

HLA-DQA1 Blocking Peptide (C-term)

Synthetic peptide Catalog # BP21386b

Specification

HLA-DQA1 Blocking Peptide (C-term) - Product Information

Primary Accession

P01909

HLA-DQA1 Blocking Peptide (C-term) - Additional Information

Gene ID 3117

Other Names

HLA class II histocompatibility antigen, DQ alpha 1 chain, DC-1 alpha chain, DC-alpha, HLA-DCA, MHC class II DQA1, HLA-DQA1

Target/Specificity

The synthetic peptide sequence is selected from aa 188-202 of HUMAN HLA-DQA1

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

HLA-DQA1 Blocking Peptide (C-term) - Protein Information

Name HLA-DQA1

Function

Binds peptides derived from antigens that access the endocytic route of antigen presenting cells (APC) and presents them 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 membrane 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 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 system 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 removal 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 membrane 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.

Cellular Location

Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane; Single-pass type I membrane protein. Golgi apparatus, trans-Golgi network membrane; Single-pass type I membrane protein. Endosome membrane; Single-pass type I membrane protein. Lysosome membrane; Single-pass type I membrane protein Note=The MHC class II complex transits through a number of intracellular compartments in the endocytic pathway until it reaches the cell membrane for antigen presentation

HLA-DQA1 Blocking Peptide (C-term) - Protocols

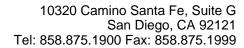
Provided below are standard protocols that you may find useful for product applications.

• Blocking Peptides

HLA-DQA1 Blocking Peptide (C-term) - Images

HLA-DQA1 Blocking Peptide (C-term) - Background

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HLA-DQA1 Blocking Peptide (C-term) - References

Chang H.-C.,et al.Nature 305:813-815(1983). Schenning L.,et al.EMBO J. 3:447-452(1984). Auffray C.,et al.Nature 308:327-333(1984). Schiffenbauer J.,et al.J. Immunol. 139:228-233(1987). Ellis M.C.,et al.Immunogenetics 46:410-417(1997).