

In Vivo Star Anti-Human HLA-DR/DP/DQ Antibody

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| Catalog Number: | 518501, 518502, 518503 |
| Size: | 1 mg, 5 mg, 25 mg |
| Target Name: | Human HLA-DR/DP/DQ |
| Regulatory Status: | RUO |

PRODUCT DETAILS

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| Clone: | F3.3 |
| Application: | Direct ELISA, functional assay, Flow Cytometry |
| Reactivity: | Human |
| Format: | Liquid |
| Product Description: | In vivo Grade Recombinant Anti-Human HLA-DR/DP/DQ Monoclonal Antibody |
| Isotype: | Mouse IgG1 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: | 4°C |
| 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, HLA-DP, and HLA-DQ are the classical human leukocyte antigen (HLA) class II molecules that play a central role in the adaptive immune system by presenting extracellular peptide antigens to CD4⁺ T helper cells. Encoded within the MHC class II region on chromosome 6, these molecules are expressed primarily on professional antigen-presenting cells such as dendritic cells, macrophages, and B cells. Their main function is to initiate immune responses against pathogens, maintain tolerance to self-antigens, and regulate immune-mediated signaling within tissues.

Structurally, HLA class II molecules are heterodimers composed of two non-covalently associated transmembrane glycoprotein chains: an alpha (α) chain and a beta (β) chain. The α 1 and β 1 domains form the peptide-binding groove, which accommodates peptides typically 13–25 amino acids long. Each HLA-II is encoded by distinct gene pairs: HLA-DRA and -DRB for HLA-DR, HLA-DPA and -DPB for HLA-DP, and HLA-DQA and -DQB for HLA-DQ. These molecules assemble in the endoplasmic reticulum and are stabilized by the invariant chain (Ii), which prevents premature peptide binding. In late endosomes, Ii is degraded, and peptides generated from extracellular antigens are loaded onto HLA-II molecules with the assistance of the peptide editor HLA-DM before being presented on the cell surface.

The ligands for HLA-DR, DP, and DQ are peptide antigens derived mainly from exogenous proteins internalized by antigen-presenting cells. These peptide-MHC II complexes are recognized by the T cell receptor (TCR) on CD4⁺ T helper cells, triggering their activation and cytokine release. This interaction bridges innate and adaptive immunity, shaping both humoral and cell-mediated responses.

Polymorphisms in HLA-DR, DP, and DQ genes have profound effects on disease susceptibility. Specific allelic variants are associated with autoimmune disorders, including type 1 diabetes (DR3-DQ2, DR4-DQ8), rheumatoid arthritis (DRB1*04), celiac disease (DQ2 and DQ8), and multiple sclerosis (DRB1*15). These associations arise because certain variants preferentially present self-peptides that activate autoreactive T cells. Conversely, HLA-II polymorphism contributes to protection against infections by broadening the range of peptides that can be presented to the immune system.

In therapeutic contexts, HLA-DR/DP/DQ typing is critical for transplantation compatibility, as mismatches can lead to graft rejection or graft-versus-host disease. Beyond transplantation, HLA-II molecules are vital in predicting immune responses to vaccines and designing antigen-specific immunotherapies. Advances in peptide-based vaccination and autoimmune disease treatments increasingly rely on identifying the precise peptide-HLA-II combinations that drive protective or pathogenic immune activation. Thus, HLA-DR, DP, and DQ remain central to understanding immune recognition, tolerance, and therapeutic modulation in human health and disease.

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