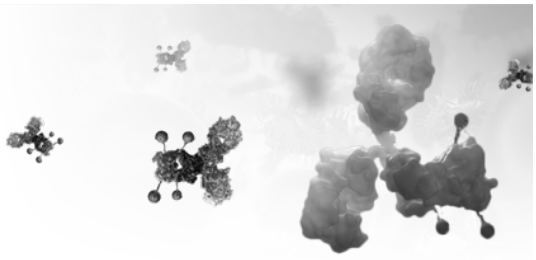

Rat Anti-Mouse TIM-3 (CD366) Monoclonal antibody, clone RMT3-23 (CABT-L4412)

This product is for research use only and is not intended for diagnostic use.

PRODUCT INFORMATION

Product Overview	The RMT3-23 monoclonal antibody reacts with mouse TIM-3 (T cell immunoglobulin and mucin domain-3) also known as CD366. TIM-3 is a 60 kDa member of the TIM family of immune checkpoint receptors and exists as a type I transmembrane glycoprotein with a mucin-like domain in its extracellular portion and a tyrosine phosphorylation motif in its cytoplasmic portion. TIM-3 is specifically expressed at high levels on the surface of Th1 lymphocytes whereas Th2 lymphocytes express TIM-1 and TIM-2. TIM-3 activation occurs via binding to the cell-associated C-type lectin galectin-9. Upon binding TIM-3 induces apoptosis of Th1 cells. Inhibition of TIM-3 signaling in mice has been shown to exacerbate experimental autoimmune encephalomyelitis, promote IFNγ production and Th1 cell proliferation. Tim-3 has also been shown to be required for the induction of tolerance, as both TIM-3 knockout animals and mice treated with TIM-3-Ig fusion protein display defects in the induction of antigen-specific tolerance. Additionally, TIM-3 signaling is currently being explored as a cancer immunotherapy target as CD8 T cells which express both TIM-3 and PD-1 exhibit greater defects in both cell-cycle progression and effector cytokine production than cells that express PD-1 alone. The RMT3-23 antibody acts as a TIM-3 receptor antagonist and has been shown to have functional activity including suppressing tumor cell growth in a murine sarcoma model.
Target	Mouse TIM-3 (CD366)
Immunogen	Recombinant mouse TIM-3
Isotype	IgG2a, κ
Source/Host	Rat
Species Reactivity	Mouse
Clone	RMT3-23
Purification	Protein G purified.

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	Purity>95%. Determined by SDS-PAGE
Conjugate	Functional Grade
Applications	in vivo TIM-3 neutralization, in vitro TIM-3 blocking, FC
Molecular Weight	150 kDa
Format	0.2 μM filtered liquid. Purified from tissue culture supernatant in an animal free facility
Concentration	Lot specific
Size	5 mg
Buffer	PBS, pH 7.0. Contains no stabilizers or preservatives. [low endotoxin azide-free] Endotoxin level: <2EU/mg (<0.002EU/μg). Determined by LAL gel clotting assay Related dilution buffer: CABT-LB04
Preservative	None
Storage	The antibody solution should be stored undiluted at 4°C, and protected from prolonged exposure to light. Do not freeze.
Ship	Wet ice

BACKGROUND

Introduction	The protein encoded by this gene belongs to the immunoglobulin superfamily, and TIM family of proteins. CD4-positive T helper lymphocytes can be divided into types 1 (Th1) and 2 (Th2) on the basis of their cytokine secretion patterns. Th1 cells are involved in cell-mediated immunity to intracellular pathogens and delayed-type hypersensitivity reactions, whereas, Th2 cells are involved in the control of extracellular helminthic infections and the promotion of atopic and allergic diseases. This protein is a Th1-specific cell surface protein that regulates macrophage activation, and inhibits Th1-mediated auto- and alloimmune responses, and promotes immunological tolerance. [provided by RefSeq, Sep 2011]
Keywords	HAVCR2;hepatitis A virus cellular receptor 2;TIM3;KIM-3;TIMD3;Tim-3;TIMD-3;HAVcr-2;kidney injury molecule-3;T-cell membrane protein 3

GENE INFORMATION

Official Symbol	hepatitis A virus cellular receptor 2
Synonyms	HAVCR2; hepatitis A virus cellular receptor 2; TIM3; KIM-3; TIMD3; Tim-3; TIMD-3; HAVcr-2;

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	kidney injury molecule-3; T-cell membrane protein 3
References	Liu, J. F., et al. (2018). "Blockade of TIM3 relieves immunosuppression through reducing regulatory T cells in head and neck cancer." J Exp Clin Cancer Res 37(1): 44. PubMed;Dietze, K. K., et al. (2013). "Combining regulatory T cell depletion and inhibitory receptor blockade improves reactivation of exhausted virus-specific CD8+ T cells and efficiently reduces chronic retroviral loads." PLoS Pathog 9(12): e1003798. PubMed;Zelinskyy, G., et al. (2011). "Virus-specific CD8+ T cells upregulate programmed death-1 expression during acute friend retrovirus infection but are highly cytotoxic and control virus replication." J Immunol 187(7): 3730-3737. PubMed;

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