

Poster presentation

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DC-SIGN as a Receptor for HTLV-I Binding, Entry and Infection

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Human T cell leukemia virus type 1 (HTLV-1) has been identified as the etiologic agent of adult T cell leukemia (ATL) and HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP). Numerous studies have demonstrated that patients diagnosed with HAM/TSP exhibit rapid activation and maturation of dendritic cells (DCs) while ATL is associated with their maturation defect. In addition to T cells, HTLV-1 is known to infect DCs. HTLV-1 infection of DCs could alter general DC function or the specific processing and/or presentation of HTLV-1-specific peptides, potentially playing a major role in the course of HTLV-1-associated disease. In this regard, we have demonstrated that an important antigen receptor on DCs, DC-SIGN serves as a receptor for HTLV-1 binding using a quantum dot-based fluorescent binding assay. We have also demonstrated that gene silencing of DC-SIGN inhibits the infection of DC in a DC/T cell co-infection system. Furthermore, expression of DC-SIGN in B cells enhances viral binding, integration, and infection. These investigations, which consider the involvement of DC surface molecules in HTLV-1 pathogenesis, are the first explorations of the intricate mechanisms that underlie the interactions between DCs and HTLV-1.