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In Vivo Star Anti-Mouse CD79b Antibody

Catalog Number:	512201, 512202, 512203
Size:	1 mg, 5 mg, 25 mg
Target Name:	CD76b, Igb, Ig-beta, B29
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	HM79b
Application:	ELISA, WB, Flow cytometry, IHC, ICC, animal model study
Reactivity:	Mouse
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-mouse CD79b Monoclonal Antibody
Isotype:	Armenian Hamster IgG Lambda
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

CD79b, also known as Igβ or B29, is an essential component of the B cell antigen receptor (BCR) complex and plays a critical role in B cell development, activation, and signaling. CD79b is expressed almost exclusively on B lineage cells, from the pre-B cell stage through mature peripheral B cells, and remains associated with surface immunoglobulin throughout the B cell lifespan. By transducing signals from antigen-bound immunoglobulin, CD79b enables B cells to respond appropriately to antigenic stimulation.

Structurally, CD79b is a type I transmembrane glycoprotein belonging to the immunoglobulin superfamily. Its extracellular region contains a single Ig-like domain that associates noncovalently with membrane-bound immunoglobulin. CD79b forms a heterodimer with CD79a (Igα), and together these two signaling subunits pair with antigen-specific immunoglobulin heavy and light chains to form the complete BCR complex. The cytoplasmic tail of CD79b contains an immunoreceptor tyrosine-based activation motif (ITAM), which is essential for downstream signaling.

Unlike many immune receptors, CD79b does not bind a ligand directly. Instead, it functions as a signal-transducing subunit for the BCR, which recognizes a wide range of antigens through its associated immunoglobulin component. Upon antigen binding and BCR

clustering, the ITAMs within the cytoplasmic tails of CD79a and CD79b become phosphorylated by Src family kinases. This triggers recruitment of Syk and activation of multiple signaling pathways, including PI3K, MAPK, and NF-κB, leading to B cell activation, proliferation, differentiation, or tolerance depending on context.

Alterations in CD79b expression or signaling are implicated in several diseases. Mutations affecting CD79b can impair BCR signaling and result in immunodeficiency due to defective B cell development or activation. Conversely, chronic or dysregulated BCR signaling involving CD79b contributes to the pathogenesis of B cell malignancies, including diffuse large B cell lymphoma and other non-Hodgkin lymphomas. In these cancers, sustained signaling through the BCR promotes malignant cell survival and proliferation.

Therapeutically, CD79b has emerged as an important target in B cell–directed therapies. Antibody–drug conjugates targeting CD79b exploit its restricted expression on B cells to deliver cytotoxic agents selectively to malignant cells, minimizing off-target effects. In addition, modulation of BCR signaling pathways downstream of CD79b has become a cornerstone of therapy for B cell cancers. These strategies underscore CD79b's central role in B cell biology and its clinical relevance in immune-mediated disease and oncology.

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