): sc-29655, Ah Receptor shRNA Plasmid (h): sc-29654-SH, Ah Receptor shRNA Plasmid (m): sc-29655-SH, Ah Receptor shRNA (h) Lentiviral Particles: sc-29654-V and Ah Receptor shRNA (m) Lentiviral Particles: sc-29655-V.

Molecular Weight (predicted) of Ah Receptor: 96 kDa.

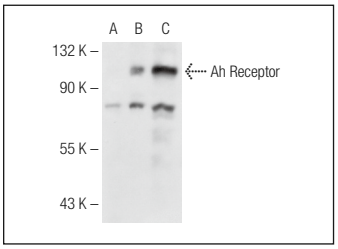
Molecular Weight (observed) of Ah Receptor: 122 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, PC-3 cell lysate: sc-2220 or Ah Receptor (m): 293 Lysate: sc-178266.

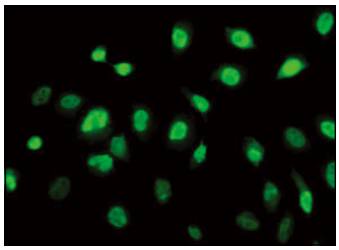
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



Ah Receptor (L-15): sc-101104. Western blot analysis of Ah Receptor expression in non-transfected 293: sc-110760 (**A**), mouse Ah Receptor transfected 293: sc-178266 (**B**) and PC-3 (**C**) whole cell lysates.



Ah Receptor (L-15): sc-101104. Immunofluorescence staining of paraformaldehyde-fixed HeLa cells showing nuclear localization.

SELECT PRODUCT CITATIONS

1. Sun, F., et al. 2015. A novel prostate cancer therapeutic strategy using icaritin-activated arylhydrocarbon-receptor to co-target androgen receptor and its splice variants. Carcinogenesis 36: 757-768.
2. Borghi, M., et al. 2019. Targeting the aryl hydrocarbon receptor with indole-3-aldehyde protects from vulvovaginal candidiasis via the IL-22-IL-18 cross-talk. Front. Immunol. 10: 2364.
3. Ravell, J.C., et al. 2020. Defective glycosylation and multisystem abnormalities characterize the primary immunodeficiency XMEN disease. J. Clin. Invest. 130: 507-522.
4. Gong, K., et al. 2020. EGFR inhibition triggers an adaptive response by co-opting antiviral signaling pathways in lung cancer. Nat. Cancer 1: 394-409.
5. van de Veerdonk, F.L., et al. 2022. Anakinra restores cellular proteostasis by coupling mitochondrial redox balance to autophagy. J. Clin. Invest. 132: e144983.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.