

HLA-DR (LDR): sc-51947

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- α/β 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 and -DP) 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. HLA-B belongs to the HLA class I heavy chain paralogs. Class I molecules play a central role in the immune system by presenting peptides derived from the endoplasmic reticulum lumen. HLA-B and -C can form heterodimers consisting of a membrane anchored heavy chain and a light chain (β -2-Microglobulin). Poly-morphisms yield hundreds of HLA-B and -C alleles.

REFERENCES

1. 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.
2. Siegmund, T., et al. 1999. HLA-DMA and HLA-DMB alleles in German patients with type 1 diabetes mellitus. *Tissue Antigens* 54: 291-294.
3. Arndt, S.O., et al. 2000. Functional HLA-DM on the surface of B cells and immature dendritic cells. *EMBO J.* 19: 1241-1251.
4. Brunet, A., et al. 2000. Functional characterization of a lysosomal sorting motif in the cytoplasmic tail of HLA-DO β . *J. Biol. Chem.* 275: 37062-37071.
5. Doebele, C.R., et al. 2000. Determination of the HLA-DM interaction site on HLA-DR molecules. *Immunity* 13: 517-527.
6. Louis-Plence, P., et al. 2000. The down-regulation of HLA-DM gene expression in rheumatoid arthritis is not related to their promoter polymorphism. *J. Immunol.* 165: 4861-4869.
7. Toussiro, E., et al. 2000. The association of HLA-DM genes with rheumatoid arthritis in Eastern France. *Hum. Immunol.* 61: 303-308.

SOURCE

HLA-DR (LDR) is a mouse monoclonal antibody raised against CESS human cell line.

PRODUCT

Each vial contains 100 μ g IgG_{2a} in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

RESEARCH USE

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

APPLICATIONS

HLA-DR (LDR) is recommended for detection of HLA-DR of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)].

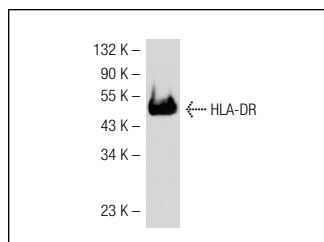
Molecular Weight of HLA-DR mature chain: 30 kDa.

Positive Controls: Ramos cell lysate: sc-2216, NAMALWA cell lysate: sc-2234 or BJAB whole cell lysate: sc-2207.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use goat anti-mouse IgG-HRP: sc-2005 (dilution range: 1:2000-1:32,000) or Cruz Marker™ compatible goat anti-mouse IgG-HRP: sc-2031 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048.

DATA



HLA-DR (LDR): sc-51947. Western blot analysis of HLA-DR expression in BJAB whole cell lysate.

PROTOCOLS

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