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In Vivo Star Anti-Mouse CD16/CD32 Antibody

Catalog Number:	511201, 511202, 511203
Size:	1 mg, 5 mg, 25 mg
Target Name:	CD16/32, Fcγ R III/II, Ly-17
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	2.4G2-mFab
Application:	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-mouse CD16/CD32 Monoclonal Antibody
Isotype:	Mouse Fab Fragment
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
RRID:	AB_3739386
GeneID:	14131, 14130

BACKGROUND INFORMATION

CD16 (FcγRIII) and CD32 (FcγRII) are receptors for the Fc region of immunoglobulin G (IgG) and are key mediators of antibody-dependent immune responses. They are expressed on a variety of immune cells, including natural killer (NK) cells, monocytes, macrophages, neutrophils, dendritic cells, and B cells. By linking antibody recognition to cellular effector functions, CD16 and CD32 serve as critical bridges between humoral immunity and innate immune mechanisms.

Structurally, both CD16 and CD32 are members of the immunoglobulin superfamily but differ in affinity, signaling capacity, and cellular distribution. CD16 exists in two main forms: CD16a (FcγRIIIa), a transmembrane receptor expressed on NK cells and myeloid cells that associates with signaling adaptor chains containing immunoreceptor tyrosine-based activation motifs (ITAMs), and CD16b (FcγRIIIb), a glycosylphosphatidylinositol (GPI)-anchored receptor expressed primarily on neutrophils. CD32 includes several isoforms, most notably CD32a (FcγRIIa) and CD32b (FcγRIIb), which are transmembrane receptors with distinct cytoplasmic

signaling motifs.

The ligands for CD16 and CD32 are IgG antibodies bound to antigen, forming immune complexes. CD16 primarily binds IgG1 and IgG3 and is a major mediator of antibody-dependent cellular cytotoxicity (ADCC), particularly by NK cells. CD32a transmits activating signals via an intrinsic ITAM, promoting phagocytosis, cytokine release, and inflammatory responses. In contrast, CD32b contains an immunoreceptor tyrosine-based inhibitory motif (ITIM) in its cytoplasmic tail and functions as a negative regulator, dampening B cell receptor and Fc receptor signaling to maintain immune balance.

Altered expression or function of CD16 and CD32 is implicated in a range of diseases. In autoimmune disorders such as systemic lupus erythematosus and rheumatoid arthritis, dysregulated Fcγ receptor signaling contributes to immune complex–mediated inflammation and tissue damage. In infectious diseases, these receptors are essential for efficient clearance of antibody-opsonized pathogens. Genetic polymorphisms in CD16 and CD32 influence IgG binding affinity and are associated with variability in susceptibility to disease and responses to antibody-based therapies.

Therapeutically, CD16 and CD32 are central to the mechanism of action of many monoclonal antibody drugs. Engagement of CD16 on NK cells is critical for ADCC-mediated tumor cell killing, making it a key determinant of antibody efficacy in cancer therapy. Engineering antibodies to enhance Fcγ receptor binding is an active area of drug development. Conversely, targeting inhibitory CD32b signaling is being explored to modulate immune responses in autoimmunity and to improve vaccine efficacy. Together, CD16 and CD32 represent pivotal regulators of antibody-driven immunity with broad clinical relevance.

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