Cynomolgus EDAR Protein (Fc Tag)

Catalog Number: **90111-C02H**

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Biological Solution Specialist

General Information

Gene Name Synonym:

DL; ECTD10A; ECTD10B; ED1R; ED3; ED5; EDA-A1R; EDA1R; EDA3; HRM1; dl; ED1R; ED3; ED5; EDA-A1R; EDA3

Protein Construction:

A DNA sequence encoding the cynomolgus EDAR (Met1-Ala187) was expressed, fused with the Fc region of human IgG1 at the C-terminus.

Source: Cynomolgus

Expression Host: Human Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Glu 27

Molecular Mass:

The recombinant cynomolgus EDAR is a disulfide-linked homodimer. The reduced monomer comprises 404 amino acids and has a calculated molecular mass of 44.6 KDa.The apparent molecular mass of the protein is approximately 60-64 KDa respectively in SDS-PAGE.

Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

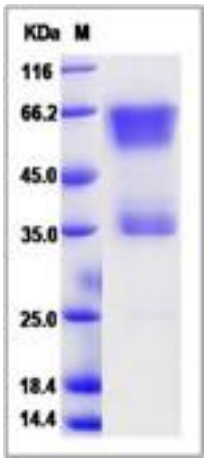
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Tumor necrosis factor receptor superfamily member EDAR is a Single-pass type I membrane protein. Edar was expressed reiteratively in signaling centers regulating key steps in morphogenesis. activin signaling from mesenchyme induces the expression of the TNF receptor edar in the epithelial signaling centers, thus making them responsive to Wnt-induced ectodysplasin from the nearby ectoderm. This is the first demonstration of integration of the Wnt, activin, and TNF signaling pathways. Defects in EDAR are a cause of ectodermal dysplasia anhidrotic (EDA), 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.

References

1.Elomaa O, *et al.* (2001) Ectodysplasin is released by proteolytic shedding and binds to the EDAR protein. Hum Mol Genet. 10 (9): 953-62.
2.Koppinen P, *et al.* (2001) Signaling and subcellular localization of the TNF receptor Edar. Exp Cell Res. 269 (2): 180-92.
3.Chassaing N, *et al.* (2006) Mutations in EDAR account for one-quarter of non-ED1-related hypohidrotic ectodermal dysplasia. Hu. Mutat. 27 (3): 255-9.