

**HLA-DQA1 Antibody (C-term)**  
**Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP21386b**

**Specification**

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**HLA-DQA1 Antibody (C-term) - Product Information**

Application	IF, WB, FC,E
Primary Accession	<a href="#">P01909</a>
Reactivity	Human, Mouse
Host	Rabbit
Clonality	polyclonal
Isotype	Rabbit IgG
Calculated MW	27805

**HLA-DQA1 Antibody (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**

This HLA-DQA1 antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between 188-222 amino acids from the C-terminal region of human HLA-DQA1.

**Dilution**

IF~~1:25  
WB~~1:8000  
FC~~1:25

**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 (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**HLA-DQA1 Antibody (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

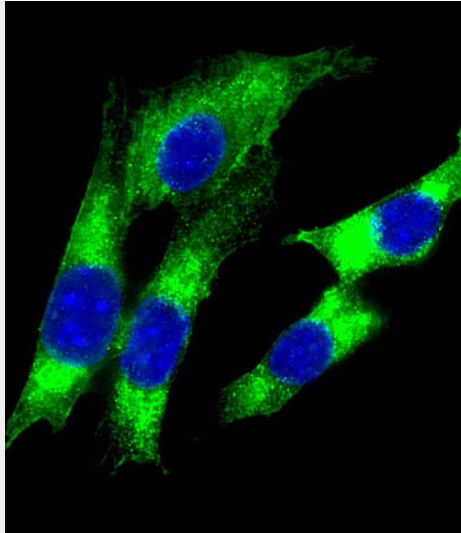
#### **HLA-DQA1 Antibody (C-term) - Protocols**

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

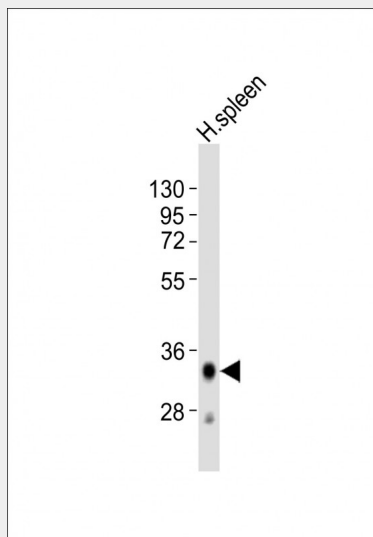
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

#### **HLA-DQA1 Antibody (C-term) - Images**

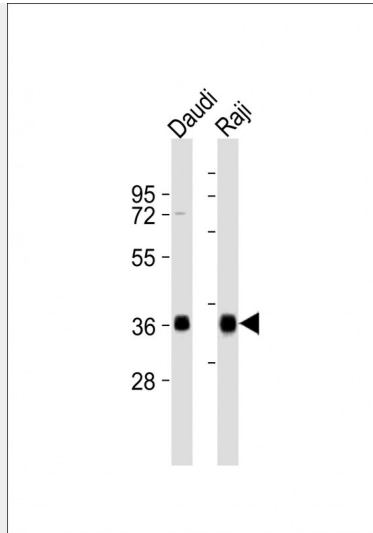




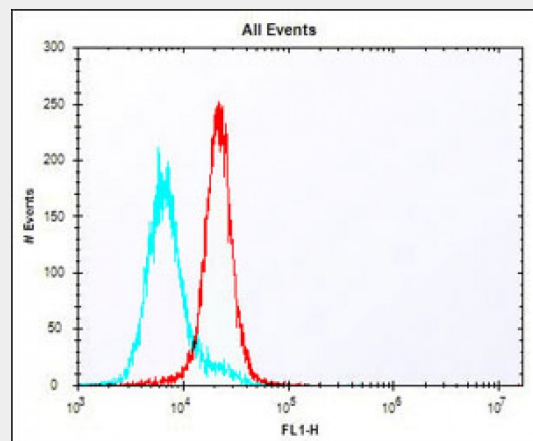
Immunofluorescent analysis of 4% paraformaldehyde-fixed, 0.1% Triton X-100 permeabilized NIH/3T3 (mouse embryonic fibroblast cell line) cells labeling HLA-DQA1 with AP21386b at 1/25 dilution, followed by Dylight® 488-conjugated goat anti-rabbit IgG (NK179883) secondary antibody at 1/200 dilution (green). Immunofluorescence image showing cytoplasm staining on NIH/3T3 cell line. The nuclear counter stain is DAPI (blue).



Anti-HLA-DQA1 Antibody (C-term) at 1:2000 dilution + human spleen lysates Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution Predicted band size : 28 kDa Blocking/Dilution buffer: 5% NFD/MBST.



All lanes : Anti-HLA-DQA1 Antibody (C-term) at 1:8000 dilution Lane 1: Daudi whole cell lysates  
 Lane 2: Raji whole cell lysates Lysates/proteins at 20  $\mu$ g per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution Predicted band size : 28 kDa Blocking/Dilution buffer: 5% NFDN/TBST.



Overlay histogram showing K562 cells stained with AP21386b (red line). The cells were fixed with 2% paraformaldehyde (10 min) and then permeabilized with 90% methanol for 10 min. The cells were then incubated in 2% bovine serum albumin to block non-specific protein-protein interactions followed by the antibody (AP21386b, 1:25 dilution) for 60 min at 37°C. The secondary antibody used was Alexa Fluor® 488 goat anti-rabbit IgG (H+L) (1583138) at 1/400 dilution for 40 min at 37°C. Isotype control antibody (blue line) was rabbit IgG1 (1 $\mu$ g/1x10<sup>6</sup> cells) used under the same conditions. Acquisition of >10, 000 events was performed.

### HLA-DQA1 Antibody (C-term) - 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-DQA1 Antibody (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).