Recombinant Human EDA-A1/Ectodysplasin A1

Catalog Number: 3944-ED

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Ser160-Ser391 & Lys178-Ser391 Accession # Q92838
N-terminal Sequence Analysis	Ser160 & Lys178
Structure / Form	Homotrimer
Predicted Molecular Mass	24.1 kDa & 22.2 kDa (monomers)

SPECIFICATIONS

SDS-PAGE	30-40 kDa, reducing conditions
Activity	Measured by its ability to compete with biotinylated human EDA-A1 for binding to immobilized rhEDA R/Fc Chimera.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 10 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Ectodysplasin is a 45 kDa type II transmembrane TNF superfamily protein that is associated with X-linked hypohidrotic ectodermal dysplasia (HED), a disorder of hair, tooth, and sweat gland development (1 - 4). The human EDA-A1 cDNA encodes a 41 amino acid (aa) cytoplasmic region, a 21 aa transmembrane segment, and a 329 aa extracellular region that contains a terminal TNF homology domain, a collagenous domain, and a stalk region (3, 5, 6). Within the collagenous and TNF homology domains, human EDA-A1 shares greater than 97% aa sequence identity with bovine, canine, mouse, and rat EDA-A1. Multiple alternately spliced EDA variants have been described (4, 7). The dominant variant, EDA-A2, has a deletion of two amino acids that changes the receptor binding selectivity from EDAR to XEDAR (4, 7, 8). The collagenous domain of EDA-A1 mediates non-covalent homotrimer formation (5, 6). Shedding of the collagenous and TNF homology domains of EDA-A1 is accomplished by a furin-like protease. The released fragment maintains its trimeric state and ability to bind EDAR (9, 10). Some EDA-A1 polymorphisms found in HED patients alter the protease recognition site and prevent shedding (9). EDA-A1 is expressed in developing hair follicles, epidermis, teeth, sweat glands, salivary glands, and forebrain (6, 8, 11 - 13). It regulates ectodermal appendage formation and is critical to the patterning and morphogenesis of hair follicles, partially through the induction of Lymphotoxin beta (5, 12, 14). Receptor and ligand expression are regulated by factors involved in many aspects of tissue morphogenesis. EDA-A1 expression is induced by Wnt6 (12, 13), while the expression of EDAR is induced by Activin βA and inhibited by BMP-2, -4, and -7 (13, 15).

References:

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