Recombinant Mouse TIM-3/HAVCR2 Protein (aa 20-191, His Tag)

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

Catalog Number: PKSM041276



Description Species Mouse Mol Mass 20.2 kDa Accession O8VIM0 Not validated for activity **Bio-activity Properties** > 95 % as determined by reducing SDS-PAGE. Purity < 1.0 EU per µg of the protein as determined by the LAL method. Endotoxin Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 Storage °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at $< -20^{\circ}$ C for 3 months. This product is provided as lyophilized powder which is shipped with ice packs. Shipping Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Formulation Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual. Reconstitution Please refer to the printed manual for detailed information.

kDa	MK	R
120 90 60		
40		
30		
20		
14		

Background

Data

T cell immunoglobulin and mucin domain-3 (TIM3), also called hepatitis A virus cellular receptor 2 (HAVCR2), is a transmembrane glycoprotein of the TIM family of immune regulating molecules and plays an important role in the Th1-mediated immune response. TIM3 is expressed on the Th1 cells, CD8 T-cells, monocytes, and dendritic cells, but not on Th2 cells. TIM3 expressed by monocytes and dendritic cells facilitates phagocytosis of apoptotic cells and up-regulates cross-presentation of apoptotic cell-associated antigens through interaction with phosphatidylserine. Engagement of TIM3 by its ligand galectin-9 induces a range of immunosuppressive functions which enhance immune tolerance and inhibit anti-tumor immunity. Stimulation of TIM3 with an agonistic antibody promotes inflammation through the activation of innate immune cells. TIM3 is also regarded as a potential target molecule for immunotherapy. TIM3 and programmed cell death 1 (PD-1) as two important coinhibitory regulators of T cell responses, have been implicated with the T-cell dysfunction or exhaustion associated with chronic HBV infection including HBV-related HCC.

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