

# XEDAR Antibody

Catalog No: #24427

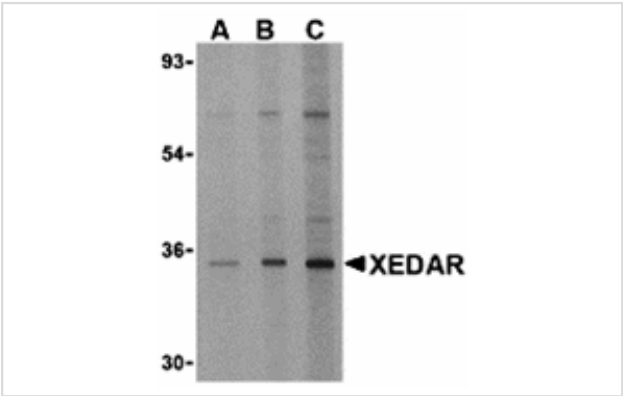


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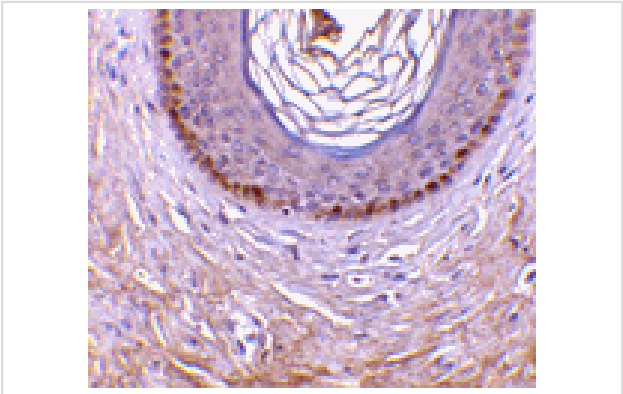
## Description

|                       |   |
|-----------------------|---|
| Product Name          | XEDAR Antibody  |
| Host Species          | Rabbit  |
| Clonality             | Polyclonal  |
| Purification          | Affinity chromatography purified via peptide column   |
| Applications          | ELISA WB IHC  |
| Species Reactivity    | Hu  |
| Immunogen Type        | Recombinant protein   |
| Immunogen Description | Raised against recombinant human XEDAR.   |
| Target Name           | XEDAR   |
| Other Names           | X-linked ectodysplasin-A2 receptor, EDA-A2 receptor, TNFRSF27   |
| Accession No.         | Swiss-Prot:Q9HAV5Gene ID:60401  |
| Uniprot               | Q9HAV5  |
| GeneID                | 60401;  |
| Concentration         | 1mg/ml  |
| Formulation           | Supplied in PBS containing 0.02% sodium azide.  |
| Storage               | Can be stored at -20°C, stable for 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. |

## Images



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



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

## Background

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- $\kappa$ B 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.

Note: This product is for in vitro research use only