

**HLA-DQA1 Antibody (Center)**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP12234c****Specification**

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**HLA-DQA1 Antibody (Center) - Product Information**

Application	<b>WB, FC,E</b>
Primary Accession	<a href="#">P01909</a>
Other Accession	<a href="#">NP_002113.2</a>
Reactivity	<b>Human</b>
Host	<b>Rabbit</b>
Clonality	<b>Polyclonal</b>
Isotype	<b>Rabbit IgG</b>
Calculated MW	<b>27805</b>
Antigen Region	<b>57-84</b>

**HLA-DQA1 Antibody (Center) - 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**

This HLA-DQA1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 57-84 amino acids from the Central region of human HLA-DQA1.

**Dilution**WB~~1:1000  
FC~~1:10~50**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-DQA1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

**HLA-DQA1 Antibody (Center) - 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 heteronamer. 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**

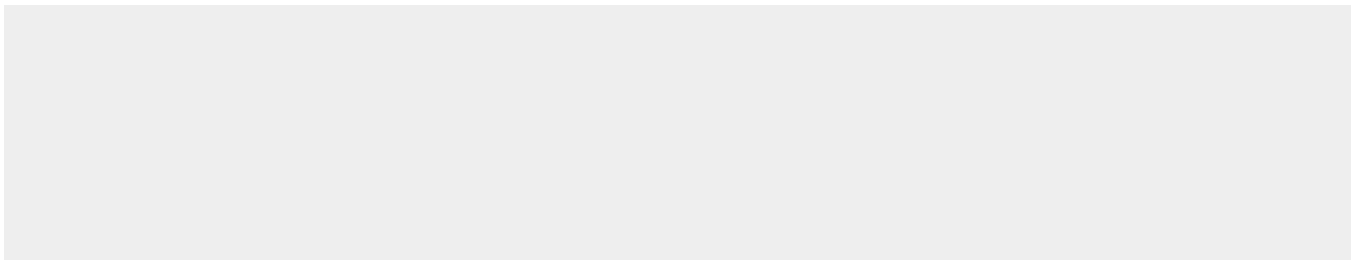
Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane; Single-pass type I membrane protein. Golgi apparatus, trans-Golgi network membrane; Single-pass type I membrane protein. Endosome membrane; Single-pass type I membrane protein. Lysosome membrane; Single-pass type I membrane 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

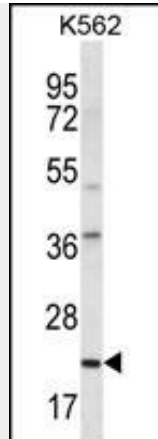
#### **HLA-DQA1 Antibody (Center) - Protocols**

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

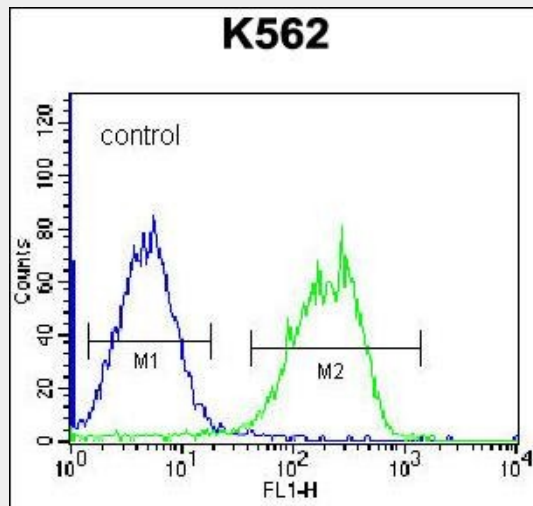
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### **HLA-DQA1 Antibody (Center) - Images**





HLA-DQA1 Antibody (Center) (Cat. #AP12234c) western blot analysis in K562 cell line lysates (35ug/lane). This demonstrates the HLA-DQA1 antibody detected the HLA-DQA1 protein (arrow).



HLA-DQA1 Antibody (Center) (Cat. #AP12234c) flow cytometric analysis of K562 cells (right histogram) compared to a negative control cell (left histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

### HLA-DQA1 Antibody (Center) - Background

HLA-DQA1 belongs to the HLA class II alpha chain paralogues. The class II molecule is a heterodimer consisting of an alpha (DQA) and a beta chain (DQB), 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 alpha chain is approximately 33-35 kDa. It is encoded by 5 exons; exon 1 encodes the leader peptide, exons 2 and 3 encode the two extracellular domains, and exon 4 encodes the transmembrane domain and the cytoplasmic tail. Within the DQ molecule both the alpha chain and the beta chain contain the polymorphisms specifying the peptide binding specificities, resulting in up to four different molecules. Typing for these polymorphisms is routinely done for bone marrow transplantation.

### HLA-DQA1 Antibody (Center) - References

Donaldson, P.T., et al. J. Hepatol. 53(6):1049-1053(2010)  
Noble, J.A., et al. Diabetes 59(11):2972-2979(2010)  
Ferreira, R.C., et al. Nat. Genet. 42(9):777-780(2010)  
Ovsyannikova, I.G., et al. PLoS ONE 5 (7), E11806 (2010) :  
Xun, Y.H., et al. Zhonghua Shi Yan He Lin Chuang Bing Du Xue Za Zhi 23(6):430-433(2009)