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XEDAR Antibody

Catalog # ASC10433

Product Information

Application	WB, E, IHC-P
Primary Accession	Q9HAV5
Other Accession	AAQ89953, 37222208
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	32759
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	XEDAR antibody can be used for the detection of XEDAR by Western blot at 0.5 - 2 g/mL. Antibody can also be used for immunohistochemistry starting at 10 g/mL.

Additional Information

Gene ID	60401
Other Names	XEDAR Antibody: XEDAR, EDAA2R, EDA-A2R, TNFRSF27, XEDAR, UNQ2448/PRO5727/PRO34080, Tumor necrosis factor receptor superfamily member 27, X-linked ectodysplasin-A2 receptor, EDA-A2 receptor, ectodysplasin A2 receptor
Target/Specificity	EDA2R;
Reconstitution & Storage	XEDAR antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	XEDAR Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	EDA2R
Synonyms	TNFRSF27, XEDAR
Function	Receptor for EDA isoform A2, but not for EDA isoform A1. Mediates the activation of the NF-kappa-B and JNK pathways. Activation seems to be mediated by binding to TRAF3 and TRAF6.
Cellular Location	Membrane; Single-pass type III membrane protein.

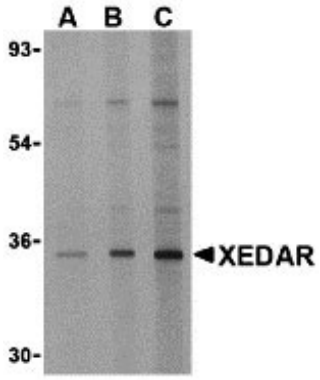
Background

XEDAR Antibody: X-linked ectodysplasin-A2 receptor (XEDAR) is a recently isolated member of the tumor necrosis factor receptor family that is highly expressed during embryonic development and binds to ectodysplatin-A2 (EDA-A2). Two predominantly expressed isoforms, XEDAR-s and XEDAR-L, differ by only a 21-amino region at the juxtamembrane region of the cytoplasmic domain. Neither isoform possesses a death domain and both have been shown to act mainly through TRAF3 and TRAF6 to activate the NF-κB and JNK pathways. Cells transfected with XEDAR and treated with EDA-A2 cause the assembly of a secondary complex containing FADD, caspase-8 and caspase-10, leading to the activation caspase-8 and caspase-3, and finally apoptosis. The EDA-A2-induced apoptosis is dependent on caspase-9 activation, as various pharmacological and genetic inhibitors of caspase-8 blocked apoptosis following EDA-A2 treatment.

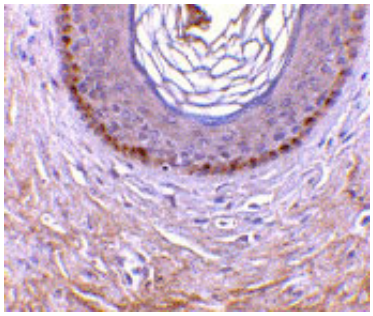
References

Yan M, Wang LC, Hymowitz SG, et al. Two-amino acid molecular switch in an epithelial morphogen that regulates binding to two distinct receptors. Science 2000; 290:523-7.
Sinha SK, Zachariah S, Quinones HI, et al. Role of TRAF3 and -6 in the activation of the NF-κB and JNK pathways by X-linked ectodermal dysplasia receptor. J. Biol. Chem. 2002; 277:44953-61.
Sinha SK and Chaudhary PM. Induction of apoptosis by X-linked ectodermal dysplasia receptor via a caspase 8-dependent mechanism. J. Biol. Chem. 2004; 41873-81.

Images



Western blot analysis of XEDAR in 293 cell lysate with XEDAR antibody at (A) 0.5, (B) 1 and (C) 2 µg/mL.



Immunohistochemistry of XEDAR in human skin tissue with XEDAR antibody at 10 µg/mL.