Scope and Purpose of the HIV Molecular Immunology Database

HIV Molecular Immunology (formerly called HIV Molecular Immunology Database) was added as a companion volume to the NIAID, Division of AIDS-funded Human Retroviruses and AIDS Genetic Sequence Compendium in 1995. This publication, the 2000 issue, is the printed version of the Web-based HIV Immunology Database (http://hiv-web.lanl.gov/immunology). Included herein are T-cell epitope tables and maps on HIV proteins, alignments, and annotation, as well as a map of linear B-cell epitopes and a summary of monoclonal antibodies with discontinuous epitopes. The protein alignments highlight the sequence heterogeneity among international isolates in well-characterized CTL epitopes; helper T cell and antibody epitope alignments are available only on our web site at http://hiv-web.lanl.gov/immunology. The annotation includes information such as how specific epitopes were experimentally defined, HLA specificities for T-cell epitopes, isotypes of monoclonal antibodies, the initial antigenic stimulus immunogen, and brief notes describing the context in which a given epitope was studied. The compendium begins with review articles relevant to the immunology of HIV. Comments on the database or requests for the hard copy can be sent via email to immuno@t10.lanl.gov.

Citing the Database

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The Cover

The cover illustration by Vincent Detours, Theoretical Biology and Biophysics, Los Alamos National Laboratory, shows the steps in the generation of CTL epitopes (reviewed in [1]). Viral proteins are cleaved by proteasomes [2]. The N-terminal ends of the resulting peptides may be trimmed by other proteases (not shown, [3]). Transporters associated with antigen processing (TAP) then translocate peptides into the endoplasmic reticulum for loading onto MHC class I molecules [4]. The groove of MHC molecules accommodates only peptides which are 8–11 amino-acids in length, and with sequence matching the anchor residues motif characteristic of the host MHC background [5]. Loaded MHC migrate to the cell surface where they become available for T cell receptor (TCR) binding [6]. Viral escape mutations may affect various stages of the epitope generation process [7].