## Recombinant Mouse TIM-3/HAVCR2 Protein (Fc Tag)

Catalog Number: PKSM041277



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

O8VIM0

Description	
Species	Mouse
Mol_Mass	46.3 kDa

**Bio-activity** Not validated for activity

Pro	pe rtie s

Accession

**Purity** > 95 % as determined by reducing SDS-PAGE.

Endotoxin < 1.0 EU per µg 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 of PBS, pH 7.4.

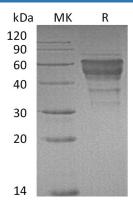
Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants

before lyophilization.

Please refer to the specific buffer information in the printed manual.

**Reconstitution** Please refer to the printed manual for detailed information.

## Data



## Background

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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