

## Anti-EDAR [EDAR3] Standard Size Ab00784-10.3

This antibody was created using our proprietary Fc Silent™ engineered Fc domain containing key point mutations that abrogate binding to Fc gamma receptors.

This chimeric human antibody was made using the variable domain sequences of the original Mouse IgG1 format, for improved compatibility with existing reagents, assays and techniques.

**Isotype and Format:** Human IgG1, Fc Silent™, Kappa

**Clone Number:** EDAR3

**Alternative Name(s) of Target:** ectodysplasin A receptor; Tumor necrosis factor receptor superfamily member EDAR; Anhidrotic ectodysplasin receptor 1; Downless; Ectodermal dysplasia receptor; Ectodysplasin-A receptor

**UniProt Accession Number of Target Protein:** Q9UNE0

**Published Application(s):** agonist, SPR, WB, ELISA

**Published Species Reactivity:** Chicken, Dog, Rat, Human, Mouse

**Immunogen:** EDAR3 was prepared by immunizing female OVE1B mice (with the Edar gene deleted) subcutaneously with human EDAR-Fc and positive hybridoma clones were screened for binding hEDAR by ELISA.

**Specificity:** EDAR3 recognises and binds to CRD1+CRD2 of the extracellular domain of human EDAR. EDAR3 cross-reacts with EDAR derived from mouse, dog, rat and chicken when EDAR is fused to the glycosylphosphatidylinositol anchor of TRAILR3. EDAR is the receptor for the TNF family ligand EDA1, which is a type II transmembrane protein possessing a collagen-like domain and a C-terminal TNF-homology domain. EDAR is important for the correct development of skin appendages including hair, teeth and eccrine sweat glands. LoF mutations in the Eda gene is known to cause XLHED (X-linked hypohidrotic ectodermal dysplasia), and results in abnormal development.

**Application Notes:** EDAR3 can be used in surrogate reporter assay in which Fas-sensitive cells were transfected with a construct formed by fusing the ectodomain of mouse or human EDAR to the intracellular domain of Fas. Binding of EDAR3 resulted in the induction of apoptosis in these cells, confirming the agonistic activity of the antibody. (EC50 (dose required to kill half of EDAR:Fas-expressing Jurkat cells) hEDAR:Fas ~ 100 ng/ml; EC50 mEDAR:Fas ~ 30 ng/ml - both were less active than DEAI-Fc). The agonistic activity was shown to be cross-reactive with EDA-deficient dogs, in which dentition was corrected when the antibody was administered intravenously at 2 and 14 days old. Lacrimation was also improved in the 2 day old dogs. Tail-hair formation was also rescued after administration of EDAR3 to newborn EDA-deficient

Tabby mice, with 0.18 mg/kg required for half-maximal tail-hair reversion. EDAR3 can also be used in ELISA and as a Fab fragment (generated by ficin digestion) in SPR experiments. EDAR is recognized by EDAR3 in WB under non-reducing conditions.

**Antibody First Published in:** Kowalczyk et al. Molecular and therapeutic characterization of anti-ectodysplasin A receptor (EDAR) agonist monoclonal antibodies. J Biol Chem. 2011 Sep 2;286(35):30769-79. [PMID:](#)

**Note on publication:** Describes the generation and characterization of anti-EDAR antibodies that mimic the action of EDA1 in stimulating EDAR both in vitro and in vivo.

## Product Form

**Size:** 200 µg Purified antibody.

**Purification:** Protein A affinity purified

**Supplied In:** PBS with 0.02% Proclin 300.

**Storage Recommendation:** Store at 4°C for up to 3 months. For longer storage, aliquot and store at -20°C.

**Concentration:** 1 mg/ml.

Important note – This product is for research use only. It is not intended for use in therapeutic or diagnostic procedures for humans or animals.