SANTA CRUZ BIOTECHNOLOGY, INC.

HLA-DQ1/3 (HL-37): sc-51615

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Major histocompatibility complex (MHC) class II molecules destined for presentation to $CD4^+$ helper T cells is determined by two key events. These events include the dissociation of class II-associated invariant chain peptides (CLIP) from an antigen binding groove in MHC II-$\alpha\beta$ dimers through the activity of MHC molecules HLA-DM and -DO, and subsequent peptide antigen binding. Accumulating in endosomal/lysosomal compartments and on the surface of B cells, HLA-DM, -DO molecules regulate the dissociation of CLIP and the subsequent binding of exogenous peptides to HLA class II molecules (HLA-DR, -DQ, -DP and -DR) by sustaining a conformation that favors peptide exchange. RFLP analysis of HLA-DM genes from rheumatoid arthritis (RA) patients suggests that certain polymorphisms are genetic factors for RA susceptibility. The α1 chain of HLA-DQ1 class II molecule (Ia antigen) complex can bind peptides and present them to $CD4^+$ T lymphocytes.

REFERENCES

1. Corte, G., et al. 1981. Human Ia molecules carrying DC1 determinants differ in both α- and β-subunits from Ia molecules carrying DR determinants. Nature 292: 357-360.
2. Horejsi, V., et al. 1986. Characterization of seven new monoclonal antibodies against human DR, DR + DP and DQ1 + DQ3 antigens. Tissue Antigens 28: 288-297.
3. Momburg, F., et al. 1987. B cell lymphomas of high-grade malignancy frequently lack HLA-DR, -DP and -DQ antigens and associated invariant chain. Int. J. Cancer 40: 598-603.
4. Kropshofer, H., et al. 1998. A role for HLA-DO as a co-chaperone of HLA-DM in peptide loading of MHC class II molecules. EMBO J. 17: 2971-2981.
5. Siegmund, T., et al. 1999. HLA-DMA and HLA-DMB alleles in German patients with type 1 diabetes mellitus. Tissue Antigens 54: 291-294.
6. Toussirot, E., et al. 2000. The association of HLA-DM genes with rheumatoid arthritis in Eastern France. Hum. Immunol. 61: 303-308.
7. Brunet, A., et al. 2000. Functional characterization of a lysosomal sorting motif in the cytoplasmic tail of HLA-DOβ. J. Biol. Chem. 275: 37062-37071.
8. Doebele, C.R., et al. 2000. Determination of the HLA-DM interaction site on HLA-DR molecules. Immunity 13: 517-527.
9. Louis-Plence, P., et al. 2000. The downregulation of HLA-DM gene expression in rheumatoid arthritis is not related to their promoter polymorphism. J. Immunol. 16: 4861-4869.

CHROMOSOMAL LOCATION

Genetic locus: HLA-DQB1 (human) mapping to 6p21.32.

SOURCE

HLA-DQ1/3 (HL-37) is a mouse monoclonal antibody raised against Burkitt's lymphoma cell line Raji of human origin.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PRODUCT

Each vial contains 100 µg IgG_3 in 1.0 ml of PBS with < 0.1% sodium azid and 0.1% gelatin.

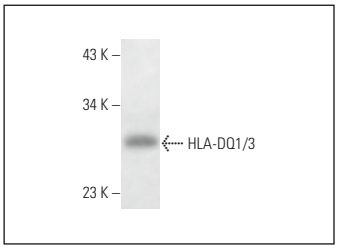
APPLICATIONS

HLA-DQ1/3 (HL-37) is recommended for detection of polymorphic determinant on HLA-DQ1 and HLA-DQ3 of human origin by Western Blotting (non-reducing) (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and flow cytometry (1 µg per 1 x 10^6 cells).

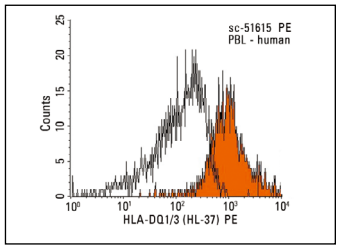
Molecular Weight of HLA-DQ1/3: 29 kDa.

Positive Controls: Raji whole cell lysate: sc-364236.

DATA



HLA-DQ1/3 (HL-37): sc-51615. Western blot analysis of HLA-DQ1/3 expression in Raji whole cell lysate.



HLA-DQB1 (HL-37): sc-51615. Indirect FCM analysis of human peripheral blood leukocytes stained with HLA-DQB1 (HL-37), followed by PE-conjugated goat anti-mouse IgG: sc-3738. Black line histogram represents the isotype control, normal mouse IgG_3: sc-3880.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.