Datasheet for ABIN1881422

anti-HLA-DQB1 antibody (N-Term)

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Overview

Quantity:	400 µL
Target:	HLA-DQB1
Binding Specificity:	AA 13-39, N-Term
Reactivity:	Human
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This HLA-DQB1 antibody is un-conjugated
Application:	Western Blotting (WB)

Product Details

Immunogen:	This HLA-DQB1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 13-39 amino acids from the N-terminal region of human HLA-DQB1.
Isotype:	Ig Fraction
Purification:	This antibody is purified through a protein A column, followed by peptide affinity purification.

Target Details

Target:	HLA-DQB1
Alternative Name:	HLA-DQB1 (HLA-DQB1 Products)
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

Target Details

	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 an 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 miroenvironment has been implicated in the regulation of antigen loading into MHC II molecules, increased acidification produces increased proteolysis and efficient peptide loading.
Molecular Weight:	29991
NCBI Accession:	NP_001230891, NP_002114
UniProt:	P01920
Pathways:	TCR Signaling, Production of Molecular Mediator of Immune Response, Cancer Immune Checkpoints, Human Leukocyte Antigen (HLA) in Adaptive Immune Response

Application Details

Application Notes:	WB: 1:1000
Restrictions:	For Research Use only

Handling

Format:	Liquid
Buffer:	Purified polyclonal antibody supplied in PBS with 0.09 % (W/V) sodium azide.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	4 °C,-20 °C
Expiry Date:	6 months

Publications

Product cited in:

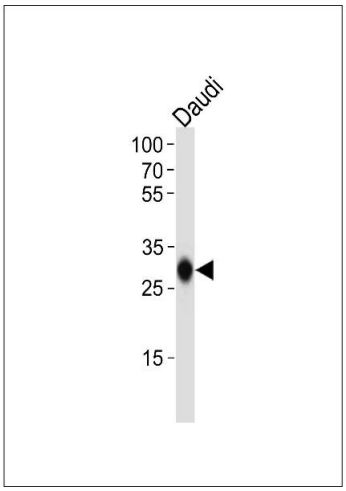
Mehta, Vazquez, Kulkarni, Kerrigan, Atwal, Metsugi, Toppmeyer, Levine, Hirshfield: "Polymorphic variants in TSC1 and TSC2 and their association with breast cancer phenotypes." in: **Breast cancer research and treatment**, Vol. 125, Issue 3, pp. 861-8, (2011) (PubMed).

Hoogeveen-Westerveld, Exalto, Maat-Kievit, van den Ouweland, Halley, Nellist: "Analysis of TSC1 truncations defines regions involved in TSC1 stability, aggregation and interaction." in: **Biochimica et biophysica acta**, Vol. 1802, Issue 9, pp. 774-81, (2010) (PubMed).

Mieulet, Lamb: "Tuberous sclerosis complex: linking cancer to metabolism." in: **Trends in molecular medicine**, Vol. 16, Issue 7, pp. 329-35, (2010) (PubMed).

Guo, Ying, Zhang, Yuan, Qian, Wang, Yang, He: "Tandem affinity purification and identification of the human TSC1 protein complex." in: **Acta biochimica et biophysica Sinica**, Vol. 42, Issue 4, pp. 266-73, (2010) (PubMed).

Liu, Wu, Chen, Ter-Minassian, Asomaning, Zhai, Wang, Su, Heist, Kulke, Lin, Liu, Christiani: "A Large-scale genetic association study of esophageal adenocarcinoma risk." in: **Carcinogenesis**, Vol. 31, Issue 7, pp. 1259-63, (2010) (PubMed).



Western Blotting

Image 1. HLA-DQB1 Antibody (N-term) (ABIN1881422 and ABIN2843450) western blot analysis in Daudi cell line lysates (35 µg/lane).This demonstrates the HLA-DQB1 antibody detected the HLA-DQB1 protein (arrow).