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HLA-DRB1 Blocking Peptide (Center)

Synthetic peptide
Catalog # BP20577c

Specification

HLA-DRB1 Blocking Peptide (Center) - Product Information

Primary Accession	P04229
Other Accession	Q30154

HLA-DRB1 Blocking Peptide (Center) - Additional Information

Other Names
HLA class II histocompatibility antigen, DRB1-1 beta chain, MHC class II antigen DRB1*1, DR-1, DR1, HLA-DRB1

Target/Specificity
The synthetic peptide sequence is selected from aa 113-127 of HUMAN HLA-DRB1

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-DRB1 Blocking Peptide (Center) - Protein Information

HLA-DRB1 Blocking Peptide (Center) - Protocols

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

- Blocking Peptides

HLA-DRB1 Blocking Peptide (Center) - Images

HLA-DRB1 Blocking Peptide (Center) - Background

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.

HLA-DRB1 Blocking Peptide (Center) - References

Tonnelle C.,et al.EMBO J. 4:2839-2847(1985).
Bell J.I.,et al.Proc. Natl. Acad. Sci. U.S.A. 82:3405-3409(1985).
Coppin H.L.,et al.J. Immunol. 144:984-989(1990).
Raymond C.K.,et al.Genome Res. 15:1250-1257(2005).
von Salome J.,et al.Immunogenetics 59:261-271(2007).