



HLA-DQA2 Mouse mAb

Catalog No	YP-mAb-19498
Isotype	IgG
Reactivity	Human,Mouse,Rat
Applications	WB
Gene Name	HLA-DQA2 HLA-DXA
Immunogen	Synthesized peptide derived from human HLA-DQA2. AA range:120-190
Specificity	This antibody detects endogenous levels of HLA-DQA2 at Human, Mouse,Rat
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Monoclonal,Mouse,IgG
Purification	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
Dilution	WB 1:500-2000
Concentration	1 mg/ml
Purity	≥90%
Storage Stability	-15°C to -25°C/1 year(Do not lower than -25°C)
Synonyms	HLA-DQA2 HLA-DXA;HLA class II histocompatibility antigen, DQ alpha 2 chain (DX alpha chain) (HLA class II histocompatibility antigen, DQ (6) alpha chain) (HLA-DQA1) (MHC class II DQA2)
Observed Band	
Calculated Molecular Weight	28kD
Cell Pathway	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 . 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.
Tissue Specificity	Restricted to skin Langerhans cells , although some expression at low levels may occur at the surface of B lymphoblastoid cells.
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.

Background

matters needing attention

Avoid repeated freezing and thawing!

Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

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