10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

EDAR Rabbit pAb

EDAR Rabbit pAb
Catalog # AP55050

Product Information

Application	IHC-P, IHC-F, IF
Primary Accession	Q9UNE0
Predicted	Rat, Dog, Human, Mouse, Rabbit
Host	Rabbit
Clonality	Polyclonal
Calculated MW	48582
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human EDAR
Epitope Specificity	141-230/448
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Membrane; Single-pass type I membrane protein (Probable).
DISEASE	Defects in EDAR are a cause of ectodermal dysplasia anhidrotic (EDA) [MIM:224900]; also known ectodermal dysplasia hypohidrotic autosomal recessive (HED). Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. EDA is characterized by sparse hair (atrichosis or hypotrichosis), abnormal or missing teeth and the inability to sweat due to the absence of sweat glands. Defects in EDAR are the cause of ectodermal dysplasia type 3 (ED3) [MIM:129490]; also known as ectodermal dysplasia hypohidrotic autosomal dominant or EDA3. ED3 is an autosomal dominant condition characterized by hypotrichosis, abnormal or missing teeth, and hypohidrosis due to the absence of sweat glands.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	The tumor necrosis factor receptor (TNFR) superfamily represents a growing family of type I transmembrane glycoproteins that are involved in various cellular functions, including proliferation, differentiation and programmed cell death. These proteins share homology for cysteine-rich repeats in the extracellular ligand binding domain and an intracellular death domain. Members of the TNFR superfamily transmit signals through protein-protein interactions, and these signals can lead to the activation of either the caspase and Jun kinase pathways, which promote cell death, or the NFκB pathway, which results in cell survival. The ectodermal dysplasia receptor (EDAR) promotes all three of these pathways and mediates ectodermal differentiation. EDAR is encoded by the downless gene and is mutated in ectodermal dysplasia syndromes, which are characterized by impaired hair, teeth and sweat gland development. Ectodysplasin A (EDA) is a type II membrane protein that is encoded by the Tabby gene and produces many splice variants, the longest of which, EDA-A1, serves as the ligand for EDAR. EDA-A2, which differs from EDA-A1 by the deletion of two amino acids, binds only the X-linked ectodysplasin-A2 receptor (XEDAR). Both EDAR and XEDAR

exhibit homology with TROY.

Additional Information

Gene ID	10913
Other Names	Tumor necrosis factor receptor superfamily member EDAR, Anhidrotic ectodysplasin receptor 1, Downless homolog, EDA-A1 receptor, Ectodermal dysplasia receptor, Ectodysplasin-A receptor, EDAR, DL
Dilution	IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,ELISA=1:5000-10000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	EDAR
Synonyms	DL
Function	Receptor for EDA isoform A1, but not for EDA isoform A2. Mediates the activation of NF-kappa-B and JNK. May promote caspase- independent cell death.
Cellular Location	Membrane; Single-pass type I membrane protein
Tissue Location	Detected in fetal kidney, lung, skin and cultured neonatal epidermal keratinocytes. Not detected in lymphoblast and fibroblast cell lines

Background

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.