

Recombinant Human CD40 Protein(Fc Tag)

Catalog Number: PDMH100257

Note: Centrifuge before opening to ensure complete recovery of vial contents.

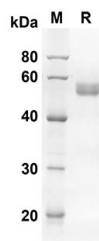
Description

Species	Human
Source	Mammalian-derived Human CD40 protein Glu21-Arg193, with an C-terminal Fc
Calculated MW	43.9 kDa
Observed MW	55 kDa
Accession	P25942
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human CD40 proteins, 2 µg/lane of Recombinant Human CD40 proteins was resolved with an SDS-PAGE under reducing conditions, showing bands at 43.9 KD

Background

For Research Use Only

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Rev. V1.7

This gene is a member of the TNF-receptor superfamily. The encoded protein is a receptor on antigen-presenting cells of the immune system and is essential for mediating a broad variety of immune and inflammatory responses including T cell I-dependent immunoglobulin class switching, memory B cell development, and germinal center formation. AT-hook transcription factor AKNA is reported to coordinately regulate the expression of this receptor and its ligand, which may be important for homotypic cell interactions. Adaptor protein TNFR2 interacts with an this receptor and serves as a mediator of the signal transduction. The interaction of this receptor and its ligand is found to be necessary for amyloid-beta-induced microglial activation, and thus is thought to be an early event in Alzheimer disease pathogenesis. Mutations affecting this gene are the cause of autosomal recessive hyper-IgM immunodeficiency type 3 (HIGM3). Multiple alternatively spliced transcript variants of this gene encoding distinct isoforms have been reported.