

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse EDAR in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse EDAR Glu27-Ala187 Accession # Q9R187
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Blockade of Receptor-ligand Interaction	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

EDAR is a type I transmembrane protein which is a member of the TNF Receptor Superfamily (TNFRSF). The extracellular domain contains 14 cysteine residues, six of which approximate the TNFRSF cysteine-rich region, the cytoplasmic domain contains a region with homology to the death domains found in other TNFRSF members. Mouse EDAR is a 488 amino acid (aa) protein with a predicted 30 aa signal, a 159 aa extracellular domain, a 22 aa transmembrane domain, and a 277 aa cytoplasmic domain. The human and mouse EDAR homologs share 91% identity. Within the TNFRSF, EDAR shares the highest homologies with XEDAR and TNFRSF19/TROY. EDA-A1 is the EDAR ligand. EDA and EDAR have been associated with hypohidrotic ectodermal dysplasia (HED). HED is characterized by abnormalities in hair, teeth and eccrine sweat gland morphogenesis. HED was initially found to associate with two gene loci, tabby and downless. Tabby was later identified as the gene for EDA and downless as the autosomal EDAR gene. EDA has two splice variants, EDA-A1 and EDA-A2 which differ by only two amino acids. Despite this minor difference, the EDA isoforms display strong receptor specificity. EDA-A1 only binds to EDAR, whereas EDA-A2 binds to XEDAR, an X-linked TNFRSF member with high homology to EDAR. Mutations in EDA, EDAR and XEDAR have been associated with HED.

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