

HLA-DRB4 Antibody (N-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP13690a

Specification

HLA-DRB4 Antibody (N-term) - Product Information

Application WB,E **Primary Accession** P13762 Other Accession NP 068818.4 Reactivity Human Host **Rabbit** Clonality **Polyclonal** Isotype Rabbit IgG Calculated MW 29941 Antigen Region 43-72

HLA-DRB4 Antibody (N-term) - Additional Information

Gene ID 3126

Other Names

HLA class II histocompatibility antigen, DR beta 4 chain, MHC class II antigen DRB4, HLA-DRB4

Target/Specificity

This HLA-DRB4 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 43-72 amino acids from the N-terminal region of human HLA-DRB4.

Dilution

WB~~1:1000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

HLA-DRB4 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

HLA-DRB4 Antibody (N-term) - Protein Information

Name HLA-DRB4

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

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. Late endosome 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

molecules, increased acidification produces increased proteolysis and efficient peptide loading.

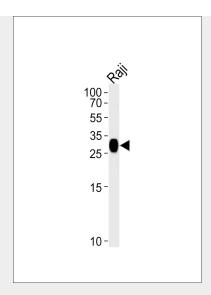
HLA-DRB4 Antibody (N-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

HLA-DRB4 Antibody (N-term) - Images





Western blot analysis of lysate from Raji cell line, using HLA-DRB4 Antibody (N-term)(Cat. #AP13690a). AP13690a was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody. Lysate at 35ug per lane.

HLA-DRB4 Antibody (N-term) - Background

HLA-DRB4 belongs to the HLA class II beta chain paralogues. This class II molecule is a heterodimer consisting of an alpha (DRA) and a beta (DRB) chain, both anchored in the membrane. It plays a central role in the immune system by presenting peptides derived from extracellular proteins. Class II molecules are expressed in antigen presenting cells (APC: B lymphocytes, dendritic cells, macrophages). The beta chain is approximately 26-28 kDa and its gene contains 6 exons. Exon one encodes the leader peptide, exons 2 and 3 encode the two extracellular domains, exon 4 encodes the transmembrane domain and exon 5 encodes the cytoplasmic tail. Within the DR molecule the beta chain contains all the polymorphisms specifying the peptide binding specificities. Typing for these polymorphisms is routinely done for bone marrow and kidney transplantation. DRB1 is expressed at a level five times higher than its paralogues DRB3, DRB4 and DRB5. The presence of DRB4 is linked with allelic variants of DRB1, otherwise it is omitted. There are 4 related pseudogenes: DRB2, DRB6, DRB7, DRB8 and DRB9.

HLA-DRB4 Antibody (N-term) - References

Awa, W.L., et al. Eur. J. Endocrinol. 163(1):97-104(2010) Christiansen, O.B., et al. J. Reprod. Immunol. 85(1):9-14(2010) Amoli, M.M., et al. Dis. Markers 28(1):49-53(2010) Gomez-Tortosa, E., et al. Dement Geriatr Cogn Disord 30(1):8-11(2010) Wei, B., et al. Zhonghua Liu Xing Bing Xue Za Zhi 30(11):1143-1146(2009)