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Tim-3 Recombinant Protein

CATALOG NUMBER: 90-550

Specifications

SPECIES:	Mouse
SOURCE SPECIES:	CHO cells
SEQUENCE:	The extracellular domain of mouse Tim-3 (aa 21-189) is fused to the N-terminus of the Fc region of human IgG1.
FUSION TAG:	Fc Tag
APPLICATIONS:	This recombinant proteins is for research use only.
BIOLOGICAL ACTIVITY:	Immobilized recombinant human Galectin-9 at 500ng/ml can bind Tim-3 (mouse):Fc (human) (rec.) with an apparent KD <10nM. Optimal dilutions should be determined by each laboratory for each application.

Properties

PURITY:	>98% (SDS-PAGE)
PHYSICAL STATE:	Lyophilized
BUFFER:	Lyophilized from 0.2um-filtered solution in PBS.
STORAGE CONDITIONS:	Stable for at least 1 year after receipt when stored at -20˚C. Working aliquots are stable for up to 3 months when stored at -20˚C.

Additional Info

ALTERNATE NAMES:	TIM3, TIMD3, Hepatitis A Virus Cellular Receptor 2, HAVcr-2, T Cell Immunoglobulin and Mucin Domain-containing Protein 3
ACCESSION NO.:	AAI06852
PROTEIN GI NO.:	76825173

Background

The TIM (T cell/transmembrane, immunoglobulin and mucin) family plays a critical role in regulating immune responses, including allergy, asthma, transplant tolerance, autoimmunity and the response to viral infections. The unique structure of TIM immunoglobulin variable region domains allows highly specific recognition of phosphatidylserine (PtdSer), exposed on the surface of apoptotic cells. Tim-3, a type I transmembrane protein, contains an immunoglobulin and a mucin-like domain in its extracellular portion and a tyrosine phosphorylation motif in its cytoplasmic portion. TIM-3 is preferentially expressed on Th1 and Tc1 cells, and generates an inhibitory signal resulting in apoptosis of Th1 and Tc1 cells. TIM-3 is also expressed on some dendritic cells and can mediate phagocytosis of apoptotic cells and cross-presentation of antigen. Tim-3 functions to inhibit aggressive Th1-mediated auto- and alloimmune responses. Tim-3 pathway blockade by administration of Tim-3:Fc fusion protein accelerates diabetes in nonobese diabetic mice, causes hyperproliferation of Th1 cells and Th1 cytokine release in an experimental autoimmune encephalomyelitis (EAE) model and prevents acquisition of transplantation tolerance induced by costimulation blockade.

FOR RESEARCH USE ONLY

December 14, 2016