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InnoCyto Inc.

15375 Barranca Pkwy, Suite I-103
Irvine, CA 92618
Email: info@innocyto.com
Phone: 1-949-418-7543

In Vivo Star Anti-Mouse CD154 (CD40L) Antibody

Catalog Number:	512001, 512002, 512003
Size:	1 mg, 5 mg, 25 mg
Target Name:	CD154, CD40L, gp39, TRAP, T-BAM, TNFSF5, CD40 Ligand
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	MR1
Application:	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-mouse CD40L (CD154) Monoclonal Antibody
Isotype:	Armenian Hamster IgG
Antibody Type:	Recombinant
Purity:	>95% by reducing SDS-PAGE
Endotoxin:	< 1 EU per 1 mg of the protein by the LAL method.
Storage Conditions:	4ºC
Grade:	In vivo
Recommended Usage:	This product is suitable for in vivo animal use. Optimal amounts need to be determined empirically for each experiment.
Hidden Synonyms:	InVivoMab, InVivoPlus, GoInVivo, In Vivo Gold

BACKGROUND INFORMATION

CD154, also known as CD40 ligand (CD40L) or TNFSF5, is a key immunoregulatory molecule that plays a central role in communication between T cells and antigen-presenting cells. It is most prominently expressed on activated CD4+ T helper cells, but can also be found on activated CD8+ T cells, platelets, B cells, and other immune cell types under certain conditions. CD154 is essential for coordinating adaptive immune responses, particularly those involving B cell activation and antibody production.

Structurally, CD154 is a type II transmembrane glycoprotein and a member of the tumor necrosis factor (TNF) superfamily. It consists of a short N-terminal cytoplasmic domain, a single transmembrane region, and a C-terminal extracellular domain that forms homotrimers, a characteristic feature of TNF family ligands. Trimerization of CD154 is required for effective binding and activation of its receptor, enabling efficient signal transduction in CD40-expressing cells.

The principal ligand for CD154 is CD40, a receptor expressed on B cells, dendritic cells, macrophages, monocytes, and various non-hematopoietic cells. Engagement of CD40 by CD154 delivers a potent activation signal to antigen-presenting cells. In B cells, this interaction is required for immunoglobulin class switching, germinal center formation, somatic hypermutation, and the

development of memory B cells and long-lived plasma cells. In dendritic cells and macrophages, CD154–CD40 signaling enhances antigen presentation, cytokine production, and the ability to prime naïve T cells.

Defects in CD154 expression or function have profound clinical consequences. Genetic deficiency of CD154 causes X-linked hyper-IgM syndrome, characterized by impaired immunoglobulin class switching, recurrent infections, and defective humoral immunity. Conversely, excessive or prolonged CD154 expression contributes to pathological inflammation in autoimmune diseases such as systemic lupus erythematosus and rheumatoid arthritis, as well as in transplant rejection and atherosclerosis. CD154 expressed on platelets has also been implicated in thromboinflammatory processes.

Therapeutically, the CD154–CD40 pathway has attracted significant interest. Blocking CD154 or CD40 can dampen pathogenic immune activation and is being explored as a strategy for treating autoimmune disease and preventing transplant rejection. However, early anti-CD154 therapies were associated with thrombotic complications due to platelet expression of CD154, prompting the development of safer approaches targeting CD40 instead. Conversely, agonistic strategies that enhance CD154–CD40 signaling are being investigated to boost immune responses in cancer immunotherapy and vaccine development. Together, these approaches highlight CD154’s critical role in immune regulation and therapeutic intervention.

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