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, 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


CHROMOSOMAL LOCATION

Genetic locus: HLA-DQB1 (human) mapping to 6p21.3.

SOURCE

HLA-DQ1/3 (6D464) 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.