Recombinant Human CD40 Protein(His Tag)

Catalog Number: PDMH100223

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

°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.	Description		
Calculated MW18.9 kDaObserved MW25-35 kDaAccessionP25942Bio-activityNot validated for activityPropertiesPurity> 90% as determined by reducing SDS-PAGE.Endotoxin< 1.0 EU/mg of the protein as determined by the LAL method	Species	Human	
Observed MW25-35 kDaAccessionP25942Bio-activityNot validated for activityPropertiesPurity> 90% as determined by reducing SDS-PAGE.Endotoxin< 1.0 EU/mg of the protein as determined by the LAL method	Source	Mammalian-derived Human CD40 proteins Glu21-Arg193, C-terminal His	
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		Mannitol.	
Reconstitution It is recommended that sterile water be added to the vial to prepare a stock solution of	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.		0.5 mg/mL. Concentration is measured by UV-Vis.	

Data

	М	R	
kDa	11		
80	-		
60	-		
40	-		
30	-	-	
20			
12			

SDS-PAGE analysis of Human CD40 proteins, 2 µg/lane of Recombinant Human CD40 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 18.9 KD

Background

The protein encoded by this gene is a member of the TNF-receptor superfamily. This receptor has been found to be essential in mediating a broad variety of immune and inflammatory responses including T cell-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 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.

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