

## HA2Q Antibody

Catalog No: #34737

Package Size: #34737-1 50ul #34737-2 100ul

Orders: [order@signalwayantibody.com](mailto:order@signalwayantibody.com)Support: [tech@signalwayantibody.com](mailto:tech@signalwayantibody.com)

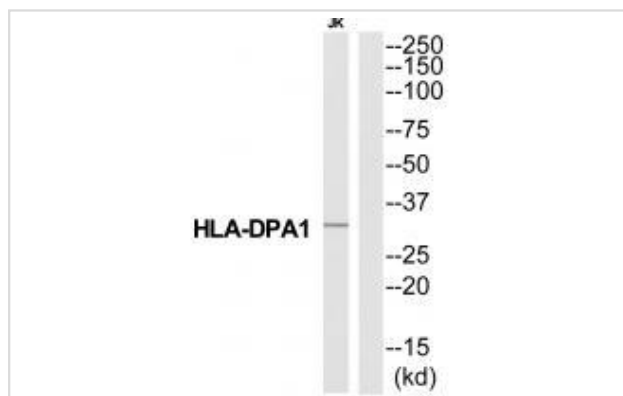
## Description

Product Name	HA2Q Antibody
Host Species	Rabbit
Clonality	Polyclonal
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Applications	WB
Species Reactivity	Hu
Specificity	The antibody detects endogenous levels of total HA2Q protein.
Immunogen Type	Peptide
Immunogen Description	Synthesized peptide derived from N-terminal of human HA2Q.
Target Name	HA2Q
Other Names	DP(W3); DP(W4); DPA1; HLA class II histocompatibility antigen; DP alpha 1 chain
Accession No.	Swiss-Prot: P20036NCBI Gene ID: 3113
Uniprot	P20036
GeneID	3113;
SDS-PAGE MW	30kd
Concentration	1.0mg/ml
Formulation	Rabbit IgG in phosphate buffered saline (without Mg <sup>2+</sup> and Ca <sup>2+</sup> ), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Storage	Store at -20°C

## Application Details

Western blotting: 1:500~1:3000

## Images



Western blot analysis of extracts from Jurkat cells, using HA2Q antibody #34737.

## 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 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.

Lawrance S.K., Nucleic Acids Res. 13:7515-7528(1985).

Gustafsson K., J. Biol. Chem. 262:8778-8786(1987).

Young J.A., Hum. Immunol. 23:37-44(1988).

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Note: This product is for in vitro research use only