**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). Polymorphisms yield hundreds of HLA-B and -C alleles.

**REFERENCES**


**CHROMOSOMAL LOCATION**

Genetic locus: HLA-DRB1 (human) mapping to 6p21.32.

**SOURCE**

HLA-DRβ (TAL 14.1) is a mouse monoclonal antibody raised against HLA-DRβ of human origin.

**PRODUCT**

Each vial contains 200 µg IgG2a kappa light chain in 1.0 ml of PBS with <0.1% sodium azide and 0.1% gelatin.

HLA-DRβ (TAL 14.1) is available conjugated to either phycoerythrin [sc-53316 PE] or fluorescein [sc-53316 FITC], 200 µg/ml, for IF, IHC(P) and FCM.