

Recombinant Human HLA-DQA2 Protein (24-214 aa), His-tagged

Cat. No. HLA-DQA2-1279H **Lot. No.** (See product label)

SPECIFICATION

Product Overview	Recombinant Human HLA-DQA2 Protein (24-214 aa) is produced by E. coli expression system. This protein is fused with a 6xHis tag at the N-terminal. Research Area: Immunology. Protein Description: Partial.
Species	Human
Source	E.coli
ProteinLength	24-214 aa
Description	Binds peptides derived from antigens that access the endocytic route of antigen presenting cells (APC) and presents th on the cell surface for recognition by the CD4 T-cells. The peptide binding cleft accommodates peptides of 10-30 residues. The peptides presented by MHC class II molecules are generated mostly by degradation of proteins that access the endocytic route, where they are processed by lysosomal proteases and other hydrolases. Exogenous antigens that have been endocytosed by the APC are thus readily available for presentation via MHC II molecules, and for this reason this antigen presentation pathway is usually referred to as exogenous. As mbrane proteins on their way to degradation in lysosomes as part of their normal turn-over are also contained in the endosomal/lysosomal compartments, exogenous antigens must compete with those derived from endogenous components. Autophagy is also a source of endogenous peptides, autophagosomes constitutively fuse with MHC class II loading compartments. In addition to APCs, other cells of the

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gastrointestinal tract, such as epithelial cells, express MHC class II molecules and CD74 and act as APCs, which is an unusual trait of the GI tract. To produce a MHC class II molecule that presents an antigen, three MHC class II molecules (heterodimers of an alpha and a beta chain) associate with a CD74 trimer in the ER to form a heterononamer. Soon after the entry of this complex into the endosomal/lysosomal syst where antigen processing occurs, CD74 undergoes a sequential degradation by various proteases, including CTSS and CTSL, leaving a small fragment termed CLIP (class-II-associated invariant chain peptide). The roval of CLIP is facilitated by HLA-DM via direct binding to the alpha-beta-CLIP complex so that CLIP is released. HLA-DM stabilizes MHC class II molecules until primary high affinity antigenic peptides are bound. The MHC II molecule bound to a peptide is then transported to the cell mbrane surface. In B-cells, the interaction between HLA-DM and MHC class II molecules is regulated by HLA-DO. Primary dendritic cells (DCs) also to express HLA-DO. Lysosomal microenvironment has been implicated in the regulation of antigen loading into MHC II molecules, increased acidification produces increased proteolysis and efficient peptide loading.

Form Tris-based buffer,50% glycerol

Molecular Mass 25.6 kDa

AA Sequence EDIVADHVASYG VNFYQSHGPSGQYTHEFDGDEEFYVDLETKETVWQLPMFSKFIS
FDPQSALRNMAVGKHTLEFMMRQSNSTAATNEVPEVTVFSKFPVTLGQPNTLICLV
DNIFPPVVNITWLSNGHSVTEGVSETSFLSKSDHSFFKISYLTFLPSADEIYDCKVEH
WGLDEPLLKHWEPEIPAPMSE

Purity > 90% as determined by SDS-PAGE.

Notes Repeated freezing and thawing is not recommended. Store working aliquots at 4 centigrade for up to one week.

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Storage

The shelf life is related to many factors, storage state, buffer ingredients, storage temperature and the stability of the protein itself. Generally, the shelf life of liquid form is 6 months at -20 centigrade/-80 centigrade. The shelf life of lyophilized form is 12 months at -20 centigrade/-80 centigrade.

Concentration

A hardcopy of COA with reconstitution instruction is sent along with the products. Please refer to it for detailed information.

GENE INFORMATION

Gene Name

[HLA-DQA2 major histocompatibility complex, class II, DQ alpha 2 \[Homo sapiens \]](#)

Official Symbol

[HLA-DQA2](#)

Synonyms

HLA-DQA2; HLA DXA; HLA-DQA1; DX alpha chain; HLA-DXA; DX-ALPHA;

Gene ID

[3118](#)

mRNA Refseq

[NM_020056](#)

Protein Refseq

[NP_064440](#)

MIM

[613503](#)

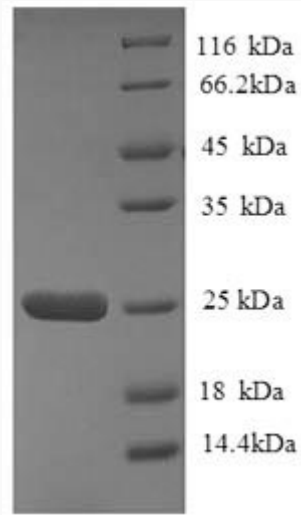
UniProt ID

[P01906](#)

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(Tris-Glycine gel) Discontinuous SDS-PAGE (reduced) with 5% enrichment gel and 15% separation gel.

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