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In Vivo Star Anti-Human HLA class II DR/DQ Antibody

Catalog Number:	518901, 518902, 518903
Size:	1 mg, 5 mg, 25 mg
Target Name:	Major Histocompatibility complex II, human leukocyte antigen, HLA, MHC class II
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	9.3F10
Application:	Direct ELISA, functional assay, Flow Cytometry
Reactivity:	Human
Format:	Liquid
Product Description:	In vivo Grade Recombinant Anti-Human HLA class II DR/DQ Monoclonal Antibody
Isotype:	Mouse IgG2a Kappa
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 in in vitro functional assays or in vivo on human cells used in animal models. Optimal amounts need to be determined empirically for each experiment.
Hidden Synonyms:	InVivoMab, InVivoPlus, GoInVivo, In Vivo Gold

BACKGROUND INFORMATION

HLA-DR and HLA-DQ are members of the major histocompatibility complex (MHC) class II family, which play a central role in orchestrating adaptive immune responses. These molecules are predominantly expressed on professional antigen-presenting cells (APCs), including dendritic cells, macrophages, and B cells, and are responsible for presenting extracellular peptide antigens to $CD4^+$ T helper cells. This interaction is essential for initiating immune responses against pathogens, maintaining self-tolerance, and shaping adaptive immunity through cytokine signaling and T cell activation.

Structurally, both HLA-DR and HLA-DQ are heterodimeric glycoproteins composed of an alpha (α) and a beta (β) chain, each anchored in the cell membrane. The α1 and β1 domains form the peptide-binding groove, which accommodates peptides typically 13–25 amino acids in length. Unlike MHC class I, which binds peptides generated from intracellular proteins, class II molecules load peptides derived from extracellular sources. This process occurs in late endosomes or lysosomes, where the invariant chain (Ii) guides and stabilizes the complex until peptide loading occurs via the HLA-DM molecule. Once a stable peptide-MHC complex is formed, it is transported to the cell surface for recognition by the T cell receptor (TCR) on $CD4^+$ T cells.

The ligands for HLA-DR and HLA-DQ are peptide antigens derived from pathogens, commensal flora, or self-proteins. The TCR on helper T cells acts as the complementary binding partner, recognizing these peptide–MHC class II complexes with high specificity. Co-stimulatory interactions between APCs and T cells further determine whether the response leads to immune activation or tolerance, making HLA-DR/DQ pivotal in immune regulation.

In disease, specific HLA-DR and HLA-DQ alleles are strongly associated with autoimmune disorders. For instance, HLA-DRB1*03 and DQB1*02 are linked to type 1 diabetes and celiac disease, while HLA-DRB1*04 alleles are associated with rheumatoid arthritis. These genetic variants influence peptide binding preferences and may promote the presentation of self-antigens, triggering autoreactive T cell activation. Moreover, altered HLA-II expression on tumors and infected cells can impair immune surveillance. Elevated HLA-DR expression on immune cells also serves as a marker of immune activation and has been linked to inflammation and chronic infections.

Therapeutically, HLA-DR and HLA-DQ are essential in transplantation compatibility testing, autoimmune disease risk assessment, and antigen-specific immunotherapies. In cancer immunotherapy and vaccine development, understanding an individual's HLA-II genotype helps tailor peptide-based vaccines and checkpoint inhibitor strategies to optimize T helper cell activation. Likewise, therapies that modulate HLA-II expression or block autoreactive T cell recognition are being explored as treatments for autoimmune diseases such as multiple sclerosis and type 1 diabetes.

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